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Contributors

Jürgensen, Theodor Hermann, 1840-1907.
Stengel, Alfred, 1868-1939 (Translator)
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Krehl, Ludolf, 1861-1937.
Dock, George.
Royal College of Physicians of London

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Nothnagel's Practice

DISEASES OF

THE HEART

BY

PROF. TH. v. JÜRGENSEN
of Tübingen

PROF. L. v. SCHRÖTTER
of Vienna

PROF. L. KREHL
of Greifswald

EDITED WITH ADDITIONS

BY

GEORGE DOCK, M. D.
Professor of Theory and Practice of Medicine and Clinical Medicine,
University of Michigan, Ann Arbor

AUTHORIZED TRANSLATION FROM THE GERMAN, UNDER THE EDITORIAL SUPERVISION OF

ALFRED STENGEL, M. D.
Professor of Clinical Medicine in the University of Pennsylvania

PHILADELPHIA AND LONDON
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PREFACE.

THE excellence of the series of monographs issued under the editorship of Professor Nothnagel has been recognized by all who are sufficiently familiar with German to read these works, and the series has found a not inconsiderable proportion of its distribution in this and other English-speaking countries. I have so often heard regret expressed by those whose lack of familiarity with German kept these works beyond their reach, that I was glad of the opportunity to assist in the bringing out of an English edition. It was especially gratifying to find that the prominent specialists who were invited to co-operate by editing separate volumes were as interested as myself in the matter of publication of an English edition. These editors have been requested to make such additions to the original articles as seem necessary to them to bring the articles fully up to date and at the same time to adapt them thoroughly to the American or English reader. The names of the editors alone suffice to assure the profession that in the additions there will be preserved the same high standard of excellence that has been so conspicuous a feature in the original German articles.

In all cases the German author has been consulted with regard to the publication of this edition of his work, and has given specific consent. In one case only it was unfortunately necessary to substitute for the translation of the German article an entirely new one by an American author, on account of a previous arrangement of the German author to issue a translation of his article separately from this series. With this exception the Nothnagel series will be presented intact.

ALFRED STENGEL.

EDITOR'S PREFACE.

THE editor of a work is justly expected to defend his assumption of the position and to explain his method of carrying out his part. As to the former, I may say that I gladly accepted the task of editing the translation of the heart sections of Nothnagel's "Specielle Pathologie und Therapie," because I had used the originals with much interest and profit, and because I appreciated the honor of assisting in presenting the work to my English reading colleagues.

Several excellent works upon diseases of the heart have appeared within the last quarter century in Germany and it seems strange that none of them was translated into English; especially strange when we consider the many and important contributions made in that country to the normal and pathologic anatomy of the heart, the physiology of the heart and circulation, the methods of diagnosis, the pharmacology of cardiac remedies, and the application of nonmedicinal measures to the treatment of patients with heart disease. To be sure, all these discoveries were available to and utilized by American and English writers, yet it would seem of interest to see more directly how discoveries so important would affect the literature and the methods of treatment of those to the manner born. This can now be done, with all the advantages and disadvantages of collaboration, and I think most readers will agree with me when I state my belief that the lack of a simple division of the material and a common point of view is more than made up by finding in one book the sound learning and wide clinical experience of Professors von Jürgensen and von Schrötter and the deep and broad training in anatomy, physiology and pathology, as well as the excellent clinical observations of Professor Krehl.

In accordance with the wise view of the editor of the series, I have not attempted many or radical alterations or additions. I did not wish to change the native flavor of the work, but tried to secure accuracy of language and of statement, to correct the few verbal errors that had slipped into the original, and to make the medicinal preparations conform to the U. S. Pharmacopœia.

Matters of interest brought out since the original was published have been added in brackets. These include important American and English contributions which I trust will make the work still more valuable as a work of reference. I have found nothing that seemed necessary to omit, and in only one instance have modified what seemed to me the meaning of the author.

GEORGE DOCK.

ANN ARBOR, January, 1908.

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INSUFFICIENCY (WEAKNESS) OF THE HEART.

BY

THEODOR VON JÜRGENSEN, M. D.

INSUFFICIENCY (WEAKNESS) OF THE HEART

"Apud nos olim in laesa respiratione vel nunquam, vel perraro audiebantur nomina vitiatae structurae cordis et praecordiorum. Modo post observationes saepenumero institutas in mortuis eadem nomina plus nimio audiuntur ac timentur in vivis."* Thus spoke Albertini, of Bologna, in the year 1726, who, according to Morgagni,† deserves high praise for his contributions to our knowledge of diseases of the heart.

When it is remembered that Harvey's immortal work, "Exercitatio anatomica de motu cordis et sanguinis," was not published until 1628, the ignorance of earlier writers does not appear strange. Until some insight was gained into the circulation as a whole anything like a correct knowledge of diseases of the heart was impossible; for that implies not only a knowledge of the conditions in health, but also the knowledge obtained by the examination of heart patients during life and after death.

In the eighteenth century pathologic anatomy began to be developed and pointed the way to clinical investigation.

I do not consider it necessary to describe the development of our knowledge of heart disease in this work; it would be a mere catalogue of events and practically devoid of interest. For the earlier periods the work has already been done,‡ and Haeser § has given a complete presentation of the historic development of the knowledge of heart disease.

Gradually we have progressed; but, as in every other field of learning, the goal is still remote. Again and again the progress of human knowledge suggests other aspects of vital processes which compel the physician's attention. Nevertheless a description which is based upon and includes only clinical observation and clinical data must be subject to many limitations.

* "Formerly we rarely or never heard the terms injured structure of the heart and precordia in cases of impaired respiration (laesa respiratio); but since observations have been made time and again on cadavers, these same terms are far too much heard and dreaded in the living."

† "De Sedibus et causis morborum per anatomen indagatis," Epistol. xvii, art. 5, where the above quotation occurs.

‡ Martini, "Beiträge zur Geschichte der Lehre vom Herzen und den Herzkrankheiten von den frühesten Zeiten einer wissenschaftlichen Medicin bis zur Begründung der Lehre von den Herzkrankheiten durch Senac," Berlin dissertation under the direction of A. Hirsch (1869), with an accurate list of authorities and many quotations.

§ "Lehrbuch der Geschichte der Medicin," vol. ii, third edition, Jena, G. Fischer, 1881. [See also, "Historic Outline of Cardiac Pathology," etc., by C. N. B. Camac, "Bulletin of the Johns Hopkins Hospital," February, 1904.—Ed.]

Can we speak of a disturbance sui generis, *common to all forms of heart disease?* Is there such a thing as a uniform clinical picture?

Yes and no.

No, because a number of different disturbances of the respiratory system give rise to approximately the same manifestations. The reason for this is readily understood, since circulation and respiration have the same task to perform—that of providing the tissues with the blood-supply on which their activity depends and removing the products of their activity, which are not only useless, but may be even a burden and directly harmful.

The older physicians did not distinguish between diseases of the lungs and diseases of the heart,—taking these terms in their widest application,—and referred to them by the common term "respiratio laesa." In these, as in all other pathologic conditions, they viewed the clinical picture as a whole; and although in certain respects we have gained a very accurate knowledge of the conditions, we ought not to close our eyes to the justice of such a view. For what is the final result of our knowledge at the present time? What is the sum total of all special investigations as compared with what has actually been accomplished?

"Peculiar to diseases of the heart and lungs and foreign to most other diseases are those disturbances of metabolism which owe their origin to diminution of the gaseous interchange in the lungs and to slowing of the circulation. Disregarding the great variety of causes that may lead to such a condition, we shall confine our attention to the fully developed clinical picture of a non-compensated valvular lesion or profound interference with the interchange of gases in the lungs. It is immaterial whether the disease first attacks the heart or the respiratory organs, the sum total of the phenomena which we are about to study with reference to their influence on the metabolism may be designated 'dyspneic conditions.'"

So says Karl v. Noorden.*

Is it then expedient to begin our presentation of the subject with general considerations? I think it is; at least, if the clinical aspect is to be made the basis of our description of diseases of the heart.

What we *first* see at the bedside is practically always a uniform clinical picture. Of course, the picture must be analyzed as much as possible, and no one will be satisfied with a diagnosis of "insufficientia cordis," much less "respiratio laesa." Nevertheless, the general impression which the patient makes on the physician in this as in every other disease at once suggests the line of investigation that will probably reveal the seat of the trouble.

By including what is common to all forms of heart disease in a common discussion the description becomes more condensed and a better general view of the subject is possible. For this reason I shall begin with—

CARDIAC INSUFFICIENCY (WEAKNESS).

The term is used to designate functional deficiency of the heart causing such a retardation of the circulation that tissue respiration cannot be adequately performed.

The **kinetic energy of the heart depends on its muscular fibers.** These are capable of performing only a certain amount of work. If

* "Lehrbuch der Pathologie des Stoffwechsels," Berlin, Hirschwald, 1893, S. 309.

more than this is demanded of them, a disturbance results and persists until equilibrium has been established between the amount of work to be performed and the amount of work that can be performed. But the establishment of equilibrium alone does not end the matter so far as the heart is concerned—at least, not always. An important factor is that the nutrition of the heart is directly dependent on the work the organ performs. If the tension of the blood in the beginning of the aorta diminishes, the quantity of blood propelled through the coronary arteries in a unit of time and its velocity are diminished in the same proportion. The heart muscle receives less nutritive material and is less completely relieved of the accumulated products of metabolism which contain fatigue stuffs. If the demand on the heart can be diminished before the development of severe fatigue, compensation can be established: blood-supply as well as drainage corresponds to the diminished demands on the muscle. But if the amount of work cannot be diminished with sufficient rapidity, a vicious circle develops: the heart being unable to obtain a sufficiency of blood, its activity diminishes; and because its activity diminishes, it cannot obtain a sufficiency of blood.

These conditions are quite simple and suffice to show that the work of the heart is directly dependent upon the condition of the heart muscle. The theory which ascribes a much greater importance to the heart muscle itself is constantly gaining ground. Thus Engelmann* says in a critical review:

"According to recent anatomic and physiologic investigations there appears to be no doubt that the normal action of the heart pump in all animals, both in the adult and in the embryonal state, is independent of the nervous system and is exclusively due to muscular activity. The fundamental functions of automatism, rhythm, impulse conduction, and coördination, which were formerly generally ascribed to the cardiac ganglia, as well as a number of important phenomena of self-regulation of the heart-beat, now find a perfectly satisfactory explanation in certain qualities and inter-relations of the elementary muscle-cells of the heart wall. The cardiac nervous system, therefore, has merely an auxiliary, secondary significance."

It seems to me that our observations on the diseased heart harmonize quite well with these views in a great many respects and in some respects better than with the older theories. The phenomena observed after brief overexertion of the heart are better understood on this supposition. The fact that the lifting of a heavy weight may permanently impair the strength of a previously hypertrophied heart (see Fraentzel's case quoted below) can hardly be attributed to temporary insufficiency of the blood-supply. But it can readily be understood that violent compression, even of brief duration, may loosen the connections between the cunningly arranged individual fibers to such a degree that perfect coöperation is no longer possible. This would cause an impairment of the functional power of the entire organ, although the work performed by the individual fiber might be undiminished or even increased.

We know that when the body is at rest the heart liberates only a portion of the force of which it is capable; the available remainder is called

* Th. W. Engelmann, "Ueber den myogenen Ursprung der Herzthätigkeit und über automatische Erregbarkeit als normale Eigenschaft peripherer Nervenfasern," "Pflüger's Archiv für die gesammte Physiologie," Bd. lxv, S. 535 ff. The article contains an accurate critical summary of the facts and a bibliography.

the *reserve force*. The extent of this has been determined by recent investigations.* To Benno Lewy† we are indebted for some very clear explanations of great value on this subject. I quote the following from his very valuable contribution: The reserve strength of the heart enables the organ to perform thirteen times the amount of work which it exerts when the body is at rest. This increase is effected partly by an increase in the quantity of blood that is propelled during a single systole—the amplitude of contraction—and partly by an increase in the number of systolic contractions in a unit of time.

Even a moderate excess of bodily work causes an increase of that of the heart to about four times the normal. This extra amount of work can easily be performed by the normal heart year after year if the general nutrition is sufficient and the necessary periods of rest are allowed.

The daily amount of work performed by the heart when the body is resting is about 20,000 q by absolute measurement, corresponding to 133 large calories. During bodily activity these values go up to about 45,100 q and 300 calories.

Hence, according to Rubner's‡ figures, the heart requires for its own use when the body is at rest and fasting 5.8 per cent. of the total amount of energy introduced with the food, and 8.9 per cent. of the total of available energy during hard work.

It is important to determine these figures because until a few years ago the amount of work performed by the heart was considerably overestimated—on the lowest estimate more than twice as high as the above—a point which has been proved by the investigations of Tigerstedt§ and Zuntz. Although these figures are only approximate, they permit one to form an idea of the demand upon the working heart muscle and the amount of energy that it needs to enable it to work under various conditions. The question whether, in a given case, the energy introduced with the food and that furnished by the body, by the combustion of its tissues, are sufficient to enable the heart to work, should be answered on the basis of these investigations.

Another point of importance is that the heart is supplied with the blood which it requires for its nutrition during diastole, while it is resting. "Owing to the great differences in the frequency of the pulse the duration of the ventricular systole varies within much narrower limits than that of the ventricular diastole, both in the same individual and in different individuals of the same species. Hence the changes in the pulse frequency are in the main caused by variations in the duration of the diastole.‖

This is the conclusion drawn by Tigerstedt from the sum total of the data that have been obtained, and Benno Lewy** quite correctly argues that: "Since the increase in the activity of the heart which accompanies muscular work is always associated with an increase in the frequency of the pulse, and since the latter increases at the expense of diastole, it

* N. Zuntz, "Die Ernährung des Herzens und ihre Beziehung zu seiner Arbeitsleistung," "Deutsche medicinische Wochenschrift," 1892, S. 109 ff.

† B. Lewy, "Die Arbeit des gesunden und des kranken Herzens," "Zeitschrift für klinische Medicin," Bd. xxxi, S. 320 ff. (1896–97). With a list of the most important literature.

‡ Dr. Max Rubner, "Calorimetrische Untersuchungen," "Zeitschrift für Biologie," new series, Bd. iii. Whole number, Bd. xxi, S. 382 (1885).

§ Tigerstedt, "Lehrbuch der Physiologie des Kreislaufes," Leipzig, 1893, Veit & Co., p. 146, § 11.

‖ Tigerstedt, *loc. cit.*, p. 131.

** *Loc. cit.*, 389.

follows that the heart is under unfavorable conditions for nutrition during an excess of muscular work, even when the ingestion of food as a whole is adequate. For mechanical reasons, therefore, the heart can perform an increased amount of work only for a limited time. If the organ is kept on working beyond that time and remains healthy, it must work under conditions which render the duration of diastole long enough to admit a sufficient blood-supply to the myocardium; in other words, retardation of the pulse and relative heart rest must ensue."

CAUSES OF CARDIAC INSUFFICIENCY.

The causes of cardiac insufficiency can be divided into two main groups:

I. Those which directly affect the heart and injure its mechanism.

II. Those which affect the heart indirectly by preventing it from receiving the amount of blood necessary to enable it to perform the work required of it.

Both sets of causes may induce disturbances either rapidly or slowly. The differences in the clinical picture are due to the course of the disease, not to the exciting causes.

It is needless to say that impairment of the mechanism favors a diminution of the blood-supply and, conversely, if the blood-supply is insufficient, the mechanism is more likely to be disturbed. The first statement is a corollary of the proposition that the nutrition of the heart and its functional capacity are directly dependent on the amount of work it performs; while the second statement can be directly deduced from the law which holds true for all living tissues.

Let us take up the causes in detail.

I. The causes which affect the heart directly and may injure its mechanism are:

(*a*) **External violence acting on the heart,** with or without destruction of the heart walls or the vascular trunks inclosed in the pericardial sac.

A detailed discussion of these matters belongs to the realm of surgery. There are, however, a few points of general interest.

In the first place, the marked variation observed in the symptoms that follow an injury of the heart shows that the resisting power of different individuals varies. The portion of the heart affected by the injury is also an important factor; although the post-mortem findings and our knowledge of the seat and significance of the individual heart ganglia are equally inadequate to form a basis for a positive opinion.

A few examples may here be given; they have been taken from the careful collection of cases by Georg Fischer,* and the original numbers have been retained.

No. 172.—C. Latour d'Auvergne, "le premier grenadier de France," sixty-eight years of age, was wounded in the battle of Neustadt by a lance-thrust between the sixth and seventh ribs and fell to the ground, cursing the enemy who had struck him down ("la menace â la bouche contre l'ennemi, qui l'avait frappé"), and died at once. Fautrel performed the autopsy: pericardial laceration 4 to 5 cm. long; the sac contained a small quantity of blood. A very small wound at the apex of the left ventricle 3 to 4 mm. deep, non-penetrating.

In this case sudden death is explained by the profound psychic shock sustained at the time of the injury. It must also be remembered, however, that the man was old and that his heart must have been subjected to a severe strain before and during the battle.

* Dr. Georg Fischer, "Ueber die Wunden des Herzens und des Herzbeutels," "Langenbeck's Archiv für klinische Chirurgie," Bd. ix, S. 571 ff. (1868).

No. 200.—Duval, thirty-five years of age. In a duel with swords sustained an oblique wound at the level of the sixth rib on the right side. The hospital surgeon diagnosed a non-penetrating wound of the chest, as there was neither thoracic pain nor dyspnea. The pulse was quiet. Appearance of the face unchanged; the patient was permitted to return to his frigate without assistance. The witnesses said the sword was broken off about five inches from the tip, and that the piece could not be found. There was nothing to indicate that the steel had remained in the chest. In the evening (the wound was received at 10 o'clock in the morning) he was brought to the hospital on a stretcher. Dyspnea, profuse hemoptysis, coarse râles, pain, rapid, irregular pulse, weak voice. Skin hot and dry. During the night great restlessness, stertorous breathing. Death in twenty-two hours. The tip of the sword, three inches in length, had entered the right auricle at its base and injured the lung. A pint (old French measure; a little less than one liter) of blood was found in the pleural cavities. The case was reported by Dupuytren.

No. 228.—From Ambroise Paré: A nobleman, while fighting a duel, was wounded under the left nipple. After receiving the wound he made several passes at his antagonist and, when the latter fled, pursued him 200 paces and then fell dead.

The wound of the heart was so large that one finger could be introduced; a large quantity of blood was found on the diaphragm.

These are not isolated observations. They teach us that even a severely wounded heart is capable of maintaining for a time the amount of circulation required for severe bodily exertion.

Injuries of the heart also have a general interest from the fact that they give us an insight into the nature of the causes of death.

The above cases do not furnish absolute proof that sudden death, in the literal sense of the word, can take place unless it be from complete destruction of the heart or the loss of large quantities of blood at the time of the injury. Some of the cases that have been reported can, to be sure, be interpreted in this way. In addition to case No. 172, which has been cited, we may refer to case No. 128, about which it is said: "A sword-thrust pierced the left ventricle and the wounded man fell as if struck by lightning and promptly expired" ("quasi fulmine ictus concidit, moxque extinctus est"). But such statements, like those contained in other similar reports, are so indefinite in the matter of time that it is impossible to determine whether or not death was in part caused by the escape of blood either externally or into the pericardium or pleura. The important question to determine is whether concussion of the heart alone is sufficient to cause death. That it plays an important part in bringing about such a result is not disputed. But Fischer, in discussing the clinical course,* states that instant death coincident with the injury is very rare. It is quite true that the numerous cases of those who die on the battle-field from heart wounds necessarily cannot be taken into account.

Richard Bernstein† also collected a number of cases among which he was unable to find a single instance of sudden death which could be attributed to simple concussion of the heart.

Riedinger‡ attempted to settle the question by experimentation and reached the following conclusion: "In a case of injury of the thorax due to external violence, severe symptoms or even death must, in the absence of any severe injury to organs, be attributed to arrest of the heart resulting from intrathoracic irritation of the vagus and in part to a more than temporary diminution of the vascular tone in the peripheral vessels, producing circulatory disturbances in the brain."

* *Loc. cit.*, 720.

† "Ueber die durch Contusion und Erschütterung entstehenden Krankheiten des Herzens," "Zeitschrift für klinische Medicin," Bd. xxix (1896), S. 518 ff.

‡ "Verletzungen und chirurgische Krankheiten des Thorax und seines Inhaltes," "Deutsche Chirurgie," Lieferung 42 (1888), S. 18.

This would seem to show that the process is quite complicated and that the heart is only indirectly involved through vagus inhibition and diminished blood-supply.

Fatal hemorrhage, that is, the escape of blood from the vessels or from the heart, may produce death because the blood remaining in the body is insufficient for the needs of the entire organism, and also because, owing to the cardiac injury, the circulation of the blood is also insufficient. There is nothing surprising about this. But the case is different when the blood which escapes from the heart or from the vascular trunks surrounded by the pericardium accumulates within the pericardial sac.

Morgagni spoke of compression of the heart brought about in this way and believed it to be the cause of sudden death. Cohnheim* has given us an accurate description of what happens when large quantities of blood rapidly accumulate within the pericardial sac. He showed that effusions into the pericardial space, if they attain a certain size, interfere with the filling of the cardiac chambers. This is due to the tension of the pericardial sac produced by the pressure of a rapid effusion. The tension obstructs or inhibits the entrance of the blood into the venous trunks inclosed in the pericardial sac.

Animal experiments show that, as soon as the pressure in the pericardial sac attains a certain height, the pressure in the systemic veins suddenly rises, while the pressure in the systemic arteries as suddenly falls. This is because the right heart is insufficiently filled with blood—the blood-supply it obtains through the venæ cavæ, which are surrounded by the tense pericardial sac, being interfered with. The negative pressure normally present within the pericardial sac is rendered positive by the pressure of the exudate. Depending on the difference in pressure between the veins outside and inside of the sac,—in other words, the portions of the venæ cavæ which are inclosed in the pericardial sac and those on the outside of it,—either no blood at all will enter the heart or there will be merely a slowing of the blood current. It is obvious that the increase of pressure in the venous trunks incident to the accumulation of blood in front of the heart favors the movement of blood contained in these venous trunks toward the heart. The flow will persist so long as the pressure exerted by the tense pericardial sac and its contents is less than the pressure exerted by the veins; but the flow must also diminish in proportion to the lessening of the difference between the two pressures, and must become equal to zero as soon as there ceases to be any difference in pressure. The pressure within the pericardial sac is about −3 to 5 mm. of mercury; the pressure in the jugular and subclavian veins is +0.2 and −0.1 respectively on the right side, and −0.1 and −0.6 mm. respectively on the left side. These are the measurements obtained by Jacobson† in a sheep. Hence it only requires an increase of pressure equivalent to a few millimeters of mercury to make it impossible for the blood to flow into the right auricle. Arrest of the heart in systole, entirely preventing diastolic relaxation, which is probably what was formerly meant by compression of the heart, does not occur. Life is extinguished long before the pressure reaches the point necessary to arrest the heart in systole. If a smaller quantity of blood enters the right heart, a correspondingly smaller quantity will also enter the left heart. The result is a fall of the arterial pressure in the greater circulation, possibly down to zero. In any case the circulation must be diminished in force and velocity corresponding to the obstacle to the entrance of blood into the right side of the heart.

Cohnheim estimates the quantity of blood which is sufficient in cases of intrapericardial rupture to arrest the circulation at 150 to 200 cm.

It is needless to say that the loss of such a small proportion of the blood contained in the circulation cannot be called a fatal hemorrhage.

On the other hand, we must inquire how far the results obtained by experiment can be applied to man.

* "Vorlesungen über allgemeine Pathologie," 2. Aufl., Bd. i, Berlin, Hirschwald, 1882, S. 20 ff.

† Comp Tigerstedt, *loc. cit.*, 432.

In the first place, the thorax is opened and the animal is kept alive by artificial respiration while the experiments are performed. When the thorax is closed every inspiratory expansion causes a diminution of the pressure within the thoracic cavity. This decrease of pressure also affects the pericardial sac and its contents. Hence the absolute values that make it impossible for the blood to enter the venous trunks which are enclosed by the pericardium must necessarily be different. They must be larger than when the thorax is opened. But on the whole the conditions are not sufficiently altered to preclude the application of the results to the human subject.

The effect of an injury by which the pericardial sac and the heart itself are opened varies in the individual case, depending on whether the pericardial wound is so large as to render the structure incapable of elastic tension.

This is undoubtedly the case when the laceration is large, but by no means necessarily always in cases of stab wounds. Thus Fischer has reported cases in which the entrance of a needle into the heart caused a fatal hemorrhage (No. 2; in this case effusion into the pericardial sac is specially mentioned). It is also quite possible that much larger openings may close up at first and later, after some days, open up again, so that a sufficiently large effusion of blood may again take place into the pericardial sac. Some very curious things happen in these cases and are very difficult to explain.

One of the most curious cases is the following:

No. 308, of Fischer.—"Suicide. Wound at the upper edge of the sixth rib on the left side, two inches from the sternal border, of the size of a lead-pencil. No wound of exit. Slight hemorrhage. Chill, thirst, vomiting; extremities icy cold; pulse and heart-beat can barely be felt; increasing cyanosis; death after three hours. Lying on the pericardium, which was uninjured, was a small leaden bullet; the sac contained 12 to 15 ounces (360 to 450 cm.) of blood. There was a small round opening at the apex of the right ventricle, which formed the beginning of a channel extending into the left ventricle and corresponding in size to the bullet. No blood in the pleural cavities." (Heidenreich, "Bayerisches ärztliches Intelligenzblatt," 1865, No. 51.)

It is a remarkable fact that injuries of the heart by external violence may be followed after a variable interval by the development of a disease which very probably bears a causal relation to the injury. This occurs not only in cases in which there is already an old lesion, usually of the endocardium, in which case endocardial inflammation develops. Cases have also been reported in which acute endocarditis developed some time after the injury, although no signs of previous alteration could be found (compare case 112 in R. Bernstein's collection, p. 537). A heart already damaged at the time of the injury may become permanently disabled, a fact of some medicolegal importance in cases of accidents and damage suits.

(*b*) **Spontaneous Rupture of the Heart.**—Rupture of the heart without any external violence occurs only when the muscular structure is severely damaged. The subject will, therefore, be discussed in greater detail in connection with diseases of the heart muscle. At this point I shall merely give a short description of the symptoms in a case observed by myself.

Personal Observation 1.—Woman, sixty years of age. Up to her fifty-fifth year she had no symptoms worth mentioning, although there was dyspnea on severe exertion and loss of working power; there was also obesity.

In her fifty-eighth year she had rather sudden attacks of pain and oppression in the lower portion of the thorax and in the epigastric region, with palpitation and nausea. These symptoms gradually increased and later assumed the form of genuine attacks of angina pectoris.

Admitted November 3, 1884. On the morning of the second—a few days before she had already suffered from dyspnea, severe cough, want of appetite, and nausea—she still felt comparatively well. She could attend to her work as a midwife, went to see some of her patients, and even "forced" herself in the afternoon to attend a christening, although she had felt pain in the chest toward noon. When she returned home from the christening "it suddenly came upon her." She had hardly gone up the (low) steps leading to the door of her house when she became very red in the face, could scarcely breathe, and felt a frightful stabbing pain in the region of the heart and epigastrium. Rest in bed failed to relieve the pain and she did not feel better until one and a half hours later. During the following night she had palpitation from time to time but no actual attack; in fact, she did not have another until the forenoon of the third, and then it lasted only half an hour. The vertigo persisted and was so severe that the patient remained in bed until the morning of the fourth. Now she is able to get up and make a few visits.

The history during the decisive period of the case is as follows: "Suddenly about midnight, between the fourth and fifth of November, she had a very violent attack and the physician was sent for early in the morning. As I entered the room at 5.45 in the morning the patient's groans could be heard as far as the door. When I asked her how she was, she answered brokenly that she was going to die. She would not lie quiet, but constantly turned from one side to the other, raised herself in bed, and lay down again on her back. The forehead was wrinkled, the lips and cheeks were slightly cyanotic; the hands and feet cold. The pupils were slightly contracted and reacted perfectly. A short time before she had vomited several times, the vomited material consisting chiefly of mucus. The tongue was slightly coated. The respirations were 42 to the minute, superficial, and seemed to be rather labored. When asked to do so, however, the patient was able to take a fairly deep breath. The pulse was 78, small and compressible. The heart-sounds everywhere were faint but pure. The lungs were slightly overdistended and there was impaired (band-box) resonance; catarrh of the large bronchi was also present. The abdomen was negative and the stools had been regular.

"The pain and feeling of oppression were referred to beneath the lower portion of the sternum. The patient said she felt as if something had been torn in that spot. The patient begged me 'for God's sake' to help her and said 'she could not stand it any longer.' An injection of 1.5 cm. of a 3 per cent. solution of morphin was given under the skin of the breast, and the subjective symptoms improved in a few minutes. The improvement continued. At 10.30, when I made my regular rounds, the patient was very much quieter, the pain was much less, the pupils were contracted; it was evident that the morphin had taken effect. Examination showed cyanosis; quiet breathing, respirations 26; pulse 116, small and imperfectly filled. The heart dulness could be definitely made out only by light percussion and in the erect posture. It was slightly enlarged upward and to the right; the apex beat was in the fifth intercostal space, a little outside of the nipple-line, and barely palpable. Auscultation: Over and a little inside of the apex-beat a distinct systolic murmur, audible only in the dorsal position and disappearing when the patient sits up. The diastolic sound over the pulmonic artery is accentuated. Otherwise the heart-sounds are pure, only somewhat faint.

"After the examination had been concluded, the patient lay down quietly and answered questions in a perfectly rational manner; she even smiled a little. About ten minutes after I had left she suddenly raised both arms above her head as if in a convulsion and, without anything further happening, death ensued."

The autopsy,* which was performed three hours later by Prof. Ziegler, revealed a laceration 3 cm. in length of the muscles of the left heart. The pericardial sac contained about 250 c.c. of blood. The cause was myomalacia following occlusion of the corresponding branch of the coronary artery.

In this case the laceration appears to have been rapidly followed by death; it was probably due to the overexertion of the heart incident to the changes in the position of the body during the examination and the muscular effort which these changes necessitated.

* The pathologic report of this case is given by Beck, "Zur Kenntniss der Entstehung der Herzruptur, u.s.w.," Tübingen Dissertation. 1886.

(*c*) **Rapidly Forming Inflammatory Effusions into the Pericardium.**—The tension of the pericardium caused by the effusion is, as has been said, more important than the quantity of fluid. Hence a gradual accumulation of fluid up to a liter is much less dangerous than a rapid effusion of only a few hundred cubic centimeters. It may be remarked, however, that a rapidly developing purulent effusion hardly ever occurs unless the muscle of the heart is also involved, hence the weakening of the muscular power from myocarditis must also be taken into account in these cases. The last-mentioned factor is not so important in large but slowly forming exudates. Perhaps it is not of much importance anyway. At any rate, the most important factor is the obstruction to the flow of blood to the heart by the distended and elastic pericardial sac. One can see, as I have, symptoms of the most profound cardiac weakness disappear almost immediately after artificial evacuation of the pericardial sac; this is conclusive.

(*d*) **Accumulation of Large Quantities of Air within the Heart.***—Under certain circumstances the atmospheric air may enter a vein that has been opened and thence make its way into the right side of the heart. The necessary conditions for this accident are negative pressure in the vein and failure of its walls to collapse at the point of injury. We know from experience that during operations air most frequently enters the internal jugular, the subclavian, and the axillary veins.

"The stretching of the parts previous to dividing them at an operation may keep the large vein from collapsing at the moment when it is opened, and if, as usual, the blood is removed by sponging, the air gets into the vein" (Heineke).

The entrance of air through the uterus is perhaps equally frequent.

"After the placenta has come away, the veins are widely opened, owing to the rigid condition of the uterine wall, and air is very apt to enter if it has had an opportunity to get into the uterus, which happens not very infrequently in operative procedures, especially when giving injections" (Heineke).

The entrance of air into other veins is very rare.

I here give Heineke's† brief account of what happens when air enters a vein:

"Ulrich describes a case which came under his treatment about as follows: In removing a tumor from the neck of a man, fifty-four years of age, the internal jugular vein was cut so that the opening in the gaping and empty vein could be plainly seen. Physicians who were watching the operation said they heard a hissing sound; immediately frothy blood was seen to ooze from the vessel; the patient became unconscious, had slight convulsions of the facial muscle, and immediately afterward opisthotonos; the face became pale; the pulse small and tremulous, and the breathing stopped. After a few respirations, separated by long intervals, life was extinguished."

"In a case observed by Girbal in which a tumor had been extirpated under general anesthesia, a bubble of air entered one of the veins of the neck as the operator was about to remove a small piece of the tumor which had been overlooked after the patient had come out of the anesthesia. A hissing sound was heard; the patient's face at once became pale; he gave a plaintive cry; the extremities became cold; the pulse could not be felt; a cold sweat covered the chest; consciousness, sensation, and movement were lost. Over the heart a gurgling noise which obscured the heart-sounds was heard for about one minute. Altogether the symptoms lasted only three to four minutes. After an hour pulse and respiration had become normal and auscultation revealed nothing unusual."

* Comp. W. Heineke, "Blutung, Blutstillung, Transfusion nebst Lufteintritt und Infusion," "Deutsche Chirurgie," Lieferung 18, Stuttgart, 1885, Enke, S. 64 ff.

† *Loc. cit.*, p. 66.

As no postmortem report is given, it is impossible to say whether death, which occurred thirteen hours after the operation, had any connection with the entrance of air into the vein; but it probably did not.

The severity of the disturbances caused by the entrance of air depends not so much on the absolute quantity of air that enters the circulation as on the rapidity with which the entrance is effected. Rapidly entering air collects within the right heart, distends the ventricle, and prevents the entrance of blood from the systemic veins as well as the escape of blood into the pulmonary artery. The result is emptying of the arterial system, accompanied by a fall of arterial tension in some cases down to zero, exactly as when the pericardial sac is distended.

The mechanism may be tentatively explained as follows:* The air is an elastic gas; the blood, a non-compressible fluid. Fluids tolerate differences in pressure; gases strive to overcome such differences by expanding more or less or, in other words, they only change their volume. Hence when air enters the veins, the blood in the right heart is replaced by gas, and the kinetic energy furnished by the right heart is no longer exclusively employed in driving the blood which it contains, as part of this energy—perhaps the greater part—is expended in compressing the air and is, therefore, not available for propelling the blood. Hence the blood can be only partially removed from the right heart; but if less blood is introduced into the greater circulation, the tension in the systemic arteries must fall.

Air continues to enter the right heart so long as the force which propels the air is able to overcome the elastic resistance offered by the walls of the ventricle, which become more and more tense as the air continues to accumulate. The air is driven by the same forces as those which are responsible for the movement of the blood in the venous portions of the greater circulation, and the most important among these forces is inspiration.

The air which enters the right heart along with the blood there separates from the blood and occupies the highest point in the heart. If the accumulation of blood in the right heart becomes so great that the tricuspid, owing to the distention of the walls of the right ventricle, is unable to close, air escapes through the open valve; this is propelled by the force of the right heart into the systemic veins, and there causes a positive venous pulse.

The individual data on which this explanation is based have been verified by numerous experiments performed by a great many investigators. Observations on the human subject yield similar results.

The theory that death after aspiration of air must always be attributed to embolism in the lesser circulation was recently advocated anew by A. Hauer, who worked under Knoll.† This is not the place to discuss this theory in detail, but I may say that I do not believe it to be correct.

If the filling and distention of the right heart from the entrance of air into the circulation are less marked, the muscle retains the power to propel

* See von Jürgensen, "Luft im Blute—Klinisches und Experimentelles," "Deutsches Archiv für klinische Medicin," Bd. xxxi (1887), S. 468. In this article I quote Couty's interpretation based on investigations of his own and of Magendie, Bouillaud, Muron, and Laborde. The article also contains a complete bibliography. I may add that Virchow should be mentioned prominently among the earlier names. (See Virchow's "Gesammelte Abhandlungen zur wissenschaftlichen Medicin," Zweiter Abdruck, Hamm, Grothe, 1862, S. 305 ff.)

† "Ueber die Erscheinungen im grossen und kleinen Kreislauf bei Luftembolie," "Zeitschrift für Heilkunde," Prague, Bd. xi (1890).

the accumulated air through the pulmonary capillaries into the left heart. From there it enters the greater circulation and may continue to advance along its channels.

My own experiments were the first to show how far this is possible. I took a dog weighing 43.5 kg. and injected in the course of two and one half hours 3650 c.c. of air into the femoral artery (in the direction of its capillaries) under a hydrostatic pressure of three meters (or about 220 mm. of mercury). Only slight disturbances of the heart action and of the respiration were observed.*

Other results,† such as any ultimate injury to the body, cannot be discussed in detail because the subject does not belong within the province of this book. One general fact should be mentioned, however; air circulating in the blood causes an increase of resistance in the capillaries and a diminution of the amount of oxygen that combines with the hemoglobin.

In my above-mentioned experiment (No. 8) the amount of oxygen in the arterial blood after the injection of 1130 c.c. of air, which amount had been introduced within a space of an hour, was 11.12 per cent. by volume; after the forced introduction of 3650 c.c. of air, performed within two and one half hours, it had fallen to 6.8 per cent. by volume. The normal value according to Pflüger is 22.2 per cent. by volume.

It is possible that this diminution of the oxygen percentage which was obtained by such a crude experiment may be present to a certain extent even when small quantities of air circulate in the blood. In the heart it cannot fail to produce a diminution of the quantity of oxygen necessary in a unit of blood for the maintenance of nutrition. This deficiency the heart might make good by increasing its own work. Whether or not a serious nutritional disturbance develops would depend on the ability of the heart to increase its work sufficiently to meet the new conditions.

Dilatation of the left heart by an accumulation of air probably does not occur in a healthy subject. At least, experiments on the dog‡ show that in order to produce dilatation such an increase of pressure is necessary as can be produced only by the will of the experimenter.

(*e*) **Heart Thrombi.**—Blood-clots may form in the cavities of the heart, and very exceptionally may be large enough to interfere directly with its action.

We distinguish:

Pedunculated thrombi: These are firmly attached to the heart wall and project from their point of origin into the cavity of that portion of the heart to which they belong.

Ball thrombi: These thrombi have become detached from the heart wall and are freely movable within the cardiac cavity.

According to R. A. Pawlowski, who has collected all the cases contained in the world's literature, amounting only to 25, 19 occurred in the left auricle, 4 in the right auricle, and 2 in the left ventricle. Corresponding to the seat of the thrombus,—the foramen ovale was frequently found to be the starting-point of pedunculated thrombi or "true polyps,"—the symptoms frequently were those of marked interference with the flow of the blood from the left auricle to the left ventricle with the clinical phenomena of mitral stenosis. Some cases, however, merely presented the picture of cardiac weakness, and even that was not always distinct.

* *Loc. cit.*, experiment 8, p. 458.

† Comp. Heller, Mayer, v. Schrötter, "Ueber arterielle Luftembolie," "Zeitschrift für klinische Medicin," Bd. xxxii. Supplementheft (1897), S. 113 ff.

‡ Comp. the work of Heller, Mayer, v. Schrötter, referred to above.

Death may occur from sudden occlusion of the mitral or tricuspid orifice, but it may also occur with the symptoms characteristic of cardiac insufficiency in general.

It is obvious that the action of the heart must necessarily be interfered with in a manner and to a degree depending on the seat and size of the thrombi.*

(*f*) **Sudden Occlusion of a Large Portion of the Pulmonary Artery.**—Here again the essential factor in the production of the cardiac disturbance which ensues, aside from the caliber of the occluded branches, is the rapidity with which the occlusion takes place. The occlusion in most cases is due to the entrance of an embolus from the veins or from the right heart. Portions of tumors or of parasites (echinococci) are rare forms of emboli. Whenever a foreign body of this kind occludes a portion of the pulmonary artery, a greater amount of work is thrown upon the right heart because the resistance to the passage of the blood in the unobstructed portions of the vessel increases. If the right heart is unable to perform the amount of work required by this increased resistance, the blood accumulates in the right ventricle as well as in the veins of the systemic circulation, while the quantity of blood in the systemic arteries diminishes. In this way death may ensue rapidly.

Lichtheim's† investigations have apparently upset Virchow's ‡ interpretation of these processes, which is as follows: "The interference with the passage of blood through the pulmonary vessels is evidently the primary cause of the disturbance." In these words he summarizes the results of his observations, experiments, and reasonings. Lichtheim found that about three-quarters of the total extent of the pulmonary arteries can be occluded without causing a fall of pressure in the carotid artery. If we may apply this observation to man, we must find some other explanation for the cases of sudden death after embolism of the pulmonary artery, because such cases occur when the obstruction is far less than that stated by Lichtheim. However, the propriety of applying the results of Lichtheim's experiments to the human subject has been attacked in various quarters and, as it seems to me, with very good reason. v. Recklinghausen § urges the following general objection:

"There is a great difference between a healthy dog with good nutrition and vigorous circulation, in which embolism of the pulmonary artery has been artificially produced, and a patient who has been ill and has perhaps been confined to his bed for some time and whose nutrition and cardiac action have in some way become defective. Conic foci have been produced by occlusion of branches of the pulmonary artery only in a few cases and after previously weakening the animal."

More important are the objections that have been urged against the technic adopted in conducting the experiment. Lichtheim worked with curarized animals with the thorax laid open, kept alive by artificial respiration. In this way he created conditions which differ materially from those which obtain in a closed thorax.‖ The importance of this difference has been directly shown by Landgraf ** in the following experiments: In a young rabbit the pressure in the carotid at the beginning of the experiment was 70 mm. of mercury. (It is to be remarked that Lichtheim obtained the same results in rabbits as in dogs.) After ligation of the left pulmonary artery the pressure fell to 30 mm. of mercury. The trunk of the pulmonary artery, and later the right ventricle, also became distended and the left auricle became paler.

* In regard to the possibility of making a diagnosis see v. Ziemssen, "Zur Pathologie der gestielten und Kugelthromben des Herzens," "Verhandlungen des Congresses für innere Medicin," Bd. ix (1890), S. 281 ff.

† "Gesammelte Abhandlungen," S. 307.

‡ "Die Störungen des Lungenkreislaufes," Berlin, Hirschwald (1876).

§ "Handbuch der allgemeinen Pathologie des Kreislaufes und der Ernährung," "Deutsche Chirurgie," Lieferung 2 and 3, Stuttgart, Enke (1883), S. 150.

‖ For the pressure conditions in a normal lung within the closed thorax see Tigerstedt's review.

** "Klinisches und Experimentelles zur Lehre von der Embolie der Lungenarterie," "Zeitschrift für klinische Medicin," Bd. xx (1892), S. 181 ff.

In this experiment the pleura was not opened; the animal was placed under the influence of chloral hydrate, and artificial respiration was unnecessary.

Landgraf,* in summing up, expresses himself as follows: "These experiments have convinced me that, when the anmial is breathing naturally, the conditions are quite different from those which obtain during artificial respiration. In both cases we find conditions which harmonize with those observed in man, namely, a fall of pressure in the aortic system, engorgement of the venous circulation, swelling and dilatation of the right heart."

Tigerstedt† also concludes his very readable review of this entire question with the following words: "This appears to show that Lichtheim's results are correct only within certain narrow limits."

When obstruction of the pulmonary artery develops more slowly its effects on the heart are much less marked and the clinical symptoms are sometimes very slight or even barely noticeable. If the first onset of the storm can be weathered and the heart muscle is not too much weakened, compensation may be established in a comparatively short time. Gerhardt‡ says very aptly: "Although an attack of this kind creates the impression of a profound and exceedingly severe disturbance of the respiration and circulation, severe enough, for example, suddenly to strike down a strong man, the entire clinical picture undergoes a marked change within a short time—at most, two days. The unobstructed portion of the pulmonary system enlarges, the pulse again becomes fairly full, the temperature of the skin and the color of the face are restored, dyspnea is reduced to a very tolerable degree and only returns on very rapid movement."

A few observations on the human subject may be appropriately given here.

Virchow,§ Case 7 (Abstract).—Male, age twenty-four. Typhoid fever of moderate severity. In the middle of the second week death occurred, preceded by the following symptoms:

At the evening visit the patient felt quite well, was cheerful and entirely free from pain. The pulse, on the other hand, as well as the heart-beat, was intermittent without being particularly weak, the intermission occurring regularly after the second or third pulse-beat. Pulse 80, after deducting the missing beats. The patient was perfectly quiet until 11 o'clock in the evening and slept well. About this hour he awoke, asked for the bedpan, and had a copious, liquid stool without any special trouble. He also seized his urine glass and evacuated about 12 to 15 ounces of pale yellow, clear urine. He had hardly done this and said a few words to his neighbor when he suddenly made two or three flail-like movements with his arms, followed by several deep, snorting respirations, and after a few minutes was dead." (Friedreich's report.)

At the section Virchow found the following:

"The main branch of the pulmonary artery in the lower lobe of the lung is occluded by a piece of thrombus 1½ inches in length, lying free within the vessel. It closely resembles in appearance another clot which is found adherent to the wall of the right iliac vein, but presents a ragged surface. A number of other obstructions of branches of the pulmonary artery are also found, as well as numerous thrombi in the pelvic veins of the left side; even the common iliac vein contains a kind of cavernous tissue which indicates former attacks of a similar nature. The heart is flaccid."

The exertion incident to raising himself and emptying the bowel and bladder in all probability caused the embolus to be loosened from its attachment and swept into the blood stream.

Virchow || Case 6 (Abstract).—Female, aged twenty-three years. Light case of walking typhoid. Death occurred about the end of the second week; the time could not be determined exactly.

* *Loc. cit.*, p. 198.

† *Loc. cit.*, p. 451.

‡ C. Gerhardt, "Der Hämorrhagische Infarct," R. Volkmann's "Sammlung klinischer Vorträge—Innere Medicin," No. 31, S. 7.

§ *Loc. cit.*, p. 346 ff.

|| *Loc. cit.*, p. 341, *et seq.*

The following account is given in the history:

"It is said that the patient was busy with fancy work during the afternoon and got up in the evening to make her bed, complaining of chilliness and great lassitude. She was so fatigued that she could not sleep, was restless, and complained of a stitch in the side on deep inspiration. She did not complain of being thirsty or of feeling hot. At 6 o'clock the next morning she ate a little soup for breakfast and felt quite cheerful. About 7 o'clock she sat up to urinate, but suddenly became pale and went into syncope, at the same time uttering a succession of short groans as though she had singultus. She soon recovered, however, but became very red in the face. At 7.30 o'clock she had a second violent attack, with anxiety and pressure on the chest (according to the statement of a surgeon who was busy near the patient), cold sweat, coldness of the extremities, pallor of the face, disappearance of the pulse, and weakness. Consciousness was not lost. After rubbing, brushing, the administration of emetics, and the application of mustard plaster and the like, she came to again with a deep sigh, but the breathing was very shallow, the movements of the thorax being barely perceptible. The face became bluish red, but the entire body remained cold and pulseless; there was great anxiety, the voice was extremely weak, with occasional groans, as during the first attack. There were three convulsive upward movements of the thorax, the limbs were extended, the eyes rolled up, and with a deep sigh the patient expired. When Dr. Reuss (the attending physician) got there at 8 o'clock he found only a corpse."

The autopsy, which was performed by Virchow, showed the following:

"Thrombus in the vena sacra media, from which a clot had extended into the vena cava about an inch. The right auricle contained a plug about as thick as the little finger and five and one half inches in length, evidently of an older formation. It lay quite free in the cavity and was squeezed together in various places, showing that it had evidently been moving. Finally, the main trunk of the right pulmonary artery was filled with a thrombus which was also compressed and lying free within the lumen and was exactly of the same nature as the plug in the heart; it was four inches in length. Behind it was fluid blood, and there were four smaller thrombi in secondary branches of the pulmonary artery. The wall of the vessel showed no changes." The changes found in the heart itself were not significant and had nothing to do with the course of the disease.

The embolus in the trunk of the pulmonary artery was probably carried there during the first attack, but had not produced complete occlusion, which occurred only at the second attack and was then rapidly followed by death.

When branches of the pulmonary artery are gradually occluded, a very large portion of the lumen may be rendered impermeable without causing death. This may take place even in cases of pronounced cardiac weakness due to tissue changes; in fact, perhaps even more likely than in the case of a vigorous heart. This apparent anomaly is readily understood when it is remembered that a feeble heart will propel the plug more slowly instead of wedging it fast at once. This affords time for the development of compensation, in which the bronchial arteries, and through them the left heart, are also concerned. (See the anatomic relations of the pulmonary circulation.)

To what extent this is possible is shown by the following case.

Personal Observation 2.—Male, age sixty years. Has had dyspnea for some years. In the course of the last six weeks this has become worse, and finally very troublesome. Admitted February 11, 1877.

Strongly built, comparatively well-nourished man; marked cyanosis; severe dyspnea; respirations 40; pulse 88, weak, irregular, and distinctly intermittent. Temperature 37.4° (99.3° F.).

Lungs somewhat distended [emphysematous—Ed.]; no signs of focal disease; everywhere vesicular breathing with a few sibilant râles. Heart: Both sides enlarged, particularly the right; sounds weak and pure. The pulmonic sound is extremely weak as compared with the aortic. The peripheral arteries are atheromatous.

On the afternoon of the twelfth of February the patient's face suddenly became distorted, speech became indistinct, and the breathing at the same time irregular. At the evening visit, two hours later, slight paresis was observed in the left side of

the face; the respirations were 44 and irregular; pulse 100, irregular and intermittent. The temperature had risen to 39.2° (102.2° F.).

February 13th: Indistinct signs of focal disease in the lungs; pulse 108 to 112, stronger and less intermittent. Respirations 42 to 46. During the night he had a profuse sweat.

February 14th: Bloody sputum resembling that of pneumonia; possible focus in the left apex. Pulse distinctly stronger. In the afternoon, about 4 o'clock, as the patient, who had been perfectly conscious up to that time, was about to get into bed again after urinating, death suddenly occurred with convulsions.

The autopsy, performed by Schüppel, showed the following (abstract):

The main trunk of the left pulmonary artery is completely occluded by a pale, grayish-red thrombus an inch in length and loosely adherent to the wall of the vessel.

To this thrombus, which has an old appearance, adhere more recent thrombotic masses which occlude all the branches of the artery but are very loosely adherent to the wall.

The main trunk and larger lateral branches of the right pulmonary artery are also filled with completely occluding thrombi of imperfectly laminated structure for a distance of two to three inches. The thrombi are partially adherent; some parts show an older formation, consisting of pale gray and even puriform masses; other portions are evidently recent.

The branches of the pulmonary artery show a considerable degree of atheromatous degeneration.

The heart is enlarged by fully half in the horizontal diameter, and is also longer than normal; the muscle is flabby. The right ventricle contains several old thrombi among the columnæ carneæ, with globular vegetations partly broken down; these thrombi may have formed the origin of the occlusion of the pulmonary arteries. The auricle is free from old clots. The tricuspid and pulmonary valves are normal. The wall of the right ventricle is moderately thickened; the cavity is dilated. The muscle of the right ventricle is pale, brownish gray in color, and friable; the left ventricle is moderately hypertrophied; the myocardium is extremely soft and friable and of a dirty brown color. The valves of the left side present no special disturbances. There is moderate atheroma of the aorta and of the large vascular trunks.

The lungs present old cicatricial retractions and emphysema, edema, small infarcts, and in places collapse or flaccid infiltration. The brain shows nothing but marked edema. Acute miliary tuberculosis: nodules in the lungs, liver, kidney, and the retroperitoneal glands.

The results of the autopsy indicate that both main trunks of the pulmonary artery must have been for some time the seat of clots which interfered with the circulation. These clots gradually spread and thus caused more and more extensive obstruction of the vessels. In spite of this extensive obstruction and the weakness of the heart, it held out simply because the process developed so slowly. It is quite possible that the clots in the pulmonary arteries were apposition (secondary) thrombi, the nucleus having been derived from the right heart. It is by no means positive that sudden death was directly connected with thrombosis of the pulmonary artery. In addition to the circulatory disturbance which had followed thrombosis of the pulmonary artery, there was also miliary tuberculosis. If the heart had been stronger and its muscle had not been damaged, it might have held out longer and certainly would not have given out so suddenly.

Interference with the circulation in the pulmonary artery may be caused by a variety of diseases of the lungs, pleura, or mediastinum. But in every case there are also other disturbances which influence the action of the heart; hence the condition will not be discussed at this point.

(*g*) **Sudden Marked Relaxation of the Vascular Tone.**—A severe blow on the abdomen causes a sudden change in blood distribution and an accumulation of the greater portion of the blood in the veins of the abdominal viscera. The blood-supply to the heart is thereby diminished; the right side receives less blood and, therefore, cannot send as much to the left side of the heart. There is no question whatever that these observations, which are based on Goltz's experiment (noting the effect of blows), are applicable to the human subject.

It is possible that the irritation of the brain incident to violent emo-

tional excitement may act in a similar manner through the medium of the vasomotor nervous system. This is the best explanation for the cases of profound syncope or even death which sometimes occur from psychic causes.

Acute cardiac insufficiency after perforation of an abdominal viscus, allowing the escape of large quantities of fluid of a very irritating character, probably belongs in the same category. Another form is that which follows the ingestion of strong acids or of lye, causing extensive cauterization of the gastric mucous membrane.

The common feature of all these disturbances is that they offer a purely mechanic obstacle either to the entrance of blood into the right ventricle or to its escape from that cavity, so that the left heart no longer receives the necessary supply of blood. As a direct result the blood-supply of the entire heart becomes insufficient and the nutrition of the organ suffers. Even the phenomena which occasionally follow wounds of the heart are often very difficult to interpret. (See above, p. 21.) The truth is, we must take account of other disturbances besides those which external violence may produce on a pumping mechanism; we must also take into account the histologic structure of the heart, with its wonderful arrangement of muscle-fibers, which, like the nervous system with which they are connected, are subject to conditions of which we know very little. It is all the more important that we should try to gain some insight into these disturbances. For this purpose we must take into consideration:

(*h*) **What Occurs When the Passage of Blood Through the Coronary Arteries is Interfered With.**—This subject will be discussed at length in another portion of this work, and I shall, therefore, confine myself to a few remarks in the present connection.

In the first place it is well known that atheroma—the anatomic foundation of impeded circulation in the great majority of cases—is hardly ever limited to the coronary arteries, but extends to a greater or less degree over the entire arterial system. It, therefore, permanently increases the labor of the heart. This is not unimportant because, unless hypertrophy of the left ventricle ensues, the heart, which is constantly working to overcome an increased resistance, has less reserve strength at its disposal. But hypertrophy by no means occurs in every case. Indeed, the stenosis of the coronary arteries caused by the arteriosclerosis, if it is marked, interferes with the development of hypertrophy. Hence the functional power of the heart is somewhat diminished even when there are no demonstrable structural changes in the muscle-fibers. The weakness shows itself chiefly when an extra demand is thrown on the organ, because this necessitates the passage of a certain quantity of blood through the heart in a unit of time. If the stenosis of the coronary arteries interposes an increased resistance, the blood-supply, and with it the functional power, may diminish. When these conditions are permanent, the case is sufficiently clear; but not so in those cases in which we have attacks of angina pectoris. These cannot be directly attributed to deficient work on the part of the heart, for it has been maintained by such authorities as v. Leyden,* A. Fraenkel, and O. Vierordt†—to name only a few—that signs of cardiac weakness may be entirely absent during the attacks.

* "Sklerose der Coronararterien und davon abhängige Krankheitszustände," "Zeitschrift für klinische Medicin," Bd. vii (1884), S. 559.

† "Angina pectoris." A. Fraenkel and O. Vierordt, Referees in the "Verhandlungen des Congresses für innere Medicin," Bd. x (1891), S. 228 ff.

Other factors are not only concerned, but in fact play the most important part. Though opinions differ widely in some respects, there is perfect agreement on one point, namely, that these other causal factors must be sought in that portion of the nervous system which controls the heart.

It is because of this influence of the nervous system that the structural changes which are often found do not harmonize with the functional disturbance. Curschmann,* for example, was unable to find any direct cause of death in most of the cases that he observed and examined post-mortem. The gross tissue disturbances that are found consist of myomalacia or fibroid change (Ziegler).

The explanation of what occurs when no actual changes can be demonstrated in the heart may possibly be furnished by Kronecker's† discovery. In the case of a dog, he found that by inserting a needle into the heart at a certain point of the ventricular septum, corresponding to the boundary of the upper third, about 1 cm. underneath the surface, he caused immediate arrest of the heart-beat and fibrillar twitchings of the ventricle, which continued until the animal's unavoidable death by heart failure. This fibrillar twitching was identical with that observed by Cohnheim‡ and v. Schulthess-Rechberg after occlusion of the coronary arteries. In view of the similarity of the phenomena observed, Kronecker interpreted his experiments by assuming that "this dangerous spot in the ventricular septum contains a nerve-center which supplies the vessels of the coronary arteries with nerve paths. This vascular nerve-center, when stimulated either indirectly or reflexly, may cause anemia of the heart walls and paralysis of the coördinating conduction paths, just as anemia of the brain may produce syncope followed by coma, while the muscles show the Kussmaul-Tenner convulsions." Kronecker, it should be stated, does not admit that the normal stimulation of the heart is conducted through the musculature. It seems to me that this personal explanation of his obviates many of the difficulties connected with his experiment.

(*i*) A similar explanation may be offered for the **phenomena that follow the action of substances to which we attribute toxic effects on the heart.** Heart poisons, in the narrower sense of the term, when ingested in sufficient quantity, are capable of completely abolishing the action of the heart. The dose need not be large, and very small amounts are capable of influencing the action of the heart in certain ways. None of these substances, even when they produce death, cause any recognizable tissue disturbances. We speak of—

Molecular Effects.—All that is meant by this is that, since we are unable to demonstrate any changes in the structure of the tissues in spite of the fact that definite alterations in the vital manifestations of the affected structures are present, it follows that the "poisons" must exert some peculiar influence.

The theory which is most in accord with our present conception of these matters is that the disturbance consists in some change in the relative position of the smallest particles.

The chemic view as to the arrangement of atoms in relation to the various molecules may be taken as a prototype, as it does for the term "biologic affinity," which is also used in this connection. This term "biologic affinity" suggests the explanation that the poisons which circulate in the blood in minute quantities are subject to a special form of attraction on the part of the cell groups which they

* "Verhandlungen des Congresses für innere Medicin," Bd. x, S. 276.

† "Ueber Störungen der Coördination des Herzkammerschlages," "Verhandlungen des Congresses für innere Medicin," Bd. xv (1897), S. 524 ff.

‡ "Ueber die Folgen der Kranzarterienverschliessung für das Herz," "Gesammelte Abhandlungen," S. 623 ff.

influence. It is conceivable that when these poisons are taken out of the blood they accumulate in their sphere of activity, and this may explain why certain definite quantities of the poison exert such a marked influence on minute functional units, such as nerve-centers, for example. These theories are simply rendered necessary by man's craving for a knowledge of causes.

A certain peculiarity of some of the heart poisons ought to be specially mentioned, because it tends to show the intimate connection existing between the muscular and nervous elements of the organ. I refer to digitalis, which affects at least the heart muscle and the pneumogastric nerve.

There are also certain other poisons which have been shown to produce tissue disturbances in the heart, particularly in the myocardium. Phosphorus may be taken as the type of these poisons. The fatty degeneration which this substance produces is sufficient to explain the diminished power of the heart muscle.

Finally, a word in regard to the injury that may be done to the heart by the abuse of alcohol and tobacco. In the case of alcohol at least, the explanation is by no means simple; not only the quantity, but also the method in which the substance is ingested, is important, and the question can be approached from various points of view.

I shall take this point up again in a different connection, and for the present shall merely refer to the important work of Bollinger in reference to the "beer heart."

The effect of the abuse of tobacco will be discussed in connection with cardiac neuroses.

(*k*) **Changes in the Heart in the Infectious Diseases.**—In the main, what we have to deal with in infectious diseases is also the effect of a poison on the heart. In the severest intoxications, such as may be produced by diphtheria, scarlet fever, and smallpox, the diminution of the work of the heart may be as rapid and complete as in cases of actual poisoning. No visible tissue changes are produced.

The case is different when the effect of the injurious substance is prolonged. Profound tissue disturbances may then take place, not only in the parenchyma, but also in the connective tissue of the heart muscle, the nervous portions, the endocardium, and the pericardium. Other causal factors that enter into the question besides the actual poisoning itself, such as increase in body-temperature, disturbances of nutrition on account of insufficient anabolism, may here be disregarded. We know, at all events, especially through the splendid work of Ernst Romberg,* that the doctrine of an acute infectious myositis, first promulgated by Frenchmen (Hayem, Martin), deserves full consideration.

In a special class belong those forms in which the cause of the disease has its seat especially in the heart itself—that is, those inflammations which are more frequently produced by pus cocci than all the other pathogenic microbes.

(*l*) **Changes in the Heart Due to Blood Dyscrasiæ.**—Hemorrhage should be mentioned first. It is evident that in cases of fatal hemorrhage the heart, being deprived of its nutrient material, dies along with the other vital organs. If a patient recovers after the loss of considerable quantities of blood, the symptoms of inadequate functional power will be proportionate to the diminution in the supply of oxygen due to the re-

* "Ueber die Erkrankungen des Herzmuskels bei Typhus abdominalis, Scharlach und Diphtherie," "Deutsches Archiv für klinische Medicin," Bd. xlviii (1891), S. 369 ff., und Bd. xlix (1892), S. 413 ff.

duction of the number of red blood-corpuscles. The same thing occurs in all diseases of the blood associated with a decrease of the red blood-corpuscles, of which leukemia may be taken as the type. Permanent dyscrasia leads to degenerative processes in the heart muscle, which develop early in proportion to the amount of work the heart is compelled to do.

On the other hand, the symptoms are somewhat more obscure in the conditions which are included in the group of so-called "wasting diseases"; for in these cases the conditions which are responsible for the disturbance can by no means always be recognized with absolute certainty, as, for example, in diabetes mellitus, a disease in which heart paralysis due to degeneration or atrophy of the muscle is sometimes the cause of sudden death. "That the heart muscle is the seat of rapid metabolism as well as of a degenerative process is evidenced by the finding of numerous young glycogen-containing muscle-fibers beneath the endocardium and in other places." So says Frerichs,* who qualifies his statement as follows: "It is possible, and indeed probable, that other as yet unknown factors are also concerned, and that during moments of special exertion, which almost always precede sudden, unexpected death, these unknown factors render the organism incapable of responding to the increased demand for oxygen and metabolism, and thus interrupt the continuation of life." These other factors suggested by Frerich in cases of diabetes mellitus sometimes become much more prominent than the cardiac changes, so that death is brought on through some cerebral disturbance. Why these variations when the fundamental changes are the same? That is a question which cannot as yet be answered.†

It should be pointed out that similar phenomena of sudden disturbance of cerebral activity are observed in other wasting diseases, such as carcinoma, chronic nephritis, cirrhosis of the liver, pernicious anemia, characterized by a diminution of the hemoglobin in the the blood, a symptom that is usually not present in diabetes mellitus. Hence the theory advanced by Senator and Riess must, for the present, be accepted as the most probable one. According to this theory these conditions are to be attributed not only to profound nutritive disturbances in the blood itself, but also to some form of poison elaborated within the body.‡ This view finds considerable support in Fr. Müller's investigations on the metabolism of cancer patients.§ These investigations prove that a poison may be formed in cases of carcinoma and may cause degeneration of the protoplasm. It is not difficult to understand how the heart may also be involved in such a process.

With becoming reserve Fr. Müller says: "An increase in the destruction of albumin appears to be common to all diseases associated with cachexia, and it seems also to play an important part." If that is the case, it will help us to gain a clearer insight into the question. For on

* "Ueber den Diabetes," Berlin, Hirschwald, 1884, S. 120, 121.

† See C. v. Noorden, *loc. cit.*, S. 406 ff.; H. Senator, "Ueber Selbstinfection durch abnorme Zersetzungsvorgänge, u.s.w.," "Zeitschrift für klinische Medicin," Bd. vii (1884), S. 235 ff.

‡ See L. Riess, "Ueber das Vorkommen eines dem sogenannten Coma diabeticum gleichen Symptomencomplexes ohne Diabetes," "Zeitschrift für klinische Medicin," Supplement zum 7. Bde. (1884), S. 34 ff.

§ "Zeitschrift für klinische Medicin," Bd. xvi (1889), S. 496 ff. See C. v. Noorden, *loc. cit.*, S. 458 ff.; Ernst Grawitz, "Klinische Pathologie des Blutes," Berlin, Enslin, 1896, S. 315 ff.

that theory the heart, being supplied with blood of lowered efficiency and its principal portions—those which contain albumin—being prone to degeneration, must be regarded as being in greater danger than any of the other organs. For its work, which is always associated with the consumption of tissue, is indispensable; it continues and must continue throughout the individual's life.

(*m*) **The heart performs its work by contracting; hence whatever interferes with contraction injures its functional power.** Any deposition of non-contractile elements in the muscle tissue or on the walls of the heart may be a disturbing factor. Whatever is deposited within or upon the heart must follow the movements of the viscus. Hence, to effect the necessary diminution in size during systole, the heart must exert itself enough to overcome the inertia and tension of the non-contractile portions embedded in its substance. They are inert masses that are moved with the moving muscle and must adapt themselves to its shape. This is a mechanic necessity, and it is evident that a certain portion of the kinetic energy of the heart is, therefore, diverted from its proper function of propelling the blood.

In addition it must be remembered that the seat of these foreign bodies, as we may term them in a single word, may also be of importance. If they are situated near the coronary branches, they may cause permanent narrowing of these vessels. To what extent their proximity may disturb the nervous structures in the heart or interfere with the regular contractions of the organ is a question that may be asked but cannot as yet be positively answered.

I remember a case of sudden death in which a small syphiloma situated in the septum of the heart was all that was found at the autopsy. The patient, a prostitute, had collapsed while dancing.

Diastolic relaxation is perhaps also somewhat impeded, but this is a less important factor. Large foreign bodies projecting into the interior of the heart diminish the space intended for the reception of the blood and may in that way diminish the amplitude of contraction. The same effect may be produced because the distending force of the blood-stream meets with increased resistance due to the presence of non-contractile material in the muscle tissue.

Finally, it must not be forgotten that some of the injurious conditions about to be mentioned directly interfere with the nutrition of the heart muscle.

Such conditions, we learn from the pathologists,* are infectious granulomata (tubercles, syphilomata, actinomyces); tumors (sarcoma, lymphosarcoma, fibroma, lipoma, myxoma, rhabdomyoma—metastatic carcinoma); parasites (cysticerci and echinococci).

In addition, deposits of fat on and within the heart muscle, the various forms of muscular degeneration, especially the formation of extensive fibroid patches, should be mentioned.

This division also includes the changes which are brought about by adhesion of the heart to its covering, which has become rigid [thickened and adherent pericardium—Ed.], or to neighboring structures. In this condition a considerable portion of the kinetic energy of the heart muscle is diverted from its proper function, which is to subserve the circulation.

(*n*) There remain finally the **disturbances of the functional power**

* See Ziegler's "Lehrbuch der speciellen pathologischen Anatomie."

of the heart due to defective innervation. These have in part been referred to elsewhere, because it is not practicable to draw sharp lines of distinction between the special province of the muscular and that of the nervous system. Nevertheless, the chief part of this subject remains to be discussed, and this will have to be done briefly because a detailed discussion would lead us too far away from our subject.* Suffice it to say that the cardiac nerves may suffer a functional injury at any point along their course, from their origin to their termination, and that any such injury must affect the action of the heart. Injuries, as from traumatism, pressure by tumors, etc., acting on the center for the inhibitory nerve-fibers in the medulla oblongata and on the trunk of the vagus, are most readily recognized. Injury to the accelerator fibers is more difficult to detect because we have no accurate knowledge of the center for these fibers, although it is probably also situated in the medulla oblongata. In addition, there are all the various reflex influences to which these nerve-centers are exposed. There is no doubt that these reflexes may originate in many different quarters and that Tigerstedt's† view, expressed in the following words, is quite justifiable: "It seems to me more correct to regard the cerebral cortex and the other portions of the brain lying in front of the medulla, so far as their influence on the heart's centers and in general on all the vegetative centers of the medulla is concerned, as peripheral organs with reference to the medulla. The centers in the medulla receive reflex stimuli from the cortex in exactly the same way as they are stimulated through centripetal fibers coming from the other portions of the body.

"According to this view, the true center for the inhibitory cardiac nerves is situated only in the medulla. This center may be influenced by a great many nerves which must be regarded as centripetal with reference to this center, situated in the skin, in the heart itself, in the abdominal viscera, in the lungs, in the organs of special sense, and in various portions of the brain."

The older teaching that the heart ganglia are automatic centers for the movement of the heart is no longer tenable. The heart ganglia are exclusively sensory.

E. Romberg,‡ who in association with the younger His determined this point, has this to say in regard to its significance: "Nothing definite is as yet known in regard to their individual functions. It is possible, perhaps probable, that they transmit to the central nervous system the minutely graduated, unconscious sensations which reflexly regulate the action of the heart through the vagus and accelerator nerves and control the caliber of the blood-vessels. Hence the activity of the cardiac ganglia, to borrow Nothnagel's words, must still be regarded as 'a *conditio sine qua non* for a normal circulation,' but not in the same sense as we have until now believed. They control the rhythm of the heart not directly, but indirectly, through reflex paths. It is due to the action of the cardiac ganglia that the heart is able so completely to respond to the various demands upon its functional power."

Here, again, we therefore have to deal with probable reflex effects which, as elsewhere, may become operative without any other than a functional stimulation taking place in the part where they originate.

* See Tigerstedt, *loc. cit.*, p. 193. † English translation, p. 195.

‡ "Beiträge zur Herzinnervation," "Verhandlungen des Congresses für innere Medicin," 1890, Bd. ix, S. 363.

In the present state of our knowledge we must be content to forego any further inquiry into the nervous processes which influence the functional power of the heart muscle. No one can doubt that these processes do exert a marked and far-reaching influence on the heart muscle. It may be possible occasionally, in an individual case, to gain some further understanding of the process; but in the main this difficult question remains to be solved by future investigations.

Let us now turn to the **second principal division:**

II. In the following we shall discuss those causes of cardiac insufficiency which involve an insufficient blood-supply to the heart for the work which they impose upon the organ.

The determining factor in this group is that the work imposed upon the heart brings with it conditions which produce an insufficient blood-supply. That is to say, there is a disproportion between the demand and the possible supply of effective and nutrient blood, and this disproportion is caused by the work of the heart itself.

Let me repeat once more Benno Lewy's fundamental dictum:

"Since the increased action of the heart incident to muscular exertion is regularly associated with an increase in the number of pulse-beats, which in turn is effected at the expense of diastole, it follows that during excessive muscular exertion, even when the amount of nourishment taken up by the digestive organs is otherwise adequate, the heart labors under unfavorable nutritive conditions. Hence for mechanic reasons the heart can only perform an increased amount of work for a limited space of time. After that, if it is to remain healthy, the diastole must be long enough to permit of a sufficient blood-supply to the myocardium, that is to say, retardation of the pulse and relative heart rest must ensue."

While this applies chiefly to temporary overexertion of the heart and its consequences, due to muscular work, the principle can nevertheless be extended to other conditions as well. I refer to those conditions in which the work of the heart is permanently increased, and adequate recuperation is rendered difficult or impossible.

There is another reason for this subdivision. Whatever interferes with the work of the heart most commonly develops gradually and not all at once, so that an opportunity is afforded for the heart to increase its musculature and, by dilating its cavities, to enlarge its amplitude of contraction. In this way compensation may be brought about and the heart may be enabled more or less completely to do its work again, at least for a time. It is almost needless to say that a heart which has undergone such a change requires a much larger blood-supply, and this determines the limit beyond which it cannot increase its work.

Like every other attempt at classification, the one here suggested has its weak points.

The conditions observed in suddenly developing diseases of the lungs or pleura must be judged differently, because in these cases hypertrophy of the heart muscle, in the beginning at least, is not present.

Cases of sudden dilatation of the heart produced by excessive muscular exertion might more properly be included in the first principal division—among those disturbances which, acting directly on the heart itself, injure its mechanism.

Other objections might be urged; but it seems to me that, in order to obtain general points of view, the details may be sacrificed.

I shall, therefore, consider together:

1. The effects on the heart of excessive general muscular exertion (heart strain).

2. Alterations in the valvular apparatus and orifices of the heart on account of which the kinetic energy furnished by the muscle is in part diverted from its function of subserving the circulation.

3. Permanent increase of resistance in the greater and lesser circulation.

4. Diminution of the motive power furnished by the auxiliary organs and at the disposal of the circulation; that is to say, everything that impedes the movement of the lungs within the closed thoracic cage.

1. Let us first take the simplest of these conditions: **the effect of excessive muscular exertion on the heart.**

We must distinguish between what occurs when the demand on the heart is increased only for a short time and the effect on the organ of increasing its labor for a longer period. I shall first cite a few examples:

Allbutt reports an experience of his own in the following words:* "In the summer of 1868 I began to walk in the Alps a little too soon for good training. After three days, walking on lower levels, but for longish distances, K. and I ascended the Galenstock and the next day crossed the Oberaarpass. Instead of starting from the Grimsel, we remained at the Rhône glacier, crossed the Grimselpass from thence, ascended the Sidelhorn before settling down to the day's work. At the end of the day again, instead of dropping down on Viesch, we determined to seek the better quarters of the Aeggischhorn; and had accordingly to mount that sturdy little Alp by a somewhat rapid ascent. Hitherto I had been in good condition; but the new call for combustion to meet the demand for the additional force required to lift 11½ stone (1 stone is equal to 6.35 kg. or 14 pounds avoirdupois) to a height of, say, 2000 feet, threw a great stress upon the right heart; and I was rather suddenly seized with a strange and peculiar *'besoin de respirer,'* accompanied by a very distressing sense of distention and pulsation in the epigastrium. On placing my hand over my heart, I felt a laboring diffused beat all over the epigastrium. I at once opened my shirt and ascertained by percussion that the right ventricle was very greatly dilated. I, therefore, threw myself at length upon the grass, with my shoulders raised, and had the satisfaction, in a few minutes, of finding the distention, the oppression, and the dulness recede. I was then able to rise and sit down, or even to move about on the level, but, curiously enough, the instant I began to ascend, the symptoms returned.

"I was, therefore, obliged to send K. forward and to proceed myself with great caution. When I got up to the height of the inn and had only to walk a mile or two by the waterway, I ceased to suffer, as I felt no general fatigue whatever, and was able to dine well on my arrival. In the night, about 3 A. M., I was suddenly awakened by a severe and distressing palpitation in the epigastrium, with great dyspnea; there was not, however, the same extension of dulness over the sternum. I went to the window, and drew a few long respirations, which gave me ease, and I lost my ailment altogether. No doubt the pressure of a full abdomen against the diaphragm while recumbent had again embarrassed the overtaxed right ventricle.

"Christian Almer, to whom I described my symptoms, said that the same thing had occurred occasionally to himself and to other guides when cutting a number of steps on steep slopes."

This case shows conclusively that increasing the work of the heart for a certain length of time causes weakening of the heart muscle. The weakness, however, is not absolute. It manifested itself only when a greater demand was made on the heart, which was already suffering from an insufficient blood-supply as compared with the original, by no means inconsiderable, demands upon it. The increased demand in itself need not by any means be very great. A very slight increase suffices to bring on insufficiency of the muscle, which has already almost reached the limits of its functional power. The muscle is not permanently injured; indeed, it retains its efficiency within certain limits, for by exercising great care and

*See Seitz, "Die Ueberanstrengung des Herzens," VI. Abhandlungen, Berlin, Hirschwald, 1875, S. 9. "Ueber die Folgen der Einwirkungen von Ueberanstrengung und Gewalt auf das Herz und die grossen Blutgefässe."

walking slowly, with frequent periods of rest, Allbutt succeeded in reaching the highest summit. Another interesting point is that complete recovery takes place in a short time.

The same thing happened to the guides—and this is a point that deserves to be specially emphasized, in spite of the fact that they were accustomed to the heaviest kind of muscular work, they were subject to temporary attacks of heart weakness.

A single overexertion of the heart is capable of causing serious permanent injury to the organ.

Thus Fraentzel* reports the following case:

"On the twelfth of October, 1877, J. B., workman, thirty-seven years old, was admitted to my wards suffering from severe dyspnea. His previous history was that he had gone through the last three campaigns in perfect health and had never suffered from swelling of the joints. He gave up his original occupation of boatman some time ago and became a brick-carrier. He spent the day transferring bricks from boats in the river (Spree) in a so-called tray to the shore and loading them on a wagon, and in doing so excelled all his fellow-workmen by his great bodily strength, which was untiring. While the latter carried thirty-six bricks (two bricks weighing fifteen pounds) at a time, he carried six more, or, in other words, he transported 315 pounds while his fellow-workmen carried only 270. One day in 1876, in a spirit of bravado, to show off his enormous strength, he loaded a few more bricks on his hod than usual; he is not quite sure, but he thinks it was six. On attempting to raise this load from the bottom of the boat to his shoulder he felt such an intense pain in the left side of the chest, radiating into the left arm, that he was obliged to drop bricks and hod and was unable to continue his work. Dyspnea and a feeling of unrest in the chest came on at the same time, and he was barely able to get home. He could get no rest either day or night and, after an interval of a few weeks, during which he had become dropsical, he applied for admission to the Moabit Hospital, where he spent sixteen weeks. When he left the institution he felt quite well and returned to his former occupation. But he found that his working capacity had greatly diminished—he could only carry half as many bricks as before. Gradually his strength returned, and in the course of a few months he was as strong as ever and felt quite well.

"On the twelfth of October, 1877 he again tried to carry more than his usual number of bricks, which was forty-two, although he is not quite sure of the actual figures. At the very instant when he tried to raise the load he felt the same pain in the region of the heart and of the left arm as at the first attack. But this time the pain was so severe that he collapsed and it was about an hour before he recovered sufficiently for two of his friends to take him to his house, which was nearby. Here he suffered from frightful dyspnea, and on the same day dropsical swelling of the face and of the limbs also developed. On the next day cough and a boiling feeling in the chest made their appearance, and in the evening he applied for relief at the Charité."

On the morning of the fourteenth of October Fraentzel found the patient in an exceedingly dangerous condition:

"The man, who is of Herculean build, is sitting upright in bed and complaining of dyspnea; his voice is almost completely gone. The sensorium is unaffected. The expression of the face is that of profound collapse; the forehead is covered with a few drops of cold perspiration; the nose and extremities are cold; there is slight edema of the face, nose, and forearms, and more marked edema of the legs.

"Extreme objective and subjective dyspnea, 38 respirations in the minute. Loud, groaning expiration and occasionally tracheal rhonci which can be distinctly heard in the room. All the auxiliary muscles of respiration are working vigorously and there is marked inspiratory retraction of the lower intercostal spaces. Active expiration. Short cough at brief intervals; sputum: about eight tablespoonfuls of a thin, fine, frothy, somewhat reddish fluid. Examination is very difficult because the slightest movement places the patient in imminent danger of asphyxia. The thorax presents the typical barrel shape. The percussion-note in front on both sides as far as the fourth rib is loud and deep and of the same quality posteriorly as far as the seventh rib; from that point on there is moderate dulness, gradually increasing

* "Vorlesungen über die Krankheiten des Herzens," Berlin, Hirschwald, 1889, Bd. i, S. 112 ff. The history of the case is here given in condensed form.

downward. Over the lungs everywhere vesicular breathing is heard, with fine mucous râles, which vary greatly in number at different times; over the area of dulness these auscultatory phenomena are also present, but appear to be less distinct. The apex-beat is not visible, but can at times be felt in the fifth intercostal space in the nipple-line.

"The heart dulness begins at the left sternal border at the upper edge of the fourth rib; it cannot be separated below from the liver dulness, and extends to the left 2.5 cm. beyond the nipple-line, and to the right 2 cm. beyond the right border of the sternum.

"The heart-beat is very irregular—from 148 to 160 to the minute.

"The heart-sounds are replaced by a feeble, hollow sound heard during systole, which appears to be a murmur.

"The radial arteries are very small and the tension is diminished; the pulse is quite irregular, the pulse-wave varying greatly in height. There is marked delirium cordis.

"The tongue is very cyanotic, but clean and moist. There are great thirst and complete anorexia. The abdomen is greatly distended, very tense, and exceedingly sensitive to pressure in the epigastrium and at the lower border of the ribs on the right side. The liver dulness is greatly enlarged; the outline cannot be accurately determined on account of the patient's condition.

"The percussion-note over the lower portions of the abdomen from the level of the umbilicus down is intensely dull. The bowels are sluggish. The urine is very scanty (since the patient's admission the day before he has barely voided 250 c.c.), brownish red, with a heavy brick-red sediment, contains small quantities of albumin, but microscopic examination shows no morphologic elements and only uric acid and its salts.

"Temperature 37.5° C. (99.5° F.)."

The subsequent course was as follows: The worst symptoms subsided in a few days. At the end of about two weeks the patient was quite comfortable so long as he remained quietly in bed; but the slightest movement—even walking only a few steps to the closet—brought on the most frightful dyspnea and the face and extremities became deeply cyanotic.

Not until the middle of December was the patient able without considerable discomfort to walk from his bed to an armchair in the ward and sit in it for a few hours. Although the heart dulness had considerably diminished and the heart-sounds had become pure, albeit hollow, the irregularity of the heart's action persisted. The same condition was found when the patient was examined on the first of July, 1878, although the heart dulness had still further diminished. The patient was unable to go upstairs and permanently quite helpless and unable to work.

He was discharged on the twenty-fourth of February, 1879, but was again admitted on the thirty-first of March in a very sad condition: beginning gangrene of the toes and perityphlitis with fever. From this he recovered; but the history, which was brought to a close in the year 1889, contains the following note:

"The condition of the heart, especially the irregularity of the heart's action, has so far undergone no change whatever; the patient is unable to work and his disability is permanent."

This case is of the greatest importance. Oscar Fraentzel's interpretation of the symptoms appears to me to be unassailable; it may be summed up in the following words: A single, momentary overexertion of the heart was the cause of a severe, persistent, and indeed permanent injury of the organ. The two attacks were at first entirely similar, and are to be regarded as a sudden failure of the heart, which until then had possessed unusual power. The cause was dilatation of the left portion of the heart, a condition that may be produced by the enormous increase in pressure that takes place in the aorta after a momentary excessive muscular effort. It is very remarkable that all the effects of cardiac insufficiency, especially general anasarca, developed in such a short time—in the case of the second attack within a few hours.

The first time the heart recovered completely, albeit after a considerable interval, and was again able to respond to the former extraordinary demands made upon it. The second time it did not recover. Although

the heart dulness diminished,—showing that the dilatation had subsided,—the heart was, nevertheless, permanently disabled by the single overexertion. How is that to be explained? In my opinion as follows: The intense dilatation unquestionably produced a structural disturbance in the tissue of the heart, and this disturbance interfered with the regular coöperation of the individual muscle-fibers of the heart that takes place under normal conditions. The disturbance may have been due in part to nervous influences or perhaps solely to some change in the relative position of the muscle-fibers. This is a point that cannot be definitely settled. But, however that may be, after the first attack a restoration of the original conditions was effected by means of rest, which allowed the heart to be sufficiently supplied with blood. After the second attack the heart, in spite of rest and care, never recovered sufficient power to restore the degree of pressure in the aorta necessary for its own nutrition. The first attack was not followed by an increase in the mass of the muscle or hypertrophy, because the increased demand was not permanent, nor was there dilatation. For the heart had returned to its normal condition and was able to perform the amount of work required of it without becoming fatigued. After the second attack, when the heart became permanently unable to contract and relax sufficiently, one would naturally have expected dilatation to occur. Why it did not develop it is difficult to say. It might be suggested that most of the blood was retained in the veins of the greater circulation and that the amplitude of contraction became so small that only very small quantities of blood were set in motion; in other words, there resulted a permanent diminution of the difference in pressure in the arteries and veins. It is, of course, assumed that the right side of the heart also lost some of its power; for there can be no possible doubt of that.* The whole history of the case helps us to understand what happens when the demand on the heart is permanently and excessively increased.

The most valuable data bearing on this question are derived from observations made on soldiers after unusual exertions in the course of their regular duties. Da Costa† had an opportunity to see a large number—more than 300—of patients of this kind during the Civil War and he quite justly remarks:

"It is very possible that from inherent circumstances our war furnished more material of the kind than is likely soon to be met with again; for so many men called, by the tap of the drum, from civil pursuits, and sent without previous training into the field, is not a state of things likely often to happen."

The contributions of all those physicians who have busied themselves with this matter present practically the same feature: At first the phenomena of heart fatigue which, if the course is entirely favorable, are followed by complete recovery, in less favorable cases by organic changes in the heart, namely dilatation and hypertrophy; that is to say, a permanent injury, the results of which vary in the individual case according to external conditions.

It should be emphasized that Da Costa mentioned among the factors which specially favor the occurrence of this form of cardiac disturbance fever and diarrhea. These conditions, which reduce the general nutrition of the body as a whole, were present—the former in 17 per cent., the latter

* See the section on Hypertrophy and Dilatation.

† "Ueberreizung des Herzens," "Eine klinische Studie über eine Form von functioneller Störung des Herzens und ihre Folgen," bei Seitz, *loc. cit.*, S. 41 ff.

in 30.5 per cent., of the cases; that is, taken altogether, in almost one-half of all the cases (200), and preceded the cardiac symptoms.

In addition to these facts I shall quote Allbutt's * observations, which, from a practical standpoint, are very important:

"Let us now pass from well-nourished individuals to those who, either because of carelessness or poverty, did not get sufficient food. Among cases of this kind I find two which were described by my friend, and former teacher Dr. Paget, in Cambridge. Dr. Paget says, in a letter to me:

"'Two cases are set down in my memory in which dilatation of the heart existed in a high degree without valvular disease and in which I could make out no cause for the disease except the habit of taking long and active exercise fasting. Both the patients were men of active, energetic habits and high courage and endurance; both were tall and strong, one of them rather stout. They were in the habit of going without food from breakfast until a late dinner. I satisfied myself that no other cause was assignable except the long-continued exertion and the neglect of due supply of nourishment. I have met with other cases, as you no doubt have, in what seemed to be the beginning of a like state of the heart which has been arrested by the simple expedient of a sandwich box and a flask.'"

These impressions of an independent, as well as a keen observer are further confirmed by the observations of English physicians among the young men in the universities of Oxford and Cambridge, who are such enthusiastic athletes, that without good food and careful training heart fatigue may easily ensue. The same thing is shown by what occurs when new recruits are trained, at least, in the German army.

To sum up, the following general rule may be formulated: The better the nutrition of the body as a whole, which cannot be separated from the nutrition of the heart, and the more the heart is trained, the less likely is cardiac fatigue to develop. But there is a maximum which varies for each individual case and which must never, under any circumstances, be exceeded. Let us now examine those cases in which the heart is for years subjected to a strain. The importance of this group of cases has only lately been properly appreciated.†

Why? To answer this question I shall cite v. Leyden's very apt remarks: Among German and French writers, at least, stress has been laid on the changes of the heart which can be demonstrated by physical examination—the most frequent of which is dilatation. The etiology was considered a matter of secondary importance.

This method has been followed by German and French authors in their study of heart diseases in general ever since the founding of physical diagnosis.

There is no doubt that this method has been the means of greatly increasing our knowledge of exact scientific diagnosis, but it also has its disadvantages. Its defect is that it makes a disease out of what is merely a pathologic condition of a single organ.‡ v. Leyden also emphasizes the importance of recognizing the causal factors in judging the course of the disease and in laying down the treatment. This point of view is particularly important in the case of disturbances of the heart, because it enables us to take a comprehensive view of the entire subject.

The above-mentioned cause of heart disease is being recognized more and more generally. Observation has taught us that the special demands on the heart are not necessarily associated with definite occupations. The fundamental condition in every case is an increased demand to which the

* See Seitz, *loc. cit.*, p. 41 *et seq.*

† See Leyden, "Herzkrankheiten in Folge von Ueberanstrengung," "Zeitschrift für klinische Medicin," Bd. xi (1886), S. 105 ff. Johannes Seitz, "Zur Lehre von der Ueberanstrengung des Herzens," nach Beobachtungen auf Herrn Prof. Biermer's Klinik (Zürich), "Deutsches Archiv für klinische Medicin," Bd. xi and xii (1873-74); reproduced in Seitz's monograph, which has been referred to.

‡ *Loc. cit.*, p. 110.

heart cannot respond permanently. One must agree with v. Leyden* when he says: "It would be a mistake to suppose that the laboring portion of the population enjoyed the sorry privilege of becoming ill as the result of hard work; for the same heart diseases are met among those in better circumstances and even in the highest circles. The better classes also work and expose themselves to excessive exertion, either in their occupations or voluntarily during athletic pursuits. I have seen a considerable number of cases of this form of heart disease among army officers of high rank, sportsmen, foresters, and others of that class. Merchants, officials, and physicians also develop similar conditions. Again, the female sex is by no means exempt. I remember a well-known actress who died early of cardiac dilatation and who had sown the seed for her disease by going through the exertion and excitement of a starring performance in a convalescence from diphtheria."

It may be objected that the theory of overexertion of the heart is in danger of becoming too general, not to say altogether vague, by such a wide application of the term.

To this we may reply: The essential consideration is that the heart is unable to respond to the demands made upon it. The amount of kinetic energy furnished by the heart muscle varies quite as much in the individual case as does the demand which an individual makes upon his heart. It seems to me that in this discussion, which is confined to functional disturbances and takes no account of anatomic changes, it makes little difference whether a structural change is present in the heart or not; in the last-mentioned case of v. Leyden's there probably was. Tissue changes merely cause a diminution of the available energy. If the demand had been less, impairment of the continued function need not have resulted. But, conversely, permanent impairment of function would have occurred even if the heart muscle had been entirely intact, provided the demand made upon it had been in excess of its functional power.

It is impracticable in my opinion to attempt a sharp distinction between these things—it is quite enough to call attention to differences in the conditions present in the individual case.

As an example of the way in which the mode of life of a certain class of the population may favor the occurrence of cardiac insufficiency I shall describe the conditions found among the vine-dressers in Tübingen.†

These people work hard and are not particularly well nourished. They are mostly field hands, and the women also have to work steadily to help earn a living. A few small and usually only moderately productive vineyards, a few acres of poor, unproductive land, and a few head of cattle, besides an occasional day's work, constitute their entire source of income. It need hardly be pointed out that these people are usually insufficiently nourished for the amount of energy they use up. The food they eat is hard to assimilate, consisting of potatoes, coarse bread, and very little meat. Their chief beverage is cider; they also drink some beer, but very little brandy.

The fact that the land they cultivate is situated on hillsides of varying grades and not on a level plain makes their work especially hard. They are compelled to use up an unusual amount of energy in tilling their land. They not only have to carry the manure up on their backs, but also get their produce home by the same laborious method.

How much work does a man have to perform in such an occupation?

* *Loc. cit.*, p. 109.

† See Dr. Wilhelm Münzinger, "Das Tübinger Herz; ein Beitrag zur Lehre von der Ueberanstrengung des Herzens. Aus der Tübinger Poliklinik," "Deutsches Archiv für klinische Medicin," Bd. xix (1877), S. 449 ff.

With a heavy load of manure in a hod on his back, hung from the shoulders by two straps, the farmer climbs up the hillsides. The load on his back forces him to bend far over in order to keep his center of gravity well forward. To neutralize the forward and downward pull of the straps he has to draw his shoulders well together in front. The arms are crossed over the chest. In order to give the shoulder muscles a better purchase, the thorax is held in the inspiratory position so that the glottis is opened as little as possible. The abdominal muscles are vigorously contracted as in raising any load, hence the abdominal cavity is under high pressure, which reacts on the lower surface of the diaphragm. The large amount of muscular exertion necessary to raise a load of about 40 kg. (88 pounds), besides the weight of the man's body, a vertical height of 30 to 70 meters (32.8 to 76.3 yards) requires a large amount of air for respiration. To take deep breaths with widely opened glottis and large excursions of the diaphragm would necessitate a constant change in the position of the center of gravity and constant compensatory movements of the body muscles to prevent falling. For this reason alone the man, as he climbs up the slope and at the same time carries his load, is forced to content himself with superficial breathing; besides, frequent short respirations effect a greater interchange of air in the same unit of time than deep inspirations at long intervals. The reciprocal effect of these conditions is to produce a permanent inspiratory distention of the thorax, in which all the portions of the lung are not exposed to the same pressure. The apices are exposed to less pressure from without and, on the other hand, have to receive the air which is under greater tension in the other portions of the lung. They are usually permanently distended and indeed actually emphysematous.

Under these conditions an increased demand is made on the heart. Not only does the expenditure of muscular energy necessitate the elimination of enormous quantities of carbon dioxid that are formed as the result of the increased consumption of oxygen by the overactive muscles; but the mechanic conditions created by the act of hill-climbing, which have just been explained and which diminish the extent of pulmonary surface available for the interchange of gases, necessarily force the right heart to increased exertion. The respiratory muscles are in part employed in raising the load, so that the gaseous interchange required for tissue respiration must be effected by the heart forcing a larger quantity of blood over the pulmonary surface at a higher velocity.

Similarly, the work of the left heart is also increased. Traube long ago pointed out that muscular contraction brings with it obliteration of the capillaries contained in the contracted muscle, diminution of the diameter of the arterial system, and increased resistance within it—all of which increases the work of the left heart.

Let us examine our vine-dresser more closely. The man has covered considerable ground. The breathing becomes more and more rapid, betraying his increased need for air. He is forced, on account of air-hunger, to rest for a time before he can continue his climb up the hill. At last he arrives at the top, panting and gasping, with violently beating heart.

It is not too much to say that it requires a naturally vigorous and constantly well-nourished heart to respond to such a demand, repeated day after day.

It seems to me that the question may be raised whether heredity may not enter into the problem.* The mode of life of these Tübingen vintagers has been the same for centuries. Generations after generations have climbed up the hills, carrying heavy loads on their backs. The people usually marry among themselves and new blood is rarely introduced.

It is evident, from what I have said in regard to their customary food, that the general nutrition is very deficient. A workingman is able to perform a uniform quantity of work, and his muscles retain their functional power only so long as the supply from without is sufficient to make good the loss. This applies to the heart muscle and to the muscles of respiration even more than to all the other muscles of the body, because they have to work harder and more continuously. Another factor is that boys, as well as girls, but chiefly the former, are required to do the hardest kind of work quite early in life —about the fourteenth or fifteenth year. Hence it is not astonishing that the effects of heart fatigue show themselves so frequently.

I had never seen any conditions of this kind in my former sphere of activity among the inhabitants of Kiel. There people also worked hard, but they lived on level ground and were better nourished. Even among the stevedores, who were employed to unload the vessels and had to carry heavy sacks to the warehouses, such an occurrence was unknown, or at least so rare that other explanations could be found in the individual case. As for the facts being such as I represent them, I can offer the testimony of Bartels, for I showed him specimens of the Tübingen heart in patients and at the autopsy table, and to him, experienced physician that he was, the condition was entirely new.

There is one more point that I must touch upon: "Constantly repeated severe muscular exertion frequently, although not always, gives rise to other alterations which develop *pari passu* with the direct overexertion and of themselves permanently increase the labor of the heart, even when the body is at rest. I refer to atheromatous degeneration of the arteries and distention and emphysema of the lungs. The heart changes observed under the above-described conditions of life were formerly attributed to these conditions. Atheroma was held responsible for the disturbances in the left heart, while the phenomena observed in the right heart were ascribed to emphysema. To be sure, such an interpretation is perfectly justifiable, but the exciting cause of these gradually developing tissue-changes is the same as that which directly produces the overexertion of the heart, namely, hard physical labor. The effects of hard work on the heart are much more marked when emphysema and atheroma have developed, for in the presence of these changes the heart tires more rapidly because the constant demand made upon it is increased. Nevertheless, the doctrine that the heart can be weakened by excessive exertion remains unconfounded.

2. and 3. We have now to consider those **changes in the heart itself which permanently impair its functional power, namely, disturbances affecting the valvular apparatus and the orifices.** As this subject will be discussed at length in another place, we may confine ourselves here to a few general remarks.

What is the function of the valves? To regulate the flow of blood as it enters and leaves the heart in such a way as to make certain that the blood moves in the proper direction and that the kinetic energy furnished by

* Lichtheim is not disinclined to answer this question in the affirmative. ("Verhandlungen des Congresses für innere Medicin," Bd. vii (1888), S. 43.)

the heart muscle is completely utilized. If the valves fail to close properly, a portion of the blood corresponding to the degree of insufficient closure escapes in the wrong direction, or at least the amount of blood sent in the proper direction is less than under normal circumstances. This results in the work of the heart muscle being less perfectly utilized for the purpose for which it is intended. In order to restore the circulation to its normal condition the work of the muscle must be increased. That the same thing must occur when the orifices through which the blood escapes from the heart are narrowed requires no special explanation.

As this diminished utilization of the cardiac power is permanent whenever valvular disturbances or stenosis have developed at the orifices, it follows that the increase in the work of the heart must also be permanent. Hence the possibility of heart fatigue is *à priori* greater than under normal conditions. And this is actually the case. It may be stated positively that a heart with changes of this kind at its orifices and valves is less able to respond to an increased demand,—which is practically always in the form of external work,—especially when this increased demand has to be met continuously for a considerable length of time.

The same reasoning applies to anything that causes a permanent increase of the resistance in the greater, as well as in the lesser, circulation. To overcome this resistance the work of the heart also has to be increased and therefore the same sequelæ must develop.

The increased demand first affects either the left or the right side of the heart, depending on whether the increased resistance is in the greater or in the lesser circulation. Indirectly the other side is also involved. Benno Lewy* gives an elaborate explanation of these conditions. He points out that in order to overcome an obstacle, the muscle which has to supply the power necessary to do so must work harder; and in order to work harder it requires a larger quantity of blood. Hence this larger quantity of blood must be supplied and the entire mass of blood passing through the circulation in a unit of time must also be increased by an amount corresponding to the quantity of extra blood required; in other words, exactly the quantity required by the myocardium for performing this extra amount of work. "As the same quantity of blood has to pass through the capillaries of both circulations, the flow of blood must also be increased in the pulmonary circulation, which is not directly affected by the obstacle. Hence any obstacle to the circulation must influence both circulations in every case; when the obstacle is situated in the greater circulation, the flow of blood through the lungs, and with it the work of the right heart, must also be increased; but the amount of increase due to this cause must not be overestimated."

It seems to me that this fact deserves careful consideration: since the right heart is naturally less powerful, an increased demand—small in itself—may, if it is permanent, become a matter of importance in the course of time.

4. The **relation of the lungs to the circulation is of great importance.** The functional power of the heart is in a great degree dependent on the lungs, not only because of the interchange of gases, but also because of the assistance rendered to the circulation by the respiratory muscles, which is effected through the agency of the lungs.

We are far from having a clear insight into the actual conditions even under normal circumstances—still less in pathologic conditions.

* *Loc. cit.*, p. 363 *et seq.*

It will be well to first consider the factors which help to produce the variations in the aortic pressure that take place synchronously with the respiratory movements. Thus writes Tigerstedt,* from whom I borrow the following table:

Natural Respiration.	Inspiration.	Expiration.
1. Aspiration of the blood into the right heart	Increased +	Diminished —
2. Diastole of the heart	Facilitated	Impeded
3. Systole of the heart	Impeded	Facilitated
4. Width of the pulmonary vessels	Increased +	Diminished —
5. During the transition from one respiratory phase to another, blood either remains behind in the pulmonary vessels or is expelled from them	Remains behind	Is expelled
6. During the respiratory phase the flow of blood in the pulmonary vessels is	Facilitated	Impeded
7. Effects of four to six as regards the supply of blood to the left heart	At first diminished, then increased	At first increased, then diminished
8. The frequency of the heart when the pneumogastric nerves are intact	Increased +	Diminished —
9. The vessels of the greater circulation	Dilate	Contract
10. Intra-abdominal pressure	Increased +	Diminished —

Tigerstedt comments on this table as follows: "Nos. 1, 2, 6, 8, and 10 among the above factors during natural respiration cause an increase, and Nos. 3, 5, and 9 a decrease, of the aortic pressure during inspiration. When the breathing is moderately accelerated, the second group of factors is more than compensated for by the first and the pressure rises during inspiration; not at once, however, but only after a certain time—after the fall in the aortic pressure, which is chiefly due to factor 5, has ceased and the favorable effect of inspiration on the return flow of blood into the veins (factor 1) has had time to influence the supply of blood to the left heart.

"On the other hand, when the respiration is rapid, the dilatation of the pulmonary vessels during inspiration can be recognized only by the blood which remains behind (factor 5); and the element which increases the pressure, namely, the enlargement of the vessels, does not make its appearance until the end of inspiration. In the same way during expiration only the first factor that produces an increase of pressure has time to manifest itself before another inspiration takes place.

"When I add that during rapid breathing the frequency of the pulse in the various respiratory phases does not vary appreciably, it will be readily understood that under these circumstances the pressure falls during inspiration and rises during expiration."

The plethysmogram and the sphygmogram show that these experimental results, which were obtained on the dog, are also applicable to man.

We see, therefore, that even when the mechanism is not disturbed, the conditions are exceedingly complicated. In a few morbid disturbances and under certain conditions it is possible to gain some insight into the mechanism. As an example I may mention what occurs when the larynx becomes occluded with false membranes; but, on the whole, we must content ourselves with the following axiom, which is the result of experience:

Everything that hinders the respiratory activity necessary for the needs of the body at the time increases the work of the heart by impeding the expansion of the lungs.

Hence under these circumstances there is a greater possibility of the heart tiring soon and at last giving out. The proofs of the correctness of this fact are sufficiently obvious:

In slowly developing, permanent pathologic conditions, be they em-

* *Loc. cit.*, pp. 460 and 461.

physema, the loss of pulmonary tissue, or extensive adhesion of the two layers of the pleura, we regularly find an increase in the muscle of the right heart. This is a proof that the demand made upon it has been not only increased, but permanently increased.

In rapidly developing diseases of the pulmonary tissue—the pneumonias, extensive catarrh of the smaller air-passages and large effusions of fluid or air in the pleural cavities, the labor of the heart increases in proportion to the interference with the expansion of the lungs. But in these conditions other causes which increase the need for oxygen are present: the febrile increase of body-temperature and the constant increase in the work of the respiratory muscles. I need only refer to the increase in the number of heart-beats and the shortening of the diastolic period for rest and nutrition which always occur is these conditions. While a naturally vigorous heart is able to withstand all this and, in addition, the damage done to its tissues by the intoxication in the case of infectious diseases, for example—an already weakened heart may succumb even to the slight disturbance incident to bronchial catarrh not necessarily extensive or attended with much fever. We know from our daily observation how rapidly the symptoms of heart weakness follow bronchial catarrh whenever the heart is near the limit of its functional capacity. And why? I do not think a positive answer is possible. While it is true that the last drop causes a full cup to overflow, that does not explain what really happens.

Possibly the following may help us to a precise, although only a general conception:

The blood gives off carbon dioxid and takes up oxygen in the lungs. In order that this may be done to satisfy the requirements of the body a definite quantity of blood must come in contact with a definite quantity of air in a unit of time. The movements of the air and of the blood are controlled by the respiratory muscles and by the heart, which in turn are regulated through the nerve-centers. In response to nervous stimuli the respiratory muscles can be made to assist the heart and vice versâ; so that, even if one of them is not able to do its full share of the work, the required quantity of blood, nevertheless, meets the required quantity of air in the lung. But this is possible only to a certain extent, and always presupposes increased activity on the part of either the heart or the respiratory musculature, whichever is helping the other. This undeniably results in an increased consumption of oxygen and equally increased formation of carbon dioxid. Although this cannot be proved by actual figures, we may safely assume that in conditions of severe dyspnea uninterrupted by actual intervals of rest this increase must be quite considerable.

It is quite conceivable that a time may come when, in spite of the muscular work performed, the gaseous interchange is insufficient on account of the restriction of the pulmonary surface. The results of the increase in the work of the muscles control the situation; the extra amount of oxygen required cannot be supplied, the carbon dioxid formed by the action of the muscles cannot be disposed of, and the resulting unfavorable mixture of gas in the blood makes itself felt, first, in the working muscles themselves, and **later also in the central organs.**

The assistance furnished by the latter becomes weaker because the central organs tire just as the muscles tire. Unless the trouble can be remedied by diminishing the amount of work performed by restricting the consumption of oxygen, and with it the formation of carbon dioxid, collapse of the entire machinery is only a question of time. To all this

there are frequently superadded in chronic heart patients changes in the blood-vessels of the lung, brown induration, etc., which permanently increase the resistance to the movement of the air as well as of the blood, and thus narrow down the limits of fatigue.

If there is elevation of temperature, which in itself causes "heat dyspnea" and aggravates the above-described processes, even speedier fatal termination may be looked for. We need not hesitate to add what happens when a heart patient is forced to bodily exertion. A few steps suffice to bring on dyspnea, although the work involved is surely greater than that of respiration when all the auxiliary muscles are brought into play.*

Another condition that belongs to this group is an accumulation of fluid or air within the peritoneal cavity. The size of the effusion is less important than the degree of tension. The same conditions obtain as in the case of pleurisy or pericarditis. Large accumulations of fluid may produce dyspnea and seriously embarrass the circulation by preventing the descent of the diaphragm quite as much as an excessive accumulation of air in the intestines. The most alarming symptoms are observed in perforation-peritonitis when large quantities of gas escape into the peritoneal cavity.

It is generally thought that the disastrous consequences in severe cases are due to the complete absence of the inspiratory descent (flattening) of the diaphragm. In the very worst cases this is not all; the pressure is transmitted from the peritoneal cavity through the relaxed diaphragm to the thoracic cavity, so that the lower portions of the lung are actually compressed. The respiratory surface is thus diminished, with all the consequences of that condition.

Probably there is also some indirect interference with the movement of the blood, at least, in the sphere of the inferior vena cava. Here, however, the conditions are very complicated.

I shall have to content myself with giving a brief account of certain experiments which I performed with a different object in view. Large quantities of atmospheric air were forced into the closed peritoneal cavity of dogs under high pressure and allowed to remain for hours.

In all these cases circulatory as well as respiratory disturbances were observed. The degree which these disturbances may attain is shown by the following experiment:

Vigorous dog, weighing 28 kg. The pressure of the air in the peritoneal cavity during a period of three hours fluctuated between 76 and 88 mm. of mercury, after which blood was withdrawn from the femoral artery, which had been previously exposed. But the blood flowed so slowly that the intra-abdominal pressure had to be reduced to 52 mm. of mercury before the blood could be collected over mercury without undergoing coagulation.

The percentage of gas contained in this blood was considerably less than normal (Prof. v. Hüfner's analysis). For purposes of comparison I give Pflüger's average figures in a parallel column:

IN 100 VOLUMES.

	Blood Used in Experiment.	Normal.	Difference.
Total quantity,	30.50	58.30	48 per cent.
Carbon dioxid,	19.51	34.30	43 "
Oxygen,	10.55	22.20	52 "
Nitrogen,	0.44	1.80	76 "

* See also the section on Cardiac Dyspnea.

It is obvious from these figures that the slowing of the circulation caused marked impairment of metabolism. This develops comparatively rapidly and disappears with equal rapidity as soon as the high positive intra-abdominal pressure has been again reduced.

It may possibly be of interest to give a brief account of another observation. In the same animal the pressure in the abdominal cavity was +80 mm. of mercury four hours after the beginning of the experiment—at 3.30 P. M.

When the femoral artery was opened, only a scanty flow of dark blood was obtained. By letting out air, the pressure was reduced to zero; at 3.35 P. M. air was again injected, so that at 3.42 the pressure was again +80 mm.

This was followed by a copious flow of bright-red blood. At 3.45, the pressure being still +80, the blood from the femoral artery was black and flowed very slowly. I may remark that only a few cubic centimeters of blood were removed each time.

At the autopsy (the animal, which had been anesthetized, was, of course, not allowed to bleed to death) both sides of the heart were found empty; the large veins were engorged, especially the superior vena cava.

Even a tension of 36 mm. of mercury is sufficient to produce serious disturbances. I do not know whether any measurements have ever been made on man to determine the intra-abdominal pressure in peritonitic processes. That the degree of tension must be considerable will be doubted by no one who has liberated gas from the intestine or the free peritoneal cavity under such circumstances. But I will not venture to say how far my experiments are applicable to man.

SYMPTOMATOLOGY (SUMMARY).

When the heart stops beating suddenly and forever, death ensues in a very short time. The individual may have time to carry his hand to his chest and cry out that he is choking or dying; consciousness is immediately lost, the face becomes pale, the radial pulse cannot be felt, the heart-sounds become more and more dull and feeble, and the contractions irregular. Occasionally there are slight convulsions—and then the end. Or death may be even more rapid, sudden collapse without any indication that the victim has felt anything.

I have already cited cases of laceration of the heart, the entrance of air into the heart, and embolism of the pulmonary artery, and have given details in those connections.

Even when the symptoms are most alarming, however, the heart action may be restored, as in the second case of entrance of air into the heart reported on page 26. The accident is more frequently observed after shock, after severe hemorrhage, and especially after intense emotional excitement in those who are not endowed with a resistant nervous system. The length of time an interruption of the heart's action—to the observer at least complete—can be borne without causing death varies greatly in individual cases; at most it is but a few minutes. If the case is to end favorably, the observer first feels a faint pulsation in the precordial region; presently, as the heart-beats gain strength and regularity, the heart-sounds begin to be audible—at first an indistinct, dull sound, probably produced by the muscle, and after a time a louder, clearer tone, which gradually approaches in quality the normal heart-sound. The pulse again becomes perceptible, the pallor disappears, and consciousness returns.

In the subacute form of heart failure, which develops more slowly, the disturbances at first are the same, but the course is less stormy and the highest degree of cardiac insufficiency is not attained in every case. Pallor of the face, and coldness of the hands and feet, the tip of the nose, and ears are present, and gradually the entire skin becomes cool and pale. This is because the surface circulation is weak. The pulse is small; perhaps it can barely be felt at the radial artery; it is usually somewhat fre-

quent. The heart-sounds are dull; the respirations are often accelerated and irregular, but may be deep and slow; consciousness is at least somewhat clouded and not infrequently lost altogether. In febrile cases there is usually a fall of temperature, and even in non-febrile cases there may be a drop of more than a degree [centigrade.—Ed.]. The question of life or death depends on whether the heart's action becomes stronger again or is extinguished altogether.

The clinical picture of protracted and possibly permanent heart weakness is in the main fairly definite.

The earliest and most constant sign is that work beyond a certain limit can no longer be performed without inconvenience. This first betrays itself by fatigue of the skeletal muscles coming on earlier, and by a diminution of the energy they are able to furnish in a unit of time. Heart patients have to rest at short intervals and cannot do as much work as before. In fact, this is the constant complaint of those who have to make their living by the work of their hands. Another symptom is shortness of breath, which develops much earlier than in healthy individuals whenever a little extra exertion is called for. In patients previously exempt a great tendency to bronchial catarrh soon manifests itself. The digestive processes may also be somewhat disturbed. The patient says he can no longer eat everything; he complains of distention in the epigastrium, with a sense of pressure some time after eating; constipation is a very frequent complaint. In brain-workers there is often a slight but unmistakable loss of mental power, besides a change in the disposition, showing itself either in ill humor or possibly in a pessimistic tendency.

If the patient's circumstances are such that he can devote himself to the care of his health, and if he really does so, he may live on in tolerable comfort for years. Except for the impairment of his strength, the greater vulnerability of the bronchial mucous membrane and the necessity of adhering to a careful diet, there are no disturbances. By planning his mode of life with due reference to all these things and demanding no more of his strength than the altered conditions permit, the patient may save himself much serious trouble.

Unavoidable accidents, it is true, may at any time cause a breach in the rampart that can never be repaired. Febrile diseases, especially those which involve the respiratory organs,—the pneumonias,—are a serious menace, not to mention those which affect the heart directly. Even if life is preserved, a distinct change for the worse in the permanent disease can hardly be prevented.

The case is different when an already weakened heart is daily compelled to exceed the limit of its functional power. Troublesome palpitation very often develops even during rest; sleep is frequently disturbed by it, and especially is the patient unable to go to sleep. Resistance to fatigue is diminished; the appetite suffers, and other digestive disturbances soon make their appearance. Sooner or later the patient is forced to take to his bed.

It may still be possible, by absolute rest and suitable feeding, to bring about considerable improvement, but usually it is too late, particularly in the case of patients who work with their hands.

The fully developed clinical picture is as follows: The face is bluish or pale; the cheeks, lips, and scleræ distinctly cyanotic. The veins in the neck are distended, fail to empty themselves properly during inspiration, swell during expiration, and exhibit pulsations synchronous with the

heart-beat. The muscles of the shoulder-girdle, the sternocleidomastoids, trapezii, and scaleni, are tense and continuously active. When dyspnea becomes intense, the patient sits erect and grasps a support with both hands in order to get the benefit of additional muscles, the pectorals, etc., in expanding the thorax. In some cases, as when there is extensive bronchial catarrh or some other obstruction in the air-passages, the auxiliary muscles of expiration—the abdominal—are also called into forced activity. The breathing in general is always labored. The surface of the body is cool, especially the parts most distant from the heart, as the hands and feet, the tip of the nose, and the ears. Not infrequently edema develops, beginning about the ankles and gradually ascending, more marked on the posterior than on the anterior surfaces; effusions take place in the peritoneal cavity and sometimes also in the pleural sacs. The liver is often considerably enlarged, sensitive to pressure, and may present a true venous pulse. The spleen is also enlarged.

The outline of cardiac dulness varies, as it must, according to the cause of the cardiac weakness. Marked fluctuations in the extent of the cardiac dulness may be observed within a very short time because the tension of the weakened muscular walls undergoes rapid changes.

With regard to the heart's action, marked differences are observed. The variations are so great, not only in the same, but also in different, cases that general statements can only be made with the greatest reserve. Two fundamental types may, however, be distinguished:

1. The heart-beats are frequent or infrequent, regular or somewhat irregular, but always feeble; the sounds are weak and hollow, but pure.

2. The heart-beats are constantly frequent, but the contractions follow one another always at irregular intervals and vary greatly in strength.

Some are exceedingly feeble and barely drive enough blood into the partially filled aorta to produce a distinct pulse-wave in the radial artery. These are followed by other contractions, very much more vigorous than the former, which may be so strong that they seem to exceed in power the contractions of a normal left ventricle. The heart-sounds in this form are very variable. They are very apt to be replaced, or at least accompanied by murmurs.

The excretion of urine is cases of heart weakness in general is usually much diminished; it may be only 100 c.c. in twenty-four hours, although in most cases several hundred cubic centimeters are evacuated. The urine is dark and of high specific gravity; on cooling, it throws down a precipitate consisting chiefly of urates. Albumin is frequently present, at least at times, but always in small quantities.

The appetite is always diminished, and in severe cases almost abolished. Other digestive disturbances—vomiting, obstinate constipation—also develop later. A tendency to constipation is practically constant, while diarrhea is rarely observed.

The mental activity varies. As a rule, the patients complain of dizziness, a sense of pressure and tension in the head, and are incapable of sustained and serious thought. With rare exceptions they are depressed, surly, and irritable. The need of mental as well as bodily rest is very great, but refreshing sleep is usually absent. As the heart weakness increases, hebetude, somnolence, and finally actual loss of consciousness and coma develop. Violent delirium usually does not occur, or at most is of very short duration.

The body-temperature is always somewhat diminished; sometimes it

is several degress below the normal unless from one cause or another fever is present. The nutrition of the body suffers, and in severe cases actual emaciation with its consequences develops. Bed-sores and also thrush are difficult to prevent. Death may occur unexpectedly, but in most cases dissolution is gradual.

In the fully developed clinical picture the local changes in the heart itself are not marked. This is practically always the case during this period of the disease, and it is often difficult or even impossible to decide whether there is a lesion in the valves or at the orifices, or whether the disturbance affects only the muscle.

Individual Symptoms in Chronic Cardiac Insufficiency.

1. **The Heart Itself.**—The working power of the heart depends on the condition of its muscle. The truth of this simple proposition was for a long time unheeded. Clinicians preferred to busy themselves with the changes at the orifices and in the valves which regulate the direction of the blood current than with those parts which actually furnish the work. The purely mechanic conceptions which are the result of this tendency must be stigmatized as one-sided, for we know that the proper working of the valvular apparatus depends very much on certain specially arranged muscle bundles in the heart.

The principal reason why the muscle-fibers of the heart were neglected is that marked tissue disturbances, particularly in large foci, seem to be rare in the myocardium. Examination with ordinary methods failed to reveal anything definite, and without the evidence of distinct changes a hesitation was felt in approaching the question of the functional significance of the muscle. The signs of inflammation were apparently absent.

In all my postmortem reports of fatal cases of genuine pneumonia, in most of which the autopsy was made by my colleague Schüppel, and which I published at the time, the note is found again and again with variations,* "Fatty and albuminous degeneration," so that I thought it would be of little interest to give the details. I hoped to obtain by chemic means† some data which might indicate, if only roughly, the pathologic condition, but my attempts in that direction were fruitless.

Many agreed with Rühle‡ that: "Before venturing to insert among those forms of heart disease which have an anatomic foundation a group characterized solely by functional changes, it is necessary to show that with the aid of all our present methods of investigation it is impossible to demonstrate any anatomic changes that might serve to explain the functional disturbances; and this has certainly not been done in cases of 'overexertion of the heart.' At least, something similar to it ought to be demonstrated in other transversely striated muscles." In the article quoted, Rühle gives brief directions for the finding of small myocarditic foci, which he obtained from Köster, who had a special gift for recognizing these changes macroscopically. Rühle himself gives a detailed clinical description of his own observations in these conditions made on the anatomic basis of "myocarditis." The picture which he presents is practically

* "Croupöse Pneumonie," "Beobachtungen aus der Tübinger Poliklinik," Tübingen, Laupp, 1883, S. 288.

† *Loc. cit.*, pp. 288, 289.

‡ "Zur Diagnose der Myocarditis," "Deutsches Archiv für klinische Medicin," Bd. xxii (1878), S. 98.

identical with Münzinger's* description of the Tübingen heart, which he took from my cases in the Poliklinik. Even at that time, after several years' observation of these cases, which are so common in Tübingen, I formed the opinion that the chief lesion was in the heart muscle. It is quite possible to gain a clear insight into the condition from a purely clinical standpoint by taking the anatomic finding, "degeneration of the heart muscle," and its immediate consequence, "disturbance of the heart action," as the starting-point for one's investigation of the subject. Compared with the primary, fundamental conception, the presence or absence of a valvular lesion is of secondary importance, although it may be important in the diagnosis of an individual case. Valvular lesions, that is, disturbances of the valvular apparatus, merely form a link in the morbid process which terminates in cardiac insufficiency, or permanently insufficient work on the part of the heart.† I felt no hesitation in selecting this view as the basis of the description of diseases of the heart in my "Lehrbuch der speciellen Pathologie und Therapie,"‡ for, as the years went on, I became more and more convinced that this view is both the simplest and the one which best fits the case.

That this is justified has been demonstrated by anatomic and physiologic facts as well as by the results of the histologic investigations carried out on the diseased heart by means of modern improved methods. These investigations have effected something more. Köster, in his fine contribution,§ published a number of years ago, showed that a large proportion of the changes occurring in the heart in small disseminated foci must be attributed to infection. He also showed that these changes are not limited to the coverings of the organ nor to the muscles, but, as a rule, involve different parts of the heart at the same time. These discoveries have been abundantly confirmed and still further elaborated by the Leipzig school (Krehl,‖ Romberg,** Kelle††). We now know that pericardium, endocardium, and myocardium are all involved together in the morbid process, as well as the blood-vessels—as for the nervous constituents of the heart, nothing definite can be said as yet.

It would, therefore, be more correct—even judging from clinical observation—always to speak of a pancarditis, although it is needless to say this does not relieve the physician of the responsibility of trying to find out which portions of the organ are chiefly diseased. In my opinion, this means more than a mere change of nomenclature. The accepted terms are certainly not calculated to help the inexperienced physician or the student to gain a clear insight into the problem.

The description of the anatomic aspects of cardiac disturbances does not belong to the present subject and will be discussed in another place. On the other hand, there are a number of individual clinical phenomena that require more detailed description.

First, as regards the extent of the cardiac dulness.

We now know that it may exhibit marked variations within a very

* "Deutsches Archiv für klinische Medicin," Bd. xix (1877).

† Münzinger, *loc. cit.*, p. 457.

‡ Section 130 of the first edition (1886), "Insufficienz des Herzens."

§ "Ueber Myocarditis," Bonn Programm, 1888.

‖ "Beitrag zur Pathologie der Herzklappenfehler," "Deutsches Archiv für klinische Medicin," Bd. xlvi (1890), S. 454 ff.

** "Ueber die Erkrankungen des Herzmuskels bei Typhus abdominalis u.s.w.," *ibid.*, Bd. xlviii (1891), S. 369 ff.

†† "Ueber primäre chronische Myocarditis," *ibid.*, Bd. xlix (1892), S. 442 ff.

short time. Rapidly developing dilatation from excessive bodily exertion may subside with equal rapidity if the heart is healthy and remains healthy. In proof of this I have already cited the observation made by the English physician Allbutt on himself while climbing a mountain. (See p. 40.) That the course of events may be different, however, is shown by an important case described by Oscar Fraentzel. (See p. 41.) I shall cite the following as an example of what sometimes occurs when the heart has been diseased for some time.

Personal Observation 3.—Hypertrophied heart, slight insufficiency of the mitral valve and stenosis of the left auriculoventricular orifice. Cardiac weakness after bronchial catarrh; dropsy. Sudden severe symptoms of dyspnea and relaxation of the heart; simultaneous formation of a small hemorrhagic infarct. Acute dilatation, especially of the right heart, subsiding in a short time. Intestinal hemorrhage. Sudden death. The case was under clinical observation for seven months.

Female, fifty-four years of age. Since her youth she has worked hard, especially in the fields, and has suffered from palpitation and dyspnea "for many years." Pneumonia seven and five years ago.

Toward the end of 1888 she was treated in the Poliklinik for ascites and cutaneous edema, which had been preceded by bronchial catarrh. Discharged in the beginning of March, 1889. Has not been able to do as much work since then. In June she again noted swelling of the hands and feet and the dyspnea and palpitation increased. Admitted June 25, 1889; the same findings as before.

Sudden change for the worse on the first of July at 4 o'clock in the morning. At 11 o'clock the following notes were dictated:

The woman sits upright in bed; there is extreme cyanosis of the lips and a rather yellowish-green discoloration of the forehead. Positive venous pulse in the neck, extreme dyspnea,—60 respirations in the minute,—labored expiration; epigastric pulsation. The radials are tortuous and hard; the pulse 120 beats in the minute, regular, and accompanied by marked and extensive pulsations of the thorax. Marked edema of the hands, also of the entire legs and of the abdominal wall. Moderate ascites, the dulness extending a hand's breadth below the umbilicus.

Thorax not typically barrel-shaped. The percussion-note is not distinctly deep tympanitic. The lower border of the lungs posteriorly at the spinous process of the eleventh dorsal vertebra; in front, at the level of the fifth rib.

At the left base the lower border of the lung is about 1 cm. higher than on the right side, and in the posterior axillary line merges with a dull area of rectangular shape 5 cm. in the vertical, and 8 cm. in the horizontal, diameter. This dull area, which is fairly resistant, is quite sensitive to pressure. About 3 cm. above the upper border of the dulness in the axillary line a sound is heard during respiration which may perhaps be interpreted as a rub. Numerous râles are heard scattered over both lungs. The apex impulse is in the fifth intercostal space and anterior axillary line. Cardiac dulness: upper border, second intercostal space; right, about one finger's breadth beyond the right sternal border in the upper part, below, at the lower border of the lung, in the mammillary line.

Systolic murmur at the apex; the other heart-sounds are pure and, on the right side, remarkably loud. There is frequent vomiting.

An analysis of the course of the disease reveals the following: Fluctuations in the extent of the cardiac dulness. The cardiac dulness began to diminish on the second of July, and this diminution continued until the sixth, *when the cardiac dulness was smaller by 4.5 cm. than on the first of July.* The apex impulse moved inward a corresponding degree. The diminution to the right of the sternum was insignificant. On the eighth of July an *increase of about 1 cm. in the horizontal diameter was demonstrable;* after that there was no further change.

The heart action began to quiet down on the second. The pulse averaged 97 beats in the minute, the maximum being 106 and the minimum 90.

Respiratory rate: medium, 34; maximum, 40; minimum, 32. The temperature only once—on the evening of the first of July—rose to 38.1° C. (100.6° F.). The dull area on the left persisted until the end; as early as the second of July distinct friction could be heard over this area and later also ringing râles. There was no bloody sputum. The ascites and anasarca increased, but the subjective condition of the patient improved.

On the sixteenth of July she conversed with the attendants as usual. At 9

o'clock she went to the commode, suddenly collapsed, was with great difficulty brought back to bed, and immediately died.

I shall give only the principal points in the autopsy notes, which were dictated by Professor v. Baumgarten:

Great anasarca; marked cyanosis of the face; about four liters of fluid removed from the abdominal cavity; pleural cavities contain only a small quantity of clear fluid.

Pericardium intimately adherent to the heart. The heart measures from right to left 16.5 cm., the length from the insertion of the large vessels to the apex being 13 to 14 cm. The apex extends to the center of the sixth intercostal space.

After the pericardium is separated the heart appears enormously enlarged and somewhat conic in shape. The longitudinal groove deviates to the left so that the right ventricle forms two-thirds of the anterior heart wall. Coronary vessels enormously swollen. The adhesions between the pericardial layers are quite thin.

The left auricle contains abundant "currant-jelly" clots.

The mitral easily admits two fingers. The auricular surface of the mitral is incrusted with irregularly nodular calcareous masses. The curtains of the mitral are thickened and tendinous and somewhat retracted; the flaps are adherent to one another in both diameters.

The other valves are intact. The wall of the right ventricle in some places measures as much as 8 mm. in thickness; the right ventricle is greatly dilated. Its lumen extends to the apex; the left ventricle ends above the apex. The apex of the right auricle contains a thrombus.

The upper portion of the left lung is emphysematous, while the lower is atelectatic. Below there is an adhesion between the most anterior portion of the lung and the diaphragm. This area involves the lower lobe and part of the upper lobe. Over the region of the adhesion the pleura is covered with villous excrescences.

At the tip of the lingula and in a portion near the posterior border of the lower lobe are firm dark-red masses which cause bulging of the tissues. The one near the posterior pulmonary border is as large as a walnut and when cut proves to be a hemorrhagic infarct. The right lung contains nothing special.

The spleen is somewhat enlarged and firm; the cut surface is dark red and full of blood (congested spleen). It occupies a vertical position and the tip is covered by the twelfth rib, leaving a space about as wide as two fingers between the spleen and the diaphragm.

Congested kidneys. In the liver the acini are distinctly seen; the centers are dark red, the peripheral portions yellowish; but the organ does not present the appearance of the cyanotic, nutmeg liver.

Gastro-intestinal mucous membrane extremely hyperemic. The duodenum contains some blood-tinged mucus; the small intestine is deeply injected and the lowest portions of the ileum contain large interstitial hemorrhages. The intestinal contents at this point also show an abundance of fresh blood.

Aside from the anatomic changes in the mitral valve, which are quite considerable, the heart in this case presents in the main the features of a hypertrophied heart which has become permanently disabled by protracted bronchial catarrh. The sudden change for the worse may possibly have some connection with the small hemorrhagic infarct in the lung, but this is not certain. There is also a possibility that the heart failure may have been due to some other cause, unknown, and that the infarct played a very secondary rôle.

From a diagnostic standpoint it is rather interesting that the extent of the dulness at the point where the infarct was assumed to exist and where it was actually found underwent some change during the time the case was under observation, so that the portion below the diaphragm appeared to vary in size from time to time. The exact measurements are of no importance. The presumption that the spleen was concerned finds some support in the unusual position in which it was found at the autopsy. The atelectatic portion of the lung, which at the autopsy was found to be about as large as a walnut, was too small to be detected by percussion, although it was peripherally situated and at first was probably larger on account of the constant edema in the immediate neighborhood.

A very important point is the change in the extent of the cardiac dul-

ness and the reduction which took place in its size, which, as was demonstrated, amounted to 4.5 cm. At the same time the apex-beat moved toward the median line, the heart quieted down, and the pulse diminished in frequency by more than twenty beats. The respirations also diminished by twenty to the minute. But the improvement in the action of the heart was only temporary; after two days the heart dulness again increased by 1 to 1.5 cm. and the dropsy increased.

The significance of this variation in the extent of the heart dulness is obvious. If it spreads, the heart itself is larger; if it contracts, it is a sign that the viscus has again diminished in size. A change occurring in so short a time can only be due to a diminution of the blood-supply, which again depends on the resistance offered by the heart muscle to the inflow of blood during diastole. Hence the increase or decrease in the size of the heart dulness is a useful index of the functional power of the heart muscle.

It is, therefore, necessary, if possible, to determine whether a change has taken place, and if so the character of the change; for this obviously is of great importance in a prognostic sense. When the fluctuation is as great as in this case, this determination is not difficult; but, as a rule,—at least in my own experience,—the variations in the size of the cardiac dulness are confined within much narrower limits and it requires a careful examination by a practised hand to detect the differences.

I shall not enter upon a detailed discussion of the physical methods of examination in general and shall confine myself to describing the method which I am in the habit of employing in such cases. First of all, the height of the diaphragm is determined on the right side in the mammillary and axillary lines and posteriorly at the vertebral column. The greatest errors may be committed if the extent of the lungs, which, as we know, is subject to great variations, is not sufficiently taken into account. When the lungs are distended, a thicker layer of air-containing tissue is interposed between the heart and the chest wall. This modifies not only the percussion-note, but also the resistance to the palpating finger. On the other hand, retraction of the elastic lung, either because the breathing is superficial or for any other reason, has the opposite effect. The level of the diaphragm in the majority of cases affords a fairly reliable indication as to which of these two conditions is present; for the level of the membrane is chiefly determined by the expansion of the lungs—chiefly, but not exclusively. Among the many other factors concerned the varying pressure within the peritoneal cavity—in other words, the pressure acting on the lower surface of the diaphragm—requires special attention. Distention of the stomach and intestines with gas, especially of the large intestine, dropsy, enlargement of the liver—all these may come and go within a short time.

Further information is obtained from the character of the percussion-note elicited over the lungs, particularly the occurrence of a pure tympanitic note indicating retraction, or hyperresonance indicating distention (emphysema).

The movements of the heart itself, which can be seen and felt over a greater or less extent, also merit attention, although they are ambiguous and must, therefore, always be interpreted in connection with the other phenomena. The same caution applies to the closure of the pulmonary artery, as determined by palpation, and indicates an increase of the work of the right ventricle when palpably more distinct, a lessening when it

disappears, but which may also indicate retraction or distention (emphysema) of the left lung. Displacement of the apex-beat outward or inward, upward or downward, is subject to the same consideration.

How is percussion to be performed? I employ palpatory percussion as taught by Wintrich and Traube, and agree with Ebstein, who has specially cultivated this method, that it yields reliable results. These can be obtained even when a thick layer of emphysematous lung tissue is interposed between the heart and chest wall, as I have determined to my own satisfaction by examinations on the cadaver with the kind aid of my deceased colleague, Schüppel. My opinion is that light percussion is better than deep percussion, because the former enables the examiner more readily to appreciate the changes in resistance by the sense of touch. I am perfectly ready to admit, however, that any one can obtain satisfactory results with whatever method he is most proficient in. But I do not think it is allowable to make a direct comparison between the limits of dulness determined by two different observers; if there is a difference in such a case, it is not fair to conclude that there was a change in the dilatation of the heart, unless the differences are very great.

The practice of marking the outline of the heart dulness on the chest wall with a colored pencil has now become practically universal, not only in clinical work, but also in private practice. The busy practitioner soon finds that he thereby saves much time for a careful examination.

We have another and less well-known procedure in the Poliklinik* for the purpose of carefully controlling topographic relations. After the outlines have been found by palpation or percussion and marked on the skin with colored pencils, we take a piece of tracing-paper, lay it on the chest, and, while the paper is drawn taut, indicate certain definite landmarks, as, for example, for the anterior surface of the chest, the upper and lower portions of the sternum, the nipples, and the ribs as far as the axillary line. The outline of the heart dulness, which has been marked on the thorax, is then traced on the tracing-paper and may be later transferred to an ordinary sheet of paper or applied directly to the record of a former examination [for purposes of comparison.—Ed.]. In this way the results of two successive examinations can be directly compared and measurements can, of course, also be made. Any changes in the portions of the body under observation can be followed with ease and certainty. [Outlines made on the record-sheets by rubber stamps and filled in according to the marks on the thorax are more convenient and practically as accurate, especially if the exact dimensions of the area are recorded.—Ed.]

It is obvious, from the relation of the heart to the surface of the chest, that changes in size are more readily demonstrated in the right half, which has a larger area of contact with the chest wall, than in the left. The wall of the right heart is thinner and favors dilatation; whether for the same reason the dilatation also disappears more slowly is another question. As a matter of fact, changes in the width of the cardiac dulness are found frequently. But these cannot always be referred to the right half of the heart. When the left side of the heart is dilated, the left ventricle pushes itself considerably forward, the apex-beat is displaced downward and outward, and in that case there is also an increase in the length of the heart dulness.

* See "Duodeno-Jejunal-Hernie mit Erscheinungen von Darmverengerung," von Dr. Otto Staudenmayer, "Mittheilungen aus der Tübinger Poliklinik," Bd. i, Stuttgart, E. Schweizerbart, 1886, S. 213.

Proof of the fact that dilatation of both sides of the heart may subside in a short time is found in the investigations of the brothers Augustus and Theodore Schott, undertaken to determine the effect of "resistance exercises" on the heart.*

Let us now discuss the *changes in the activity of the heart which show themselves in the weakened heart itself. These include the behavior of the valves and orifices, the rhythm, and the amplitude of contraction.*

Changes in the valves and orifices: abnormal sounds. The importance of the part played by the cardiac muscle in the action of the valves is becoming more and more recognized. The details of the mechanism have been mentioned elsewhere; here I shall merely state that certain peculiarly arranged muscle bundles play such an important part in the closure of the mitral and tricuspid (atrioventricular) valves, as well as of the aortic and pulmonary arteries (arterial valves on both sides of the heart), that perfect closure of these valves is impossible without their coöperation. In the same way the papillary muscles are indispensable to perfect closure of the mitral and tricuspid valves. It must also be remembered that the diastolic unfolding of the semilunar valves cannot be complete unless the origins of the two large arteries have been compressed during the preceding systole by the muscle-bundles specially provided for that purpose. It follows that abnormalities in the action of the valves may be caused not only by structural disturbances in the valves themselves, but also by disturbances in those muscular portions of the heart whose special function it is to reinforce the valvular apparatus. If the muscle is damaged, we may expect to find all the phenomena observed in disturbances depending on structural changes in the valves or orifices. One point, however, must be emphasized at once: the signs produced by irregularity in the action of the muscle—the valves being intact—are in many, not to say in most, cases extremely variable; while the opposite is true of valves which are permanently damaged.

If the present teaching is correct and the muscle plays such an important part in the closure of the valves, the variable character of the signs finds its readiest explanation in variations of the nutrition and, therefore, of the functional power of the muscle-bundles involved. In support of this view it may be said that the symptoms of general heart failure quite generally coincide with fully developed or suddenly occurring signs of a disturbance of valve closure. As for the actual tissue changes in the muscle-fibers that take part in the action of the valves and their relations to changes in other portions of the heart, these are questions that still await solution.

Insufficient closure at any one of the orifices which connect the heart with the vascular system is usually recognized chiefly by the auscultatory phenomena that are produced. To a certain extent these are decisive, but they should always be interpreted with caution. One thing should never be forgotten: the rules for the diagnosis of disease of the valves or orifices are the outcome of experience; they are empirical rules and cannot be directly deduced from general propositions in regard to the normal physiology of the heart. Imperfect closure due to muscular insufficiency occurs chiefly in the mitral and tricuspid valves and therefore affects the systolic sound.

The systolic sound is primarily a muscle sound; but with it are associated other sounds, produced by the vibrations either of the mitral and

* See section on Treatment, p. 157.

tricuspid or of the semilunar valves, or by vibrations within the blood.* Since even the normal conditions are so complicated, and what was said in the year 1844 by Kürschner† in regard to the theory of the heart-sounds still holds good to-day, namely—"It would be difficult to find a chapter in physiology that has been more worked over without any great result and that has given rise to more controversies with little prospect of final solution";—it is not only justifiable, but indeed imperative, for the scientific physician to follow his clinical and anatomic experience, and this is a field in which the student never graduates.

It is impossible to determine with certainty whether a sound heard during the cardiac systole is due to a permanent ("organic") or to a temporary ("inorganic") change by the character of the sound alone as heard at a single examination. Even after repeated examinations it is rarely possible to form a positive judgment based on the auditory impression alone. It is like the individual links of a chain, each one of which must be separately tested for its soundness before the strength of the entire chain can be trusted.

That is not an easy matter. The signs which justify a provisional diagnosis of "permanent disturbance" will be discussed at length in another place. Here I shall merely state the following general proposition:

In cases of cardiac weakness every kind of murmur that can be heard in cases of permanent structural changes in the valves and orifices may be present.

And not only murmurs are found. Friction sounds, such as occur in pericarditis, are also heard, even when there is no trace of inflammation or roughness about the pericardium. It is true that friction sounds are rare, but still they do occur. According to my own experience, I should enumerate the signs heard in cases of cardiac weakness in the following order, according to their frequency: systolic murmurs, friction sounds, diastolic murmurs. It is needless to say that the diagnosis of spurious pericardial friction sounds must never be made unless all the signs of genuine pericardial sounds are present; that is to say, a sound having the peculiar character of a friction sound and synchronous with the closure of the valves, but not associated with it. It is true that I have heard these spurious sounds in cases of cardiac weakness only during systole, never during diastole, hence to that extent there is a difference between this spurious sound and what is heard in extensive inflammation of the pericardium. But in the beginning of such a condition the friction sound not infrequently is heard only with systole or part of the systole, just like the spurious friction sound. I am inclined to believe that irregular, violent contraction of the heart is apt to simulate this sound; in other words, that it is really a muscle sound.

I have never observed the phenomenon when the heart was acting quietly, and especially when the action was slow. Besides the audible, there is also a palpable spurious friction which sometimes gives one the impression as if a portion of the heart were slipping by under one's finger at a very rapid rate.

The friction sound was discussed at length a number of years ago by Johannes Seitz. It was often observed among the patients in Biermer's clinic, and was, indeed, more frequent and more marked there than I have ever seen it. After this time the phenomenon seems to have attracted

* Tigerstedt, "Lehrbuch," S. 60.
† Tigerstedt, *loc. cit.*, S. 55, 56.

less attention, and I shall, therefore, give an abstract of case 20,* which is reported at length and is absolutely convincing:

Boatman, twenty-six years old. Has always been well and unusually strong, being able to carry as much as 300 pounds.

The first disturbances in the heart occurred without any obvious cause in the beginning of January, 1871: Fatigue brought on by his usual work and after rapid marching; also palpitation. Gradual loss of appetite. After stopping work for a few days on account of an increase in the symptoms, he had an apoplectic attack with left-sided paralysis in the night from the twenty-seventh to twenty-eighth of March, and was admitted to the Zürich clinic.

First period in hospital from twenty-eighth of March to sixth of April. Examination of the heart: "Relative cardiac dulness third to sixth rib; from the middle of sternum to about 1 cm. outside of the left nipple-line. Very faint cardiac movements visible in the fifth intercostal space immediately within the left mammillary line over an area of about 5 cm. in diameter, and nowhere else.

"The heart-sounds from time to time are rough and very irregular. Is this the result of irregular heart action or reduplication of some of the sounds? Murmurs are present, but it is difficult to determine their point of origin, whether the valves or pericardium, and also difficult to state their relation to the phases of the cardiac cycle."

On the day of discharge the following notes were made: "Heart action still irregular and in the region of absolute cardiac dulness the peculiar sounds persist, although somewhat diminished in force. It is still difficult to decide whether the murmurs are systolic or diastolic, and whether they are pericardial or valvular in character. It seems most probable, however, that they are pericardial sounds, and that the patient is suffering from pericarditis with very little exudate."

Second period in hospital from May 22d to July 17th. The man returned to his hard work, but his strength was broken. On admission, slight edema of the legs, hydrothorax, and ascites.

Examination of the heart: "The relative cardiac dulness extends from the third to the sixth rib and its width is enlarged; it begins at the right border of the sternum and extends to the left, 1 cm. beyond the left mammillary line. Moderately strong pulsation is seen in the epigastrium, with slight impulse over the lower portion of the sternum and adjoining portions of the ribs; slight edema, but nowhere distinct cardiac movements.

"On auscultation, the principal sound heard over the heart is a fairly loud, blowing systolic murmur audible from the mammillary line to the sternum and from the second to the third rib. It is soft, has exactly the same character as a valvular murmur, and is not in the least pericardial. It can be heard two, four, or six times in succession, after which one or two or more short, faint sounds of a slightly scratching character are heard. It is impossible to say whether these sounds are systolic or diastolic, single or double, and it is doubtful whether they can be attributed to the valves or to the pericardium."

During the night from the fifth to the sixth and on the morning of the sixth the patient complained so much of pain in the chest and of oppression that morphin had to be given.

"The heart dulness is increased in width, possibly owing to an effusion into the pericardial sac. The marked systolic murmur, exactly like that of mitral insufficiency, is still present, and in addition other sounds which must be described as pericardial friction sounds. The sounds are becoming gradually weaker."

A summary of the case dated the thirteenth of May is as follows:

"Marked improvement in the cardiac symptoms. The murmurs have diminished, particularly that which was thought to be due to mitral insufficiency and which has disappeared. The murmurs are unquestionably pericardial in character."

This change in the murmurs, which was carefully recorded, continued until the patient was discharged. Constriction and stabbing pains in the precordia occurred from time to time, but subsided on the application of wet-cups to the precordia. At the time he was discharged, when his general condition and his nutrition were greatly improved, the following notes were made about the heart. "The condition of the heart has also become more favorable. The murmur at the apex, which had suggested a mitral insufficiency, has ceased; there remains only a faint

* Johannes Seitz, "Zur Lehre von der Ueberanstrengung des Herzens," "Deutsches Archiv für klinische Medicin," Bd. xii (1874), S. 296 ff., and in the monograph, p. 254 ff.

rubbing, scraping, or scratching below and to the left of the sternum, indicating pericarditis, which we have no doubt exists; for, in addition to the murmur, pure, loud sounds can be heard everywhere both during systole and during diastole."

Third period in hospital from August 18th to 23d, the day of his death.

The patient in the mean time had lived quietly, but his condition had become steadily worse. In addition to troublesome palpitation and constriction at the slightest exertion, there were loss of appetite with impaired nutrition, cough with expectoration and headache.

On admission, some edema of the legs, hydrothorax, bloody sputum as in pulmonary infarct. Severe dyspnea.

"Heart dulness considerably increased in width; extends from the third to the sixth rib and from the right sternal border to about 3 cm. outside of the left mammillary line. The heart-beat is feebly felt in the precordial region, being most marked in the fourth intercostal space and parasternal line, and in the fifth intercostal space and mammillary line.

"The heart-sounds themselves are normal and pure, and can be heard with more or less ease all over the precordial region. They are somewhat hollow and indistinct, and are constantly accompanied by pericardial sounds. Usually it is no more than a slight grazing sound; at times accompanying quite a number of systoles and again only a single contraction. Occasionally the grazing sound occurs during diastole or between diastole and systole. The friction murmur is, in the main, feeble, being best marked in the region of the sternum, especially along its lower edge on the left side. In the region of the apex it is less loud and weaker."

Death occurred suddenly without any discoverable cause.

We felt almost sure of our diagnosis when we went to the autopsy. It could only be a pericarditis: "no other thought could be nor was entertained."

But it was found that the pericardium was absolutely free and that there was only a slight enlargement and dilatation of the heart, particularly of the left ventricle. There were no changes worth mentioning in the endocardium, nor could any marked changes be demonstrated in the heart muscle. Eberth "satisfied himself that there was no distinct and significant pathologic alteration." The methods of examination known at that time were inadequate.

Any one who has carefully followed cases of this kind during the period when we still adhered to the traditional teachings in regard to the diagnosis of heart diseases must have had the same doubts and experienced the same disappointments.

In this case the other symptoms—the behavior of the apex-beat and of the heart dulness—undoubtedly contributed their share to the misinterpretation of the murmurs.

Seitz offers the following explanation for the production of these "pericardial" murmurs: "The murmurs can only be explained by assuming that they were produced in the absence of pericarditis or irregularities on the surface of the viscus, merely by the heart grazing adjacent structures. The production of the murmur in this way was due in part to the enlargement of the heart and in part to the character of its movements; for the heart was overexerting itself, although to no purpose. Gendrin, Pleischl, and Mettenheimer* also believed that friction murmurs might be caused by violent contraction of the heart."

* This is not quite correct. Mettenheimer, "Ueber pericardiale Reibungsgeräusche ohne Pericarditis" ("Archiv des Vereines für wissenschaftliche Heilkunde," Leipzig, Denicke, Bd. ii, S. 423 ff., 1866), reports a case in which the most violent friction murmurs, such as are only heard in pericarditis, were both felt and heard shortly before death. The heart's action, which had been quite feeble, had suddenly become most violent and assumed a rolling, boring character. At the autopsy partial rupture of the heart with an effusion of blood into the fatty degenerated myocardium was found. Numerous ecchymoses were present under the inner and under the outer layer of the pericardium. The pericardial sac contained neither blood nor serum.

Mettenheimer attributed the friction to the fact that the pericardium was dry and of the same character as described by Pleischl in the cadavers of cholera patients,

From these words we may infer that Seitz directly attributed the murmurs, at least in part, to overexertion of the heart—that is, to the processes going on in the muscle itself. In another place, however, he distinctly states that rubbing of the heart on its surroundings is necessary for the production of the sound: "The proximity of the three serous sacs, pericardium, the reflected portion of the left and right pleura, the pulmonary borders, and the heart, may be largely responsible for the production of the murmur, because the close proximity of so many moving membranes cannot help favoring the production of such a sound."

In support of my own interpretation that the spurious friction murmur is a muscle sound I may mention:

1. If it were due to rubbing of a vigorously contracting and enlarged heart on its surroundings, it would also have to occur in the regular contraction of a hypertrophied but perfectly competent heart. I have never heard it under such circumstances.

2. It occurs in infectious diseases when no enlargement of the heart can be demonstrated. But a certain degree of irregularity in the heart action is practically always present. Blowing murmurs similar to those which are present in cases of deficient valvular closure may accompany or alternate with the friction murmurs.

Seitz himself mentions this fact. "In the region of the sternum particularly a slight grazing was frequently heard, varying in distinctness and in duration, either lasting for some time or quite transient, so that it often puzzled me to know how to explain it. Generally it was so indistinct that it was not even possible to decide whether it was a pericardial rub or a very slight modification of the valve sound. It was present in cases in which there was not the slightest reason for assuming a cardiac affection. Indeed, I remember one case which came to autopsy and in which not the slightest cause for the peculiar modification of the first heart-sound could be found. These repeated experiences gradually gave rise to the conjecture that occasionally a normal heart may produce a slightly abnormal noise in the course of its normal movements by grazing its surroundings; and when such a noise is associated with the heart-sound, it gives it a peculiar, delicate, grazing or licking character. At the present moment there is a rather mild case of typhoid in the wards in which this impure heart-sound, especially to the left of the sternum, is very pronounced, and in which we have every reason to suppose that once more we are dealing with the murmur 'which really is nothing at all.'"

I have long paid special attention to this matter and have *heard this pseudopericardial friction sound* not infrequently in many infectious diseases.

Since we know that the myocardium is involved in all infectious diseases, although the change does not go as far as true inflammation, the theory that this is a muscle sound is quite justifiable. It is, of course, practically impossible to bring absolute proof.

in whom pericardial friction sounds were present during life. The petechiæ, like any other irregularity, might have favored the production of the rub. Finally, the peculiar boring and burrowing movement of the heart caused an increase in the contact surface between the two layers of the pericardium. He emphasizes the fact that the force of the heart, both in cholera and in this case of his, was a very important factor in the production of the friction sounds; but according to his own explanation increased heart action can only produce friction sounds when the other conditions are fulfilled. We need not hesitate even in this case, in which there was gross muscular disturbance, to assume a peculiar modification of the muscle tone.

The so-called *gallop rhythm* of the heart is a more important sign. Instead of two sounds, as in the normal heart, three are heard, which produce the same auditory impression as the sound of a horse's feet galloping on hard ground.

It is the generally accepted view that gallop rhythm is heard only when cardiac weakness is present. Considerable difference of opinion exists as to the mode of production of this peculiar phenomenon.

In regard to the succession of the individual tones, or rather the accent on each sound, much difference of opinion exists. O. Fraentzel,* after a minute description of the phenomenon and the conditions in which it is produced, found it necessary to restrict the definition within very narrow limits. He employs the term gallop rhythm only when two of the three audible tones coincide with diastole and the first is accentuated, that is to say, accentuated not only with respect to the other diastolic sound, but also as compared with the first or systolic sound. He accordingly represents the gallop rhythm graphically as follows $\underset{\text{S.}}{\smile} \mid \underset{\text{D.}}{\overset{\prime}{\text{—}}\smile}$.

Potain,† on the other hand, taught that the sequence of sounds corresponds to the anapest: ⏑ ⏑ —́ . The first two short sounds he regards as the normal heart-sounds, and the last as an adventitious sound indicating the character of the pathologic change. In the one case of this kind which Traube ‡ reports he represents the sound by a dactyl: —́ ⏑ ⏑ . Carl v. Noorden,§ as Fraentzel, represents the phenomenon as follows: ⏑ —́ ⏑ ⏑ —́ ⏑ . "Besides the systolic and diastolic sounds, there is also a presystolic sound."

For further discussion of this question the reader is referred to Ottomar Rosenbach.||

In the present state of our knowledge it is probable that the third sound is produced by contraction of the auricle and is a true muscle sound; hence it is heard before the ventricular sound, that is, it is presystolic, as stated by Traube. This practically disposes of the question. How the sounds are represented is of minor importance. Traube himself selects the dactyl to represent the sounds, although the second, short sound really belongs to the succeeding heart cycle.

Occasionally a double contraction of the heart can be detected by palpation. Traube convinced himself that the phenomenon was an apparently diastolic double sound, the second part of which was really presystolic. He represents the sounds as follows, taking account also of the intervals:

| —́ . . . . . ⏑ . . . . ⏑ . . | —́ . . . . . ⏑ . . . . ⏑ . . | —́

The cardiograms traced by Kriege and Schmall in the case of two patients with well-marked gallop rhythm but regular heart action, with a rate of only eighty beats per minute, clearly show the auricular wave. Thus by a different method of experimentation they prove that the increased activity of the auricle is in part responsible for the production of gallop rhythm.

According to the older interpretation advanced by Traube, the presystolic sound is due to the unfolding of the auriculoventricular valve (mitral or tricuspid), which is said to be brought about by the blood that is forced into the ventricle by the actively

* "Vorlesungen," Bd. i, S. 56 ff.

† See H. Kriege and B. Schmall, "Ueber den Galopprhythmus des Herzens," "Zeitschrift für klinische Medicin," Bd. xviii (1891), S. 261 ff.

‡ "Gesammte Beiträge," Bd. iii, S. 13.

§ Eulenburg's "Realencyklopädie," 3. Aufl., Bd. ii, S. 551.

|| "Die Krankheiten des Herzens," Wien und Leipzig, Urban and Schwarzenberg, 1897, S. 293 ff.

contracting auricle even before ventricular contraction takes place. At the present time observers are rather more inclined to attribute the phenomenon solely to the louder muscle sound which necessarily accompanies the more forcible contraction of the auricle.

Fraentzel, with some reserve, gives a different explanation*: "We must assume that the return flow of the blood in the aorta and the pulmonary artery, owing to deficient heart power, are no longer synchronous and that closure of the pulmonary and aortic valves takes place at different times and thus produces the gallop rhythm."

v. Leyden † says: "The murmur is most probably produced by irregular muscular contraction of the left ventricle, that is, the ventricle making two incomplete contractions instead of a single complete one." But he remarks: "The mode of origin of this peculiar phenomenon, which is both palpable and audible, has never been properly explained." O. Rosenbach ‡ also believed that increased action of the auricle is a necessary condition; but he has a peculiar explanation for the production of the sound, denying that it is a muscle sound produced in the auricle.

Judging from the reports in the literature and my own experience, I believe that the tripartition is the most important element in the phenomenon and that it makes comparatively little difference which of the sounds is accentuated. It seems to me that the phenomenon changes very much in the same patient in the course of a very short time. It should also be remembered, as Fraentzel§ has said: "When the heart action is at all tumultuous, it is difficult even for a practised examiner to distinguish which sounds belong to the systole and which to the diastole."

If it is admitted that the presystolic sound which is derived from the auricle lends the phenomenon its special character and its peculiar significance, it is natural to inquire what causes the increased action of the auricle. Is it possible to give a positive answer to this question? I think I shall have to answer "No." Accordingly I do not care to go into the hypotheses that have been advanced and shall merely refer the reader to the original sources, where he will find the views of those who have busied themselves with this aspect of the question.‖

[Potain at first explained the extra sound by the contraction of the auricle, but later** he ascribed an important part to the sudden passive tension of the distended ventricle. According to him, from the variations in place of origin and in time, one can distinguish a right and left, a diastolic and systolic, gallop rhythm. He distinguished it from reduplication of the sounds by using a rigid stethoscope (though palpation simultaneous with auscultation with a binaural stethoscope seems quite as certain). Potain called attention to gallop rhythm in scarlet fever, dyspepsia, chlorosis, and typhoid, as well as in the conditions most frequently associated with it. In Bright's disease he denied its relation with low tension, having found it in one case when the tension was 320 mm. of mercury. Bard,†† in a suggestive article, explains gallop rhythm by a dissociation of the muscular and valvular elements of the first sound.—Ed.]

What is the prognostic significance of gallop rhythm?

Fraentzel and also Krehl consider it always a baneful sign ("*signum malum*"). I am not willing to go so far as that. There is no doubt that a heart in which gallop rhythm is heard has lost some of its functional power, but it does not necessarily mean that the heart will soon have to

* *Loc. cit.*, p. 73.

† "Ueber die Herzkrankheiten in Folge von Ueberanstrengung," "Zeitschrift für klinische Medicin," Bd. xi (1886), S. 140.

‡ *Loc. cit.*, p. 300 ff.

§ *Loc. cit.*, p. 67.

‖ See Kriege and Schmall, *loc. cit.*, p. 270 u. ff.; also Ottomar Rosenbach, *loc. cit.*, p. 295 ff.

** "La Semaine médicale," 1900, p. 175.

†† "La Semaine médicale," 1906, p. 229.

stop working. As a warning I value the phenomenon very highly; whenever it is present, it is an indication to the physician to do all in his power to support the heart muscle, which is beginning to fail.

It is, therefore, important to mention the conditions that may be confounded with gallop rhythm. The chief one is the diastolic double sound in stenosis of the mitral orifice.

In regard to the differential diagnosis, Fraentzel remarks: "In stenosis one sound almost always and often both sounds are replaced by murmurs whenever the heart action is in some way rendered more tumultuous, as, for example, by the patient making some exertion; the diastolic double sounds are heard only in the region of the apex; as a rule, there are present at the same time certain other phenomena which point to disease of the mitral valve apparatus."

The diastolic double sound which occurs in hypertrophy and dilatation of the right ventricle must also be mentioned. According to the usual interpretation, it is due to the fact that the pulmonary valves close a little before the aortic valves. I believe a practised ear can detect the difference

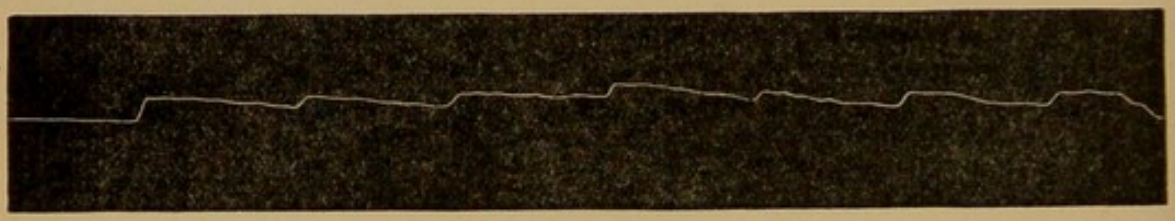


Fig. 1.

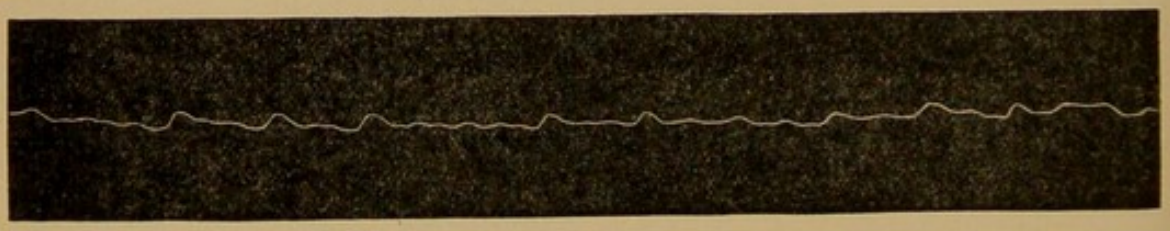


Fig. 2.

at once, although, like Fraentzel and C. v. Noorden, I am unable to give any rule that will enable one to learn to make this distinction.

I must also mention splitting of the systolic sound, which is quite common; here again the distinction is not difficult.

Alterations in the rhythm of the cardiac contractions and in the amplitude of contraction of the heart. As I have said, I think one ought to distinguish two fundamental forms, for each of which I shall now proceed to give examples.

1. The heart contracts with more or less frequency and either regularly or irregularly, but always with insufficient force. As I only wish to establish the external characteristics of the phenomenon, I shall merely give a few short extracts from case histories.

Personal Observation 4.—Male, aged fifty, plasterer. He says his present illness was caused by overexertion. Slight emphysema; digestive disturbances, loss of appetite, lassitude. Slight enlargement of the cardiac dulness: on the left side, from the upper border of the third rib passing in a flat curve to the apex-beat, which is situated in the fifth intercostal space, a little to the outside of the nipple-line, and is faintly visible and palpable. The horizontal diameter does not extend beyond the left sternal border. The heart-sounds are pure but feeble; the peripheral arteries

are neither tortuous nor rigid. The urine is free from albumin. The radial pulse was at first somewhat irregular and accelerated, and later gave the tracing shown in Fig. 1.

Personal Observation 5.—Female, aged forty-eight. Has had cough and expectoration for five years, worse during the inclement season and less troublesome in mild weather. In the beginning of February, 1891, edema developed in the feet. At the time the sphygmogram shown in Fig. 2 was taken she had been under treatment for five months. Heart dulness: Upper border of third rib; right boundary extends a little beyond the right sternal border. Apex-beat in the fifth intercostal space. Heart-sounds perfectly pure. The lungs are slightly distended and there are a few râles in the medium-sized bronchi. Some atheroma of the peripheral arteries.

The two pulse tracings suffice to show the important point, namely, the very feeble heart action.

In the second form the conditions are quite different.

2. The cardiac contractions are constantly frequent, but the beats follow one another irregularly and vary much in strength.

The following history is an important one, and is, therefore, given at somewhat greater length.

Personal Observation 6.—No changes in the valves or orifices. Muscular disturbances in the heart. Delirium cordis. Under clinical observation for about two years.

Male, fifty-one years of age at the time of death. Frequent attacks of intermittent fever and of alcoholism, in spite of which the unusually vigorous man has always been able to do hard work.

In the beginning of March, 1874, palpitation and dyspnea developed while he was engaged in doing heavy work. The symptoms gradually increased. On admission in August, 1874, the following conditions were found: Enlargement of the left heart, apex-beat 3 cm. to the outer side of the nipple-line, covering a wide area but not extending beyond the fifth intercostal space. Variable systolic murmurs at the apex. Pulse very irregular but strong, 48 to 52 pulse-beats at the radial with 68 to 72 heart-beats. Cyanosis; no edema, although it had been present just before his admission; excretion of urine normal. After a short time the patient had improved so far that he could go to work again. In the middle of June, 1875, he was admitted again. The findings were practically the same. Just before his admission he had suffered from frequent attacks of oppression, and was so weak that he had to stay in bed at times. The patient again improved, as after his former admission to the hospital, on rest in bed, digitalis, and a little wine.

After the middle of January, 1876, a marked change for the worse began. The patient was no longer able to do hard work in quarry, woods, and vineyards. Examination on the twenty-fourth of January showed marked dilatation of the heart: greatest width, 24 cm.; greatest length (measured to the base of the sternum), 20 cm.

Boundaries of the Heart Determined by Percussion.	Sternal Line.	Parasternal Line.	Nipple Line.	Maximum Extent.	Heart-sounds.
Left side.	Upper border of third rib.	Upper border of third rib.	Upper border of fourth rib.	Anterior axillary lines; upper border of sixth rib.	Hollow, dull, barely audible.
Right side.	Upper border of third rib.	Upper border of third rib.	2 cm. from the nipple-line.		No distinct murmurs either on the left or on the right side.

Radial pulse, 40; heart-beats, 100; respirations, 32. Intense cyanosis, but no edema. No elevation of temperature. Again improvement was effected in a com-

paratively short time by the same treatment as previously employed. After the pulse-rate and frequency of the heart-beats had become identical again and reduced to 70 and 80 in the minute, the man was discharged on the fourth of February.

The behavior of the urine was peculiar. From the first few days it was noticed that the urine was at times very pale, although the quantity excreted was not materially increased. Then there were other days on which the usual conditions, dark urine with high specific gravity, were observed. Albumin, when any was found, was present only in traces. The ingestion of fluid was pretty much the same on individual days, at least according to the statements of the patient and attendants; but this matter is very difficult to control in out-patient practice.

On two days the quantity of urine passed at single evacuations was received in separate vessels. The patient was not asked to urinate at definite intervals. The result was as follows: From the thirtieth to the thirty-first of January, counting from 2 o'clock in the afternoon until the same hour on the following day:

		Specific Gravity.
Evening	5 o'clock	1016
"	9 "	1018
Morning	7 "	1023
"	9 "	1025
Afternoon	1.30 "	1010

From the fourth to the fifth of February, from 3 o'clock in the afternoon until 12 o'clock the following day:

		Specific Gravity.
Evening	4 o'clock	1019
"	6 "	1008
"	9 "	1005
Night	12 "	1005
Morning	6 "	1015
Noon	12 "	1016

The daily quantity of urine from the thirty-first of January to the first of February was 1260 c.c., with a specific gravity of 1015; from the third to the fourth of February, 1650 c.c., with a specific gravity of 1010. As the autopsy showed that there were no changes in the kidneys, these changes in the quantity of the urine may be taken as an indication of the great variation in the heart action.

On the twenty-first of April, 1876, the patient was again admitted. At first the former picture was observed, but later, from the twenty-fourth to the twenty-ninth, the temperature rose and fluctuated between 40.6° and 39.5° C. (104.9° and 103.1° F.). The cause was not clear; endocarditis as well as pneumonia was thought

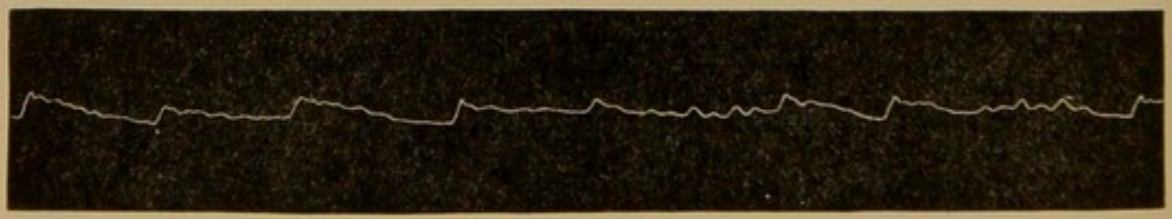


Fig. 3.

of; no positive localized signs were found. However, the fever fell by crisis. On the first of May the patient, believing himself able to work again, discharged himself.

From this time on he rapidly declined.

On the twenty-third of June the man reported with extreme dyspnea. In addition to the hard work which he was still able to perform he had drunk "in excess of his strength," as stated in the clinical history, which was obtained from his friends. This time a difference was noted in his condition in that the number of heart-beats was considerably increased—up to 150. The pulse was very irregular and feeble, as the sphygmogram (Fig. 3) shows. The respirations were 40 and extremely labored, sometimes approaching the Cheyne-Stokes type. The tracing shown in Fig. 4, which was taken with Brondgeest's pansphygmograph with the tambour applied on

the right side underneath the clavicle, gives a clear representation of the work performed during respiration by the auxiliary muscles which expand the upper portion of the thorax.

The tracing reminds one very much of Rauchfuss'* curve, the only one that he obtained in "severe paroxysms of stenosis (croup with attacks of suffocation)." Rauchfuss offers the following interpretation: "Inspiratory expansion of the thorax takes place by successive stages instead of a single effort, because the inspiratory muscles twice give out and are overcome by the tension of the greatly distended walls of the thorax before they attain the position of extreme inspiration; each time the thorax springs back to a position of incomplete expiration."

In my sphygmogram the periodic relaxation of the inspiratory muscles is seen several times—in some places five times. If the tracing were on a larger scale, it would probably show even more frequent interrruptions. Personally I am inclined to adopt the following different interpretation:

The muscles are still capable of performing the large amount of work required of them, but to do so they must be stimulated by a powerful impulse from the center. If this impulse is sent out the resistance, while quite considerable, is by no means so vigorous as when the nervous impulse is inadequate. The initial portion of the curve, which rises very little above the abscissa, shows better than anything else that the nervous impulse is the determining factor.

At this time signs of pulmonary edema were also present over both bases in this patient; with the development of the pulmonary edema the worst of the dyspnea, which always manifested itself in the above-described form, subsided. This time there was edema about the ankles and slight ascites. At times there was a transient positive pulse in the jugular veins and in the liver. The excretion of urine, as in cardiac insufficiency generally, fell to 310 c.c., with a specific gravity of 1032.

In spite of these grave symptoms the patient again became fairly comfortable. The sphygmogram on the fifth of July (Fig. 5) shows that the heart was improved. On the seventh of July the man was discharged.

He was admitted for the last time about the middle of August, and died on the twenty-eighth of August, 1876.

This last period differs from the preceding ones only by the fact that the dyspnea was less prominent, although signs of pulmonary infarct were noticed on the twenty-fifth of August.

The autopsy notes contain the following: Heart: Transverse diameter, 17 cm. Right ventricle dilated, muscle not very friable, but richly infiltrated with fat. Thickness at the

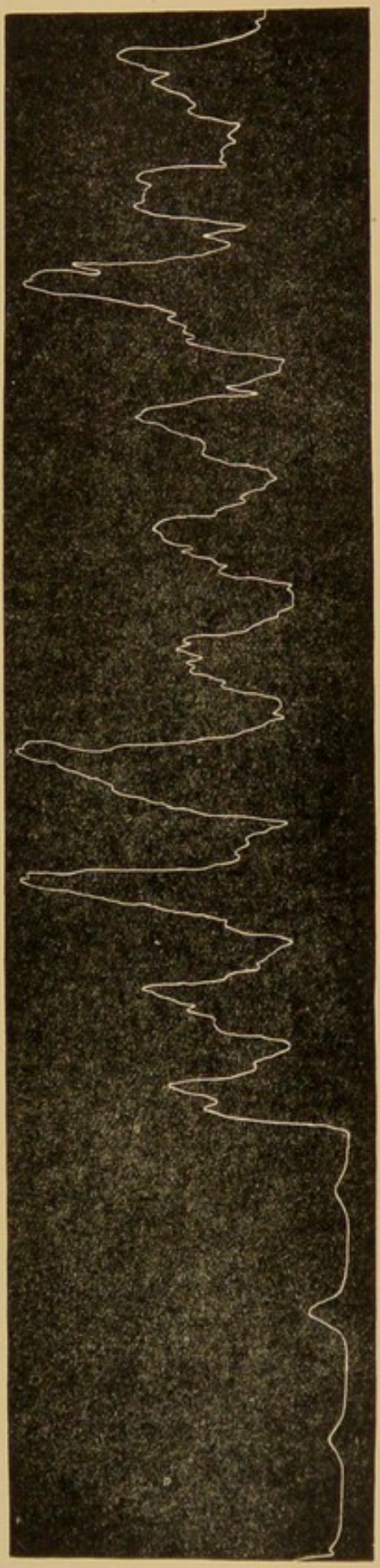


Fig. 4.

* In Gerhardt's "Handbuch der Kinderkrankheiten," Bd. iii, 2. Hälfte, S. 73, 74, Tübingen, 1878, Laupp.

apex, 4 mm. Left ventricle greatly hypertrophied; thickness of the wall, 2 cm. Here and there fibroid thickening of the endocardium is present. All the valves are intact; the aorta and pulmonary artery are slightly enlarged and exhibit some fatty degeneration.

Microscopic examination shows that the muscle-fibres in both sides of the heart are enlarged to about double their size and the nuclei very large. While some of the muscle-fibers are perfectly normal, there are a few with albuminous, and others with beginning fatty, degeneration; but they are not numerous and are distributed uniformly, not collected in groups.

Lungs: the right lung is adherent to the pericardium and contains extensive infarcts with obliteration of the supplying arterial branches. The left lung is freely movable; both are emphysematous. The other organs show the signs of congestion.

There was, therefore, no valvular disturbance of any kind in this case. The heart muscle, owing to the inadequate methods available at that time, showed but little change; it was, however, markedly incompetent, the coördination particularly being disturbed. The comparatively rapid change from a state of practically complete insufficiency to a fair degree of power is very remarkable.

It seems to be desirable, from a practical standpoint, to make a distinction between these two fundamental forms.

The first is a sign of cardiac weakness occurring in the form of fatigue. This condition of fatigue may last only a short time, and its cessation may,

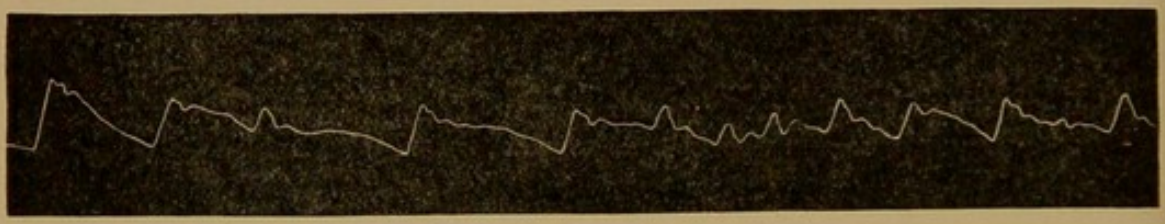


Fig. 5.

of course, be followed by the disappearance of the change in the action of the heart, which finds its expression, aside from other signs that need not be specially mentioned, in the sphygmogram. (compare Fig. 1). It may also be permanent, and in that case permanent tissue changes are present in the heart which we may designate by the general term of degeneration. These degenerations may be uniformly distributed over large areas of the heart and, if that is the case, the cardiac weakness is permanent.

The main features of the pulse are fairly constant. If an increased amount of work is momentarily thrown on the heart, the number of pulse-beats increases materially and the rhythm becomes irregular (compare Fig. 2). The volume must always be comparatively small.

The other signs are: The area of the heart dulness differs but little from the normal and may be identical with it. The area of the apex-beat may be enlarged, but the impulse is never heaving and quite frequently it is distinctly weak.

The heart-sounds are weak, hollow, and muffled; they may be so indistinct that the term murmur may be used to describe them. Such a murmur is always systolic. It is frequently heard not only at the apex, but is transmitted with the same force or, to be more accurate, with the same weakness over the entire heart.

The strength of the entire body is permanently diminished. The im-

pairment of strength chiefly affects the muscular power, which may be very greatly diminished.

The digestive apparatus requires indulgence, for even those patients who before they developed heart disease had been able to eat anything and everything become specially sensitive to any extra strain on the stomach and intestines.

A certain loss of power manifests itself in those who had been accustomed to mental work. Severe emotional excitement may be followed by grave consequences.

If the patient will avoid whatever makes an extra demand on the heart, he may continue to live comfortably for a long time, but only so long as he is willing to make sacrifices, which may have to be very considerable when marked alterations are present. It is not too much to say that he must be handled like a raw egg.

Any intercurrent disease, whether it be of the organs of respiration or digestion or a general affection that does not even give rise to any local manifestations, is a serious menace to the patient's equilibrium, which at best is extremely unstable.

A disturbance which to an individual with a sound heart would be no more than a slight indisposition gives rise to alarming symptoms in heart patients and is fraught with all the serious consequences of circulatory disturbance and insufficient tissue respiration.

The second type is in many respects quite different. The sphygmogram affords a very good indication of the condition of the heart. The sphygmogram in Fig. 5 illustrates the change in the force of the contractions and in the rhythm of the individual pulsations. Some of the pulse-beats are so slight that the lever hardly rises at all, while others send it up to a considerable height. A feeble pulse-beat may directly follow a stronger one in the same sphygmogram. We see the same thing in Fig. 3, with this difference, however, that the waves, as a whole, are absolutely lower. When this sphygmogram was taken the patient was in a very precarious condition.

This second form differs from the first, in which the cardiac weakness is continuous, by the fact that the heart at times is capable of considerable exertion. Therein lies the distinction. The heart muscle is not permanently reduced to its lowest limit of efficiency, but although at times it is capable of large amounts of work, perhaps even more than the average, it is, nevertheless, inadequate to the task imposed upon it. The number of cardiac contractions is always increased. Even during rest it exceeds the normal; when extra demands are made upon it, the number rises considerably and the pulse is often uncountable (*delirium cordis*).

The heart dulness, according to my observation at least, is always enlarged and, in fact, attains the highest known limits in this condition. The apex impulse is diffuse and heaving, unless the lung is adherent or overlaps the heart on account of extreme emphysema.

The heart-sounds are extremely variable. Sometimes the sound is a very hollow, indefinite something that can be called neither a sound nor a murmur—a scraping or scratching sound, as in pericarditis. Or the sounds may be sharp and swishing, like the whistling of a whip-lash; at other times they may be comparatively pure, or there may be murmurs like those which accompany valvular insufficiency or stenosis of the orifices. Sometimes a deep, hollow sound is heard at some distance from the patient,—one meter and more,—a sound that can be distinctly separated

from all other sounds or murmurs. It is the sound produced by the contraction of the heart muscle as a whole. It is so loud that any accompanying murmurs due to incompetent valves or narrowed orifices cannot be distinguished by direct auscultation. The sound becomes louder whenever the heart's action is increased. With the stethoscope the murmurs produced at the orifices and valves can be distinguished. I have never heard this sound except over hearts which were proved by other signs to be distinctly hypertrophic, and it disappears whenever the heart becomes permanently feeble.

Generally speaking, the auscultatory phenomena are systolic, although it is not always possible to determine this point with certainty. When the heart is greatly excited, the sounds, which are heard loudest at the apex, are also heard not only over all the anterior, but also over the posterior, surface of the chest.

The number of sounds or murmurs coincident with systole and audible over the heart is greater than the number of pulse-beats that can be felt in the radial artery. The greater the number of contractions in the unit of time, the greater will be this disproportion. I am forced to believe that the phenomenon is regularly present in this form of cardiac weakness, for I have never seen a case in which it disappeared after it had once been noted. Temporarily the same thing may occur not only in any condition of cardiac weakness, but also when the innervation is disturbed even for quite a short time—in itself it, therefore, has no significance. But when the disproportion is permanent, these fluctuations afford a useful measure of the degree of cardiac disturbance. That the pulse volume is quite variable need hardly be mentioned.

In harmony with what occurs in the heart itself the strength of the body as a whole is not uniformly and permanently diminished, but, on the contrary, is extremely variable.

A patient who, a few weeks previously, may have been in danger of his life on account of cardiac insufficiency, may be quite equal to doing the hardest kind of work.

It is also obvious that the disturbances directly due to insufficient heart action, the accumulation of blood in the veins, edema, diminution of the urinary excretions, etc., may disappear as rapidly as they had developed. These fluctuations are mathematically expressed in the quantity of urine excreted and its specific gravity. It has long been known that cardiac insufficiency is accompanied by a diminution in the excretion of urine; but another observation, which I reported many years ago,* is not so generally remembered, namely, that in this form of cardiac weakness the quantity and character of the urine passed at different times of the day shows marked variations. For this reason I shall now report a case which was accurateiy observed in this respect:

Personal Observation 7.—Building inspector, forty-eight and one half years old. In his sixth year he had septic infection which necessitated operative intervention. In his thirty-ninth year he had the first relapse; in his forty-first, a second. Until then no cardiac disturbances had been discovered. The heart disease developed gradually during the next few years, and several times led to marked symptoms of cardiac weakness: anasarca and effusions into the pleural and peritoneal cavities.

There were no positive signs of any changes in the valves or orifices; the cardiac dulness varied somewhat, especially in the transverse diameter. On admission, November 12, 1897, the heart dulness, which could not be readily determined on

* See Münzinger, *loc. cit.*, p. 468, and the figures given in Observation 6, page 69 *et seq.*

account of the interposition of the markedly emphysematous lungs, which were apparently adherent, was as follows:

On the right—mid-sternum; above—third intercostal space; apex-beat—fifth intercostal space, two fingers-breadths outside of the nipple-line.

This was the average recently; occasionally the right boundary would extend from 4 to 5 cm. beyond the sternum on the right. The apex-beat also would be displaced a few centimeters more or less to the left.

From the time the man first came under observation in 1891 the heart-sounds were feeble and hollow; at times systolic murmurs were heard; at other times not; accentuation of the diastolic sound over the pulmonary artery could never be determined.

The pulse in 1891 was still regular; but later—the exact time cannot be determined because the man was not constantly under observation—permanent irregularities in the rhythm and force of the heart developed. Sometimes the irregularity in both respects was quite marked. The disturbances increased unmistakably as the years went by. Whenever the patient again came under observation for cardiac weakness, the result of having again gone to work, the pulse at the wrist was at first often uncountable, only a few individual beats being felt. Hence there was a gross disproportion between what was felt at the wrist and what was heard over the heart. But even at times when the cardiac condition temporarily permitted him to go back to work, the irregularities did not disappear; although the pulse became considerably less frequent, being reduced by one-half.

The following sphygmogram (Fig. 6) shows the picture of the radial artery at a time when the strength of the heart was relatively good.

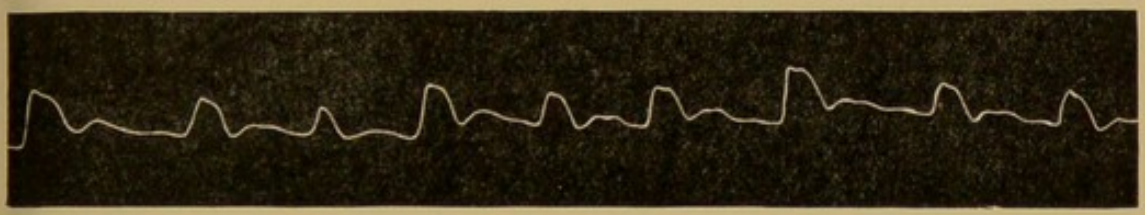


FIG. 6.

This time the patient had returned to the hospital on the twelfth of November, 1897, with moderate anasarca which was severe only in the legs, a slight degree of ascites, catarrh of the medium sized bronchi, and diminution of the quantity of urine to less than 600 c.c. The urine had a specific gravity of 1026 and contained an abundance of albumin—about 0.25 per cent. The pulse could not be counted. Rest in bed, a few drams of the infusion of digitalis, and wine, with appropriate diet, rapidly brought on improvement. By the twenty-second of November the patient was passing abundant quantities of urine and the albumin had disappeared. On the twenty-sixth the edema had almost completely subsided.

From noon on the first to the same hour on the second of December the patient was instructed to keep each urination separate. The result is shown in Fig. 7, which also gives the specific gravity of each evacuation. Note the changes in the specific gravity when the quantity of urine excreted was almost the same.

Thus in the very beginning we have the following:

120 c.c. with a specific gravity of 1013.
135 c.c. with a specific gravity of 1023.
140 c.c. with a specific gravity of 1012.

I thought it would be of some value to determine, in addition to the excretion of urine, the quantity of fluid ingested and the time when it was ingested. The amount of water taken along with the solid food, unfortunately, had to be disregarded.

From 9.30 in the morning of the seventh until the same hour on the eighth of December this was carried out, with the result shown in Fig. 8. Note that during the first part of the day there was no increase in the excretion of urine to correspond in time with the ingestion of fluid. The maximum and minimum almost coincide. Later there was some change in this respect; but the quantity of urine seems to increase as compared with the normal relatively soon after the ingestion of fluid. This means that the heart has sufficient strength to increase the pressure in the renal

arteries rapidly after the introduction of fluid into the circulation. This is also shown by the fluctuations in the specific gravity, which in the main correspond with the quantities of urine excreted. On this day the differences were much less than on that of the first observation; for it is not astonishing that with such a copious evacuation of urine as took place at 11.30 o'clock in the forenoon and 7.30 o'clock in the evening the specific gravity should be very low.

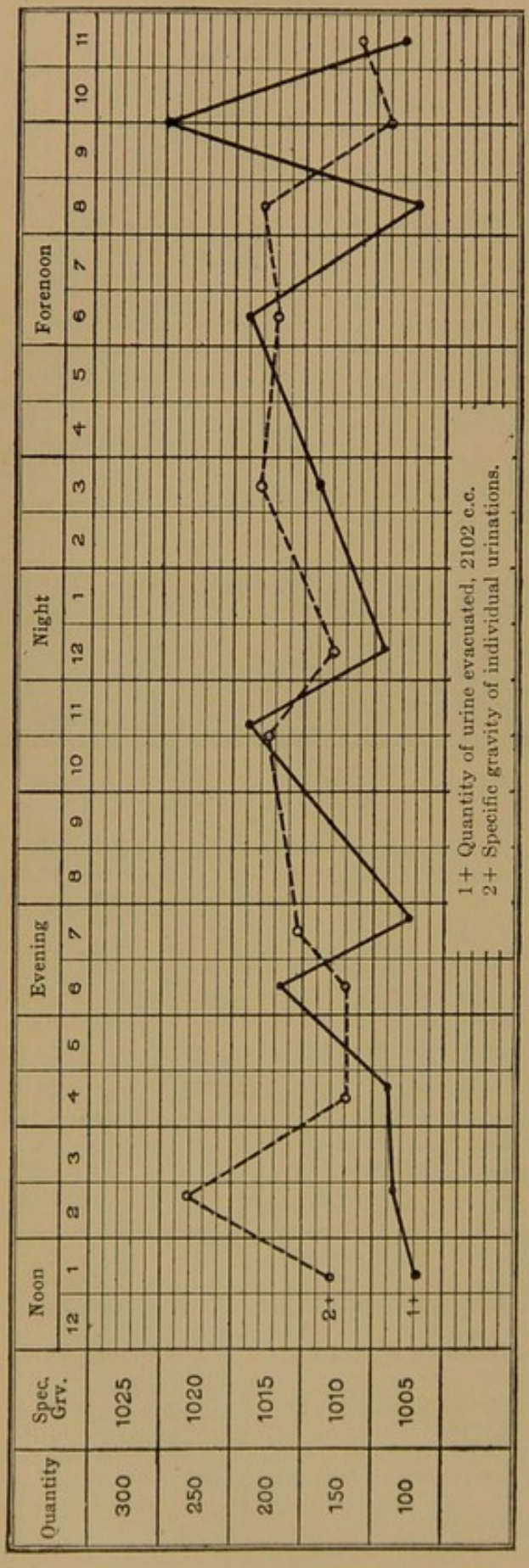


FIG. 7.

These observations lend further support to my contention that marked changes in the working ability of the heart may be present in this form of cardiac weakness, and that these changes may be completed within a relatively short time.

What takes place in these cases may be explained about as follows:

In a heart of this kind the fibers are not all equally efficient. Quite often there has been in the past a true hypertrophy, but in addition there have also occurred inflammatory or degenerative changes. Some fibers are normal, others are hyperefficient, while still others have an efficiency less than normal. Under such circumstances it is not easy for the muscle as a whole to do uniform work, because that implies that the individual fibers, each for itself, undergo a definite degree of contraction within a very short time—enough, in fact, to make the systolic change in shape complete. Under normal conditions all the fibers that are intact and equally nourished are equally irritable; the irritability may be subject to variations, but these variations affect all the fibers

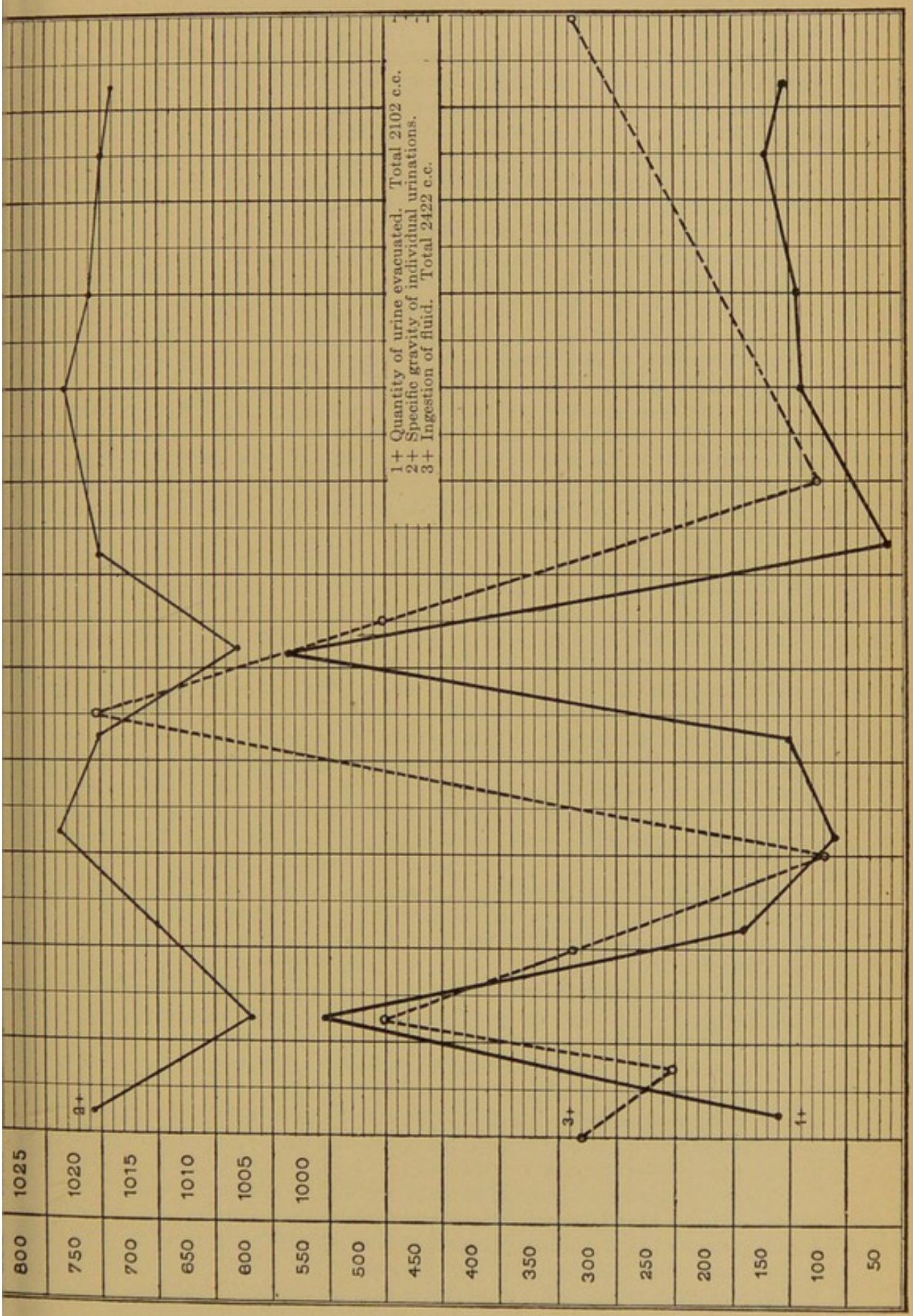

1+ Quantity of urine evacuated. Total 2102 c.c.
2+ Specific gravity of individual urinations.
3+ Ingestion of fluid. Total 2422 c.c.
1+
2+
3+
800
750
700
650
600
550
500
450
400
350
300
250
200
150
100
50
1025
1020
1015
1010
1005
1000

FIG. 8.

to the same degree. In the condition under discussion this is not the case.

We do not know whether a hypertrophic fiber naturally possesses the same irritability as a normal or degenerated fiber, nor is the question an important one in the present connection; for the amount of work of all the fibers in a diseased heart is necessarily variable. A hypertrophic fiber in contracting does more work than a normal fiber, and a normal fiber more than one that is degenerated. In the same way their susceptibility to fatigue is different.

But a fatigued fiber is less irritable. As soon as the amount of work constantly demanded of the heart exceeds the limit of that which the weakest fibers can perform without exertion, the altered irritability of the individual fibers must make itself felt—the more strongly, the greater the amount of work demanded. But the irritability of the fatigued, weaker fibers is less than that of fibers naturally more vigorous. Hence, uniform stimulation of the entire heart has become impossible.

The strength of the stimulus is another factor. Either the stimulus is sufficient to cause the fatigued and functionally weaker fibers to contract along with the others, or it fails to do so. In the former case a vigorous contraction of the entire heart results because the powerful stimulus induces a correspondingly powerful contraction in those fibers which have retained their functional power. In the second case some of the fibers fail to respond to the stimulus by contracting, and in that case the total work of the heart is probably diminished, particularly as the fibers which do not contract form a dead, inert mass interposed among those that do contract and have to be moved and compressed in the process of contraction in order that the systolic change of shape may be effected.

It should also be pointed out that the nutrition of the heart, and hence its irritability and functional power, depend on the amount of blood that flows through the organ. It is probable that a single vigorous systole is much more effective than a number of feeble contractions. On this point, however, we lack precise information, nor do we know what influence the interposition of degenerated or inflamed fibers has on the movement of the contraction wave which is propagated within the heart muscle itself.

Although the conditions are extremely complicated, one definite conclusion can be drawn, which is that the heart works less economically under these conditions. For feeble and often repeated systoles are unable either—

1. To propel the blood adequately, as shown by the fact that the pulse is no longer palpable at the wrist, although the heart continues to contract; or—

2. To supply the heart itself with a sufficient quantity of blood. When the systoles take place in rapid succession, the diastolic pause is extremely short. The necessary result of this is a fruitless expenditure of strength. Force producers must be present, for how otherwise could a heart which is almost paralyzed regain its functional power in so short a time?

We may, therefore, say that fatigue of the weaker fibers, which is attended by a rapid diminution, first, of their own power, and, later, by a similar loss of power on the part of the stronger fibers, is the chief factor in the pathologic process. If the weaker fibers fail to functionate altogether; if their contraction is so feeble and insufficient that they become a mere inert mass which has to be carried along by the vigorous fibers

which bring about the systolic change of shape, the stronger fibers are also much more exposed to exhaustion. Under such circumstances rest and the accompanying improvement in the nutrition of the entire heart may restore the functional power because the weaker fibers are again able to do their part of the work.

The fluctuation in the excretion of urine which is, so far as we can see, independent of external conditions,—at least, of any that we are able to recognize,—possibly indicates the constant presenec of some conditions which exercise a controlling influence on the work and rest of such a heart; but of these conditions we know absolutely nothing.

As physicians we must bear in mind the daily recurring experience that a heart of this kind will retain its functional power longer if it is not overtaxed, and that overexertion in a very short time brings on exhaustion beyond recovery.

2. **The Vessels.**—The *arterial* phenomena that manifest themselves in cases of heart weakness are quite simple. Insufficient filling of the arteries and alterations of the rhythm—these are the important points.

In the case of the *capillaries* and the *veins*, the conditions are not so simple. The nutrition of the tissues, and their functional activity, which depends on it, are directly controlled by the circulation in the capillaries. In the last analysis every functional and nutritive abnormality that occurs in the body of a heart patient must be referred to some disturbance of the capillary circulation. But this question cannot be treated as a whole; it must be taken up in connection with the effects of cardiac weakness on the individual organs.

The effect on the veins may be discussed more briefly.

Any reduction worth mentioning in the work of the heart is necessarily followed by a more or less marked accumulation of blood in the veins of the greater circulation. This accumulation is first seen in the superficial veins of the neck, especially the external jugulars. The vessels appear like blue cords underneath the skin, collapsing during inspiration and swelling during expiration. In itself this is a normal process; but in cases of cardiac weakness it becomes distinctly visible because the greater distention and overfilling of the vessel make it possible for the column of blood to perform movements on a larger scale.

The right auricle is filled by the two venæ cavæ under higher pressure; and because it is more distended, the further entrance of blood from the venæ cavæ and their branches is interfered with. The latter are continually overfilled and are, therefore, more widely opened, which in itself renders the contents more movable. It is also obvious that the greater mass of fluid inclosed by the venous walls also develops more kinetic energy as it moves. In order at once to dispose of all the important points that affect the movement of the blood in the jugular veins, it should be mentioned that—

1. In general pulsating movements are more visible on the right side of the neck than on the left. This is a necessary result of the course of the vessels.

On the right side the innominate (brachiocephalic) vein passes with a slight curve into the superior vena cava, while the jugular veins almost appear like simple continuations of the cava.

On the left side the innominate (brachiocephalic) vein forms an angle at its junction with the superior cava, and the two jugular veins also have a less direct course than on the right side.

Hence there is more resistance to the movement of the blood on the left side than on the right, because the change in the direction of the current due to the greater angle at the junction of the branches absorbs a part of the kinetic energy developed.

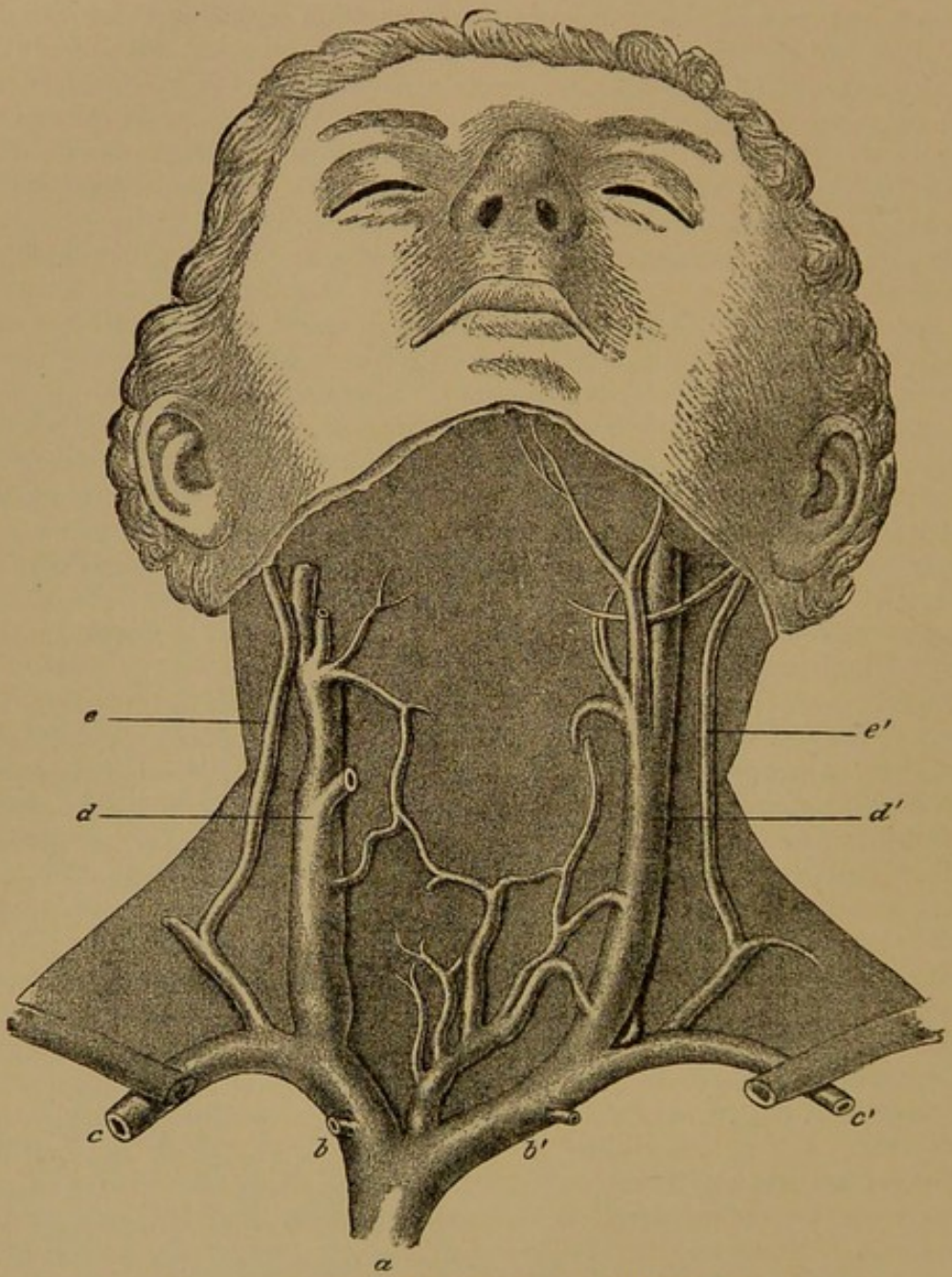


FIG. 9.—(Luschka, "The Anatomy of the Human Neck.")

a, Vena cava superior; *b*, vena anonyma brachiocephalica dextra; *b'*, vena anonyma brachiocephalica sinistra; *c*, vena subclavia dextra; *c'*, vena subclavia sinistra; *d*, vena jugularis interna dextra; *d'*, vena jugularis interna sinistra; *e*, vena jugularis externa dextra; *e'*, vena jugularis externa sinistra.

2. The valves in the jugular veins affect the upward movement of the blood.

Henle* says, in regard to the valves of the internal jugular vein:

"Before joining the subclavian vein it forms an enlargement which is more con-

* "Handbuch der Gefässlehre des Menschen," Vieweg, Braunschweig, 1868, S. 330.

spicuous on the right than on the left side. This second enlargement, the inferior bulb of the jugular vein, is closed above by a single or bicuspid valve with the free border directed downward. The single valve is attached to the outer wall of the vein: the bicuspid bounds an oblique cleft, the anterior extremity of which is generally directed laterally.

"The bulb is oviform and, depending on the position of the valve (its greatest distance from the junction of the internal jugular vein with the subclavian is 25 mm.), more or less extended. Sometimes the valve is situated as low as the angle of junction of the internal jugular and subclavian veins; in such a case the valve is usually single and extends from the subclavian vein downward into the lumen of the internal jugular. Sometimes both single and double valves are present, the upper one being double and the lower single. In such a case the latter is so placed that its free border crosses the cleft of the double valve at a right angle.

"The valves hinder the backward flow of blood from the innominate vein into the veins of the skull; they are more important on the right side of the body, where the innominate vein lies in the course of the superior vena cava, than on the left. Accordingly, these valves are more frequently absent or inadequate on the left side than on the right, and this explains why the inferior bulb of the internal jugular vein is also less constant."

The above description shows to what great variations the valvular apparatus is subject. Although it is better developed on the right side, yet whenever the vein is overfilled, the valves are unable to prevent the backward flow of blood, which is favored by the arrangement of the vessels.

In regard to the valves of the external jugular vein, Henle * says: "The external jugular vein has a double valve at its mouth, or immediately above it, and frequently a second valve about the middle of the neck; rarely a third between the two others."

These valves have probably but little influence on the movement of the blood when the vein is overfilled.

The visible vascular phenomena in the veins depending upon the respiration usually do not extend beyond the jugular, although both Eichhorst† and myself have in a few instances seen distinct respiratory fluctuations in the veins of the arm, the face, the chest, and the abdomen.

Let us now examine these movements of the blood in the veins which are dependent upon the heart.

1. *The Physiologic Venous Pulse.*—Riegel‡ laid the first necessary foundations for the proper understanding of this question.

He very properly emphasized the fact that we should follow Friedreich in fixing the term "venous pulse," altogether apart from the direction of the current, and that we should speak of a venous pulse in all cases when an independent pulsation occurs in a vein due to, and isochronous with, the action of the heart, and either single or multiple.

Adhering to this principle Riegel laid down the following rules, based on animal experiments as well as on clinical observations:

In the region of the superior, as well as of the inferior, vena cava there is a constant pulsation, coincident in time with the movements of the heart.

This pulsation is not due to any movement of the blood beginning in the right heart and passing toward the left heart, but merely represents

* *Loc. cit.*, p. 348.

† "Lehrbuch der physikalischen Untersuchungsmethoden," Bd. ii, S. 109, 3. Auflage, Friedr. Wreden, Berlin, 1889.

‡ "Ueber den normalen und pathologischen Venenpuls," "Deutsches Archiv für klinische Medicin," Bd. xxxi, 1882, S. 1 ff.

the flow of blood from the vena cava to the right auricle, which is more or less impeded at different times.

The contraction of the auricle impedes, its expansion favors, the emptying of the veins.

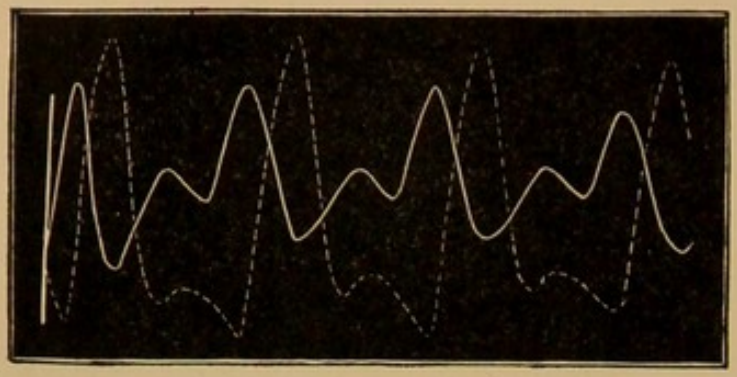


Fig. 10.

On the accompanying sphygmograms of the external jugular in man, taken from Riegel, the carotid curve from the other side of the body is also indicated in dotted lines.

As the carotid sphygmogram represents the systole and succeeding diastole of the left ventricle, this makes it possible to determine the general relations of the venous pulse to the movements of the ventricle. That is the important point; for we already have a sufficiently accurate knowledge of the time relations of the contraction of the entire heart, including both the auricles and the ventricles.

A study of the accompanying sphygmograms from Riegel (Figs. 10–14) shows that the pulse in the jugular vein falls with the beginning of ventricular systole.

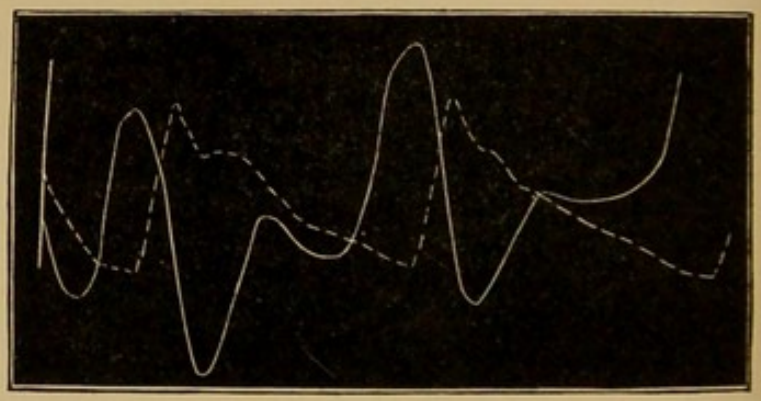


Fig. 11.

The beginning of auricular diastole coincides with the beginning of ventricular systole, the latter being expressed in the carotid tracing by an abrupt rise in the curve.

Since we know that the contraction of the ventricle begins as the auricle goes into diastole, we therefore have a measure of time for the beginning of the descending limb of the venous sphygmogram. The interpretation is obvious: As soon as the auricle begins to dilate, it offers less resistance to the blood contained in the mouths of the venæ cavæ. The blood, therefore, flows more easily from the venæ cavæ into the auricle; the blood contained in their tributaries, the peripheral veins, follows on and fills the venæ cavæ,

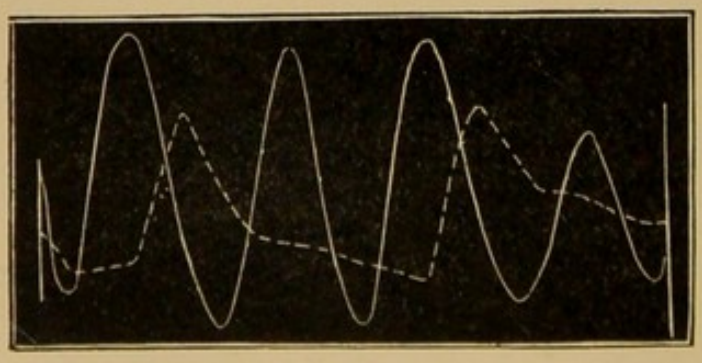


Fig. 12.

and the distention of the peripheral veins therefore diminishes. The collapse of these veins causes the pulse lever on the jugular vein to descend.

The converse of this happens during auricular systole, which follows ventricular diastole. As the auricle contracts, the blood contained in it is subjected to a greater pressure than prevails at the orifices of the venæ cavæ. This prevents the blood in the venæ cavæ from flowing into the auricle, and in the same way prevents the flow of blood from the more remote systemic veins into the venæ cavæ. The vessels are more distended and the lever rises, forming the ascending limb of the sphygmogram.

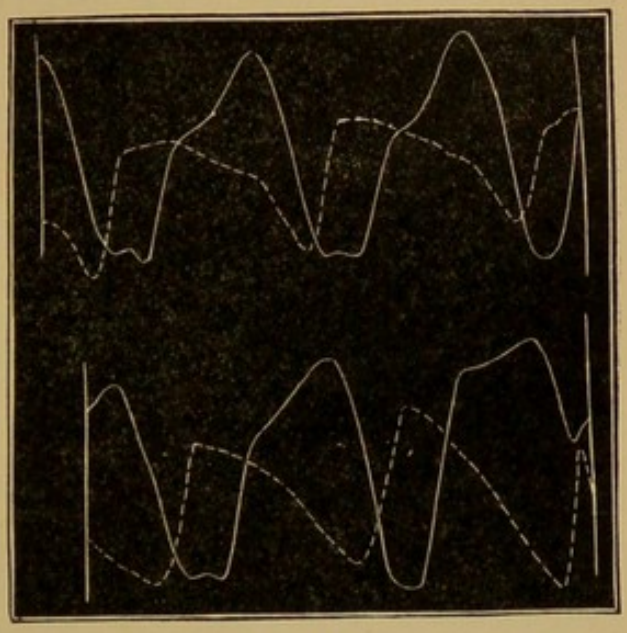


Fig. 13

It is evident that under these circumstances the venous valves can be of no importance whatever. The movement of blood toward the right auricle is merely interrupted—its direction is not altered; there is no reason why the valves should unfold, since they only prevent the flow of blood from the auricle upward.

It is, of course, admitted that the auricle, in contracting, directly forces a little blood into the orifices of the venæ cavæ. In fact, the possibility of a recurrent wave, which the theory requires, must be taken into account. But it should also be distinctly stated that under normal conditions the quantity of blood which thus flows back must be very small.*

The main thing is to establish the fact that the venous pulse is such as has just been described; that the venous sphygmogram cannot be positively interpreted in all its various parts is of comparatively minor importance.

I fully agree with Tigerstedt that the questions involved are very complicated.



Fig. 14.

The article of D. Gerhardt referred to contains an accurate critical discussion of the subject, with a wealth of personal observations and complete review of the literature. Any one who desires more information will find this paper most instructive. [The article in Sahli's "Lehrbuch" (or translation) can be consulted with profit, and also Chapter XVII of Mackenzie's "Study of the Pulse."—Ed.]

I shall merely mention a few more points of clinical importance.

* See also Riegel: D. Gerhardt, "Klinische Untersuchungen über Venenpulsationen," "Archiv für experimentelle Pathologie und Pharmakologie," Bd. xxxiv, 1894, S. 402 ff.

A study of Riegel's tracings (Figs. 10–14) shows that the form of the venous sphygmogram varies. As a rule, the normal venous pulse is anadicrotic catamonocrotic. The anacrotic limb corresponds to cardiac diastole and auricular systole, the catacrotic to cardiac systole.

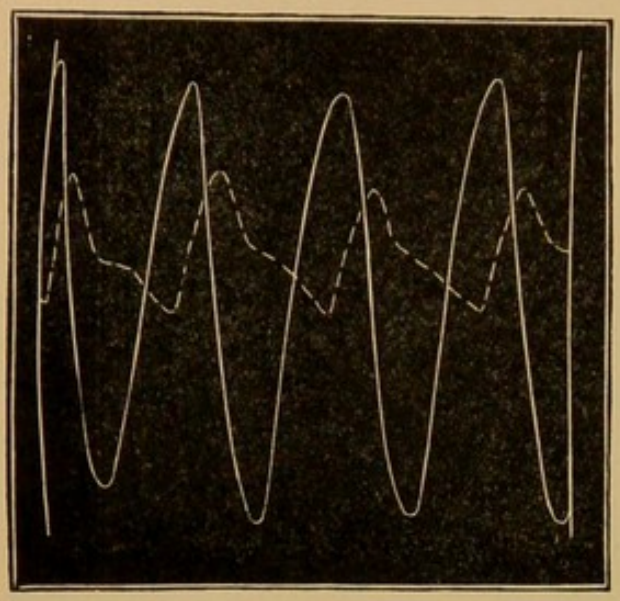


Fig. 15.

Not the first, but the second, limb of the anadicrotic wave, which is shorter in duration but usually more abrupt, corresponds to auricular systole. The catacrotic limb corresponds to ventricular systole.

In cases of extreme cardiac weakness anadicrotism is not rarely absent from the venous pulse; in that case the anacrotic limb may be monocrotic. Fig. 15 is an example of this.

It is taken from a cachectic woman, forty-four years of age, with pneumonia and a small exudate in the pericardium. Extreme cardiac weakness; very small, frequent, and extremely irregular pulse. Marked pulsation in the veins of the neck, which persisted after the exudate had been absorbed. The sphygmogram shows hardly a trace of anadicrotism.*

"In diseases associated with overfilling of the venous system and of the right heart, the venous pulse often attains a considerable size without otherwise—especially in respect to its time relations—differing from the normal venous pulse. As a rule, only the auricular wave is considerably enlarged"—probably reinforced by the greater quantity of blood thrown back into the venæ cavæ from the auricle.

Riegel gives an illustration of the truth of this statement also. (See Fig. 16.)

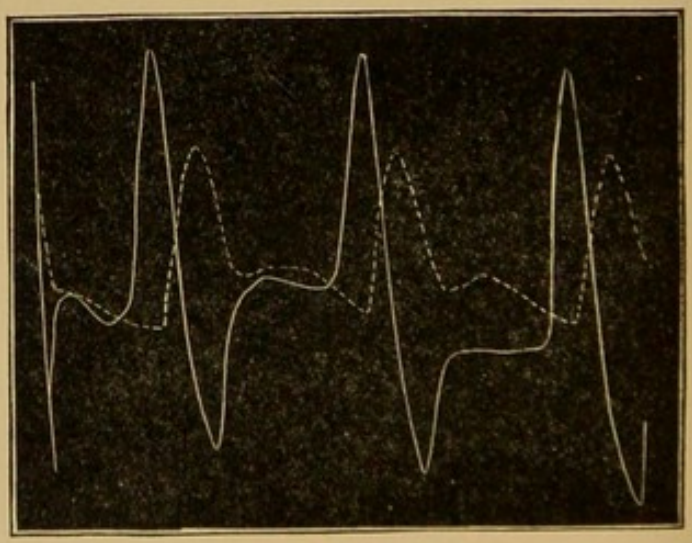


Fig. 16.

The patient was a man, thirty-five years old, who attributed his heart trouble to overexertion five years previously. Marked increase in the width of the cardiac dulness; no distinct apex-beat; distinct systolic murmur at the apex; second pulmonary sound accentuated. At times the heart action was very irregular. Pulse medium-sized, easily compressible. On the right side very distinct venous pulse in the internal jugular.

We see that the second elevation in the anacrotic portion is distinctly enlarged. There was a large accumulation of fluid in the systemic veins, which was kept back

* For the details of this case see Riegel, *loc. cit.*, p. 48.

by the auricular systole; while from the overfilled, vigorously contracting auricle a larger quantity of blood was thrown back into the venæ cavæ. This caused the forcible stroke.

2. *The Pathologic Venous Pulse.*—The pathologic venous pulse is produced by incompetence of the tricuspid valve. During the systole of the right ventricle blood is thrown back into the right auricle and thence into the venæ cavæ and their distal branches, thus producing a wave synchronous with ventricular systole and traveling in a direction from the right heart toward the capillaries. The venous pulsation is visible at some distance from the heart, depending on the strength of the wave, the presence or absence of valves in the venous channels, and their competence or incompetence.

The hepatic veins have no valves and empty into the equally valveless ascending vena cava. Hence, if the contraction of the right ventricle is adequate, pulsation of the entire liver synchronous with ventricular systole may be produced—hepatic venous pulse. The cutaneous veins of the abdomen or the legs may also exhibit actual pulsation.

If the valves in the inferior bulb of the internal jugular vein are competent and remain so, the pulsation caused by the recurrent wave appears only in that portion of the vessel which extends as far as the valves—ventricular systolic-jugular bulb pulse. But if the valves have become incompetent, the pulse-wave extends higher up into the branches of the internal jugular, and if the external jugular empties directly into the internal, the cutaneous branches of the former also exhibit pulsation.

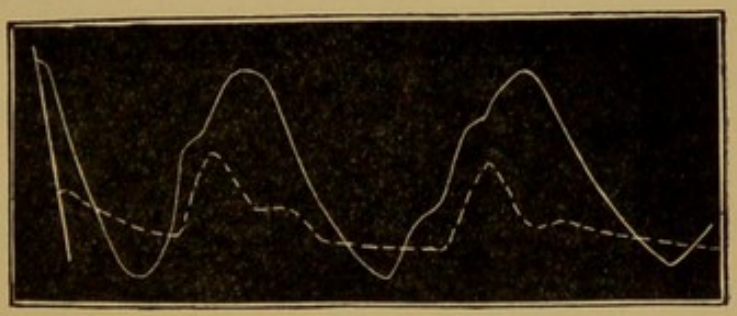


Fig. 17.

The wave may even enter the subclavian and set up pulsations in some of the veins of the neck, thorax, and upper extremities.

The distinguishing feature of this venous pulse is its time-relation to ventricular systole.

The accompanying tracings of the venous pulse in tricuspid insufficiency exhibit only one constant feature: the elevations coincide in time with ventricular systole and the depressions with ventricular diastole. The apex of the carotid pulse does not coincide perfectly with that of the venous sphygmogram, the latter being a trifle later. Its appearance is simultaneous with the first secondary wave of the carotid sphygmogram.

Similarly, the first elevation of the venous pulse is perceptible before the appearance of the arterial elevation. [It begins in the "closing time" of the ventricles, while the arterial pulse begins in the expulsion time.—Ed.] Hence the venous pulse is presystolic-systolic—not, as when the tricuspid is competent, diastolic-presystolic.

The form of the sphygmogram is quite variable, and the peculiarities of the individual case must govern its interpretation in each instance. It is least difficult in a case like that illustrated in Fig. 17. In this the first elevation corresponds to the contraction of the auricle; the second to that of the ventricle; while the explanation of the third is variable.*

* D. Gerhardt, *loc. cit.*, p. 430 *et seq.*

This variety of venous pulse, which Riegel calls the typical venous pulse, presupposes that the heart is still capable of contracting vigorously.

Without going into details, I shall merely point out that the factors which determine the shape of the venous sphygmogram are: the force of the auricular and ventricular contractions; the quantity of blood accumulated in the systemic veins and in the heart; and possibly also the variations in the tone of the venous walls.

I purposely confine myself to these fundamental principles of the doctrine of venous pulsation because I believe they are fairly well established and because a thorough mastery of them will always enable one to interpret the conditions found at the bedside. A few additional points should, however, be briefly mentioned. How can the various forms of venous pulse be recognized without the use of graphic methods, as usually has to be done in actual practice?* One source of error must be borne in mind: the transmission to the vein of the pulsations of a contiguous artery; or, to be more exact, the transmission of the carotid pulse to the jugular vein.

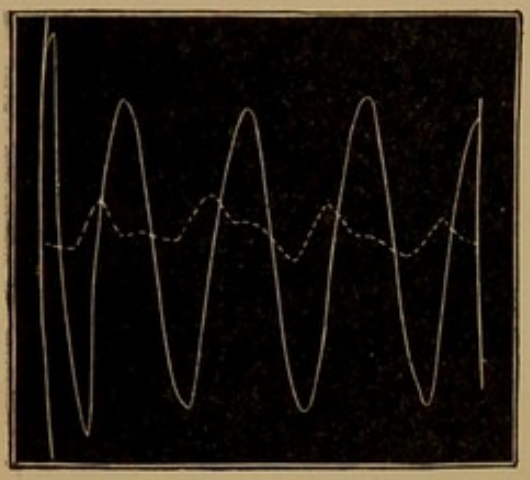


FIG. 18.

I agree with D. Gerhardt† that a transmitted venous pulse of this kind is not very often observed if the precaution is taken to note whether "the alternate distention and collapse really are confined to the vein or involve all the surrounding structures." To arrive at an absolutely definite conclusion, the carotid should be compressed as near as possible to the heart, and the vein at the same time pushed to one side. Sometimes compression of the carotid alone suffices. Other authorities‡

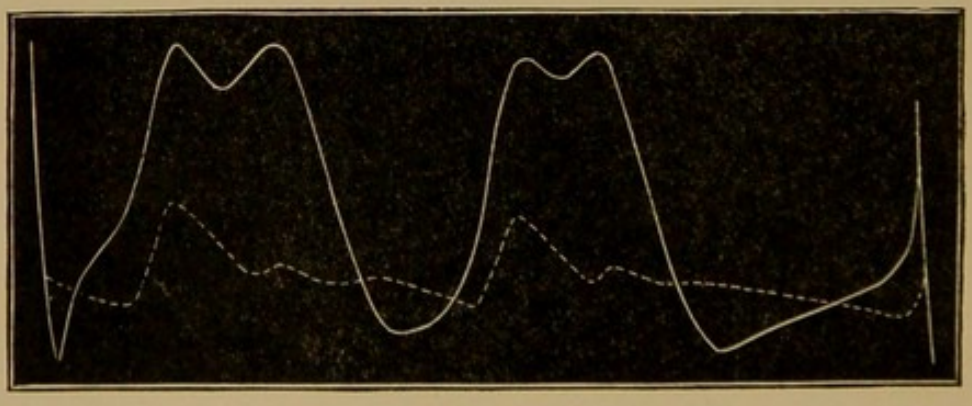


FIG. 19.

advise compressing the vein at the middle of the cervical portion. They say that the portion to the cardiac side of the point of compression collapses

* [The author's remarks are valuable, but the student of venous phenomena will find it impossible to draw accurate conclusions without controlling his observations with some apparatus that records simultaneous tracings of the venous as well as the arterial (radial, carotid) pulse.—ED.]

† *Loc. cit.*, p. 439.

‡ See Eichhorst, *loc. cit.*, p. 110.

and no longer transmits the pulsations; while in the upper, peripheral segment the distention increases, and transmits the arterial pulsations. If the pulsations follow one another in rapid succession, this expedient is indispensable; but when the rhythm is slow, attention to the character of the pulse suffices. The rapid rise and more gradual fall of the arterial pulse, when transmitted to the vein, communicate to the latter the characteristic arterial movement. When the carotid pulse is distinctly dicrotic, the phenomenon also appears in the jugular, and there is no longer any doubt as to the origin of the venous movement (Eichhorst).

D. Gerhardt* has described special modes of origin of a venous pulse caused by the arteries. They can be recognized only by the use of the graphic method and are, therefore, of no great value in actual practice.

Is it possible to distinguish positively the systolic venous pulse which is produced in tricuspid incompetence from the normal venous pulse, reinforced by the accumulation of blood in the right heart and large venous trunks, when it is not possible to record the movements of the blood derived from the left ventricle or the movements of the ventricle itself (apex-beat)?

The answer is—not always.

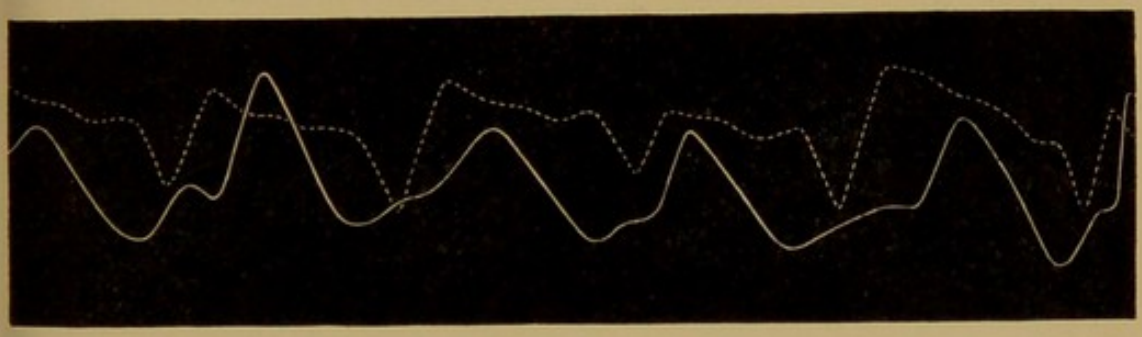


FIG. 20.

In general the criterion is the number of pulsations in a unit of time. If this number is small, it is possible to determine whether or not the collapse of the jugular vein coincides with the swelling of the carotids or with the apex-beat. But when the pulse-rate is high, it is practically impossible to do this. A very disturbing factor is the respiratory alteration of the quantity of blood in the veins; the more so, the higher the respiratory rate. In these cases all that can be seen is the alternate swelling and collapse of the cervical veins; the individual movements cannot be distinguished.

A common expedient is to compress the swollen vein at about the center of its course. If this arrests the movement of the vein in the central portion, true venous pulsation is absent; if the movement persists, it is a true venous pulse. In this way what was formerly called "undulation" was distinguished from true pulsation. Undulation is practically synonymous with normal diastolic-presystolic venous pulsation. When the venous trunks near the heart are only moderately filled and the strength of the auricular contraction is small, it is quite true that the venous movements cease beyond the point of compression; but not when the auricle is distended with blood and contracting vigorously, and the valves of the jugular veins are no longer competent. Under these circumstances the pulsation, it is true, is only diastolic-presystolic so long as the

* *Loc. cit.*, p. 440 ff.

tricuspid remains competent; but the pulsation gives rise to a true recurrent wave, and the blood is driven into the peripheral segment of the vein beyond the point of compression. Hence the origin of the pulsation cannot be positively determined by compressing the vein.

If the venous valves at the inferior bulb of the internal jugular are competent, the movement of the blood which escapes from the contracting auricle is arrested by the closing valves. During expiration especially the portion of the jugular above the valves in the bulb rises and falls rhythmically with the heart movements, and this movement can also be felt unless the heart-beats follow one another in rapid succession, when it is sometimes impossible to decide whether the elevation and distention of the vein coincide with the apex-beat.

This presystolic jugular pulse, vigorous though it may be, cannot always be distinguished from the systolic venous pulse in tricuspid insufficiency without the aid of a tracing.

Another method of examination that may be thought of is auscultation. When the valves in the bulb are competent and close properly, a short clapping sound is heard as they are unfolded by the vigorous recoil of the blood-wave. The sound is the same in quality as that heard during closure of the semilunar valves, although, of course, it is not so loud. When the finger is placed on the vein, an impulse may be felt similar to that felt over the pulmonary valves in a case where the pulmonary artery is not covered with lung.

If the valves in the bulb are incompetent, a murmur may be heard instead of the normal sound.

Closure of the valves is effected by the backward-flowing blood. It is to be borne in mind that in the presence of a presystolic jugular pulse the following auscultatory phenomena may be heard by listening over the internal extremity of the clavicle on the right side:

1. A sound corresponding to the unfolding of the valves in the bulb and coinciding with auricular systole, hence presystolic in time.
2. The systolic heart-sound, which coincides with closure of the tricuspid valve.
3. The diastolic heart-sound, transmitted by the aorta (probably not by the pulmonary artery).

If there is a systolic jugular pulse, the systolic sound should be replaced by a murmur, which should be synchronous with the unfolding of the jugular valves produced by the ventricular systole. The murmur originates at the incompetent tricuspid valve.

It is actually possible sometimes to hear the three sounds which ought to be produced by the presystolic jugular pulse when the valves are competent, *i. e.*, ⌣ ⌒ ⌣. This, however, presupposes that the auricular contraction is vigorous and not too rapid. If the valves in the vein are incompetent, a practised ear may be able to detect the presystolic murmur. I believe I have heard it, but hesitate to demonstrate it clinically and should not care to depend on it for an accurate determination of the time. This is because the murmur is longer than the brief, sharp click of the valve, hence the latter makes a much more definite impression on the ear.

In the case of a systolic jugular pulse without insufficiency of the valves I have never been able to determine the time relation of the phenomena by auscultation, any more than in cases of genuine systolic pulsation of the internal jugular when the valves in the bulb were incompetent.

D. Gerhardt* heard a loud, deep, and very hollow sound over every part of the veins which were the seat of systolic pulsation; but only when the anacrotic limb of the sphygmogram was very steep. He explained the production of this sound by the sudden stiffening of the previously relaxed venous wall. The value of this sign for determining the time of the venous pulse I will not venture to decide.

The following general conclusions may be stated: Pulsation in the

* *Loc. cit.*, pp. 438, 439.

tributaries of the superior vena cava, visible in the neck or higher up, does not always indicate positively whether true incompetence of the tricuspid valve is present or not.

On the other hand, it may be definitely stated that hepatic vein pulsations are always systolic and occur only in tricuspid insufficiency, when —and this additional provision is important—the strength of the right ventricle is fairly good.*

Is it important in actual practice to ascertain the true origin of a venous pulse?

The various forms of venous pulse unquestionably indicate how far failure of the heart has progressed. This at first leads to a greater accumulation of blood in the systemic veins (*first stage*), and the vague movements in the incompletely emptied cervical veins—feeble undulation, according to the old terminology—is a sign of this excessive accumulation. The diastolic-presystolic pulsating movements which emanate from the overfilled auricle may be regarded as the *second stage*, while the *third stage* is represented by the presystolic-diastolic, pathologic (true) venous pulse. But as the latter can occur only when the tricuspid has become incompetent, and as tricuspid insufficiency is much more frequently relative, that is. due to changes in the muscle of the right ventricle and not to permanent tissue changes in the valves, its appearance may be regarded as proof that the right ventricle is no longer able to perform its task.

If the true venous pulse persists, the functional power of the heart is not only permanently reduced, but, as a rule, continues to diminish more and more. Finally, when the work of the heart is just sufficient to maintain life, the pulse may disappear. But it seems to me that this disappearance of the pulse is rarer than its persistence until death.

It appears, therefore, that the variety of venous pulsation present affords important prognostic data. Postmortem examination shows that cardiac insufficiency results in dilatation of the capillaries and veins. This is an important point, because the increase in the width of the channels which is thus effected is responsible for the diminution of the difference between the arterial and venous pressure, which takes place in spite of the fact that the strength of the heart is not diminished. Hence, to keep up this difference in pressure the heart is compelled to work harder.

Other disturbances caused by an increase in the size of the capillaries and veins in various organs traversed by them must also be taken into account. Finally, it must not be forgotten that the stretching of the walls of the veins and capillaries may impair their nutrition. Here the conditions are extremely complicated. The chief factor is probably the slowing of the blood stream in the capillaries and veins, which, as we know, depends on a number of different factors.

[The necessity of examining venous phenomena is now generally admitted, though the results as yet cannot be of immediate diagnostic value in many cases. (See Wenckeback, K. F., "Die Arhythmie als Ausdruck bestimmter Funktionsstörungen des Herzens," Leipzig, 1903. English translation ("Arhythmia of the Heart") by T. Snowball, Edinburgh and London, 1904. Mackenzie, James, "The Study of the Pulse," Edinburgh and London, 1902. "The Interpretation of the Pulsations in the Jugular Veins," "The American Journal of the Medical Sciences," July, 1907. Also numerous articles in various clinical and physiologic periodicals.)—Ed.]

* For the details see the section on tricuspid insufficiency.

3. **Blood, Lymph, Tissue-juices, and Dropsy.**—Oertel's writings called attention to the question of the effect a diminution of the work of the heart has on the blood and tissue-juices.

Oertel taught that it produced a *serous plethora*, *i. e.*, an increase in the quantity of the blood contained in the body, but a still greater increase in the percentage of water in the blood.

This is not so easy to determine as it seems. The investigations have to be carried out on human subjects, so that everything that might injure the patient has to be avoided.

The specimen of blood cannot be taken indiscriminately from any portion of the vascular circulation. The arteries are, of course, out of the question, leaving only the blood in the superficial layers of tissue—commonly called capillary blood—and that obtained from the superficial veins. In any case a small quantity only can be used. [H. Strauss has made important advances in regard to the retention of water by the use of a refractometer. See "Zeitschrift für klinische Medicin," Bd. lx.—Ed.]

Most of the investigations have been performed with whole blood. Besides the weight of the dry residue, the percentage of hemoglobin or the number of red corpuscles has been determined. The bulk of the dry residue is obtained from the constituents in suspension, and among these chiefly from the red blood-corpuscles. Hence the question arises whether the distribution of the red corpuscles in the plasma is uniform, and whether it is the same in the arteries, where the velocity of the blood is greater, as in the veins. If there is a difference, the results obtained by examining the blood specimen will not yield definite conclusions in regard to the constitution of the blood as a whole.*

Numerous experiments have been made with very different results.† Cohnstein and Zuntz explain these differences by the different methods employed in obtaining the blood, and Krüger holds the same view. Judging from animal experiments, we may assume that arterial and venous blood contain an equal quantity of red corpuscles.

Thus, in a series of twenty experiments on cats, Fr. Krüger found that the average percentage of hemoglobin determined by the spectrophotometer of Hüfner was, in the blood taken from the carotid, 100, and in the blood taken from the jugular vein, 100.1. The dry residue in the same experiments was, respectively, 100 and 100.5. His results with the blood obtained from the liver, spleen, and kidneys are somewhat different, and we need not go into these differences here.

Another important result, which is decisive in this connection, is the fact that he confirms the following proposition, advanced by Cohnstein and Zuntz.

"Congestion in any portion of the circulation, even when it is most transient, causes an increase of the percentage of hemoglobin and of the dry residue in the blood of that portion of the circulation." The fact that this point is frequently overlooked explains the difference in the results of earlier observers, who certainly cannot be accused of any want of accuracy.

* The various conditions which control this point are discussed in an important paper by Cohnstein and Zuntz, entitled, "Untersuchungen über den Flüssigkeitsaustausch zwischen Blut und Geweben unter verschiedenen physiologischen und pathologischen Bedingungen," "Pflüger's Archiv," Bd. xlii (1888), S. 303 ff.

† Cf. Friedrich Krüger, "Beiträge zur Kentniss des arteriellen und venösen Blutes verschiedener Gefässbezirke," "Zeit. f. Biologie," N. F. vol. viii; the entire series, vol. xxvi (1890), p. 452 *et seq.* An accurate bibliography will be found in the article just referred to.

But whenever the action of the heart is insufficient, congestive phenomena never fail to make their appearance, and the increase in the dry residue of blood taken from the point where congestion is present will depend on the degree of congestion. We may conclude that the deviation from the actual composition of the whole blood—in other words, the failure of the method—always lies in the direction of an increase of the dry residue in the congested blood.

How great this error may be it is, of course, impossible to say; hence any hope of deciding the question of the amount of water contained in the blood as a whole in cardiac weakness by this method must be given up from the very start. It is equally impossible to estimate the quantity of hemoglobin in the entire mass of the blood. I adhere to this statement in spite of the results and arguments which S. Askanazy* gives in his careful and comprehensive article. He usually determined the specific gravity of the whole blood, as well as that of the serum, by Hammerschlag's method, and, in addition, regularly weighed the dry residue. The question whether we have a right, in the conditions produced by circulatory disturbances, to speak of concentration of the blood at the periphery, and whether it is possible to bring direct proof of the existence of such an unequal mixture of the blood, he attempted to solve in the following manner: From six patients with uncompensated heart lesions blood was removed almost at the same time from a vein in the arm and immediately after death by puncture from the (right) heart. A slight difference was found, sometimes in favor of the venous blood from the heart and sometimes the opposite. The dry residues of the serums obtained from the two sources also showed similar differences. From this it was concluded that "there is no material difference between the amount of water contained in central as compared with peripheral blood."

I cannot admit that this conclusion is justified, and for two reasons: First, "almost at the same time" is not equivalent to "absolutely at the same time." Blood from the vein was always taken a short time, even if only the duration of a few heart-beats or a few respirations, before the blood was taken from the heart. Second, the blood in the right heart comes from the tributaries of the inferior, as well as of the superior, vena cava. That the fact of this mixture was not without effect may be seen from the **relations of the serum.** The differences in the four experiments (out of six) in which data are given are, in my opinion, not so inconsiderable as to be regarded as within the limits of a possible error, particularly as in every instance it is recorded that the dry residue of the heart-blood was slightly in excess.

The difference in the first four cases is in favor of the heart-blood.

$$+ 0.22;\ 0.20;\ 0.44;\ 0.43 \text{ per cent.}$$

The dry residue of the whole blood varies. Of the six cases, the venous blood in four is richer, and in two poorer, in dry constituents.

The figures are as follows, indicating by a *plus* sign the amount of dry substance in favor of the venous blood and retaining the order of the original:

$$+ 0.93;\ +\ 0.69;\ +\ 0.37;\ -0.25;\ -0.26;\ +\ 0.14.$$

If it be considered justifiable to deduce averages from this small series, they would be to the effect that whole blood *taken from the vein* contains 0.27 per cent. more dry residue than whole blood taken from the heart; while serum *taken from the heart* contains 0.32 per cent. more dry residue than the serum of the venous blood.

* "Ueber den Wassergehalt des Blutes und Blutserums bei Kreislaufstörungen u. s. w. (aus der medicinischen Klinik von Lichtheim in Königsberg)," "Deutsches Archiv für klinische Medicin," Bd. lix (1897), S. 383 ff.

This is not the place to go into an explanation of these facts. Suffice it to say, in order to refute Askanazy's sweeping assertion, that it does not seem justifiable, after what I have quoted, to draw positive conclusions from an examination of the blood made just before death.

Is there no other way? As the constituents in suspension can readily be separated from the serum, and the serum for our present purposes may be unhesitatingly regarded as identical with plasma, would a determination of the water-percentage in the serum yield a correct result?

To this we must reply:

There is an intimate relation between the fluid portion of the blood and the fluid in the tissues. There, too, is a circulation; for the blood plasma escapes from the capillaries into the tissues and returns from the tissues into the veins through the lymph-channels. During this process the composition is altered—the percentage of water in the lymph is appreciably greater than in the plasma of the blood. The same is true of the tissue juices. The actual values, to be sure, have never been directly determined; but the fact is well established, as, for example, through the experiments of L. von Lesser,* who added to and confirmed the statements of older investigators.

By ligating the thoracic duct in dogs, von Lesser impeded the entrance of lymph into the blood. He then removed from these dogs blood varying in quantity from 2 to 6 per cent. of their body weight. He was able to demonstrate a dilution of the serum even when the interval between two successive venesections was only twenty to twenty-five seconds. The concentration of the serum diminished in proportion to the amount of blood withdrawn. The following table shows the details of the investigation (the composition of the serum is indicated in percentages of its solid constituents):

Before Venesection (Normal Serum).	Loss of Blood Expressed in Percentage of Body Weight.			
	2–3	3–4	4–5	5–6
7.39	7.15	—	7.00	6.79
7.40	—	7.23	7.00	6.89
7.75	—	—	—	6.31
7.79	7.51	6.69	—	—
8.18	8.09	7.52	7.08	6.78

Von Lesser's investigations show another important fact. He says, quite correctly: "It seems that the alteration in the percentage of water is completed so far as it is possible under the given conditions, within a very short time. The change in the water percentage of the serum is the same whenever the amount of blood withdrawn is the same, whether the interval between the two venesections is measured by seconds or hours." From this it may be concluded that:

Since the tension in the capillaries which are immediately connected with the tissue-fluids is also reduced by the venesection, the tissue-fluid is under a higher pressure and is therefore driven into the capillaries, from which it is returned to the whole blood and diminishes the latter's concentration.

This is the basis for the explanation of the changes in the blood which

*See bibliography in Jürgensen, "Blutenziehungen," in v. Ziemssen's "Handbuch der allgemeinen Therapie," Bd. i (1880), S. 177 ff.

occur immediately after a partial failure of the work of the heart and disappear again with equal rapidity as the strength of the heart improves.

E. Grawitz* demonstrated that "when compensation is first disturbed, the fall of the blood pressure in the arterial system causes a relaxation of the smallest vessels and the transudation of tissue-fluid into them, and that, as a result, the blood in the capillaries, as far as their junction with the veins, becomes progressively thinner. This is removed as soon as the heart is strengthened and the arterial pressure raised."

In illustration of this I quote a case of Grawitz.†

Female, aged fifty-nine, with complicated valvular lesion—aortic insufficiency and mitral stenosis (from Gerhardt's clinic). At first the general condition was good and the patient was able to go about and do light work. Any physical exertion beyond her strength, such as going up stairs, or any emotional excitement brought on a disturbance of the heart action, causing acceleration of the pulse, which became small and irregular; muffling of the heart-sounds; dyspnea, but not edema. Rest in bed and small doses of digitalis always brought relief in a short time.

The results of the successive examinations are shown in the following table.

	Time.			
	October 9th. (General condition good.)	October 13th. (Heart disturbance: small irregular pulse; but neither cyanosis nor edema.)	October 19th. (For the past two days no cardiac disturbances.)	November 17th. (For several days slight disturbances without edema.)
Number of red corpuscles in millions	4.1	3.8	4.2	4.0
Dry residue of whole blood. Per cent.	20.65	19.55	21.71	20.6
Dry residue of serum. Per cent.	8.95	7.95	9.9	8.35

This observation, like the others reported by Grawitz, shows the same result; that is, the solid constituents of the serum diminished whenever the heart action became impaired and increased as the heart regained its strength.

It seems to me that these findings are susceptible of but one interpretation, the one proposed by Grawitz, namely, alterations in the physical conditions which are perfectly obvious.

But if the disturbance of the heart action is protracted, alterations in the vital processes of those tissues which take part in the formation of the tissue-fluid are superadded.

It was formerly held that the tissue-fluid is expressed from the capillaries by a process of combined pressure and filtration and that certain osmotic processes also take place between the fluid thus evacuated and that contained within the tissues. Now, it is taught, chiefly on the strength of Heidenhain's observations, that "purely physical factors, such as differences in the pressure and in the osmotic tension, do not in themselves suffice to explain all the phenomena observed in the formation of the tissue-fluid. We must, therefore, imagine that the living capillary wall takes part in the formation of the tissue-fluid through some kind of secretory process.

* "Ueber die Veränderungen der Blutmischung in Folge von Circulationsstörungen," "Deutsches Archiv für klinische Medicin," Bd. liv (1895), S. 588 ff.

† *Loc. cit.*, pp. 597 and 598.

"Nevertheless, however, purely physical factors may also coöperate, although we are as yet unable to decide how much of the work to attribute to these physical factors and how much to the activity of the capillary cells."

In these words Tigerstedt* sums up the present status of this question. It seems to me that we are perfectly justified in applying these views to pathology. They explain why, during protracted periods of impaired heart action, we do not always observe the same differences in the composition of the blood fluid. For Cohnheim long ago showed that the conditions on which the function of the capillaries depends vary in accordance with changes in the composition of the blood that flows through them and even with changes in its velocity.

Let us now inquire what the percentage of water in the blood, measured by the serum, is actually found to be in cases of persistent cardiac weakness.

Hammerschlag,† employing a method which is adequate, at least, for detecting the grosser conditions, determined the specific gravity of the plasma or of the serum in the blood taken from the finger-tip. The result was the same whether plasma or serum was used for the examination. He examined twenty-eight cases of cardiac insufficiency with or without associated valve lesions.

The normal values obtained by his method ranged from 1032 to 1029, the average being 1030, which tallies perfectly with figures given elsewhere.

Accepting these normal values, it was found that the specific gravity was below 1029 in 16 examinations—minimum, 1025; while it was 1029 and over in 9 cases—maximum, 1031.

In three of the cases observations were made at different times:

Case VII: five observations between the sixth of May and seventeenth of November; maximum, 1031; minimum, 1028.

Case XVIII: three observations in the course of a week; the fluctuations were slight—1026.5 to 1027.5.

Case XIX: two observations in the course of twelve days; again the range was small—from 1025 to 1025.5.

The total result is that in most of the cases there was a slightly larger percentage of water in the serum.‡

E. Grawitz correctly emphasized the importance of examining the same patient at different times when the action of the heart is variable, and it was by acting on this principle that he obtained the above-mentioned important results.

A few illustrations from his paper may be given to show the effect of permanent and considerable impairment of the heart action:

Female, aged thirty-eight, with mitral insufficiency and slight stenosis at the mitral orifice. When she was admitted she presented marked signs of congestion: small, irregular, frequent pulse; marked icterus; the face puffy and cyanotic; the extremities cool and edematous; severe dyspnea, hydrothorax, ascites, and enlargement of the liver and spleen. The abdomen had been tapped several times before the patient was admitted.

The results of the blood examination are shown in Table A, on p. 95.

Under treatment the dropsical effusions disappeared and the body weight diminished by twenty-one pounds. The heart action became regular and the patient was able to return to work.

Improvement was accompanied by a progressive increase in the dry residue of the serum, which finally attained almost the normal figure—10.75 per cent., according to Grawitz.

* Compare his "Lehrbuch der Physiologie des Menschen," Leipzig, S. Hirzel, Bd. i (1887), S. 337.

† "Ueber Hydrämie." (From Nothnagel's clinic.) "Zeitsch. für klin. Med.," vol. xxi, p. 475, 1892.

‡ These figures are calculated on the basis of the table in the original paper; Hammerschlag himself has not drawn his final results correctly.

TABLE A.

	Time.			
	Marked stasis. Examination soon after patient's admission on October 22d.	Improvement begins October 27th.	Improvement continues November 2d.	Marked improvement November 22d.
Number of red corpuscles in millions.....	5.1	6.0	6.7	5.05
Dry residue of whole blood. Per cent.....	21.75	22.29	23.08	23.17
Dry residue of serum. Per cent.....	7.7	8.06	8.9	10.1

Female, aged fifty-seven, with mitral insufficiency, ruptured compensation. Intense cyanosis and dyspnea; frequent quick pulse of low tension; arteriosclerosis; marked dilatation of the right ventricle, with epigastric pulsation; marked edema. Table B shows the course of the disease. This case ended fatally. The autopsy showed, in addition to thickening and retraction of the mitral leaflets, slight beginning granular atrophy of the kidneys.

TABLE B.

	Time.					
	Marked stasis; examination soon after admission September 18th.	Improvement begins September 19th.	Improvement continues September 25th.	Change for the worse October 11th.	Still further change for the worse November 10th.	After evacuation of 10 liters by incising the leg. November 23d.
Number of red corpuscles in millions.....	6.5	7.7	7.3	6.0	5.5	4 .9
Dry residue of whole blood. Per cent.....	18.13	23.0	23.0	20.15	22.34	21.08
Dry residue of serum. Per cent.....	10.30	9.99	10.25	9.72	8.5	9.1

Grawitz remarks that in this case the serum constantly contained dissolved hemoglobin in large and fairly constant quantities; no quantitative estimation was made. But the dry residue of the serum is, on the whole, too high. Possibly the individual observations are not comparable because they were merely estimations of hemoglobin. On the whole, this case also shows a constantly increasing or decreasing concentration of the serum, depending on the amount of work performed by the heart.

The following observation is somewhat different (see table C):

TABLE C.

	Time.		
	Admission September 16th.	Improvement begins September 30th.	Improvement continues October 9th.
Number of red corpuscles in millions....	3.9	4.1	4.1
Dry residue of whole blood. Per cent...	20.33	21.0	21.28
Dry residue of serum. Per cent.....	9.45	8.15	8.32

Female, aged fifty-eight. Marked dyspnea, cyanosis, edema. There is a loud systolic murmur at the apex; bigeminus and arhythmia. The pulse is small, frequent, and irregular.

After a brief period of treatment the woman was able to go back to work, the edema having almost entirely disappeared. But the dry residue of the serum had decreased materially, which Grawitz explains by assuming that the patient was anemic—not a very satisfactory explanation.

The general conclusion of Grawitz's investigations is that, on the basis of serum determinations, an increase of the water-percentage in the blood is more frequent than a decrease in cases of cardiac insufficiency. Possibly, but not always, when the heart action is permanently inadequate, the percentage of water in the blood is approximately proportionate to the functional power of the heart.

Grawitz's own conclusions are different because he laid more stress on the red corpuscles and the dry residue in the whole blood than seems to me justifiable.* He obtained the blood for his examinations by puncturing a superficial vein in the arm and also by making a superficial incision in the finger-tip or the lobe of the ear. In cases with marked stasis the blood taken from the vein was more hydremic than the "capillary" blood. From this observation he deduced conclusions which, it seems to me, lack a proper basis; for it is impossible to determine the degree of increase in the red corpuscles, and with them in the solid constituents, of congested blood at one point only in the veins or capillaries, hence the result thus obtained cannot be used as a basis for calculating the percentage of water or the number of red corpuscles in the entire mass of the blood contained in the body.

The solid constituents in the serum of the venous blood and in the blood taken from cutaneous incisions at approximately the same time were determined only twice in one and the same patient, namely, in the case cited above (table A). The result was as follows:

	Blood from Vein.	Blood from Skin Incision.
October 27th, dry residue.....	8.06 per cent.	8.7 per cent.
November 22d, " "	10.1 "	10.03 "

Hence the first time there was an excess in favor of the skin blood of 0.64 per cent.; but the second time the serum taken from the cutaneous blood contained less solid constituents by 0.07 per cent. than the serum of the venous blood. Hence the difference in the density of these two specimens requires further explanation. There is another point. The examination in the case of Mrs. J. L. (table B) on the eighth of September showed the following condition of the blood:

Red blood-corpuscles,.....	6.5 millions
Dry residue of blood,.....	18.13 per cent.
Dry residue of serum,.....	10.30 "

Another case (Mrs. D. R.)† showed:

Red corpuscles,.....	2.7 millions
Dry residue of blood,.....	18.01 per cent.
Dry residue of serum,.....	8.08 "

The difference in the number of red corpuscles in these two cases is, therefore, 3.8 millions, while the difference in the dry residue of the whole blood is only 0.12 per cent. Taking the number of red blood-cells in the case of Mrs. J. L. as 100, that of Mrs. D. R. will be 42.5. The dry residues, on the contrary, are in the proportion of 100 to 99.3.

The same thing is observed at different times in the same patient (Mrs. J. L., table B). Comparing the figures obtained on the nineteenth of September and tenth of November, the following proportions are obtained:

Red corpuscles, 100 to 71.4.

Dry residues, 100 to 97.1.

The blood in every instance was taken from the vein. I feel perfectly convinced that so conscientious an observer as Grawitz can never have been guilty of gross carelessness; but I am at a loss to decide where the mistake comes in, for without some mistake I cannot see how the discrepancy is to be explained.

* Grawitz, *loc. cit.*, p. 609.

† Grawitz, *loc. cit.*, p. 607.

Stintzing* and Gumprecht also worked with whole blood; they determined the dry residue and the percentage of hemoglobin by Gowers' method. The method of weighing a fixed amount of blood, which Stintzing has made more convenient by taking a small quantity of blood (0.2 to 0.3 gm.) with a broad surface and allowing it to dry for twenty-four hours at a temperature of 65° to 70° C. (147° to 158° F.) without waiting for complete removal of the water, is sufficiently reliable. The values obtained are, of course, not absolutely accurate, but they are very nearly so, and can readily be compared with one another. The blood was obtained from a deep puncture in the finger-tip.

Stintzing and Gumprecht determined the dry residues in twenty healthy men and thirteen women, using their own method, with the following results (normal values):

	Average of Dry Substance Per Cent.	Maximum Per Cent.	Minimum Per Cent.
Men	21.6	23.1	19.6
Women	19.8	21.5	18.4

From Table VIII (cases of heart disease with ruptured compensation) of the two investigators I abstract the following:

In men the normal mean was surpassed in one instance with 22.7, so that the normal maximum was not attained.

The normal minimum was attained or slightly exceeded in nine instances. The absolute minimum is 14.4.

Altogether, twenty-five separate observations were made on eleven different men. Five men with heart disease fell below the normal minimum of 19.6 per cent. Six exceeded it.

In women the average was surpassed twice with 20.6 and 21.4 per cent.; hence the normal maximum was not attained among women either. The normal minimum was attained twice. The absolute minimum is 15.3.

Altogether, eleven separate observations were made on six different women. Three women with heart disease fell below the normal minimum of 18.4; three exceeded it.

Stintzing and Gumprecht conclude that:

Hydremia of the blood in the sense of a relative increase in the normal percentage of water unquestionably occurs in many cases of cardiac insufficiency."

The conclusion seems to me quite justified. As I have frequently remarked, if a mistake is made in examining the blood in states of congestion for the purpose of determining the dry residue in the whole blood, it will be in the positive and not in the negative direction. The individual values obtained may not correspond to the true values; but as they can only be too high, the conclusion drawn from the total result will be correct.

Stintzing and Gumprecht also found† that, as the edema subsides

* "Wassergehalt und Trockensubstanz des Blutes beim gesunden und kranken Menschen," "Deutsches Archiv für klinische Medicin," Bd. liii (1894), S. 265 ff. This article contains an extensive bibliography.

† *Loc. cit.*, pp. 296, 297 (Tables X and XI).

and compensation is restored, the water-percentage of the previously hydremic blood diminishes; and the converse of this is also true.

The results obtained by Grawitz, which have been cited above and which were obtained by examining serum, are to the same effect. Also those obtained by Askanazy.* The latter sums up his results as follows:

"Blood-serum is always more or less diluted and this dilution in general is in proportion to the degree of dropsy. It increases as the dropsy increases and diminishes as the dropsy subsides, disappearing altogether when compensation is restored." The same was true of the specific gravity of the serum in the nineteen cases examined by Askanazy, in twelve of which a number of determinations were made.

Grawitz † calls attention to a source of error in the older investigations of the whole blood performed by determining the hemoglobin percentage of venous blood with the customary clinical colorimetric methods. In conditions of stasis the venous blood contains large quantities of reduced hemoglobin, which possesses high coloring power—much higher than that of oxyhemoglobin, for which the various apparatus are graduated, so that in making the observations the values obtained for congested blood must be too high.

Must we assume that the blood contained in the vessels is increased—that there is a true serous plethora? Stintzing and Gumprecht answer "yes." They say: the variation in the number of red corpuscles associated with changes in the concentration of the blood, which in turn depend on the increase and decrease of the work of the heart, cannot be explained by the destruction or the new formation of red blood-cells. If that were the case, the regenerative forms which occur in other severe anemias would have to be seen, for the changes are completed in a comparatively short time; but these regenerative forms are absent.

So far I agree with them entirely. But they go on to say:

Capillaries and veins, as we know, are dilated as compared with their normal caliber in cases of cardiac insufficiency attended by stasis; and since there is not a corresponding emptiness of the arteries, the blood-channels, on the whole, must be enlarged and their contents therefore increased.

In reply I would say that it has not been demonstrated that the stasis in cardiac insufficiency affects the entire domain of capillaries and veins; nor are we in a position to give anything like a definite opinion in regard to the amount of blood in the arteries *in their entire extent.* The conclusion would be justified if the premise that dilatation of the capillaries and veins takes place without any accompanying deviation from the normal in the contents of the arteries were correct. But do we know anything about this point? I think not. I therefore do not consider it proved that serous plethora exists under these conditions.

Summing up the entire question, we have the following *general conclusion:* Cardiac insufficiency is attended more often by dilution than by concentration of the whole blood. It is probable that there is some change in the distribution of the red corpuscles and that they are more numerous wherever there is retardation in the blood current or stasis. From this it should follow that, if there is no increased formation of red blood-corpuscles, the number must be less that normal in those parts of the body which are not affected by the stasis. And, what is more important, arterial blood must contain less, and venous blood more, red corpuscles than normal. Finally, it would follow that the quantity

* *Loc. cit.*, p. 409 (Table IX). † *Loc. cit.*, pp. 596, 597.

of hemoglobin in the circulation, that is, the quantity of oxygen supplied to the tissues, must decrease in proportion as the stasis—the temporary exclusion of red blood-corpuscles from the pulmonary circulation—increases. It is not necessary to explain the serious injury to the functional power of all the tissues of the body which this would entail.

This conception, which was first suggested by Oertel, does not differ in any material respect from the prevailing view. It emphasizes that view by adding to the dictum that diminution of the heart action causes a diminution of the quantity of blood supplied to the tissues in a unit of time, another—that this increased quantity of blood contains fewer red blood-corpuscles. Although the number of red corpuscles present in arterial blood in cases of stasis has never been directly counted nor its percentage of hemoglobin determined definitely,—it is hardly necessary to say that the same investigations would have to be made on the venous blood at the same time,—it seems to me we are quite justified in assuming that the red corpuscles actually are so distributed.

Retardation of the current when the blood contains insufficient quantities of oxygen is not without effect on the tissues to which the blood is supplied. Disturbances are inevitable, for the ancient truth "the blood is the life" loses none of its force even in our day. This much is certain: A part which receives too little efficient blood and, we may add, from which the blood after it has been used up is removed too slowly, dies. Death ensues within a certain time, which varies for the different organs. The higher their differentiation or, as we also say, the finer the organization of the tissue, the more indispensable is a constant and adequate blood-supply. There is always a period during which perfect recovery is possible.

The diminution of the oxygen and the increase of the carbon dioxid in the more slowly moving blood afford some indication as to the severity of the injury that may result. But they probably do not in themselves suffice to explain what goes on. From the fact that "fatigue stuffs" accumulate in a nerve or muscle which is functionating out of proportion to the supply of blood which it receives we may conclude that another factor is also concerned. This factor is produced by the automatic activity of the working organ and disappears and becomes innocuous whenever the part is adequately flushed out with blood.

The doctrine of autointoxication is firmly anchored on its foundations. Although the identity of the substances that are active in the body is as yet insufficiently or not at all known, that they exist we are hardly justified in doubting. It applies to all the organs. Further elucidation is a matter for the future. For the physician it is important to realize that injurious substances of this kind are partly concerned in disturbances which are included under the general term of stasis because retardation of the circulation is not always proportionate to the effects produced. If it were purely a question of mechanics, we should expect the changes to harmonize more fully with the rapidity or slowness with which the blood is renewed than is actually the case. There is yet another, unknown quantity in the problem, that we call the resisting power of the entire body or of one of its parts. But we must also take into account the loss of resisting power in organs which, for some time, have been insufficiently supplied with blood. This loss of resisting power I am inclined to attribute not only to a deficiency of nutritive material,—a negative cause,—but also to the positive action of the substances which are produced on

the spot, not in a distant portion of the body, and therefore develop their activities on the spot.

These substances may be produced, first of all, in the vessels themselves; in the blood-clots or thrombi which form in them. We know that two conditions are necessary for the formation of a thrombus: the intima of the vessel—its endothelium—must have its vitality impaired at the seat of the thrombus, and the circulation of the blood must be retarded at the same point. That the former may, under certain circumstances, be the result of the latter is also well known.

This is not the place to go into the doctrine of coagulation of the blood at length. It should be mentioned, however, that in heart patients thrombus formation is frequently observed in the heart itself—particularly in the right heart, next in the sphere of the pulmonary artery, and more rarely in the systemic arteries. In the capillary system of the greater circulation and in the systemic veins we might expect to find thrombosis oftener than is the case. Even when the circulation is greatly retarded for a considerable period of time thrombosis is not often found either in the lower or in the upper extremities.

I once saw a very instructive case: A man, fifty years old, who suffered from chronic bronchitis and slight cirrhosis, with numerous bronchiectases in the atelectatic lower lobe of the left lung, presented marked dilatation of the right heart with considerable hypertrophy of the right ventricle. The left side of the heart was approximately normal. During the last two months of life thrombi developed in the external and internal jugulars, innominate, brachiocephalic, subclavian, axillary, and brachial veins of the left side. The thrombosis had been preceded and caused by a chronic endophlebitis.*

It is obvious that the necessary conditions for embolism are present. It is probable that the pulmonary arteries are most frequently involved, the embolus being derived from the right heart.

Disturbances in the movement of the body-juices, which are always associated with failing heart power, must also be mentioned. We may make the general assertion that heart weakness causes slowing of the tissue-circulation.

If the retardation attains a certain degree, the tissue-fluids accumulate first in the subcutaneous connective tissue and later in the large cavities of the body—dropsy. There is no doubt that one of the chief conditions for the production of dropsy is slowing of the circulation. Wherever the slowing is greatest, edema develops most early. In the most dependent portions of the body, which are farthest removed from the heart and in which the effects of gravity are most marked, there "congestion edema" develops earliest. The edema subsides as soon as the strength of the heart improves, and gravity is made to act in a different direction—one more favorable to the circulation [by elevating the part, etc.—Ed.].

These facts are firmly established, and hence the conclusions deduced from them are probably unassailable.

On the other hand, we are far from understanding all the details of the process. Negatively, we may say that the phenomenon is due to more than a mere alteration of pressure or nutritional change in the vessel-wall, making the latter more permeable for the blood. Again, the peculiar

* The details of this case are given by Dr. Rudolph Heiligenthal, "Ein Fall von ausgedehnter marantischer Thrombose im Gebiet der Vena cava superior," Tübingen Dissertation, 1893.

properties of the capillary walls come into play; their activity is dependent upon the vital activity, and hence on the nutrition of the individual cells of which they are composed. The manner, as well as the degree, in which these are injured probably varies at different times, but for the present we know very little about the matter.

The movement of the lymph largely depends on the movements of the body, especially of the muscles. Failure of cardiac power is followed by diminution of muscular activity, which is such an important factor in the onward movement of the lymph.

How far retardation of the lymph stream may affect the physical constitution of tissues has never been determined.

Landerer* correctly pointed out that the elasticity of the tissues may be impaired by the accumulation of tissue-fluid. In cases of marked edema this is proved by the pit which remains after pressure with the finger. But even a very slight initial degree of edema, inappreciable to the eye, may have the same effect, especially when the tissues are ill nourished, as in the case of those who have long suffered from inadequate heart action. Loss of elasticity of the tissues is accompanied by diminution of tissue tension, which is one of the most important factors in the circulation of the lymph. It is quite conceivable that the development of edema may be indirectly favored in this way.

In parts of the body that are merely the seat of simple venous hyperemia there is no impediment to the free evacuation of the lymph. On the other hand, the emptying of the thoracic duct into the subclavian vein is interfered with by the presence of stasis. Thus we have two antagonistic conditions, and it is not possible to decide beforehand which of the two will predominate; in fact, this may be subject to variations.

Among the grosser injuries should be mentioned, first, diminution of the quantity of urine, which is an inevitable consequence of the insufficient perfusion of the kidneys resulting from cardiac insufficiency.†

Finally, it should be mentioned that the question‡ has been raised and is now being discussed whether the nervous system is directly involved and whether the formation of tissue-juices represents a secretory process under the control of the nervous system.

It follows from all that has been said that the manner in which the tissue-fluid is formed under normal conditions is, after all, but little understood, and that it is probably controlled by a number of conditions in addition to the mere physical factors. These other conditions are dominated by the vital activities of organized structures. If that is the case, we can understand why we are unable to clear up this very difficult and complicated question of the effects of failing heart power.

A few facts in regard to the composition of the fluid retained in the tissues should be given:

1. Edematous fluids that occur from stasis are always considerably poorer in solid constituents than the blood plasma or the blood-serum.

Unfortunately, as Runeberg§ said, no observations have been made in regard to the percentage of water in the transudates and in the serum of the same patient. But the variation from the normal and from the

* "Die Gewebsspannung in ihrem Einfluss auf die örtliche Blut- und Lymphbewegung," Leipzig, F. C. Vogel (1884).

† For details see p. 129.

‡ See Tigerstedt, "Lehrbuch der Physiologie," Bd. i, S. 338.

§ "Klinische Studien über Transsudationsprocesse im Organismus, I," "Deutsches Archiv für klinische Medicin," Bd. xxxiv (1884), S. 1 ff.

figures which are obtained in cases of insufficiency of the heart* is so great that the fact itself becomes indisputable.

According to Karl Schmidt, the blood plasma of a healthy individual contains in 1000 parts: Solid substances, 98.49, composed of: fibrin, 8.00; albumin and extractives, 81.92; inorganic salts, 8.51. The figures given below should be compared with these.

It appears that the differences in the composition of the transudate chiefly affect the albumins; the salts vary but little.†

2. The region from which the tissue-fluid is obtained has a great influence on the composition. That obtained from the subcutaneous connective tissue always contains the smallest percentage of solid constituents.

Thus, for example, Runeberg ‡ found the following amounts of albumin: in his patient No. 24 (chronic interstitial pneumonia, chronic heart lesion, arteriosclerosis) he obtained the following results, the fluid being taken from both places at the same time:

From the peritoneal cavity, 16.4 per mille; from the subcutaneous connective-tissue, 2.0 per mille. In patient No. 26 (chronic organic heart disease) the transudate (taken at the same time) from the peritoneal cavity contained 23.0 per mille, and that from the subcutaneous connective tissue 2.4 per mille.

F. A. Hoffmann§ also made a number of determinations which are shown in the following table:

No. of Table (Hoffmann).	Age; Sex; Diagnosis.	Part from which Fluid was Obtained.	Albumin Per 1000.
13.....	Male, aged fifty years, emphysema, severe stasis with rapid disappearance of disturbances.	Left thigh.	2.7
14.....	Male, aged fifty years; emphysema; degeneration of the heart muscle.	Leg.	3.0
19.....	Female, aged forty; insufficiency of the aortic and mitral valves; albuminuria.	Thigh.	4.1
20.....	Male, aged fifty, with marked stenosis of the aortic valves; dilatation of the entire heart.	Thigh.	4.3
21.....	Female, aged sixty; emphysema; bronchitis; old fibroid myocarditis.	Thigh.	4.4

The highest quantity of albumin reported I find in a paper by Reuss ‖ in a case of degeneration of the heart. The quantity of albumin per thousand in the blood obtained from the edematous thigh was, at the first puncture with a Southey's tube, 2.24; second puncture, 11.50.

This is an unusually large quantity, and as no other data are given, we shall have to take the statement on trust. However, I have no reason to doubt it because the reporter is a most conscientious investigator and is reporting an investigation of his own.

* See pp. 93–97.

† See Runeberg, "Klinische Studien über Transsudationsprocesse im Organismus, II," "Deutsches Archiv für klinische Medicin," Bd. xxxv (1884), S. 266 ff.

‡ *Loc. cit.*, p. 10 of the first article.

§ "Der Eiweissgehalt der Oedemflüssigkeiten," "Deutsches Archiv für klinische Medicin," Bd. xliv (1889); taken from the table on p. 314.

‖ "Beiträge zur klinischen Beurtheilung von Exsudaten und Transudaten," "Deutsches Archiv für klinische Medicin," Bd. xxiv (1879), S. 583 ff.

In regard to the individual cavities, whether there is any difference between the contents of the pleural and that of the peritoneal cavity, etc., is a question that has never been settled. The number of analyses of fluid taken at the same time from different cavities from the same patient is too small to form the basis of a positive opinion.

In a case of heart disease with universal dropsy Runeberg * found 8.4 per 1000 of albumin in the peritoneal effusion and 9.0 in the pleural effusion.

The difference cannot be very great, as is proved by the observations made by Eduard Neuenkirchen† on the large clinical material of the Riga allgemeinen Krankenhaus. His method, to be sure, is not above criticism, consisting in specific gravity determinations immediately after puncture, without correction for temperature. In a small number of cases the temperature of the room was taken twenty hours later. Nevertheless the results are sufficiently accurate to justify the answer to the question which has been given.

Neuenkirchen obtained in general venous stasis due to heart disease, in most cases non-compensated valvular lesions, the following values:

Specific Gravity.	Maximum.	Minimum.	Mean.
Peritoneal effusion	1016	1007	1012.4
Pleural effusion	1016	1007	1012.2

Twenty-eight peritoneal effusions taken from 10 patients and 21 pleural effusions from 14 patients were analyzed.

3. In the same patient the composition of the fluid varies at different times even when taken from the same cavity. The variation is due chiefly to the fluctuations in the quantity of albumin.

Ranke,‡ in a case of mitral insufficiency and stenosis, examined the peritoneal fluid obtained at four successive punctures with the results given in tabular form below. Méhu, in his great work, also gives a somewhat less accurate statement in regard to individual cases.

RANKE'S TABLE.

Puncture.	Specific Gravity.	Dry Residue per Thousand.	Albumin per Thousand.	Inorganic Constituents per Thousand.
December 10, 1884, 7750 c.c.	1017.2	—	—	7.79
January 12, 1885, 8060 c.c.	1016.6	48.59	35.41	8.07
May 4, 1885, 9000 c.c.	1018.4	53.32	41.34	8.05
June 20, 1885	1019.3	54.94	43.36	7.85

* Table 1, p. 282, of Article II, "Deutsches Archiv für klinische Medicin," Bd. xxxv.

† "Ueber die Verwerthbarkeit des specifischen Gewichtes und des Eiweissgehaltes pathologischer Trans- und Exsudate zur klinischen Beurtheilung derselben," Dorpat dissertation, 1888.

‡ "Ueber Punctionsflüssigkeiten," "Mittheilungen aus der medicinischen Klinik zu Würzburg," edited by Dr. C. Gerhardt and Dr. Fr. Müller, Bd. ii, Wiesbaden, J. F. Bergmann, 1886, S. 191.

With the following table by Reuss * I quote a case of Méhu's in which an effusion into the pleura due to mitral insufficiency was evacuated six times by puncture. The length of time intervening between the individual evacuations and the quantity of fluid obtained I do not find noted.

Dry Residue per Thousand.	Organic Substance per Thousand.	Salts per Thousand.
33.80	24.90	8.90
29.20	20.60	8.60
28.45	20.09	8.36
25.97	17.27	8.70
36.38	27.58	8.80
36.40	27.90	8.50

The determination of the "organic substances" affords a valuable index for the quantity of albumin contained in the blood obtained at each puncture. This shows very great fluctuations. Taking the organic substances obtained at the first puncture as equivalent to 100, the figures corresponding to the remaining punctures are 82.7, 80.7, 70.0, 110.7, 112.1.

Albumin reached its minimum after the fourth puncture and after that exceeded the original quantity.

On the other hand, if we take the salts obtained at the first puncture as equivalent to 100, the remaining figures will be: 96.6, 93.9, 97.8, 98.9, 95.5.

The fluctuations are much less; the original figure is never attained and there is no agreement with the "organic substances."

In regard to the prognosis, it is not likely that the variations in the quantity of albumin in the transudate will ever afford any valuable data. In regard to diagnosis also we must rest content with the general statement that inflammatory exudates have a higher specific gravity and a larger percentage of albumin than non-inflammatory exudates. It seems to be impossible to find any absolute values that are positively constant as regards specific gravity or the percentage of solid constituents in general, or of albumin in particular, in a unit volume of the fluid.

This is the final result of the many laborious investigations that have been made, and it does not seem to me that they afford us any accurate information as to whether the transudations are due merely to physical factors or to some conditions intimately connected with the continuance of life.

Finally, the changes in the tissues which are often encountered after venous hyperemia has existed for a long time may be briefly mentioned. In general it may be said that a certain tendency to the development of connective tissue is associated with venous hyperemia and that this tendency varies in different organs. The matter will be made the subject of special treatment elsewhere. Which of all the causes concerned in the general picture of stasis it is that causes irritation of the connective tissue we do not know.

4. **Bronchi and Lungs; Pleura.**—First of all, the peculiar relations of the blood-vessels in the bronchi and in the lungs must be discussed. There is here a double system of blood-vessels.

The bronchial arteries subserve the nutrition, while the pulmonary artery and its branches control the function of the lungs. On the whole, this view is correct, although the line of separation must not be drawn

* *Loc. cit.*, Table III a; Hydrothorax; clear transudate, p. 612.

too sharply. What we know in regard to the question can be briefly summed up, as Küttner* has done, in the following:

"1. The branches of the pulmonary artery are distributed chiefly and most extensively to the infundibula and alveoli, but also to the mucosa of the bronchi. A few branches also enter the interlobular and subpleural connective tissue.

"The function of the pulmonary artery is, therefore, chiefly, although not exclusively, secretory.

"2. The so-called bronchial arteries ramify chiefly in the outer layers of the bronchial wall, in the subpleural and interlobular connective tissue; but they also send small branches to the alveoli and to the mucosa of the bronchi. The bronchial arteries are, therefore, chiefly, but not exclusively, nutrient vessels.

"3. The vessels arising from the capillary region of the infundibula and from the mucosa of the bronchi pour their contents chiefly into the pulmonary veins, while those vessels which originate in the capillary regions in the outer layers of the bronchial wall and in the subpleural and interlobular connective tissue are called bronchial veins and empty into the azygos and superior vena cava."

Another point brought out by Küttner should be emphasized, namely, that the pulmonary artery is anatomically an end-artery in Cohnheim's sense of the term; for true arterial anastomoses cannot be demonstrated in the vessel.

But there are extensive capillary connections. The terminal branches of the artery come so close together that they are only separated by the capillaries of one or two alveoli. These capillaries themselves anastomose among themselves and, "under certain conditions, may develop into large, collateral (anastomotic) branches. There are, in addition, numerous other connections—arterial anastomoses between the pulmonary artery and the bronchial arteries. As for their capillary connections at the points where the bronchi merge in the alveolar ducts, these are all the more important as the branches of the bronchial arteries communicate among themselves by means of arterial anastomoses." Küttner comes to the final conclusion that the pulmonary artery is not an end-artery in the functional sense of the term.

These very important facts were discovered partly by anatomic investigations carried out on the lungs of animals and men with the aid of every known method, and partly by experimentation on the living animal. Küttner,† after ligating the pulmonary artery in young rabbits and dogs, slowly introduced a suspension of cinnabar into the circulation through the external jugular. The pigment was found not only in the branches of the bronchial artery, but also in the branches and ligated trunk of the pulmonary artery, the capillaries of the alveolar walls, and the pulmonary veins.

The cinnabar must have entered the system of the pulmonary artery through branches of the bronchial, tracheo-esophageal, mediastinal, and pericardiophrenic branches, making its exit through the vena azygos and the superior vena cava.

But the large quantities of cinnabar that were found in the portions of the lungs corresponding to the ligated pulmonary arteries afford an additional proof that the circulation in these portions of the lung is relatively quite marked.

The important bearing of these facts on pathology is obvious. They enable us to understand how the obstruction in the distribution of the

*"Beitrag zur Kenntniss der Kreislaufverhältnisse der Säugethierlunge," von Dr. Küttner (Pathologisches Institut, Heidelberg), "Virchow's Archiv," Bd. lxxiii (1878), S. 476. The quotation will be found on p. 502.

† For particulars in regard to the method employed see *loc. cit.*, p. 517.

pulmonary arteries, such as was present in my observation No. 2 (see p. 31), is compatible with the continuance of life. Since the occluding blood-clot enlarged gradually, time was afforded for the necessary dilatation of the collateral channels.

The significance of the vascular connection in the lungs between the lesser and the greater circulation must be borne in mind in all disturbances of the movement of the blood through the lungs. Unfortunately, it can rarely be accurately considered.

The propriety of a mechanic analysis of the changes in the pulmonary circulation which must ensue when either the right or the left side of the heart alone suffers a loss of power cannot be denied.

On the other hand, it is to be remembered that loss of power limited exclusively to one half of the heart, such as can be artificially produced on the model, probably never occurs in a patient who has had heart disease for some time, for a considerable portion of the heart muscle is common to both the left and the right side of the heart.

It is hardly to be supposed that the muscle, which forms a continuous mass of tissue, derives its nourishment from whichever half of the heart it is specially assigned to. Any disturbances of nutrition that may occur must affect the entire mass; they are not arrested by the boundary between the left and the right side of the heart. Of course, the disturbances may be more marked on one side than on the other; but they must be present on both sides. One side of the heart cannot be diseased while the other remains intact; there cannot be insufficiency of one side coincidentally with unimpaired power of the other. How far these necessary conditions hold good cannot be determined in general, but may possibly be approximately estimated in an individual case. There is no doubt, at least, that they tend to make the pathology of the condition more difficult to understand.

The very fact that the two sides of the heart have part of the muscle in common, like the peculiar vascular connections between the greater and the lesser circulation, should be a warning against a one-sided, purely mechanic conception and interpretation of the symptoms emanating from the diseased heart. Now to take up the details.

1. **Bronchial catarrh** is a frequent, as well as a dangerous, concomitant of cardiac insufficiency. It is not without good reason that it is directly attributed to the circulatory disturbance in the bronchi, hence the term "stasis catarrh" is frequently used. When the pressure in the aorta falls and a large quantity of blood accumulates in the veins of the greater circulation, this forms an impediment to the emptying of the bronchial veins and their auxiliaries into the superior vena cava. The pulmonary veins also are powerless to remedy the condition. Instead of relieving the congestion in the bronchial mucous membrane, they usually become engorged with blood from the collateral connections and thus become involved in the general stasis.

The development of bronchial catarrh is undoubtedly caused chiefly by the accumulation of blood in the superior vena cava.

Special forms of bronchial catarrh for each kind of valvular lesion have been described, so-called "mitral bronchitis" being given the first place on the list.* Hoffmann quite properly points out that this mitral bronchitis only becomes severe

* See F. A. Hoffmann, "Die Erkrankungen der Bronchien," Nothnagel's "Handbuch," Bd. xiii, S. 78, 79 (special section).

when the stasis begins to extend beyond the right heart—that is to say, when the strength of the left ventricle is impaired.

Von Basch (see p. 124) reached the same conclusion, although he investigated the question from a different standpoint.

But the mere accumulation of blood in a mucous membrane is not synonymous with inflammation. To produce inflammation a connecting link is required, which we may imagine as follows: The stasis is very soon followed by an increased desquamation of epithelial cells and a slight swelling of the congested mucous membrane. Imbibition may occur first and may be due to unfavorable nutritive conditions in the capillary walls, brought about by the retardation of the blood as it passes through the capillaries.

Denudation of the mucous membrane due to the loss of epithelial cells thereupon renders it more sensitive to the irritation of foreign bodies, chiefly dust entering with the inspired air. The severity of the inflammation produced depends on the character and composition of these foreign bodies—whether they are germs capable of development or mere foreign bodies incapable of multiplication, on their "virulence," and on their chemic or mechanic properties.

Owing to the impaired nutrition of the insufficiently nourished mucous membrane an inflammation, no matter how it is produced, spreads and lasts longer than under normal conditions.

Finally, the mucous membrane, after repeated attacks of inflammation and quite apart from the question of its nutrition, is apt to become a *locus minoris resistentiæ*. A heart patient is preëminently subject to catching cold and is scarcely ever free from bronchial trouble. The bronchitis again acts unfavorably on the heart, imposes more work on it, and brings on exhaustion, so that the final collapse occurs earlier than would have been the case otherwise—all this has already been discussed (see p. 48).

There is nothing peculiar about bronchial catarrh in cardiac insufficiency. It should be pointed out, however, that the catarrh comparatively often extends to the finer ramifications and is more severe in the posterior, lower portions of the lungs than in the anterior, upper portions. As a rule, the disease is bilateral. But either the right or the left lower lobe may be more extensively involved than its fellow. Sometimes this is due to changes of long standing, especially pleural adhesions, or to extrinsic causes, such as position in the case of bed-ridden patients. The exciting cause—diminution of the circulation due to heart weakness—always asserts itself: whatever interferes with the circulation of the blood in the lungs at any point favors the development of bronchial disease.

Similarly, there is nothing characteristic about the sputum. Quantity as well as composition is subject to the widest variation in individual cases. In only one respect is the sputum peculiar: that is, it may contain the elements which were first described by E. Wagner as "Herzfehlerzellen" [usually known in English as "heart-disease cells."—Ed.].

F. A. Hoffmann* says of these structures: "They resemble in every respect the so-called desquamated epithelial cells of the alveoli, but are distinguished by the fact that they contain yellow and brown pigment, partly diffuse, partly granular." This pigment contains iron and ought, therefore, to be designated hemosiderin (Neumann). Unchanged red blood-corpuscles or hematoidin crystals (free from iron) may occur ex-

* "Die Bedeutung der Herzfehlerzellen," "Deutsches Archiv für klinische Medicin," Bd. xlv (1889), S. 252.

ceptionally in heart-disease cells, but have nothing directly to do with their formation.

Hoffmann positively states that heart-disease cells are desquamated alveolar epithelium containing altered hemoglobin.

Sommerbrodt* also, who tried to solve the question experimentally, regards the cells as the derivatives of alveolar epithelium. His explanation of their origin differs materially from Hoffmann's; but it would take me too far afield to go into this question in detail.

On the other hand, it is believed that white blood-cells are intimately concerned in the formation of heart-disease cells. The chief exponent of this view, Lenhartz,† gives the following description: "In a pure mucous, somewhat tough and gelatinous ground-substance, clear or pale yellow, or rarely brownish, are fine and coarse granules of a yellow or brownish red color, either singly or closely aggregated. Sometimes there is only a mere superficial, somewhat dark, dust-like deposit. When such a fleck of mucus with its yellow or rather more reddish stippling is examined under the microscope, a large number of cells arranged in rows or heaps are at once seen embedded in the ground-substance, which is quite homogeneous or infiltrated with pale, myelin-like droplets.

"The cells usually have sharp outlines, range in size from that of a colorless blood-cell to five times its size, are round, oval, spindle-shaped, or polygonal in shape, and usually contain one or more vesicular nuclei. The nucleus is often partly or completely concealed by fine and coarse granules which completely fill the protoplasm. These granules, as a rule, have the characters of myelin; sometimes they are a little more highly refractive, like fat, but in the majority of cases they represent a distinct pigment. The cell is sometimes filled with diffuse, golden-yellow pigment; but more frequently the pigment is accumulated in the cell-body in the form of fine and coarse dots, crumbs, flakes, and balls. Not infrequently a few coarse granules only are seen in the protoplasm, which exhibits a myelinoid change."

Unlike Hoffmann, Lenhartz believes that the black pigment which is occasionally found in heart-disease cells is always derived from altered hemoglobin.

He also states that most, but not all, the cells which contain the pigment, when treated with potassium ferrocyanid and hydrochloric acid, give the iron reaction, except in the case of quite recent and old pigment.

After stating his reasons at length, he sums up his opinions in regard to the origin of heart-disease cells in the following words: "The cardiac lesion cells are, for the most part, migratory cells which have either taken up free pigment or have elaborated it within themselves from red blood-cells incorporated in their cell-bodies. Some of these cells are 'possibly' derived from alveolar epithelium."

The clinical significance of finding heart-disease cells is that their presence in large numbers in the sputum is an important sign of the pulmonary alteration known as brown induration. A few scattered cells are sometimes found in pneumonia and phthisis—in short, in all diseases

*"Ueber Genese und Bedeutung der sogenannten 'Herzfehlerzellen,'" "Berliner klinische Wochenschrift," 1889, S. 1025.

† "Mikroskopie und Chemie am Krankenbett," 2. Auflage, Berlin, Jul. Springer, 1895, S. 197, and his older work, "Ueber Herzfehlerzellen," "Deutsche medicinische Wochenschrift," 1881, S. 1039; also Stintzing, under whose guidance Otto Meyer's dissertation, "Die Herzfehlerzellen und ihre pathognomonische Bedeutung," Jena, 1893, appeared.

of the lungs attended with the extravasation of blood. It is only when they are present in large numbers that the phenomenon has any diagnostic significance.

Carl v. Noorden,* who proposed the name "hemosiderin cells" for the heart-disease cells, observed them in bronchial asthma. In one case they were present in large numbers and had some connection with Curschmann's spirals, which were also present in abundance; in fact, they were "intimately concerned in the formation of these structures." v. Noorden says he is "unable to agree with those who consider the presence of these brown cells as characteristic of brown or red induration of the lung in conditions of stasis; not even the presence of the cells in large numbers is any proof of the existence of brown induration."

Cases like the one described by v. Noorden may be exceptional; but it does not seem to me that his explanation deserves the severe criticism pronounced by F. A. Hoffmann.†

Brown induration is synonymous with pulmonary congestion (Stauungslunge) in the narrower sense of the term; hence the heart-disease cells are formed as the result of stasis. Owing to the slowing of the pulmonary circulation diapedesis, as well as rhexis, takes places in the capillaries. The slowing of the current also favors the desquamation of alveolar epithelium which had previously become swollen and loosened, and permits the migration of white blood-corpuscles through the vessel-walls, which are the seat of inflammatory irritation; so that, on the whole, the formation of heart-disease cells is not difficult to understand.

2. Disturbances of respiration or, to use the common term, **dyspnea** (difficult breathing), are very commonly associated with cardiac insufficiency. The weaker the heart's action, the more quickly is slight increase of muscular activity followed by dyspnea. In many cases the symptom cannot be avoided without complete rest in bed.

Aside from the debility of which the patient becomes aware by the occurrence of dyspnea, heart patients suffer from other symptoms. We may distinguish a mild form—*cardiac dyspnea*—and a grave form—*cardiac asthma*, although the two are essentially almost identical. In fact, it appears to me that simple loss of ability to work should be mentioned as an initial symptom in this connection, as by doing so the picture of the condition as a whole is rendered clearer and more real.

At first the patient feels a greater desire for air than the amount of work he is doing would seem to call for. He cannot do the same amount of work in the same time as one with a sound heart, because he gets short-winded. He is forced to stop and breathe from time to time. The subjective sense is that of dyspnea, not of cardiac insufficiency; in fact, it is exactly the same as a healthy man feels after running too fast or up-hill. Although, of course, he is aware that his heart is working more rapidly, he finds this much less troublesome than the dyspnea and the necessity to breathe more frequently or more deeply. As the condition becomes worse, the periods of efficiency are constantly shortened and the periods of rest correspondingly lengthened. Palpitation often makes its appearance at this time, although subjectively the dyspnea remains the more troublesome symptom. But at this stage the occurrence of dyspnea still depends directly on some immediately preceding muscular exertion, and if the patient knows and keeps within his limits, he can guard against the symptom. But the matter is altered as soon

* "Beiträge zur Pathologie des Asthma bronchiale," "Zeitschrift für klinische Medicin," Bd. xx (1892), S. 98.

† See "Diseases of the Bronchi" in the Nothnagel Series, p. 101.

as dyspnea begins to occur without any previous muscular exertion. The patient now suffers from dyspnea—at first only very little—without his being able to assign any cause for the symptom.

Patients who are in the habit of observing themselves closely without having hypochondriac tendencies sometimes state that, when they are absolutely at rest physically, have not specially exerted themselves mentally, and have not been annoyed, they experience a sense of restlessness which is immediately followed by a feeling of oppression on the chest. Immediately they say they draw a few deep or a number of rapid inspirations, or even both, whether voluntarily or involuntarily they are not sure. Usually the restlessness and apprehension are so pronounced that the patients are unconscious of details and especially are unable to say whether they had palpitation or not. They invariably insist, however, that deep breathing is followed by relief. These attacks may become more frequent without increasing in severity, or a few severe attacks of the most violent dyspnea—cardiac asthma—may suddenly intervene. Judging from my experience I believe that such an attack is always preceded by some injury to the heart—not so much a general muscular exertion as exhaustion of the brain from emotional overexcitement or some bodily anomaly. Sluggishness of the stomach, or perhaps even more of the intestine (constipation), very frequently precedes an outbreak.

The attack, as practically all observers agree, usually occurs at night, shortly after the patient has gone to sleep or in the midst of deep sleep. He suddenly starts up gasping for breath; sits up in bed and holds on convulsively with both hands so as to get the full benefit of the muscles of the shoulder-girdle in dilating the upper portion of the thorax; he may even leave his bed and hasten to the window. He usually objects to staying in bed and prefers to sit on a chair because he can hold on to the arms. The skin is soon covered with a light perspiration and becomes slightly cyanotic. The respirations are frequent, prolonged, and accompanied by a fine vesicular wheezing, which can be heard at some distance—"the whole chest seethes," the attendants are apt to say. After a time, not at the very beginning of the attack, a most distressing cough sets in, and a few small masses of tenacious, yellowish mucus are expectorated. The pulse frequency is increased from the beginning; the pulse is usually small and not always irregular. This clinical picture may persist unchanged for several hours and then gradually subside; or the wheezing increases and expectoration becomes more copious and watery, with a plentiful admixture of froth of a somewhat reddish color. Any alteration of the respiratory rhythm up to the fully developed Cheyne-Stokes type may be observed. The heart begins to fail; the contractions become irregular and no longer fill the arteries; cyanosis or, what is worse, pallor of the skin develops; the patient becomes unconscious and dies. There are so many intermediate forms that it is impossible to make any more definite grouping of symptoms. Two points should be emphasized: The milder attacks not infrequently recur; and the attack is always followed by a feeling of weakness.

The special characteristics of cardiac asthma are:

(*a*) The dyspnea,—in the beginning at least,—is not chiefly expiratory, as in the case of bronchial asthma. The marked pulmonary distention which belongs to bronchial asthma is not present, or observed only after the attack has lasted some time. But too much dependence

should not be placed on these signs, and great caution is necessary in diagnosis, particularly when the physician has not had an opportunity of observing the attack from the beginning. I agree with A. Fraenkel* that spasm of the bronchi may be associated with cardiac asthma, and that when catarrh of the final bronchi has been present from the beginning, the possibility of such an association is still greater. As shown by the above case by v. Noorden, the heart-cells, often contained in the tenacious substance that is expectorated at the beginning of the attack, may occur in bronchial asthma without any changes in the heart or lungs. But, on the other hand, these cells may be absent in a case of genuine cardiac asthma.

(*b*) The "seething" noise which accompanies the respiration and impresses the layman so forcibly is due to medium-sized, small, and very small râles, which extend over the entire surface of the thorax. It is stated by some authorities to be most marked at the base posteriorly; this is often, but not always, the case. The râles are produced by a watery and very movable mass which moves up and down in the bronchi as the thorax expands and contracts. Usually the fluid has escaped from the blood and is thereby characterized as edematous fluid. This is unquestionably true of the large quantities of frothy material with very little blood which are expectorated; but it is improbable that the fine, vesicular râles which are not associated with any notable amount of sputum owe their origin to the same cause.

In bronchial asthma fine vesicular râles may be heard at the end of an attack, but not at the beginning. This point may be of diagnostic importance.

(*c*) That the force of the cardiac contractions is diminished during the attack is absolutely certain. The signs by which this diminution manifests itself will depend on the previous and actual conditions of the diseased heart. When the heart muscle is hypertrophied, the signs are not the same as in the presence of dilatation. In both cases the work of the muscle is diminished; but in hypertrophy the pulse retains its tension and apparently, at least, its strength, while the heart-sounds are not exactly weak, and there is nothing to indicate directly that the strength of the heart is failing. On the other hand, in a heart which before the attack was already impaired, dilated, and the seat of diffuse degeneration, the pulse and the heart-sounds at once indicate loss of power. As a rule, the pulse-rate is increased and becomes irregular—if not at once, certainly some time after the beginning of the attack. If the attack is protracted, gallop rhythm rarely fails to develop, and the phenomenon may be present from the beginning.

This symptom, aside from the fact that it is in itself a sign of cardiac weakness (see p. 66), is especially important because, so long as it persists, there is danger of the attack being repeated. Hence it is well to be on the lookout for gallop rhythm, because the tendency to recurrence is very great and should be combated by every means in our power, not only because every attack still further weakens the heart muscle and may damage it permanently, but also because the heart may suddenly give out in the next attack and the case thus end fatally.

Sometimes, when the heart is apparently only moderately affected, a rapid succession of cardio-asthmatic attacks brings on the symptoms of fully developed cardiac insufficiency, edema and the like. It is, of

* "Asthma," in Eulenburg's "Realencyklopädie," Bd. ii, S. 388, 3. Auflage.

course, impossible to prove that these symptoms are caused by the attacks; for it is impossible to overcome the very plausible objection that the attacks themselves represent the first signs of cardiac failure. The facts observed in cases of heart strain, it is true, give some support to the theory; the conditions are similar in the two cases.

The older view that atheroma of the arteries is always the anatomic foundation of paroxysmal cardiac dyspnea has now been abandoned, but it must not be forgotten that the tendency to cardiac asthma is frequently associated with atheroma of the arteries. All other cardiac affections, however, such as muscular disease, permanent changes at the orifices and in the valves, particularly the aortic valve,—although mitral stenosis also plays an important part in the etiology,—must also be taken into account. Special emphasis should be laid in this connection on dyspnea caused by, and immediately following, acute heart strain.

It must not be forgotten that the various forms of nephritis, especially contracted kidney, favor the occurrence of cardiac asthma. Many a symptom belonging to the general picture of uremia, and no doubt often interpreted as a sign of autointoxication, unquestionably belongs to cardiac asthma.

Diabetes mellitus is another cause of cardiac asthma. Frerichs * distinctly emphasized this fact; he was heartily indorsed by C. v. Noorden,† and, with considerable reservation, by Naunyn.‡ Like v. Leyden before him, Naunyn lays more stress on the arteriosclerosis which not infrequently accompanies diabetes than on the disease itself as a direct causative factor [and thinks angina pectoris or a condition resembling it more frequent than cardiac asthma in diabetes.—Ed.].

Finally, I may mention that attacks of dyspnea which must be interpreted as cardiac asthma very frequently occur in the late stages of pulmonary phthisis. The same thing probably occurs in other wasting diseases.

What is the immediate cause of the attack and what are the details of the morbid process?

Many give the answer which Fraenkel§ formulates as follows: "There is no doubt that the attacks of cardiac asthma have their origin in the heart and depend on a loss of functional power in the left ventricle. The resistance in the vascular system continues to increase until finally a disproportion is established between the resistance and the amount of cardiac strength available for overcoming it. A temporary congestion develops in the pulmonary circulation, which, for reasons that are readily understood, leads to a disturbance of the gaseous interchange and thus becomes the immediate cause of paroxysmal dyspnea."

According to this view, the work of the left heart diminishes, while that of the right continues "the same as before." It would be more correct to say, as I may at once remark, that the action of the left heart must be diminished more than that of the right. Then it may happen that the left ventricle fails to empty itself completely and thus interferes with the escape of the blood from the left auricle. As a result the left auricle dilates, and complete evacuation of the pulmonary veins into the auricle is so impaired that the entire mass of blood expelled from the right ventricle can no longer enter the left auricle, and part of it must, therefore, remain in the pulmonary vessels. This first causes dilatation of the pul-

* "Ueber den Diabetes," S. 81 ff.

† "Die Zuckerkrankheit und ihre Behandlung," 2 Aufl., Berlin, 1898, Hirschwald, S. 123 ff.

‡ "Diabetes mellitus," Nothnagel's "System," vol. vii, part vi (1898), p. 229 *et seq.* (German edition).

§ "Ueber die klinischen Erscheinungen der Arteriosklerose und ihre Behandlung," "Zeitschrift für klinische Med.," Bd. iv (1882), S. 19.

monary veins, because the flow into the left auricle, that is, at the periphery, is obstructed. Then follows distention in the capillary distribution of the pulmonary artery caused by the right ventricle, that is, centrally, because the contractions of the right ventricle are strong enough to drive the blood with sufficient force into the pulmonary artery.

It follows necessarily from these premises that, after a certain time, the amplitude of contraction of the right heart diminishes, because the left, being more and more dilated, drives its contents into the aorta with constantly diminishing force; hence the difference in pressure between the aortic valves and the right auricle is diminished, the systemic veins become fuller, and the velocity of their current diminished. This explanation fails to take account of the connection between the pulmonary and the bronchial arteries. I refer to what I have already said in this respect and confine myself here to the prevailing views.

It is generally admitted that acute heart strain may easily lead to the development of true cardiac asthma. It seems to me that we ought to investigate whether the phenomena observed under these circumstances offer any support to the theory which I have just developed. Allbutt, in his description (see p. 40) of what he observed in himself when he overexerted himself climbing a mountain, states that he was quite suddenly seized by a peculiar "*need to breathe*," which was accompanied by a most unpleasant feeling of distention and pulsation in the epigastrium. He could feel a forcible heart-beat diffused over the entire epigastrium, and on percussing at once he found that the right ventricle was very much dilated. This dilatation persisted only a short time, nor did it reappear during the slight attack which he had the following night. Fraentzel's examination (see p. 41), it is true, was not made until some time in the course of the second day, counting from the time when his patient had done such great injury to his heart by a single foolish overexertion. But, as the patient's dyspnea was still at its height and was accompanied by pronounced pulmonary edema, we are justified in basing an opinion on the heart findings, which were as follows:

"Apex-beat not visible, but occasionally palpable in the fifth intercostal space in the nipple-line. Heart dulness begins at the left border of the sternum, at the upper border of the fourth rib, and merges below with the liver dulness; to the left it extends 2.5 cm. beyond the nipple-line, and to the right 2 cm. beyond the right border of the sternum. The heart-sounds are replaced almost constantly by a feeble, hollow systolic sound, which appears to be a murmur."

The above note was made on the fourteenth of October. On the sixteenth we find the following:

"Apex-beat in the fifth intercostal space, a little outside of the nipple line, and distinctly palpable within the line. Heart dulness begins on the left of the sternum at the upper border of the fourth rib, and, considering the typical barrel shape of the thorax, is unusually well marked; it extends to the left 1.5 cm. beyond the left nipple-line, and to the right as far as the right border of the sternum. The lower border cannot be determined with certainty.

"At the apex a blowing, systolic murmur is heard and, in addition, usually a very feeble diastolic sound; at the other valve areas two hollow sounds are heard, in regard to the purity of which it is impossible to give a definite opinion."

By the eighteenth the heart dulness had diminished even more: to the left it extended as far as the nipple-line; to the right, as far as the right border of the sternum. The apex-beat at this time was in the fifth intercostal space and in the mammillary line. Systolic murmur feeble, but pure diastolic sound at the apex; everywhere else two feeble, pure sounds.

Finally, on the thirtieth of October, the murmur had disappeared and the heart-sounds were hollow and pure.

These findings will be interpreted by any unprejudiced observer as indicating dilatation of the entire heart, both of the right and of the left ventricle. Fraentzel himself interprets them in this manner, and it is the more astonishing that he should say:*

"Owing to the disproportion between the amount of work performed by the left, very much weakened ventricle and that of the right, the strength of which was unimpaired, acute pulmonary edema had developed."

No proof is offered for the assertion that the strength of the right ventricle was unimpaired; the findings rather point to the opposite. For aside from the considerable enlargement to the right, the sounds heard over the right heart were so feeble from the beginning that the ventricle cannot possibly be said to have been vigorous. Let us take another case, described by Fraentzel as one of "chronic heart strain."† I shall only quote the essential points from the report of the case, which is very complete.

A laborer, fifty-eight years old, of strong build. Has never been ill, but worked unusually hard during the four days preceding his present illness, as a hod-carrier, carrying bricks at the erection of a house. The first symptoms were pains in the limbs and sacrum, especially when stooping. This was followed by severe dyspnea and violent palpitation, which increased on movement. Two weeks after the beginning of his illness he applied for admittance at the Charité. Signs of marked cardiac weakness: very irregular pulse, cyanosis, effusion into the peritoneal cavity, and slight edema of the feet were found. In addition there was severe dyspnea and frequent cough, without expectoration. Examination revealed no foci in the somewhat emphysematous lungs, but bronchial catarrh. The size of the heart could not be determined; the sounds were pure and weak. At first some of the symptoms, the pain and the edema improved; soon, however, there was a marked change for the worse, cardiac as well as respiratory insufficiency becoming more and more marked. These symptoms steadily increased and the patient died three weeks after admission. Erysipelas developed just before the end.

The autopsy showed no chronic changes in the heart, but both ventricles were greatly dilated. "The coronary arteries exhibit a few slight and quite circumscribed thickenings, but no other changes." Bronchitis, pulmonary edema. It should also be mentioned that the right ventricle contained more blood than the left.

Hence in this case also, in which the signs of cardiac dyspnea had been very prominent in the clinical picture, dilatation of the entire heart was found. I am not inclined to attach very much importance to Allbutt's observation, but it is, nevertheless, noteworthy that he found dilatation of the right heart only.

That hard physical work produces dilatation of the right as well as of the left heart is readily proved by experiment. Th. Schott‡ let strong, healthy men wrestle until dyspnea developed. He found that the heart dulness was enlarged to the right, as well as to the left. I had an opportunity to convince myself of this fact at the time, for Schott was kind enough to allow me to examine his wrestlers.

From all this we must conclude that under simple conditions such as obtain in cases of heart strain, there is no proof that the accompanying dyspnea is due to exhaustion of the left ventricle, and that the right goes on contracting as vigorously as before.

The results of well-known experiments by Welch and Cohnheim to determine the mode of production of pulmonary edema have, it seems to me, been some-

* *Loc. cit.*, p. 120. † *Loc. cit.*, p. 136.

‡ "Zur acuten Ueberanstrengung des Herzens und deren Behandlung," "Verhandlungen des Congresses für innere Medicin," Bd. ix (1890), S. 448.

what too freely applied to pathologic conditions. I do not mean to deny that this form of origin of congestion of the pulmonary capillaries may occur. When the left ventricle, owing to special causes, has been steadily subjected to a severe strain, and when, after it has reached the limit of its functional capacity, it is from some cause or other compelled momentarily to do more work than it is capable of, while the right ventricle has not during the same time had any excessive amount of work to perform, the explanation is no doubt correct. It is not without significance that formerly conditions associated with general atheroma of the arteries and changes in the aortic valves were most prominent in the explanations that were offered, and in these conditions the left ventricle is much more profoundly involved than the right. Not until later was it seen that the same thing may occur in other diseases of the heart, especially myocardial, and then there was a great tendency to generalize. Cohnheim * himself, it is true, distinctly states that the functional power of the right heart is not necessarily completely preserved, nor the strength of the left ventricle diminished to the minimum which just suffices for the maintenance of life; he merely requires a relative preponderance of the strength of the right heart.

In this sense the explanation may be accepted as correct. But it does not satisfy me, and I am inclined to invoke other factors,—processes in the nervous central organs.

The sudden severe dyspnea which characterizes the onset of the attack is produced directly by stimulation of the respiratory center. But how is this stimulation effected?

A. Fraenkel believes that the stimuli are peripheral and that they originate in the lungs:

"In a typical asthmatic attack the strength of the already weakened ventricle suddenly gives way, and thus at once a high grade of congestion develops in the lesser circulation."†

He goes on to say: "The manner in which the congestion which unquestionably exists produces the dyspnea is still the subject of much discussion."

He then reviews the possibilities in detail. I shall return to this presently.

On the other hand, I wish to mention, in support of the view that the attack is due to central irritation, the following argument:

1. In persons suffering from cardiac weakness the irritability of the respiratory center is permanently increased.

This is shown by the fact that a degree of muscular exertion which produces much less carbon dioxid than is required to increase the irritability of the respiratory center in a healthy individual, suffices to bring on dyspnea in a heart patient, the degree of dyspnea being in proportion to the weakness of the heart.

2. Stimulation of the respiratory center occurs whenever the composition of the blood is altered, that is, whenever it becomes richer in carbon dioxid. The alteration may be due to slowing of the blood current at the respiratory center itself. What degree of retardation is necessary it is impossible to say, because it depends altogether on the irritability of the center at the time.

3. Attacks of cardiac dyspnea occur in conditions in which there is no doubt, or at least in which it may be assumed with great probability, that the brain is being insufficiently supplied with blood of normal composition. Acute heart strain is accompanied by diminution of functional power, which must, of course, be followed by a diminution of the amount of blood flowing through the brain. In addition, the severe muscular

* Compare "Vorlesungen über allgemeine Pathologie," Bd. i, 2 ed., S. 505, 506.
† Eulenberg's "Realencyklopädie," *loc. cit.*, 387.

work which was the original cause of the heart failure induced a considerable increase of carbon dioxid in the blood. Besides, the discovery of Geppert and Zuntz* that "substances produced during muscular work enter the blood and directly irritate the respiratory center" also has an important bearing on the question.

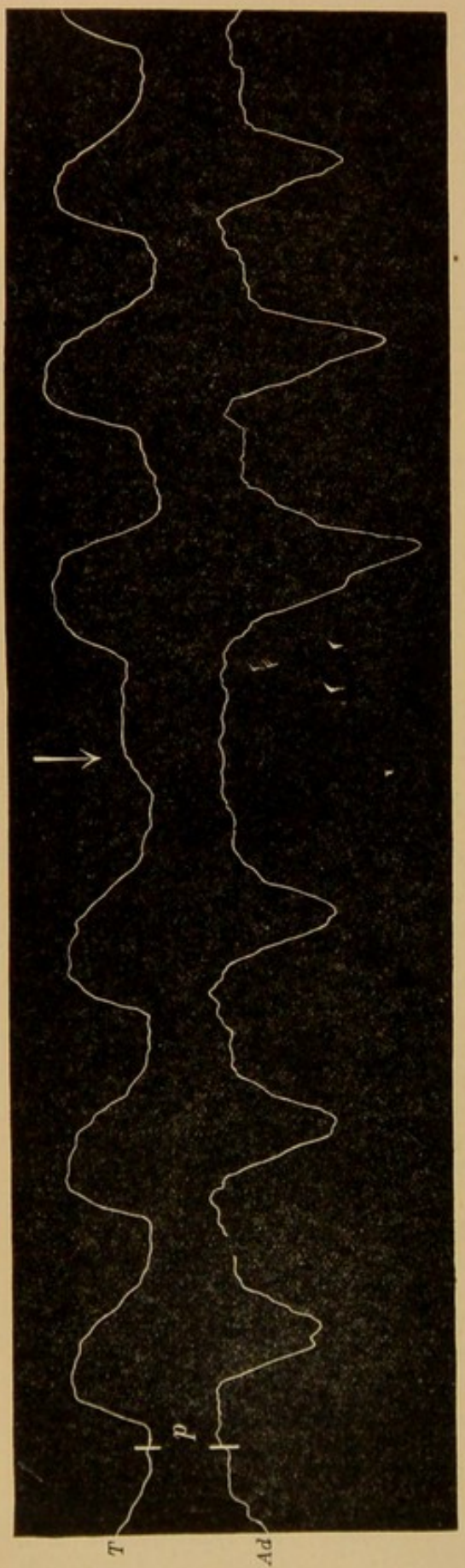


Fig. 21.

Along with the retardation of the flow of blood through the brain which accompanies the failing heart power the increase of carbon dioxid shows its effects not only directly in the respiratory center itself, but also indirectly in all kinds of reflex irritations. In addition we have the effect of the above-mentioned products of muscular work on the respiratory center, protracted by the slowing of the circulation.

What are the conditions when the attack occurs during sleep? During sleep the circulation in the brain, and with it the breathing, is diminished.† Under normal conditions this results in rest or, as we may say, diminished excitement. The investigations of Mosso have shown that even when the circulatory and respiratory organs are working normally, a slight external irritation suffices to increase the strength of the breathing. The respirations are increased still more if the sleeper is suddenly awakened. The accompanying figures, illustrating tracings taken from Mosso, afford unequivocal proof of the truth of this statement. For Fig. 21 (Fig. 27 in his own work) he gives the following explanation:

Respiratory movements during sleep. T, curve of thoracic respiration; Ad, curve of abdominal respiration,

* "Ueber die Regulation der Athmung," "Pflüger's Archiv," Bd. xlii (1888), S. 189.

† Compare Mosso: "Ueber den Kreislauf des Blutes im menschlichen Gehirn," Leipzig, 1881, Veit & Co.

taken simultaneously. The arrow indicates a noise which was not sufficient to waken the sleeper.

The thoracic curve is inverted; that is, its depression corresponds to expansion of the thorax and its elevation to collapse. The abdominal curve, on the other hand, represents a direct image of the excursions of the abdominal wall.

"The subject C. is sleeping profoundly. At the arrow I coughed involuntarily and the respiratory rate suddenly changed, although the patient had not stirred. On examining the respiratory curve we note that the expiratory movement, which had already begun, is interrupted by a short pause, and that the expiratory curve inclines a little more to the right than is the case with the preceding one.

"This is followed by an inspiration which is deeper and more rapid than the previous ones; then there is, as it were, a slight attempt at an expiratory movement, which is again followed by an expiration stronger than the preceding ones. A glance at the curves suffices to convince one that the respiratory movements of the thorax and of the abdominal walls were actually increased."

Regarding the subsequent course of the observation, Mosso reports:

"I continued to record the respiratory movements for about twenty seconds and then called C. by his name (indicated in Fig. 22 by the arrow). There was a sudden pause, which was soon followed by deepening of the respiratory movements. A few seconds after the last respirations of Fig. 22 had been recorded C. opened his eyes. I asked him whether he had heard me cough; he said he knew nothing about it, that he had slept without dreaming."

Mosso* sums up the processes as follows: "After the exertions of the day man seeks out a resting-place and goes to sleep. . . . The respiratory rate changes, and while the

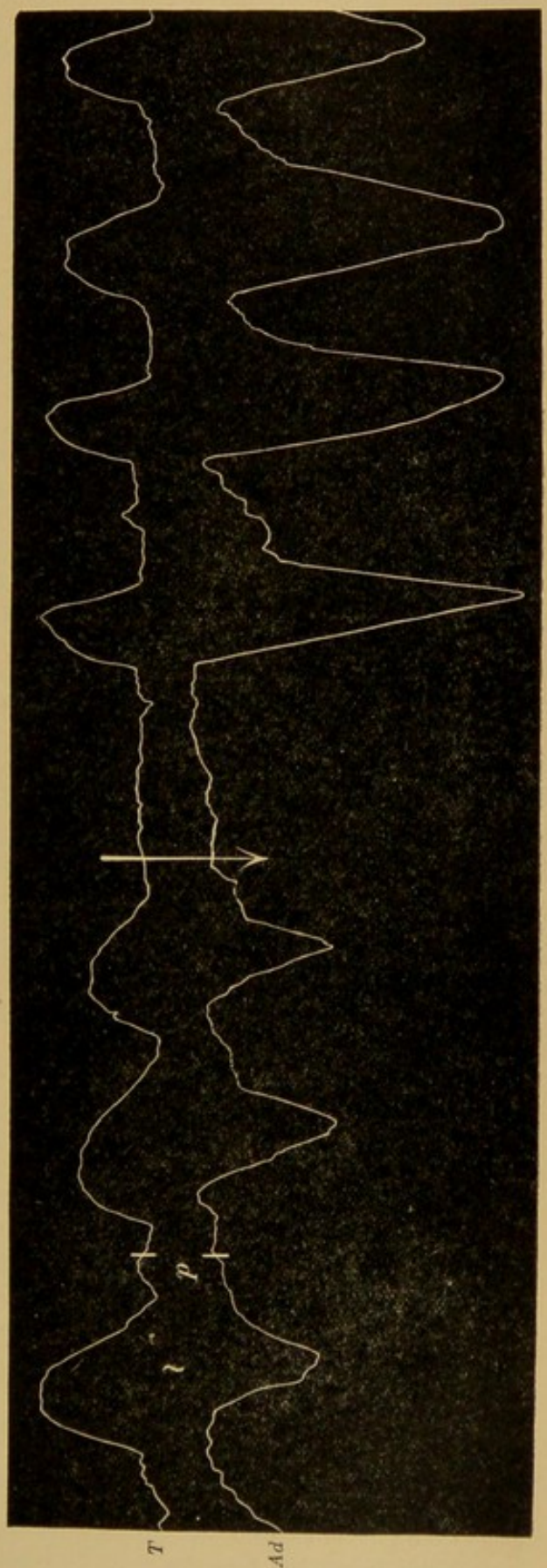


FIG. 22.

* *Loc. cit.*, p. 103.

breathing in the waking state is chiefly diaphragmatic, it becomes during sleep almost exclusively thoracic in type. The relaxation of the diaphragm may be so complete that the muscle appears to be absolutely inactive. The combustion processes in the organism are reduced to such a point that instead of seven liters of air per minute, which enter the body in the waking state, only one liter is taken up. The strength and frequency of the heart beat is also diminished; the vessels dilate; the blood pressure diminishes; and the temperature falls considerably."

When a heart patient from any cause is suddenly awakened from sleep, his breathing at once becomes stronger. But as his respiratory center is more irritable, the impulse extends more widely in every direction, and more muscles—perhaps all the muscles that are available for respiration—are called into action. The feeling of anxiety which comes over the patient causes him to assume the erect posture. He may even jump out of bed and hurry to the window. All these phenomena are associated with a sudden increase in the quantity of oxygen consumed, which is proportionate to the degree of muscular activity, and with an increase in the formation of carbon dioxid. The act of waking is also associated with an increased demand on the heart. The muscular exertion indulged in by the patient under these circumstances is in itself enough to throw a considerable strain on the heart. In addition to this there may be an important change in the regulating centers of the vessels (vasomotor centers) due to the lessening of the cerebral circulation during sleep; for these centers always require some time to adapt themselves to the altered condition brought about by the act of waking. I need only recall what has so frequently been described and observed in their own persons by those who are in the habit of observing themselves. I refer to the unpleasant sensations of oppression and palpitation brought on even when the heart is perfectly healthy by suddenly waking from sleep and starting up and jumping to one's feet. That this may lead to syncopal attacks—that is to say, general cerebral anemia—is sufficiently well known. It seems to me that these facts suffice to explain why cardiac dyspnea going on to cardiac asthma is apt to occur in heart patients whenever they are disturbed during sleep.

Finally, we may inquire how the stimulation of the respiratory center, which at first is perhaps very slight, originates. For external stimuli the answer to this question has already been given in the above-mentioned observations of Mosso. In addition to these, however, the inner processes in the brain which are excited by some insignificant change in the stimulation of peripheral nerve-endings and are reflected in the patient's dreams* must also be reckoned with. I refer to the condition known as nightmare, in which the sense of dyspnea is so pronounced.

Another mode of origin of cardiac dyspnea, the variety which is brought on by overloading the gastro-intestinal tract, is no doubt susceptible of a similar interpretation. The diaphragm, being relaxed during sleep, is forced upward by the increased intra-abdominal pressure. The lungs, as well as the heart, and perhaps also the blood-vessels within the abdominal cavity, may thereby suffer certain disturbances which tend to increase the stimuli sent out from the respiratory center. In this connection I refer to Allbutt's observation on himself, which has been quoted elsewhere. (See p. 40.)

In order to avoid any possible misunderstanding, I wish to emphasize

* Compare in this connection an interesting work by Alfred Lehmann, "Aberglaube und Zauberei," translation by Petersen, Stuttgart, 1898, Enke, p. 397.

distinctly that I am here discussing only the question of how the attack of cardiac dyspnea is brought on. In defending the theory of a central origin I, of course, leave out of consideration the changes which later develop in the distribution of the pulmonary capillaries. Nor do I wish to interfere with the interpretation of the inhibition brought about by mechanic disturbances in the latter.

Let us turn our attention once more to these mechanic disturbances. It is assumed that the strength of the right heart sooner or later begins to fail. Of the individual links in the chain of events the following should be emphasized:

1. The capillaries in the alveoli, as well as in the bronchi, contain more blood than under normal conditions.

As the force with which the blood is propelled from the left side of the heart diminishes, this engorgement must begin in the capillaries of the pulmonary system, because their evacuation is impeded. But failure of the left heart is always followed by stasis in the systemic veins: the venæ cavæ may encounter so little resistance in emptying their contents into the right auricle that the pressure in the venæ cavæ themselves does not rise for some time. But as soon as the strength of the right heart also begins to diminish, as soon as the ventricle fails to empty its contents completely into the pulmonary artery, the pressure in the venæ cavæ—the superior in this case—must increase and interfere with the evacuation of the bronchial veins. The congestion thereupon spreads further in the bronchial mucous membrane. This causes an increase of the stasis in the alveolar capillaries also. The free communications existing between these two systems permit the blood in the capillaries belonging to the pulmonary artery to flow into those of the bronchial artery. But as soon as the pressure rises in the veins of the greater circulation, the passage of blood from one system of capillaries to the other must, of course, be interfered with.

2. The congestion of the capillaries in the alveoli and in the bronchi encroaches on the space for the passage of the air.

In the larger air-passages there is ample room and the congestion is of no consequence. But in the narrower bronchial branches, in the bronchioles, and still more in the alveoli, this may cause a perceptible obstruction, which increases as the capillaries become more and more engorged with blood.

If the circulation in these capillaries is delayed for a time, their walls become permeable and extravasation of plasma and a few red blood-cells soon takes place. If this extravasation takes place from the capillaries situated in the bronchial mucous membrane, it causes swelling and still further increases the obstruction to the entrance of the air. In this way a physical, as well as clinical, picture resembling that which is seen in capillary bronchitis is produced.

When cardiac insufficiency has been present for some time, the nutrition of the blood-vessels of the lungs suffers—at least, so far as the capillary portion is concerned. The capillaries become permeable earlier than is the case under normal conditions. This conclusion is justified by the generally accepted tenets of pathology and explains why these phenomena appear earlier in heart patients than in those whose hearts are sound.

3. The type of the breathing adapts itself to the conditions created by the obstruction in the bronchi and by the stimuli sent out from the center. Hence, depending on circumstances, there may be simple accel-

eration of the breathing with accentuation of inspiration or expiration or of both, retraction of the lower lateral portions of the thorax, heaving in the flanks, and more or less pronounced disturbances of the rhythm, going on to fully developed Cheyne-Stokes breathing. The peculiar changes that may take place in the respiratory curve are shown in Fig. 4 (p. 71). The distention of the lungs corresponds to the dilatation of the thorax, provided the entrance of air is not obstructed. Aside from swelling of the bronchial mucous membrane, the obstruction may be due to a serous effusion into the alveoli or edema of the lungs, which unquestionably is a congestive phenomenon due chiefly to the disturbance of the capillary circulation.

4. The attack of cardiac dyspnea may also be associated with a true catarrh,—that is, inflammation of the bronchial mucous membrane,—a condition which may develop at various times.

Catarrh may have been present before the attack. In that case the severity of the attack may depend upon the extent of the catarrh, inasmuch as the disturbance of gaseous interchange due to the interference with the access of air to the respiratory surface is not without influence on the stimulation of the respiratory center which excites the attack.

It is also possible that during and for some time after the attack the organisms which produce inflammation and which enter with the air may find a favorable soil for the development of their activities in the sense explained in another place (p. 107). In this way we also explain what is by no means a rare occurrence, namely, that an already existing bronchial catarrh increases in severity and extent.

The reciprocal relations existing between respiration and circulation assert themselves as the catarrh develops and continues to spread. The severe dyspnea, which may persist for some time without exhibiting any distinct symptoms of paroxysmal exacerbations, and which makes its appearance in cases of cardiac weakness that go on to a fatal termination, must be attributed to this cause. For therapeutic reasons it is necessary not to lose sight of the causal connection. For even in cases in which catarrh apparently dominates the clinical picture, it is sometimes possible to relieve the patient by subjecting the heart to proper treatment. The converse of this may also occur, in which case the catarrh must be treated.

I shall now give a short account of an unusually violent attack of cardiac asthma which shows that the cardiac weakness is sometimes overshadowed by the dyspnea when that symptom reaches its full development. It also shows the possible effects of catarrh late in the course of the disease.

Personal Observation 8.—Policeman, twenty-seven years old.

Ten of his fifteen brothers and sisters are dead. There is tuberculosis in the family. The autopsy findings on one of his brothers are available. The patient served with the Guards in Coblenz and Spandau, but was invalided after a short time on account of laryngitis. This was in 1894. Since then he says his health has been good, on the whole, except that he occasionally becomes very hoarse. In the year 1895 he had an attack of severe faintness, the after-effects of which did not disappear until the next day.

For the past four weeks he has been under dispensary treatment at different times for bronchial catarrh. During the past few days he has been perfectly well.

Admitted on the seventh of December, 1897. In the morning he suddenly developed severe dyspnea with some cough. The man, nevertheless, remained on duty, but in the course of the afternoon the cough became worse and the dyspnea so severe that medical advice was sought toward evening. Examination showed a man above the average size and very powerful. Severe dyspnea; semierect posture; the face pale. When interrogated he answers with difficulty; speech is so laborious that he has to stop and rest after every second or third word. Rectal temperature

36° C. (97.1° F.); respirations 60 in the minute, very superficial and labored; all the auxiliary muscles are called into action during respiration, and particularly during expiration.

Pulse, 74; the arteries dilate rapidly and there almost seems to be a "*pulsus celer*," but the arteries are soft and not overfilled.

The breathing is accompanied by loud râles, and a copious watery, frothy secretion is expectorated. Violent paroxysms of coughing; moderate degree of pulmonary emphysema (boundary between the lung and the liver at the eighth rib in the nipple-line, and below the spinous process of the twelfth vertebra in the line of the spine). No foci were found in the lungs, but coarse and fine vesicular and in part sibilant and sonorous râles are heard all over the chest.

Heart: Upper border of third rib; to the right the limit cannot be made out, nor is the apex-beat distinctly palpable. The sounds are very indistinct. The râles and the cough greatly interfere with examination.

The patient had a very restless night, although he had received a hypodermic injection—5.0 gm. (1¼ drams) of camphorated oil and 0.02 (⅓ gr.) of morphin hydrochlorate.

Morning of the eighth of December: Cutaneous emphysema is present on both sides, from the middle of the neck about as far as the second intercostal space. The respirations again are 60 and the pulse 78. The position of the diaphragm is lower: to the right, in the nipple-line, upper border of the ninth rib; posteriorly, the level of the first lumbar vertebra. In other respects the findings are the same, except that the sputum, which is very copious, frothy, and quite thin, contains traces of blood. The apex-beat is not distinct.

There is certainly no marked enlargement of the cardiac dulness; the heart-sounds are pure.

The urine is free from albumin; specific gravity, 1022.

The subsequent course was as follows: On the thirteenth of December—the seventh day from the beginning of the attack—the emphysema had disappeared. But it reappeared on the following day in a milder form, and the normal limits of the lung were not permanently retained until the eighteenth of December, the twelfth day of the disease.

The râles slowly subsided; on the sixteenth day of the disease the auscultatory signs were such that they could not be regarded as abnormal.

The cutaneous emphysema did not continue to spread; the last traces of it could be demonstrated on the fourteenth.

After the eighth day of the disease the sputum began to increase in consistency and contained lumps of mucus. Constant and progressive diminution in the quantity of sputum until the thirteenth day of the disease, after which the expectoration was very slight. The cough ran a similar course. Blood was seen for the last time on the third and never more than a trace. Curschmann's spirals could not be found. The subjective dyspnea had disappeared on the sixteenth day of the disease, and about this time the cough had also practically ceased. The respiratory rate gradually diminished, with occasional variations.

The outline of the heart could not be determined with any accuracy until the eleventh day of the disease, when the limits were found to be as follows:

Above, the upper border of the third rib in the left sternal line. To the right, the middle of the sternum. Apex-beat, the nipple-line, fifth intercostal space. No murmurs were heard at any time; nothing but feeble heart-sounds.

The pulse-rate at the height of the disease was low. Later it varied without any obvious cause, and once, on the ninth day of the disease, reached its maximum of 96. On this day there was a slight elevation of temperature. But the deviation from the normal which shows itself in the quotient $\frac{P}{R}$—which ought to be 4.5—persisted for a long time. As late as the thirteenth day of the disease—the last of which we have any accurate figures—the ratio was determined at two different times, $\frac{72}{30}=2.4$ and $\frac{69}{25}=2.7$. The greatest deviation occurred on the first two days of the disease, when the quotient was respectively $\frac{74}{60}=1.2$ and $\frac{78}{60}=1.3$. The best quotient was found on the day when the pulse-rate was absolutely the highest, $\frac{96}{34}=2.9$. The quantity of urine could not be determined at the height of the attack—not until the fourth day of the disease. Result: considerable diminution; maximum, 1000 c.c.; positively determined minimum, 480 c.c. Average of nine determinations, 777 c.c. Specific gravity, above 1020; maximum, 1025.

The temperature at the height of the attack did not attain the level of what may be called febrile. The figures obtained are, of course, not quite normal, being a little higher than in health. True elevation—38.4° C. (100.8° F.) at noon—oc-

curred only on the ninth day of the disease, the day on which the sputum showed distinct characteristics of bronchial catarrh. This may be regarded as a febrile temperature, but on the following day and during the rest of the disease the temperature continued normal. Convalescence was quite protracted. It was not until the twelfth of January, 1898, that is, thirty-eight days from the beginning of his illness, that the man, who had been discharged "cured," was able to resume his active duties.

I examined him carefully on the twenty-seventh of October, 1898, and found that there had been no disturbances since his discharge. The lungs, which were quite normal in extent, showed no abnormal signs whatever. There was no catarrh of the laryngeal mucous membrane, but there was slight paresis of the right vocal cord. Heart: Apex-beat in the fifth intercostal space in the mammillary line. Above, upper border of the fourth rib. To the right, middle of the sternum. The heart-sounds were somewhat faint; the aortic second sound perhaps a little accentuated. The sphygmogram does not show any marked peculiarities, and, in fact, scarcely differs from the tracing taken during the subsidence of the attack.

The cause of the heart failure which ushered in this attack is not clear. During the attack there was dilatation, which beyond a doubt only affected the left heart and was very slow to subside. The dyspnea completely dominated the clinical picture. The cough, which was due to the large serous effusion into the lungs exhibiting all the characteristics of a congestive edema, was so violent that it caused laceration of the alveoli and resulted in cutaneous emphysema. This is by no means a common occurrence in a vigorous adult.

It is noteworthy that the bronchial catarrh, which occurred late, on the eighth day of the disease,—the first time mucus in any appreciable quantity was contained in the sputum,—does not coincide in time with a general change for the worse in the man's condition. The slight elevation of temperature which occurred on the next day—in the morning, 38° C. (100.4° F.), at noon, 38.4° C. (100.8° F.), and in the evening, 37.9° C. (100.2° F.)—was accompanied by a marked increase in the pulse frequency; while the respiratory rate, on the other hand, was greatly diminished. On the next day everything had returned to normal, although the expectoration of mucus continued.

Without being able to adduce any positive proof, I am of the opinion that this patient is infected with tuberculosis. I have several times seen protracted attacks of cardiac dyspnea like the present one in tuberculous patients, and once slight pulmonary edema at a time when other symptoms of the infection, which later developed rapidly, had not yet made their appearance.

The treatment, aside from the injection of camphorated oil on the day of admission, which I personally would not have given, consisted solely in the administration of morphin. Up to the tenth day of the disease 0.02 gm. ($\frac{1}{3}$ gr.) of the hydrochlorate were given hypodermically every evening, and during the day every three hours a tablespoonful of a solution of 0.06 gm. to 200 c.c. ($\frac{9}{10}$ gr. to about 6 ounces—or about $\frac{1}{13}$ gr. of morphin hydrochlorate to the dose). Heart stimulants were not needed. One liter (quart) of wine containing 5 per cent. of alcohol, distributed over the twenty-four hours, cannot be called a heart stimulant for one who is used to alcohol.

There remains to inquire how cardiac dyspnea is made to disappear.

To begin with the simplest imaginable case: If the cause is heart strain due to excessive muscular exertion, mere bodily rest will cause the symptoms to disappear in a comparatively short time, provided the heart has not suffered any permanent injury from overdistention. Bodily rest diminishes the formation of carbon dioxid, which is produced by

the action of the muscles, as well as of the irritating substances (Geppert-Zuntz), both of which stimulate the respiratory center. The elimination of these causes allows the breathing to quiet down; less work is required from the respiratory muscles; and the irritation of the center, therefore, diminishes more and more. In other words, the effects of excessive muscular exertion are removed by voluntary inhibition, and the way in which that is accomplished is obvious.

Will this explanation apply to those cases in which the attack is brought on not by a general increase of muscular exertion, but by a sudden excess of activity on the part of the respiratory muscles? As has just been explained, the increased irritability of the respiratory center is the reason why a slight irritant causes a degree of activity on the part of the respiratory muscles greatly in excess of what is necessary—that is, for effecting the gaseous interchanges in the lungs. The heart is thus directly forced to exert itself more than it is able to do in its weakened condition. This heart weakness produces disturbances in the pulmonary circulation which, by interfering with the access of air to the respiratory surface, increase the demands on the respiratory muscles and thereby also on the heart.

In this way a vicious circle is established, and unless it is broken up, death is the inevitable result. That, however, is not the invariable outcome. I should like to suggest that possibly the improvement in the favorable cases is brought about by a gradual exhaustion of the respiratory muscles, so that they do less work. If the work of the respiratory muscles is diminished, the number of central stimuli which they give off is likewise reduced. A more important result, however, is that the heart thereby gains a certain amount of rest, which helps it to recover; for there can be no doubt that the strain on the heart is relieved whenever the work of the muscles generally is diminished.

There is, of course, no doubt whatever that the subsidence of the attack is in part brought about by processes in the central organs. It should be borne in mind that the irritability of the respiratory center itself is diminished by fatigue, and that the regulatory centers for the heart and blood-vessels also contribute to the final result.

There is no doubt that an attack of cardiac asthma may be favorably influenced by a reduction in the irritability of the respiratory center. This is shown by clinical experience, which teaches us that morphin may have a most salutary effect; indeed, under certain circumstances it may cause the attack to subside in a short time. If that end is to be accomplished, the dose should not be too small; we know that morphin in doses of 0.02 to 0.03 gm. ($\frac{1}{3}$ to $\frac{1}{2}$ gr.), administered hypodermically, can be positively relied upon to diminish the irritability of the respiratory center, and in cardiac asthma the dyspnea also disappears after the administration of these doses.

I wish to point out most emphatically that whenever the weakness of the heart has produced congestive phenomena in the pulmonary vessels, it is hardly conceivable that the stasis will disappear so long as the heart continues to work under the conditions which made the stasis possible. For, if the patient continues to use his muscles to the same extent as during the time when the congestion developed, the demand on the heart will continue to be the same. The left ventricle, relaxation of which must be assumed in order to explain the development of stasis, cannot improve its nutrition if the general condition continues to develop,

because the blood which it contains is under reduced pressure and a smaller quantity of it is driven into the aorta, so that the ventricle is more likely to become fatigued. If, after fatigue has reached a certain degree, the heart failure continues to increase, the effusion of fluid into the lungs will increase at a corresponding rate and the obstruction of the respiratory surface will become more and more general.

It is, therefore, obvious that by strengthening the heart cessation of the attack is brought about, and this is true at all times. It is the most direct means of accomplishing that end; but, unfortunately, this is not always practicable, and then it becomes the physician's duty to make it practicable. In some cases this can be accomplished indirectly by diminishing the irritability of the respiratory center with morphin; in other cases directly by strengthening the heart. If it is only borne in mind that by temporarily forcing the heart to greater activity its nutrition can be improved by virtue of this increased activity, and that the efficiency of the heart is thereby prolonged, the main features of the process that must take place in a given case will be clearly understood. This point will be reverted to again in connection with treatment.

In this explanation of cardiac dyspnea I have attempted, while fully acknowledging its causal connection with the heart itself, to do justice also to the part played in the morbid process as a whole by the respiratory apparatus.

Traube * had thrown out certain hints which indicate that he held a similar view. He laid special stress on the alteration in breathing which occurs during sleep; but in his brief comment on this phenomenon he did not express a definite opinion in regard to the connection between the accumulation of carbon dioxid which is thus brought about and the onset of the attack.

A. Fraenkel † makes much of the fact that the swelling of the bronchial mucous membrane, which is a necessary result of the venous hyperemia, must offer considerable resistance to the movement of the air to and from the respiratory surface. He insists on this, as well as on the possibility that an already existing catarrh may be rendered worse, or a fresh catarrh of the bronchi may be set up.

In this latter respect the views I present in my own explanation are practically the same.

There remains to define my attitude with regard to v. Basch's doctrine of *pulmonary rigidity* and *pulmonary swelling*, which he himself originated and sought to demonstrate by numerous investigations carried out by himself and his pupils.

I shall give the shortest possible summary of this doctrine in v. Basch's‡ own words: "The change of air in the lungs, which chiefly determines the efficiency of the breathing and, therefore, the ventilation of the blood, is proportionate to the elasticity (distensibility) of the lungs, provided the forces which cause expansion and collapse of the lungs remain the same. This distensibility (elasticity) of the lungs, so long as the elasticity of the pulmonary alveoli is not altered, depends on the pressure and amount of blood in the capillaries which surround the alveoli. When these capillaries are distended and under high pressure, the lung becomes less distensible, that is to say, its expanding power is impaired; and, conversely, the lung becomes more distensible as the pressure and distention of the capillaries diminish. The condition which is produced in the lungs by a state of extreme tension of the alveolar capillaries I have designated *pulmonary rigidity*.

* "Gesammelte Beiträge," Bd. iii, S. 209.

† "Realencyklopädie," *loc. cit.*, p. 388.

‡ "Allgemeine Physiologie und Pathologie des Kreislaufes," Wien, 1892, Alfred Hölder, S. 60.

"Pulmonary rigidity acts as a mechanic obstacle to respiration, on the one hand, interfering with the respiratory expansion of the lungs, and, on the other, by preventing expiratory collapse.

"But when the alveolar capillaries are distended and the pressure in them is increased, each individual alveolus, and, therefore, the entire lung, becomes enlarged. This enlargement of the lung, which I designate *pulmonary swelling*, also impairs the respiratory change of air (ventilation) and, therefore, the efficiency of the breathing. So long as the alveolar capillaries are under moderate tension, the lung readily expands and collapses."

This explanation at first sight appears to be very plausible and is well supported by numerous experiments. But in spite of its apparent simplicity the theory has never found general acceptance.

Space will not permit more than a few general remarks on the main features of the theory.

It should be stated, in the first place, that the proposition on which v. Basch based his theory, namely, that the lungs expand as the blood enters their tissues, has been established by the author beyond a doubt. He describes his experiment as follows:*

"The lung and the heart are removed together from the thorax of a dead animal, and the pulmonary artery, as well as the left auricle into which the pulmonary veins empty, are fitted with cannulas, and arrangements made for introducing fluid under pressure from the pulmonary arteries into the lung; the trachea is then connected with a bellows, which can be made to work at a uniform rate. It will be seen that, when the bellows is started, the lung will expand to a certain definite point at each descent of the bellows. Fluid is now introduced into the pulmonary vessels. The first effect observed will be an enlargement of the lungs, and, if inflation is resumed, it will be found that the inflation of the lungs will be much less than before.

"By connecting the trachea with a water manometer before the second inflation and before the injection of fluid it can be proved that the enlargement of the lungs after the injection is not due to any escape of fluid into the alveoli; for the fluid in the manometer falls—in other words, the lung aspirates while it is enlarging, and this aspiration can only be due to an enlargement of the space inclosed by the alveolar wall."

That the facts as described really take place when the experiment is so ordered there can be no doubt. But the questions suggest themselves, how about the degree of force used? How great was the pressure used in injecting the fluid into the pulmonary artery? What was the pressure of the air which was forced into the lungs from the bellows? How great were the variations in the expansion of the lungs? I find no data on these points. In a much later report† v. Basch himself refers to the fact that no actual measurements were taken. He says:

"This experiment was followed by a large series of experiments on living animals carried out by Dr. Grossmann, which clearly show that the pulmonary swelling and the attending pulmonary rigidity represent a preliminary stage of pulmonary edema."

It seems to me that this failure to give the actual values used in the

* "Die cardiale Dyspnoe und das cardiale Asthma," "Schnitzler's klinische Zeit- und Streitfragen," Bd. i, Heft 3 und 4, Wien, Breitenstein, 1887, S. 84 ff.

† "Die Lehre von der cardialen Dyspnoe und ihre historische Entwicklung," "Wiener medicinische Wochenschrift," 1897, No. 27, 28, p. 9 of the reprint.

experiments on which the entire theory is based has given rise to the skepticism in regard to it, particularly as v. Basch's subsequent writings on the same subject suggest that the filling of the pulmonary artery with a solution of sodium chlorid—as appears from the communication first referred to—was effected under considerable pressure. He speaks of "an erection" of the capillaries and, in general, bases his arguments on artificial injections. Then he explains the difference between Donders' measurements of the traction exerted by the bloodless lung in a closed thorax in a cadaver and the measurements obtained by Jakobson and Adamkiewicz in living animals in such a way as to support his own theory: Donders' measurements, he says, were higher because of the absence of blood.

Next we have the experiments on the living animal, beginning with Grossmann's* investigation on pulmonary edema due to muscarin-poisoning. It may be asked whether the conditions in which the animals were placed were really such that the results of the experiments can be applied to the pathologic processes which develop in human subjects who have suffered for some time from cardiac disturbances. It seems to me that this question has never been decided. A. Fraenkel† has expressed his doubts, and personally I must also confess I do not consider that v. Basch's doctrine has ever been positively proved. Especially, v. Basch's contention that the entire pathologic process hinges on pulmonary swelling and pulmonary rigidity has never been clearly established. How far he goes in his argument is shown in the following statement:

"Traube's *volumen pulmonum auctum* is identical with my *pulmonary swelling*. The only difference is that Traube makes a pulmonary disease out of his *volumen pulmonum auctum*, whereas it is really a cardiac disease; for the swollen and enlarged lung merely represents the swollen vascular area between the right and the left heart."‡

So long as the original experiment fails to give any information in regard to the degree of force used I for one cannot bring myself to accept the correctness of v. Basch's teachings in their entire extent, although I am quite willing to admit that they are well worthy of careful attention.

Even granting that pulmonary rigidity and pulmonary swelling play as important a part as v. Basch contends they do, the views here presented in regard to the origin of cardiac asthma are, so far as I can see, in entire harmony with his theory.

In conditions of protracted stasis changes develop in the lungs themselves which are known under the name of *brown induration*.

Ziegler's§ description and interpretation of the processes are as follows:

"Frequent hemorrhages into the lungs, such as are quite apt to occur in heart patients with congestion in the lesser circulation, may be followed by a marked proliferation of connective tissue in the hemorrhagic areas accompanying the absorption of the blood. The connective tissue gradually leads to thickening of the interalveolar septa, and here and

* "Zeitschrift für klinische Medicin," Bd. xii (1887), S. 550.

† "Diagnostik und allgemeine Symptomatologie der Lungenkrankheiten," Wien und Leipzig, Urban & Schwarzenberg, 1890, S. 185.

‡ "Historisches und Kritisches zur Lehre von der Compensation der Herzfehler," "Wiener klinische Wochenschrift," 1893, p. 23 of the reprint.

§ "Lehrbuch der speciellen pathologischen Anatomie," 9. Auflage, Jena, 1898, Fischer, S. 665.

there even to induration of the pulmonary tissue, with obliteration of the alveoli—resulting in a condition which, on account of the abundance of pigment in the thickened tissue, is aptly termed *brown induration of the lungs.*"

Pulmonary distention and true emphysema rarely fail to develop in cardiac patients who have been ill for some time. The development of these conditions is readily explained by the frequent attacks of bronchial catarrh and the accompanying cough. With the type of cardiac weakness that develops as the result of certain definite kinds of bodily labor—the carrying of a load up-hill or otherwise against the action of gravity—there are also to be considered the conditions under which the work is performed (see p. 46). The direct consequence of these conditions is pulmonary distention, and they accordingly favor the development of true emphysema.

Hypostatic Congestion.—Hypostatic congestion, which develops when the forces that drive the blood through the lungs begin to fail, because gravity makes itself felt in a definite direction, is, of course, favored by the existence of cardiac weakness. [Chevalier-Joly believes that pulmonary congestion and edema are frequently unilateral in heart disease. They may occupy either the apex or base of the right lung, and are due to the dilated right auricle, which presses on the upper or lower branches of the pulmonary veins, according to the anatomic conditions in the body. From experiments he made he believes the congestion may occur from bending of the pulmonary veins by the distended auricle. The same author noticed the pressure on the vena azygos major in such cases, and in one of his patients there was a right-sided hydrothorax, but he did not discuss that part of the subject.—Ed.]*

That it is frequently observed is a well-known fact when the heart begins to fail in acute infectious diseases.

The area of hypostatic congestion presents various forms of lobular pneumonia in isolated or confluent foci. These pneumonias are probably, for the most part, due to invasion of the pulmonary tissue by the pathogenic micro-organisms present in the bronchi, which find a favorable medium in the congested tissues; for, owing to the insufficient supply of effective blood, the resisting power of the tissue-cells in the hypostatic portions of the lung cannot be very great.

The question whether hematogenous pneumonias, particularly the form produced by the diplococcus of A. Fraenkel, develop more readily in the presence of cardiac weakness is so complicated that I shall not venture to enter upon a full discussion of it here. Suffice it to say that pneumonia constitutes a serious menace to heart patients. This is chiefly due to the mechanic conditions brought about by the disease and to the increased demands made upon the heart by the infection itself.†

In regard to the hemorrhages into the lungs which occur in cardiac patients in the form of hemorrhagic infarcts, it may be remarked that they are not always associated with occlusion of the corresponding portion of the pulmonary artery. They may occur in the typical wedge-shape even when the arterial branch which supplies the corresponding area is perfectly unobstructed.

* "Contribution de l'Oèdem et de la Congestion pulm. dans les affect. cardiaques," "Lyons Thesis," 1901.

† In regard to this question the reader is referred to what I have said in v. Ziemssen's "Handbuch der speciellen Pathologie und Therapie," Bd. v. A brief summary is also contained in my "Lehrbuch."

In patients who have suffered from cardiac weakness for some time the pleura is rarely free from tissue changes. Disseminated opacities and thickenings in these cases are of no significance; but adhesions between the two layers of the pleura sometimes occur and, depending on their extent and location, interfere more or less with the expansion of the lungs and thus become injurious. The development of these adhesions, which originate in true inflammation, probably has some connection with the bronchial catarrh. If the latter is protracted and severe, the micro-organisms of inflammation may find their way from the bronchi to the pleura. Inflammatory exudates produced in this way are certainly not common. They occur after the development of inflammatory foci within the lungs, near or in contact with the pleura. The formation of infarcts in the lung may be coincident with a genuine inflammatory exudate into the pleura at the seat of the disease, or the exudate may be secondary to the infarct. On the other hand, simple transudation not infrequently occurs during the time the infarct is developing. If the transudate is bilateral, however, it has nothing whatever to do with the infarct.

Transudation—*hydrops pleuræ*—is quite common in the severer grades of cardiac weakness. As a rule, but subject to exception, the effusion is bilateral; exceptions occur when the layers of the pleura are adherent on one side and freely movable on the other, and when the attitude is such as to cause gravity to act more in one direction than in another—these are the conditions which are responsible for deviation from the usual bilateral distribution of the exudate.

[Stengel and Steele, in several articles,* advance views at variance with those of the author, and equally opposed to the views of the French school as set forth in the Thesis of Fabre.† Stengel and Steele believe it is not possible to separate cases of hydrothorax and pleurisy as sharply as some writers have, and believe there is usually a combination of both in the cases of pleural effusion in heart disease. The specific gravity of the fluid,—1020 in many cases,—the amount of albumin,—1½ per cent. or more,—and the thickening of the pleura seen sometimes postmortem, all point to inflammatory processes. In future observations more attention should be paid to the minute differences of inflammatory and non-inflammatory effusions in the pleura and the relative position of the fluid in cases of hydrothorax. Taking cases recognized clinically as those of hydrothorax, Steele finds that in heart disease the effusion is often greater on one side,—the right more frequently,—and in a considerable proportion ($\frac{1}{5}$) of cases unilateral. He also finds that right-sided effusions occur with enlargement of the right heart, left-sided effusions usually with enlargement of the left heart. In the former cases it is probable the enlarged right auricle presses on the root of the right lung, and obstructs the vena azygos major, as suggested by Baccelli, O. Rosenbach, and others. Stengel has shown how obstruction of this vein could cause an effusion greater on the right side. In the case of unilateral left-sided effusions the relations are not easy to explain. Rosenbach thinks the enlarged left ventricle presses on the lower lobe of the left lung and pro-

* See Steele, J. D., "Pleural Effusions in Heart Disease," "Journal of the American Medical Association," October 1, 1904, for analysis of cases and important references to the literature.

† "Contribution a l'étude de la Pleurésie chez les Cardiaques et en particulier de la Pleurésie droite," Paris, 1894.

duces atelectasis, followed by chronic congestion in the pleura, with an effusion partly inflammatory, partly transudate. The pleural effusions in heart disease may precede external edema by a considerable length of time. They are characterized further by chronicity (one and one-half to two years, Osler) and by sudden onset and quick recurrence after removal.

If the effusion is large, expansion of the lungs is interfered with. They are unable to return to their state of elastic equilibrium and even suffer compression. It is evident that the compression of the lungs and the weight imposed upon the diaphragm interfere with the breathing. Displacement of the heart and disturbances of the circulation in the large venous trunks are among the rarer complications of heart disease. Of course, when the strength of the heart has already closely approached the limits of what is compatible with the continuance of the circulation, a trifle is enough to arrest its contractions altogether. It is difficult to decide, however, whether the effusion itself is not merely a sign that the strength of the heart is about to be exhausted. The small, circumscribed hemorrhages in the pleural tissue which occur in conditions of marked stasis call for remark only because they indicate the profound disturbance of the circulation. They probably never produce clinical symptoms.—Ed.]

5. **Kidneys.**—We have first to investigate the influence of cardiac weakness in the kidneys and their functional activity. It is admitted that a certain number of causes of disease involve both the heart and the kidneys; but that is foreign to the present discussion.

There is a certain *definite alteration of the kidneys*, which is constantly found in fatal cases of cardiac insufficiency that do not run too rapid a course. The beginning of this process is known as *congestion of the kidneys* (Stauungsniere), and its most advanced stage, *cyanotic induration*. In congestion the kidney is enlarged, as a whole, but especially in its transverse diameter; the capsule is tightly stretched and strips readily. After it has been removed, the *stellulæ Verheynii* are seen through the intensely hyperemic surface of the organ, their color a deeper red even than the kidney tissue itself. On the cut surface the cortex appears bluish red; the glomeruli are darker in color; the medullary substance is also darker where it joins the cortex—that is, at the base of the pyramids; as the pelvis is approached the hyperemia diminishes somewhat. In the renal pelvis dilated veins are visible.

In *cyanotic induration* the above-described conditions are modified by the proliferation of connective tissue among the uriniferous tubules. If the venous hyperemia persists for some time, the kidney becomes small, pale, and firm; the capsule thicker and perhaps adherent. The contraction of the proliferating connective tissue may be so great that the term congested and contracted kidney (Stauungsschrumpfniere—Bollinger) may be justifiable.

During the early stages microscopic examination does not reveal any marked changes in the tissue: "When the congestion has lasted but a short time, the renal vessels are uniformly overfilled with blood; the veins and capillaries often considerably distended. The capsular space of many a glomerulus and the numerous uriniferous tubules contain some fluid, which on boiling yields a granular precipitate of albumin and occasionally also contains red blood-corpuscles. A few of the uriniferous tubules also contain hyaline, transparent, colorless masses, so-called

urinary tube-casts, which owe their formation to the escape of albuminous material along with the urinary excretion from the glomeruli and its subsequent coagulation in the uriniferous tubules. Finally, some of the epithelial cells, especially in Henle's loops, contain brown and yellow, partly crystalline, granules of pigment derived from the hemoglobin of the red blood-cells which have escaped and have become dissolved within the urinary tubules. When large masses of red blood-cells have escaped from a glomerulus immediately before the examination, the capsular space or corresponding urinary tubule is more or less completely filled with red blood-cells or the products of their decomposition."*

Ziegler describes the subsequent changes as follows:

"When congestion has persisted for some time and the kidney has become indurated, the connective tissue of the urinary tubules appears somewhat enlarged, the blood-vessels are dilated and gaping, and the capillary walls and the adventitia of the veins are thickened. Sometimes slight inflammatory cellular infiltration also occurs.

Many of the epithelial cells of the urinary tubules undergo fatty degeneration and, accordingly, contain fat-drops of various sizes, particularly the epithelium of the straight tubules in the medullary substance. The glomeruli, as a rule, show no marked changes; quite frequently, however, an individual glomerulus here and there is found converted into a homogeneous ball, and a corresponding urinary tubule is contracted, collapsed, and atrophic."

The changes in the architecture of the tissue are, therefore, by no means inconsiderable; but to what extent they affect the pathologic condition as a whole is difficult to determine. Before these changes can be brought about, a protracted period of heart weakness is necessary, which must affect the functional activity of the kidneys even before any anatomic changes are produced.

How is the function of the kidneys affected by cardiac weakness?

The answer is: The quantity of urine is diminished; a unit volume of the excretion contains less water and more solid bodies in solution. As for qualitative changes, so far as we know, none, as a rule, takes place, except that there may be a small admixture of albumin.

The question whether traces of albumin may not appear in the urine of healthy individuals has by no means been finally settled in the negative, hence the small quantity of albumin found in the urine from congested kidneys is not necessarily a pathologic sign.

Of formed constituents we find a few hyaline casts, leukocytes, and, rarely, red blood-corpuscles. The presence of these elements has no more significance than that of the albumin, which at most amounts to two parts in a thousand. In view of the absolutely small quantity of the urine the loss represented by this small trace of albumin is of no significance whatever.

Bile-pigments occur in the urine in congested kidney only when there is some disturbance in the liver. This will be referred to later. Lactic acid may occur when there is severe dyspnea.

From a clinical standpoint the reduction in the quantity of water excreted through the urine must be regarded as most important. Without going into the theories in regard to the formation of urine secretion, the fundamental fact remains that insufficient supply of arterial blood

* Ziegler, *loc. cit.*, p. 737.

and insufficient drainage of the venous blood restrict and disturb the excretory capacity of the kidneys, and both these conditions are associated with cardiac weakness.

In a general way it is true that the quantity of urine excreted in a unit of time is an index of the amount of work performed by the heart; and if the specific gravity of the urine is determined at the same time, the value of this index is enhanced. It is, of course, assumed that the kidneys are normal except for the deficiency in the quantity of blood passing through them.

For some time it has been my practice to have the daily quantity of urine measured regularly in all cases requiring a careful supervision of the heart action. I give the patients cylindric graduates, provided with a funnel, into which the urine can be poured immediately after evacuation and the entire daily amount thus collected. Among the patients who come to my Poliklinik I feel quite certain that this is carefully attended to. In this way I have collected a considerable quantity of material, and I think I am justified in emphasizing the importance of this simple method, which deserves to be employed more frequently than it is.

The above proposition is open to attack on theoretic grounds. It may be objected that the flow of blood to the kidneys may be increased without any change taking place in the work of the heart, namely, through the influence of innervation on other portions of the arterial system and on the renal artery. An increased quantity of blood may enter the kidney through its dilated arteries, either under the same or under higher pressure than before, whenever the necessary changes have occurred in the remaining portions of the arterial system. This possibility is admitted and, I am inclined to think, helps to explain the rapid fluctuation in the quantity and concentration of the excreted urine in certain forms of cardiac weakness, which have been referred to in another place.* But, on the whole, this theoretic objection, in my opinion, does not detract from the value of the rule in actual practice.

If adequate excretion of water through the kidneys is impossible, accumulation of water in the body can be avoided only when the other organs whose function it is to excrete water,—the skin, the lungs, and, in a lesser degree, the intestine,—compensate by their activity for the diminished function of the kidneys.

This compensation, as we know from experience, is not, as a rule, quite adequate. Some water is stored up in the body and collects in the subcutaneous connective tissue and in the large cavities—in a word, *dropsy* develops. In cases of cardiac weakness the order in which the different parts of the body are involved is as follows: First, those parts which are farthest removed from the heart begin to swell because the kinetic energy is used up before the blood current reaches them and the blood is more than usually under the influence of gravity. Even before the exudation of fluid begins, slowing of the blood current betrays itself by cyanosis and coldness of the parts.

Accordingly, the usual order of involvement is: the feet; the hands—the dropsy spreading always from the periphery toward the center; the external genitalia, especially those of the male; the peritoneal cavity; the thoracic cavity; and, after that, or along with the large cavities of the body, the subcutaneous connective tissue of the trunk. In the more severe grades of cardiac dropsy the influence of gravity makes itself felt more and more forcibly; the accumulation of serum tends to increase

* Observations 4 and 7, pp. 68 and 74.

in the most dependent portions of the body, and this applies to the two pleural cavities as well as to every other portion of the body. The face is always less dropsical than the rest of the body; marked swelling of the face points to some local disturbance in the domain of the upper vena cava and is, as a rule, associated with a considerable and distinctly recognizable accumulation of blood in the capillaries and veins. If the latter sign is not present, the kidneys should receive attention.

Dropsy is the terror of all those who suffer from permanent cardiac weakness—and not without good reason. The difficulty of getting about and the general helplessness associated with large effusions into the subcutaneous connective tissue, not to mention the distressing tension of the skin, in themselves are sufficiently intolerable. But, in addition, by the time dropsy has developed, the cardiac weakness is so extreme that it suffices of itself to make life a burden to the sufferer. The ascites and effusions into the pleuræ in turn interfere with the action of the heart and lungs. Finally, if the tightly stretched epidermis gives way and eczema develops, excoriation of large areas of the skin results, and the patient, who is constantly struggling for air, welcomes death as a deliverer from his sufferings.

I do not think that this mechanic effect, due to the retention of water, is the only consequence of impairment of the renal function. It is accompanied by other disturbances, which, it is true, are not generally so well marked as necessarily to attract attention. These other disturbances may be in the main ascribed to an endogenous toxicosis. That this toxicosis may occur when certain metabolic products formed in the body are insufficiently excreted by the urine may be regarded as positively proved. The interference with the elimination of gaseous respiratory excretions which occurs in heart patients is one, if not the most important factor. In addition there are other factors, which are illustrated in the following history:

Personal Observation 9.—Dilatation and hypertrophy of the right heart, with fatty degeneration of the muscle. Pleural adhesions, particularly on the right side. Anthracosis, with moderate dilatation (emphysema). Scattered foci of cirrhosis and bronchiectasis. Chronic catarrh of the larger and medium sized bronchi. Profound cachexia accompanied by cerebral symptoms. Protracted diminution of the urinary excretion amounting almost to anuria.

The case was under treatment for a number of years. Duration of the last observation, four weeks.

Male, age sixty-one, glazier. For about thirty years has suffered a good deal from cough and shortness of breath, particularly during the winter. States that he was at one time in the habit of taking a good deal of alcohol. Frequent attacks of bronchial catarrh, the last one during the latter part of July and August, 1897, when he had a severe attack with symptoms of cardiac weakness. This subsided in a comparatively short time under suitable treatment.

On the fifteenth of October he was readmitted. For three days previously he had had severe cough and expectoration without any external cause, and the legs were slightly swollen.

Examination on admission: Boundaries of the lungs somewhat extended, reaching in the back on both sides to the twelfth dorsal vertebra. Extensive bronchial catarrh. Cardiac dulness scarcely enlarged; apex-beat in the fifth intercostal space a little inside of the nipple-line. Systolic sound over the apex dull. The sounds everywhere are faint, but there are no murmurs. Pathologic venous pulsation in the neck. Effusion into the peritoneal cavity, the dulness extending to within a hand's breadth of the umbilicus. Moderate edema of the ankles. Urine scanty; specific gravity, 1020; contains a small quantity of albumin.

The sputum is copious, nummular, purulent, with a musty odor and free from tubercle bacilli.

The man is very sallow, poorly nourished, with very little fat and muscle,

although he is in good circumstances for a man of his calling. The appetite is very much diminished and he takes nothing but liquid food.

The history of the course of the disease until the tenth of November, when the man died, is as follows:

In general there was no change in the condition of the lungs; the bronchial catarrh remained about the same, neither increasing nor diminishing; dropsical effusions appeared in the pleural cavities, in the right on the eighteenth of October, and in the left not until shortly before death; but they did not materially influence the respiration. The average of 25 counts showed a respiratory rate of 32 in a minute: maximum, 40; minimum, 27.

The action of the heart was more variable. While the outlines of the heart dulness remained the same and there was no displacement of the apex-beat, a systolic murmur was heard at times over the apex. The following notable circulatory disturbances were present: extension of the venous pulse to the facial and epigastric veins and, ultimately, pulsation of the liver. With this were associated intense cyanosis and hemorrhages from the throat and nose. The radial pulse was at times so feeble that it was impossible to count it; at other times it showed a marked deviation from the apex-beat. At one time there were only 46 pulsations at the wrist to 76 heart-beats. Irregularity in the rhythm as well as the size of the arteries was constant.

The heart action was, on the whole, quite slow in relation to the conditions present. The number of pulse-beats exceeded 100 only for a short period; most of the time it varied between 80 and 90.

The changes in the urinary excretion are worth noting. It was greatly diminished; the total quantity collected in twenty-five days was only 5750 c.c.—or an average of 230 c.c. a day. A very small quantity, at most half a liter during the entire period of observation (twenty-five days), was lost. On one of the days on which there was no doubt that the entire quantity of urine was collected it amounted to only 80 c.c. The specific gravity was not always taken, but was only slightly increased, thus on one day, when 100 c.c. were excreted (nothing lost), the specific gravity was 1020.

The patient's general condition in the main corresponded with the excretion of urine. The urinary curve gives one a good idea of the conditions, which must be characterized as unusual. It was impossible to find a cause for the marked fluctuations. The pulse frequently was not high, except during the last portion of the period of increased excretion (November 1st to 3d), when it was 105 to 110 to the minute. At other times it was not possible to determine any relation between the action of the heart and the excretion of urine. Digitalis, strophanthus, and diuretin were without effect.

Disturbances of the sensorium occurred repeatedly and toward the end were quite marked. The first occurred on the twentieth of October. The history contains the following note: "During the night the patient is said at times to have been confused and flighty in his talk." On the 23d there was some stupor at times during the day. On the 29th "the patient is stuporous, and occasionally evacuates the bowels involuntarily." A small quantity of urine may have been lost with the alvine discharges. This condition continued until the third of November, on which day the patient made a distinctly better impression; stupor was less marked and at times disappeared. By the fifth, however, a change for the worse had again taken place; on the following day unconsciousness developed and continued until the man's death, becoming constantly deeper. On one day also there were again involuntary discharges of feces and urine, the latter only in small quantities—a point to which special attention was paid.

The dropsical effusions were less than the continued diminution of renal activity would have led one to expect. As the patient lay on the right side most of the time, the right half of the body was much more edematous than the left. In the left pleural cavity no fluid could be detected until just before the end, while the right contained an effusion as early as the eighteenth of October. The ascites never exceeded a moderate amount, 1 liter (a quart) of fluid being found in the cadaver. The body temperature was subnormal.

The main points of the autopsy by Professor v. Baumgarten were as follows: General emaciation; skin thin, flaccid, of a dirty grayish yellow, and in the face somewhat cyanotic. Conjunctivæ not jaundiced. Marked anasarca of the right leg and, to a lesser degree, of the left.

Marked atrophy of the subcutaneous cellular tissue, of which only the faintest traces remain. The muscles are pale, and lax.

The brain is somewhat atrophic and slightly edematous, but otherwise normal.

The pericardium presents a surface about the size of the palm; the mediastinal fat is atrophied.

The heart is in its normal position and moderately enlarged to the right; the epicardial sac contains 80 to 100 c.c. of serous fluid.

The right auricle contains a thrombus which completely obstructs it; the left is free. The mitral valve readily admits two fingers, and the tricuspid three. The valvular apparatus and the large vessels are perfectly normal. The right ventricle

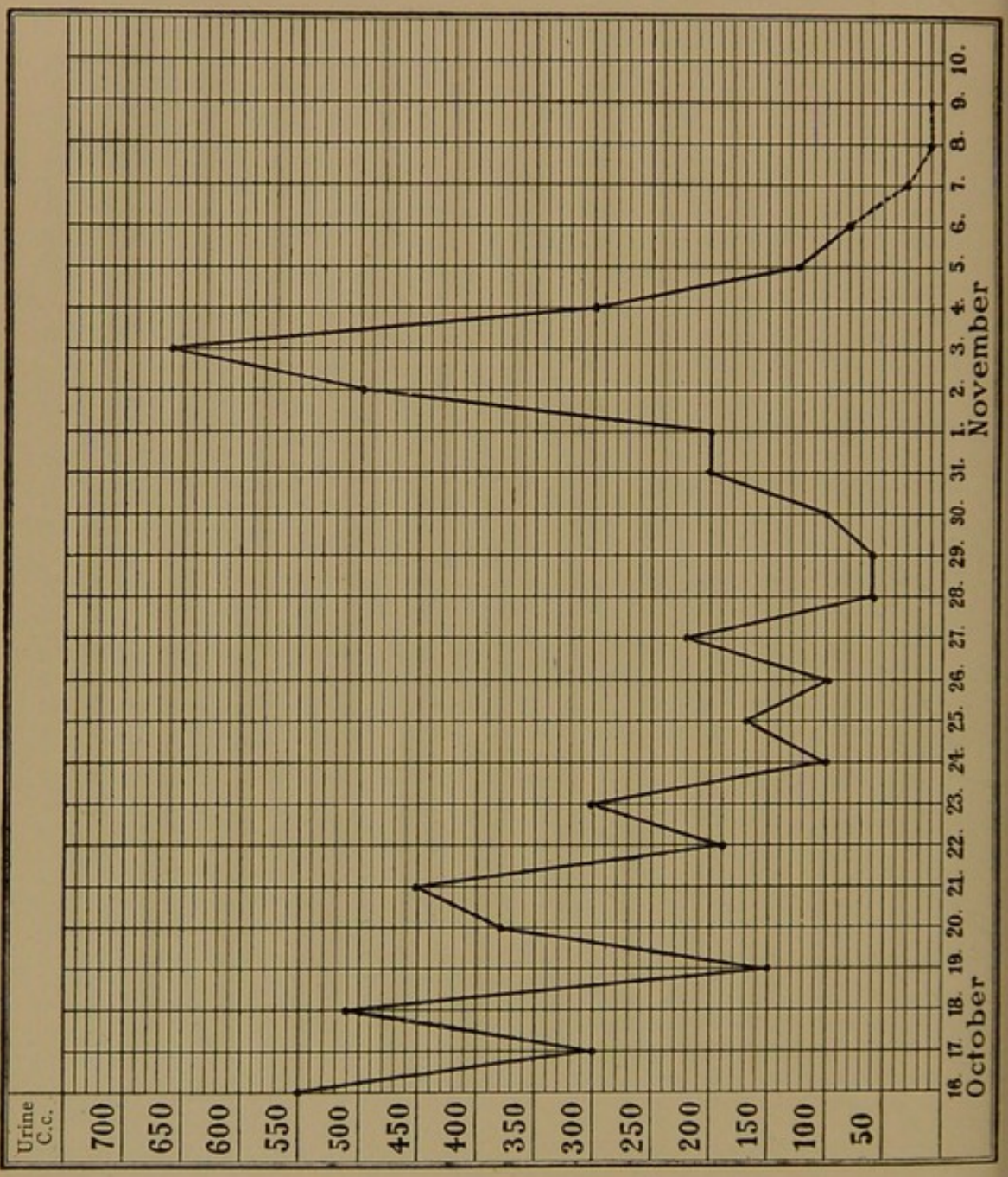


FIG. 23.

is dilated, hypertrophic, somewhat indurated, and presents marked striation and fatty degeneration of the muscle-fibers. The right auricle is also greatly dilated. The pectinate muscles are hypertrophied; the left ventricle is very flabby.

The left pleural cavity contains about 300 c.c., and the right about 1000 c.c., of a serous transudate.

The left lung is adherent to the wall of the thorax, especially in the posterior portion; it is hypervoluminous, feels downy, like an air cushion, and the anterior borders are very much rounded; there are no macroscopic emphysematous blebs.

The apex contains an indurated focus the size of a nut, which, on section, reveals itself as a coal-black nodule of connective tissue with a small cheesy deposit of long standing. The pulmonary parenchyma everywhere contains a great deal of coal pigment. The mucous membrane of the large bronchi is dark red, velvety, and covered with a mucopurulent secretion. There is no marked accumulation of secretion in the smallest bronchi.

The right lung is much more closely adherent than the left and much less voluminous; the induration of the apex is somewhat more marked. Other portions of the lung also contain coal-black fibrous nodules; the edges are sharp, and the lower lobe is the seat of compression-atelectasis. The focus in the apex is coal-black in color, and the rest of the lung also contains much more coal-pigment than the left. The bronchi contain much more secretion on the left side; they are dilated, and in places exhibit saccular enlargements (bronchiectasis). In this lung also there are no marked signs of catarrh of the finer bronchi.

Kidneys: both in a condition of moderate cyanotic induration.

Spleen: not enlarged; hard.

Liver: atrophic, cyanotic nutmeg liver.

Gastro-intestinal mucous membrane deeply injected but without much mucus; pancreas normal; peritoneal cavity contains about a liter of fluid.

In support of the theory that in this case the pathologic changes were in part due to a toxic substance, formed within the body itself and not properly excreted on account of the impaired functional power of the kidneys, I may call attention to the following points:

1. The general disturbance of tissue nutrition was unusually severe. During the time the patient was under observation he received large quantities of liquid nourishment in an easily digestible form (the various infants' foods) and daily one liter (quart) of wine. Absorption of the food was not impeded by catarrh of the gastro-intestinal mucous membrane; there was no diarrhea; neither was there fever. In spite of this, however, the patient declined steadily.

My personal impression was such that I added to the clinical diagnosis, "*carcinoma occultum*," followed, to be sure, by two interrogation marks, for there were no local signs whatever. The fact is aptly expressed in the pathologic diagnosis as universal emaciation.

2. The cerebral symptoms practically kept pace with the quantity of urine excreted. There was stupor, but no convulsions. But as the daily quantity of urine diminished, the stupor increased more and more until finally complete coma developed. But the latter subsided again whenever, as, for example, on the second and third of November, the quantity of urine increased.

This suggests the explanation that failing heart power caused anuria, and at the same time edema of the brain; but it is to be remarked that very little edema of the brain was found in the cadaver, although the quantity of urine during the last three days of life had gone down to a minimum.

Fluctuations in the elimination of carbon dioxid might be thought of, and the cerebral disturbance might be attributed to incomplete elimination. But that also is improbable, because the rate of respiration remained practically the same; the breathing itself did not present any disturbances characteristic of a central lesion (Cheyne-Stokes type), and the autopsy showed the finer bronchi free from catarrh and secretion.

It is, of course, impossible to adduce any positive proof that my view is correct; on the other hand, it seems to me that it is equally impossible to refute it. Let it suffice, therefore, to suggest the possibility of some such process.

Traube and Bartels taught that marked uremia does not occur in cases

of renal congestion (Stauungsniere), and this view, so far as I can see, has been accepted by the majority. In Husche's article, which will be mentioned later and which emanated from Gerhardt's clinic, it is stated that a heart patient (observation 4) suffered from uremic symptoms.

The composition of the urine in congestion of the kidneys (Stauungsniere) requires some discussion.

In harmony with the diminished excretion of water through the kidneys, the specific gravity is usually increased because, as has been mentioned, the reduction in the quantity of constituents in solution does not keep pace with the diminution of the water. The specific gravity may be 1035 or over; the smaller the quantity of urine, the higher the specific gravity.

There are, however, some exceptions to this rule. In the case which I have just reported at length I once found the following relations:

Quantity of Urine.	Specific Gravity.
380 c.c.	1022
100 c.c.	1020
120 c.c.	1020

In fatal cases particularly I have not infrequently seen a comparatively low specific gravity—as low as 1015—even when the quantity of urine was considerably diminished. Bartels * has made the same observation. How great the fluctuations may be in certain forms of cardiac insufficiency has been mentioned in another place (see p. 70).

The urine is generally intensely acid in reaction. The color is a reddish brown. At first the urine is clear; as it cools, a brick-red sediment forms (*sedimentum lateritium*), which can be redissolved by warming and consists of amorphous urates and possibly a few crystals of uric acid.

The quantity of urates in solution is usually relatively large; Bartels† found that it exceeded 5 per cent. Uric acid may also attain a relatively high figure, although this is not always the case. The mineral salts are subject to great fluctuations.

It is questionable whether the transition from congested kidney (Stauungsniere) to congested and contracted kidney (Stauungsschrumpfniere) can be recognized. The latter requires for its development a long-continued, severe cardiac weakness. Senator‡ has aptly remarked that contracted kidney can no longer be diagnosed after rupture of compensation has taken place and has led to a marked condition of stasis.

Renal infarcts produced by the entrance into the renal arteries of blood-clots derived from the left ventricle are to be regarded as direct consequences of severe cardiac weakness. The emboli do not often give rise to clinical symptoms because the obliterated area in the kidney is generally quite small. Hence it will rarely be possible to recognize the accident during life unless very careful microscopic examinations of the urine are made every day. If at the time of such a hemorrhage pain develops in the region of the kidney and the latter is sensitive to pressure, embolism of the renal artery on the affected side may be thought of, provided, of course, the condition of the heart is such that the formation of coagula in the left ventricle may reasonably be assumed.

* "Nierenkrankheiten," p. 188. † "Nierenkrankheiten," p. 183.
‡ "Erkrankungen der Niere," "Nothnagel's System," vol. xix, p. 271.

Traube * reports a diagnosis which was confirmed by autopsy. There was great dilatation of both ventricles; the muscle was pale and flabby; the "semilunar valves of the aorta were thickened, curled up, and covered with warty vegetations; one of the valves was torn through the middle." I have had a similar experience. In Traube's case the infarct was large—about 5 cm. on the surface; in my case it covered an area of about 3 cm.

The clinical signs were those mentioned above, with the exception of spontaneous pain; there was only pain on pressure, which, however, persisted until death—a period of about twelve days. It was one of my cases of Tübingen heart with globular vegetations in the right auricle and left ventricle. The autopsy findings were as follows (dictated by Schüppel):

"Left kidney somewhat larger than normal, very firm, and the seat of an extensive hemorrhagic infarct already become pale, occupying the entire thickness of the cortex; the base is about 3 cm. in extent. The infarct has a lemon-yellow color and is surrounded by a red areola. The capsule is firmly adherent. The cellular tissue is poor in fat and over the infarct shows inflammatory infiltration.

"The right kidney is of normal size, very firm, and shows a number of cicatricial contractions. It is anemic; the pyramids only are blood-red; the glomeruli, injected."

Even when the activity of the kidneys has been diminished for a long time, perfect recovery in every respect is possible if only the heart can be made to work more vigorously. In other words, the kidneys do not suffer atrophy from disuse. Whether this also applies to the congested and contracted kidney I will not attempt to decide.

6. **Digestive Organs—Stomach, Intestine, Liver.**—Digestive disturbances never fail to make their appearance as soon as cardiac weakness becomes at all marked. At first there are general malaise, pressure, signs of fulness after eating, frequent belching and after-taste—in short, the well-known signs of sluggish digestion.

The liver at this time, and, in fact, at any time, is but little swollen, but constipation with the resulting distention of the intestine is practically constant.

Some patients have fair appetites; but it is by no means true that beginning heart weakness is without influence on the ingestion of food. Heart patients are like other dyspeptics; some of them are so sensitive to the slightest disturbance that they at once cut down their food more than is good for them, while others are undaunted and eat what they like, being quite willing to take the consequences. C. v. Noorden† refers to this class of heavy eaters when he compares the small amount of muscular work they perform to the excessive ingestion of food in the following words: "The disproportion between the large amount of food and the limited amount of muscular exercise often results in a condition of obesity, a relationship which was emphatically pointed out by Oertel." But this combination is rarely observed among those who have to earn their bread by manual labor. In heavy eaters, on the other hand, the accumulation of adipose tissue, with or without alcoholism, is usually the first sign of disease and often precedes the symptoms of cardiac weakness, of which, indeed, it may be the cause.

As the circulatory disturbances become greater the dyspeptic symptoms also increase. I have frequently known the dyspepsia to become so prominent as to mislead a physician of only moderate experience in cases in which there was no pronounced valvular lesion and cardiac disease was entirely muscular, especially in the presence of marked impairment of the general nutrition.

* "Gesammelte Abhandlungen." vol. ii, p. 347 *et seq.*

† "Lehrbuch der Pathologie des Stoffwechsels," p. 320.

For example, in the history of a case of fatal cardiac insufficiency of purely muscular origin I find the following notes: "The patient is a vigorous man, has been treated repeatedly since August, 1875. As he complained of dyspepsia, sensitiveness to pressure in the epigastrium, and the like, gastric ulcer or even carcinoma was thought of." The latter diagnosis is very apt to be made if there happens to be a groove from constriction at the appropriate region over the liver.

The dyspeptic symptoms that occur in heart patients are not characteristic, although it may be mentioned that constipation is usually obstinate and diarrhea rarely occurs. A certain degree of cachexia, due probably to insufficient nutrition, rarely fails to develop sooner or later; but the emaciation may be disguised by the dropsical condition of the skin. But it is not only on account of their dyspeptic symptoms that the patients take less food. When the stomach and intestine are full, the movements of the diaphragm are interfered with and the feeling of oppression thus produced keeps many a patient from taking a sufficient quantity of food.

*Special Symptoms.**—1. Disturbance of hydrochloric acid secretion is observed only in pronounced conditions of cardiac weakness. In cases of universal dropsy and severe dyspnea, C. v. Noorden frequently found that the gastric juice, obtained by siphonage, contained a normal excess of hydrochloric acid after a small test-breakfast (tea with wheat bread; contents removed from three-quarters to one hour after eating), but never after a copious test-dinner containing meat (contents removed five to six hours later). The evacuation of the stomach was not delayed.

2. Absorption of food. The ingestion and assimilation of carbohydrates and albumin are the same as under normal conditions. Fat varies; assimilation may be normal, but the opposite has also been observed.†

According to the present teaching in regard to absorption from the intestine, one is not inclined to ascribe the latter directly to interference with the circulation in the intestine, and it would indeed be difficult to give reasons for such a view. We are more inclined to agree with F. Müller‡ that the absorption of fat depends chiefly on the nutrition of the epithelium in the intestine.

3. The decomposition of albumin in the intestine is not increased. Phenol and indican do not appear in increased quantities even in marked disturbances of the circulation.

Some anatomic changes are also present in cardiac weakness, but are not, as a rule, far-reaching: catarrh of the stomach and intestine; superficial hemorrhages. That is generally all. Of course, a hemorrhagic erosion may possibly develop into a gastric ulcer, but such an event must be rare; at least there is no mention of it in the literature.

Proliferation of connective tissue, which occurs elsewhere in conditions of stasis, is not known to occur in the stomach and intestine. Hemorrhages are usually insignificant and quite circumscribed, and the amount of blood lost is so small that it is not seen in the stools. Hemorrhage from the dilated veins of the rectum—at least severe hemorrhoidal hemorrhage—is by no means frequent.

Embolism of the superior mesenteric artery, with resulting infarct

* I have followed C. v. Noorden's teaching in his "Lehrbuch der Pathologie des Stoffwechsels."

† See the table given by Th. Husche, "Ueber die Stickstoff-Bilanz in den verschiedenen Stadien der Herzkrankheiten" (from Gerhardt's Clinic), "Zeitschrift für klinische Medicin," Bd. xxvi (1894), S. 66.

‡ "Ueber Nahrungsresorption bei einigen Krankheiten," "Verhandlungen des Congresses für innere Medicin," Bd. vi (1887), S. 404 ff.

of the intestinal wall, is rare; and the formation of such an infarct from disturbance of the venous flow is even more rare.

I recently observed a case of this kind. It occurred in a man of sixty, who suffered from muscular heart weakness. The patient had been in bed for a long time and had become very fat. During the last days of his life he developed abdominal pain, which constantly became more severe and was followed by watery discharges containing a large quantity of blood.

At the autopsy, which was conducted by Prof. v. Baumgarten, the following notes were made: "Abdomen distended, umbilicus obliterated. On opening the abdominal cavity all the intestines, particularly the loops of small intestine, are found to be greatly distended and covered with a fibrinous, blood-stained exudate. An old adhesion connects some of the loops of the lower ileum with the intestinal wall of the left inguinal region; the upper end of the adhesion is narrow, but is attached to the intestinal wall by a broad base. The loops of intestine are somewhat distorted by the pull of this adhesion and are thickly covered with a fibrinous, hemorrhagic exudate; they are a dark, bluish red, almost black, and the wall is thickly infiltrated.

"The mesenteric attachment of these loops is also thickly infiltrated and dark red, both on the surface and on the cut surface. Similar, but less pronounced, changes are observed in the adjacent loops of the ileum. The corresponding mesenteric veins are distended with thrombi of a dark-red color and fairly firm in texture, which totally obstruct the vessels.

"The loops of intestine on being opened are found to have a thickened, succulent, and intensely bluish-red mucous membrane, which has lost its normal glossiness and presents opaque gray spots in various places. In vertical sections taken from diseased portions of the intestine, especially the mucosa and submucosa, all the layers of the intestinal wall are seen to be the seat of a diffuse hemorrhagic infiltration.

"Along the psoas muscle on both sides of the body, but on the right side more than on the left, there is a broad, cord-like, somewhat nodular, hard mass of tissue extending as far as the *lacuna vasorum cruralium* (femoral opening).

"This hard, cord-like mass is nothing less than the subperitoneal fatty tissue of the region which has undergone a peculiar form of sclerosis. The great vessels inclosed in this tissue do not show any changes, especially no thrombosis of the femoral vein. On the other hand, the smaller veins within the sclerotic fatty tissue are all thrombosed.

"Microscopic examination of the sclerotic masses of fatty tissue from the psoas region fails to supply any satisfactory explanation for this peculiar sclerosis. Aside from the macroscopic thrombosis in the veins, more recent inflammatory proliferations of the interstitial connective tissue were found.

"The spleen is somewhat larger than normal and shows slight thickening of the capsule and trabeculæ. The splenic pulp, which in general is quite soft, contains hard, fibrous, irregularly angular foci, ranging in size from that of a pea to that of a cherry.

"The liver is the seat of diffuse, moderate fatty degeneration. The other organs of the peritoneal cavity show no noteworthy changes."

In this case there probably was a circulatory disturbance in the territory of the mesenteric veins, which was favored by the old adhesions and the marked accumulation of adipose tissue and ultimately led to marantic thrombosis. The sclerosis of the connective tissue may also have been produced by the prolonged condition of stasis.

In cases of permanent heart weakness the liver becomes profoundly involved. Shortly after the first appearance of heart failure the organ begins to swell; the tension of the capsule causes a feeling of pressure and pain, which becomes very distressing and may cause a sensitive patient involuntarily to inhibit respiration; the symptoms rapidly subside as the circulation is restored.

But if the stasis continues, the following conditions, taken from Ziegler's* excellent description, are found:

"The liver is usually somewhat diminished in size, the surface not

* "Lehrbuch der speciellen pathologischen Anatomie," 9. Auflage, S. 575, 576.

infrequently unequal, granular, and slightly nodular. The cut surface shows the typical picture of nutmeg liver, the central portions of the acini being a deep dark red and usually depressed below the cut surface, while the color of the periphery varies from dark brown to pale brown and sometimes a yellow-brown, depending on the quantity of fat contained in the cells.

"If the process goes on, the dark-purple portions preponderate over the paler tissue, which in places may be wanting altogether. At the same time the acini diminish in size—cyanotic or congested atrophy (central red atrophy) exists.

"On microscopic examination it is found that the hepatic veins, especially the central venules and the capillary region immediately adjoining, are dilated. In more advanced grades of the disease all the capillaries of the acini are dilated. The liver-cells between the dilated capillaries are always more or less atrophic and usually contain granules of yellow and brown pigment, and many of them also fat-granules. The degeneration is always most advanced at the centers and in the intermediate zones of the acini. When the circulatory disturbances are protracted and there is marked dilatation of the capillaries, some of the liver-cells may be totally destroyed, leaving only granules and fragments of yellowish and yellowish-brown pigment between the dilated capillaries.

"The periportal connective tissue of the liver usually remains intact, but it may occasionally become the seat of hypertrophy and cellular infiltration, giving rise to a special form of cirrhosis."

Thus the tissue changes are quite marked, and it may be asked whether, and if so to what degree, the function of the liver is altered by the injury which the organ suffers. We do not know much about this point.

1. The formation of urea in the liver does not seem to be materially affected. Leucin and tyrosin, the amido-acids, do not appear. The relation between the urea and the total quantity of nitrogen in the urine is not altered, and the excretion of ammonium also fluctuates within normal limits.*

2. With regard to the formation of glycogen nothing is known. In animal experiments sugar has occasionally been found in the urine, with an increase of sugar in the blood; but as no similar observations have been made in man, their interpretation cannot be allowed to affect the question.

3. Formation of bile. Whether the quantity of bile secreted undergoes any change in cases of cardiac insufficiency we do not know. *A priori* a diminution seems probable; for one of the chief conditions for the secretion of bile, namely, the ingestion of food, especially nitrogenous food, is certainly diminished in the more marked grades of heart disease.

A slight degree of yellow discoloration of the skin and conjunctivæ is quite frequent, but the urine rarely contains enough bilirubin to render Gmelin's reaction positive. On the other hand, it contains a great deal of hydrobilirubin.

In harmony with the prevailing views C. v. Noorden offers the following interpretation of these facts:

"When a large quantity of hemoglobin is liberated by the disintegrating red blood-corpuscles it is excreted by the liver. Bile secreted under these conditions is rich in bilirubin and at the same time thicker and more viscid. This interferes with the evacuation of the bile from the smaller bile-ducts, and absorption takes place.

* See Husche's figures, *loc. cit.*, p. 67 *et seq.*

That portion of the bile which is evacuated into the intestine and which contains a large quantity of bilirubin is subjected to the action of bacteria in the intestine, and these convert the pigment into hydrobilirubin, the greater part of which finds its way into the urine by absorption."

He argues,* from the condition of the bile, that "hemoglobin metabolism in congestive diseases must be very active. It is uncertain, however, whether the attack on the hemoglobin takes place in the blood itself, in the tissue, or in the liver-cell."

Whether this is a final conclusion remains to be decided. It does not appear to me that what we know of the constitution of the whole blood and the number of red blood-corpuscles which it contains is calculated to support C. v. Noorden's views.

7. **Nervous System.**—Severe anatomic changes in the brain are, on the whole, infrequent. Marantic thrombosis and embolism sometimes occur when the strength of the heart begins to fail, but, as a rule, some very grave disease of the cerebral vessels is present at the same time. Thickening and opacity of the meninges are found quite frequently, but are usually without significance or at least not of very great importance. Venous hyperemia and dropsy are often quite marked; but it is difficult to make out to what extent these conditions had already developed during life and what part is agonal.

The same is true of the spinal cord. In regard to tissue changes in the peripheral nerves nothing is positively known.

Functional disturbances, alterations in the cerebral activity, are quite the rule in the very severe grades of cardiac weakness, although vertigo and syncope, it is true, are not always present. A feeling of malaise, of bodily and mental lassitude, and irritability are practically never absent. Persons who are naturally good-natured and have good self-control of course do not act exactly like those who naturally have a less amiable disposition. The latter are a burden to themselves and their attendants when they develop heart failure. Even hypochondriasis quite often makes its appearance. In a predisposed individual a true psychosis may develop; but it is doubtful whether, as has been maintained, heart disease in itself favors the development of insanity.†

Aside from the fact that the lot of a heart patient is by no means an enviable one, and that at a time when he should still retain his powers he is more or less incapacitated for work, the somatic (systemic) changes to some extent explain the mental disturbance, which is probably attributable mainly to the congestion in the capillaries of the brain and the retardation of the cerebral circulation. To these may be added certain conditions the nature of which is not well understood and which are associated with digestive disturbances and sluggishness of the bowel.

The accumulation of carbon dioxid in the blood and in the tissues is followed by the appearance of more or less marked symptoms of carbon dioxid poisoning: drowsiness, with a cerebral form of dyspnea rather objective than subjective, and persistent coma, which gradually becomes deeper and ends in death—the usual mode of termination in cardiac insufficiency.

It is undoubtedly a true autointoxication and rarely fails to develop. But it is not certain whether it is always the only form of intoxication. I have reported a case in which renal insufficiency also appeared to be a factor of importance. It is very probable that failure of the kidneys not very rarely takes part in the final catastrophe, and whenever the

* *Loc. cit.*, p. 321.

† Compare Theodor Kirchhoff, "Lehrbuch der Psychiatrie," Leipzig und Wien (1892), Franz Deuticke, S. 59.

lungs are clear or only slightly involved, this possibility must be taken into account.

It should be noted that these conditions of torpor may subside even after they have attained a very high degree, and one must be very careful not to predict a speedy end because of their presence.

The cerebral symptoms that follow embolism and thrombosis need not be discussed in this connection.

8. **Metabolism; Temperature.**—What the complete balance of metabolism is we do not know, but a few individual items may be mentioned.

According to the teaching of the present day (Voit, Pflüger) in regard to the influence of the oxygen supply, the amount used up is increased only when the cells perform more work. As compared with a healthy individual at rest, the resting heart patient needs a larger supply of oxygen because of the greater amount of work performed by his respiratory muscles. Whether this extra amount of oxygen can be obtained is a question which has been carefully investigated by C. v. Noorden,* but without any decisive result.

The experiments fail to show "whether in chronic diseases in man, accompanied by really severe dyspnea, the body-cells are actually able until the agonal period to obtain their usual supply of oxygen by bringing all the compensatory auxiliary forces into play, and whether the agonal period begins as soon as a true oxygen deficit makes its appearance; or whether the cells adapt themselves to the altered and more difficult conditions and are able to get along with the somewhat diminished supply of oxygen." This last possibility seems to be well worth considering. The conditions are practically the same as when an athlete is undergoing training; although the amount of oxygen used and material burnt up is the same, it is more fully utilized because a larger proportion of the kinetic energy produced by the combustion is converted into work.

The following considerations are worthy of notice.

The slowing of the circulation in the lungs cannot in itself cause a diminution of the amount of oxygen taken up by the blood as it flows through the lungs, provided the ventilation of the lungs is not impaired. The cyanosis which is so frequently seen in heart patients is due to the fact that the blood flows through the capillaries and tissues more slowly and there gives off a small quantity of oxygen and takes up a larger quantity of carbon dioxid. Cyanosis, therefore, merely indicates slowing of the circulation.

"In spite of the slowing of the circulation the total quantity of oxygen supplied and the total quantity of oxygen used may be normal. We must not conclude from this that the condition does not affect the tissues; for the presence of oxygen in the capillaries at a low tension makes it difficult for the cells to obtain the necessary quantity of oxygen. This low tension they are able to overcome; but in the end the development of the cells as well as their metabolism must suffer.† This shows itself in disturbances of growth which are almost constantly present in the nails and distal phalanges in patients with chronic cyanosis, as well as in the failure of general development observed in patients who have suffered from circulatory disturbances from early youth (C. v. Noorden)."

* *Loc. cit.*, p. 315.

† Compare v. Hösslin, "Ueber den Einfluss der O_2-Spannung im Gewebe auf den O_2-Verbrauch," "Sitsungsberichte der Gesellschaft für Morphologie und Physiologie in München," 1891, S. 6.

The composition of the respired air supplied by lungs that are not everywhere equally permeable is not without influence on our estimate of the conditions in cardiac weakness; for obstruction of the finer respiratory channels almost always occurs in the more severe cases of cardiac failure. The investigations carried out by Bartels* in cases of croup, and later by Geppert† in patients suffering from emphysema and bronchitis, gave the following results.

Although the quantity of air passing in and out of the lungs in a unit of time—the coëfficient of ventilation—is adequate, the blood does not take up sufficient oxygen or give off sufficient carbon dioxid. Those portions of the lung cannot perform the gaseous interchange for the entire organism. The more extensive the impermeability of the respiratory surface, the more marked is this condition, as Geppert has directly proved.

We thus understand that the compensatory power of the respiratory muscles has a definite limit.‡

The nitrogen metabolism of heart patients is not known. The investigations that have been made are not very reliable because excretion of water through the kidneys takes place irregularly, and hence the finding of nitrogenous bodies in solution in the urine does not help us to estimate how long a time has elapsed between the ingestion of the albumin and the excretion of the nitrogen. It is, therefore, impossible to erect a metabolic equation.

It may be of interest to cite§ the results and conclusions arrived at by C. v. Noorden in a large number of investigations performed under these conditions in Gerhardt's clinic.‖ They are:

"1. When digitalis treatment is effective, very large quantities of nitrogen are usually thrown off from the body along with the water from the edematous fluid.

"2. The nitrogen flood rarely persists more than two to four days. During this time the total nitrogen output may exceed the intake by 30 to 40 grams.

"3. The nitrogen flood is not always uniform; days when the excretion is abundant alternate with others when little nitrogen is excreted, although diuresis may continue to be active.

"4. In other cases the quantity of urine only is increased. The urinary nitrogen, already in equilibrium with the nitrogen contained in the food, is not affected.

This appears to show that in the first group of cases large quantities of substances that should be excreted with the urine are deposited in the edematous tissues, and that there is also a second group of cases, in which the transudate consists chiefly of water and the excretion of nitrogenous metabolic products through the kidneys is not interfered with."

The *temperature* in bedridden heart patients is subnormal in the great majority of cases. [In a case of tricuspid stenosis (with other lesions) reported by Dock the temperature was above 98° F. only twice in two months. It was usually between 96° and 98°, sometimes 95° or slightly

* "Ueber die häutige Bräune," "Deutsches Archiv für klinische Medicin," Bd. ii (1867), S. 409 ff.

† "Untersuchungen über die Respiration bei Emphysema pulmonum," "Charité Annalen," Bd. ix, Jahrgang (1884), S. 283 ff.

‡ Compare p. 50 *et seq.*

§ *Loc. cit.*, p. 327.

‖ These are given at length by Husche in the article referred to, "Zeitschrift für klinische Medicin," Bd. xxvi (1894), S. 44 ff.

below that.—Ed.] But it may, on the other hand, be quite variable. Apparently insignificant disturbances suffice to send up the temperature, although the elevation is never very great.

THE TREATMENT OF CARDIAC INSUFFICIENCY.

General Principles.—Taking them all in all, there are not many diseases that present so many therapeutic difficulties as cardiac insufficiency. The main object is to bring about a proper relation between the amount of work performed by the heart and its functional capacity, and to avoid making any demands on the organ which it is unable to meet. As a rule, patient and physician disagree widely on the question of how much work may be demanded of the heart; and even if the patient is fully convinced that the physician is right when he tells him he must make a complete change in his mode of life, he is often compelled by the bitter necessity of providing for his family to disobey his physician's orders. Another difficulty is that a heart patient who has not yet become disabled by his disease has no accurate means of estimating how much work his heart is able to do. Even the physician often finds it difficult to determine this point. In giving his instructions he assumes that the amount of work performed by the heart in twenty-four hours shall not exceed a certain limit, which is estimated according to general principles. Unfortunately, some unforeseen accident, such as a digestive disturbance, a sleepless night, or mental excitement, may occur at any time and mar the reckoning, and the heart is at least temporarily—while the disturbance lasts—disabled and cannot perform the work demanded of it. And so the mischief is done; and although normal conditions may be restored in a few days, the disturbance may, on the other hand, be the important immediate cause of the chronic disease, which before had been only threatening the patient, becoming fully established. These accidents are particularly common in the case of patients who are not obliged to do hard physical work; for in such cases it is peculiarly difficult to determine how much work the heart is capable of.

The unfortunate tendency of all patients suffering from chronic diseases to change constantly from one medical adviser to another is particularly marked among heart patients. They furnish a rich harvest to the "specialist" who only understands, or quite frequently does not understand, "his own" organ. Every physician who has been in practice for any length of time will recall such instances. The same is true of the universal specialists—Pastor Kneipp & Co.—who fill God's acre more than they cultivate God's vineyard.

At the end of the long list of their sins may be mentioned the improper use of digitalis, from which many a heart patient has had to suffer. Not only when another physician is called in, who, not knowing what his predecessor has ordered, naturally falls into the error—it has also happened, and will probably happen again, although I have not seen such an instance during the last few years, that officious friends of heart patient A give him digitalis because it helped heart patient B so wonderfully. On the principle of "the more the better" the patient then continues to take the remedy, with the result known to every one who is familiar with the action of the drug.

What are the principles that should guide us in the management of heart patients? I repeat: a weak heart must not be allowed to over-

exert itself, and the physician must have the great danger of overexertion constantly in mind. Hence the patient's mode of life should be so ordered as to guard against any sudden strain on the already weakened heart; that is, the patient's work should be so arranged as to make it impossible for the heart to exceed the limit of its powers even for a short time. The labor curve must not present any sudden rises and falls, as  ; it must be uniform, thus ——.

The application of these principles will now be considered in detail.

Nutrition.—The ingestion of food always throws an extra demand on the heart; the greater the quantity of food ingested at any one time, the greater the amount of work performed by the heart during that time—the period of digestion. The active organs of digestion must be supplied with blood; an increased quantity of fluid is temporarily taken up by the tissues and by the blood and circulates with the latter. Distention of the stomach with food may impede the movements of the diaphragm. Hence a patient suffering from heart disease should eat several meals a day, which should be approximately equal in quantity. A single large meal either at noon or in the evening, at which enough food for the entire day is taken, must be absolutely forbidden.

Oertel* emphasizes the importance of these matters and lays down rules the observance of which may be of the greatest benefit to the patient in severe cases. One thing that he insists upon seems to me particularly important. It is that no fluid ought to be taken with a heavy meal, the patient being instructed to drink water between meals instead. With a small meal this precaution is not necessary.

The question of the diet to be prescribed must, of course, depend on individual conditions; it is impossible to give general instructions on this point. The contrast between the corpulent bon-vivant and the wretched, emaciated laborer—both suffering from muscular weakness of the heart—presents a sufficiently striking picture to show the folly of attempting to give "dietetic prescriptions for heart patients."

A number of authorities have recently stated that heart patients should be made to avoid alcoholic beverages altogether. This is certainly a mistake and may have grave consequences, as I can testify.

But we must carefully distinguish between two things. It is self-evident that a heart patient must not take any spirituous liquors that give the heart an unnecessary amount of work. On the other hand, is it certain that spirituous liquors always and under all circumstances do increase the work of the heart? If an individual is accustomed to taking a definite quantity of light wine, distributed through the day in such a manner that the temporary filling of the vessels due to the increase in fluid taken up by the tissues is never very great; if he is not conscious of any stimulating effect of the alcohol on his heart, why should he be denied his customary glass of wine, especially—and this must not be forgotten—as it diminishes nitrogen waste in the body. Really, it would be difficult to find a reason for subjecting him to such a deprivation.

It is to be remembered that in these cases one usually has to deal with habits of living and eating to which the patient has been accustomed for years and that any change demands a certain adaptability on the part of the organs.

* "Handbuch der allgemeinen Therapie der Kreislaufstörungen," Leipzig, F. C. Vogel, p. 142 *et seq.* of the fourth edition (1891).

The loss in calories caused by the withdrawal of alcohol can only be made good by giving more fat or carbohydrates, and the assimilation of these substances under all circumstances necessitates a greater amount of digestive work on the part of the body than is required for the assimilation of alcohol.

But a more important consideration is that an individual who is suddenly deprived of his accustomed quantity of wine suffers from disturbed appetite, anorexia, not infrequently insomnia, and practically always some depression; and as a heart patient is naturally inclined to be low-spirited, why should this tendency be unnecessarily increased?

It need hardly be said that no sensible person will approve of taking large quantities of alcohol from morning till night, especially in a concentrated form, even if the individual has been accustomed to such a mode of life; but there is a wide difference between permitting such immoderate indulgence and forbidding alcohol altogether. Oertel's teaching is largely responsible for the length to which men have recently gone in forbidding the use of alcohol, forgetting that Oertel's prescriptions were based on what he had seen among people who daily drank excessive quantities of beer, which was quite a different matter.

The same rules apply to *coffee*, *tea*, and *cocoa* as to alcoholic beverages. Strictly speaking, they stimulate the heart even more powerfully, and the stimulating effect does not disappear with habituation to the same extent as in the case of alcohol. It is, therefore, necessary, in regulating a patient's mode of life, to lay an embargo on strong tea or coffee.

In the matter of *tobacco* I am guided by the same principles. There is no doubt that the weed is capable of doing a heart patient great harm. In the case of immoderate smokers who, for some reason, have developed heart disease it is not infrequently observed, at least so far as an opinion may be expressed at all, that their condition progresses more rapidly than in the case of non-smokers with an equally severe form of the disease. I am distinctly of the opinion that the "nervous" symptoms which may go on to attacks of angina pectoris, and which must undoubtedly be attributed to the abuse of tobacco, are more severe in this class of patients and have a more powerful effect on the heart.

Whether the excessive use of tobacco alone suffices to produce cardiac weakness beyond what belongs to the domain of neurosis, I am prepared neither to affirm nor to deny. It seems to me difficult, if not impossible, to decide the question because many excessive smokers at the same time indulge in other excesses which threaten the integrity of the heart; they eat and drink too much, work too hard, and expose themselves to mental and emotional overexertion. In the presence of such complicated conditions I reserve my judgment. For the same reasons that I refuse to forbid alcohol altogether I am not ready to admit that a smoker who is suffering from heart disease must be forbidden to use tobacco.

One thing more should be pointed out in this connection. Quite apart from the effects of tobacco smoke itself, there are smokers who injure their stomachs by chewing the ends of their cigars and thus absorbing the products of dry distillation from the end of the cigar or possibly from a foul pipe. This is bound to lead to a genuine inflammation of the stomach and all its evil consequences. This practice must, therefore, be avoided; and if the patient is unable to break himself of the bad habit he must be made to give up smoking altogether.

If smoking causes severe pharyngeal catarrh with considerable cough or even retching and possibly vomiting in a heart patient, it must be given up altogether.

The bad effects of taking snuff are not always sufficiently appreciated. Personally I am inclined to believe that snuff-taking is almost more harmful than smoking. The snuff-taker always swallows a certain quantity of finely powdered tobacco. A glance into the throat will show the black lines and spots extending far down into the pharynx. It would be very remarkable under these circumstances if none of the snuff got into the esophagus and stomach, where it undergoes maceration and causes local irritation of the mucous membrane. Hence the local effect of snuff is added to the constitutional effect—the introduction into the body of nicotin, which is present in snuff in considerable quantities, and one who is addicted to the habit consumes a good deal of his favorite weed.

I have never seen even the most inveterate smoker continue to smoke when he feels ill, but with snuff-taking it is very different. I have often seen a patient continue to take snuff while suffering from pneumonia, for example.

It seems to me, therefore, that snuff-taking should be more strictly prohibited in cases of heart disease than smoking.

Rest and Exercise.—The question how much exercise a heart patient may be allowed is an important one. Under some circumstances absolute rest in bed is imperatively demanded, as when the strength of the heart has been reduced to such a point that it only just suffices to maintain the circulation necessary for the continuance of life. The heart is unable to meet any extra demand, as shown by the dyspnea and the marked degree of anxiety which are brought on by the slightest exertion.

The question of nursing is often a very difficult one in this stage of heart disease. These patients require the most careful nursing to guard them from bed-sores and ulcerations of the skin. When it is possible, much can be done to add to the patient's comfort by allowing him to sit in a comfortable chair for a time. But to effect the change in position without causing too much discomfort, especially when the patient is dropsical, requires strong and practised arms. Whether the patient sits up in a chair or in bed, he must be made as comfortable as possible with the aid of pillows and pads. It is hardly necessary to say that heart patients must be guarded against any loss of body-heat; unfortunately, this is not always easy, because the patient cannot tolerate anything heavy on the chest on account of the interference with the respiratory movements.

In less advanced cases the physician must decide when and to what extent the work of the heart may be increased for the purpose of stimulating and strengthening the organ. In the well-chosen words of Hoffmann: "When must absolute rest cease and exercise begin?" In my own opinion one general rule may be definitely stated: so long as the diminished action of the heart is associated with general symptoms pointing to an infection localized in the heart, absolute rest is indicated. In order to arrive at a positive opinion in these rather frequent cases, the temperature ought to be studied more accurately than is generally done. I shall return to this point later.

If there is fever due to some cause not immediately connected with the heart; if there is bronchial catarrh of any severity, a prudent physician will always order the patient to bed while the condition lasts, even if the weak heart does not appear to be involved in the morbid

process. The less the functional capacity of the diseased heart, the more must rest in bed be enforced. Even a slight disturbance, such as an attack of indigestion, diarrhea, and the like, must be taken seriously. Much good may be accomplished by observing these precautions.

In the main there is unanimity of opinion on these points. It is also generally admitted that the question whether the patient may be allowed to exercise or must be kept at rest is determined in part by the cause of the cardiac insufficiency—the form due to overexertion and the "beer-heart" being taken as the two extremes. It is also generally admitted that when any changes in the valves and in the orifices are present, their seat and extent must be given full consideration. Stenosis at the orifice of the pulmonary artery does not demand the same precautions as insufficiency of the semilunar valves of the aorta; we only mention these two extremes to show that the mechanic disturbances in the valves and orifices of the heart-pump cannot be altogether left out of consideration. Riegel* was quite right when he emphasized these matters in his monograph on "Chronic Diseases of the Heart Muscle and Their Treatment."

The question is not so much "whether," as "when and how much," exercise should be prescribed, and this question must be decided by actual trial. In judging the individual case it must be remembered that the heart is constantly in a condition of impaired efficiency as compared with the normal, and this condition can be improved only by avoiding every kind of overexertion. If that can be done, the strength may be increased by improving the nutrition of the heart, and thus gradually increasing the activity of the muscle-fibers.

In carrying out the exercises designed to accomplish this end care is necessary to see that the number of cardiac contractions does not exceed a certain maximum; in other words, the period of rest or diastole must not be shortened.

Liebermeister† suggests that a patient who has been ordered to exercise for the purpose of strengthening his heart, and who perhaps does not sufficiently heed the subjective sensations of palpitation and increased need of air, be enjoined to rest as soon as he finds that his pulse exceeds 90 or at most 100 beats in a minute.

This is a very good suggestion, particularly as a patient who is constantly observing his pulse is by that very act forced to go slowly.

Long ago certain eminent physicians (Stokes, Traube) taught that the question of exercise must be considered in the treatment of heart patients; but it is only lately that the procedure has been systematized and combined with other measures. These special procedures call for a fuller description.

Oertel's Method.‡—This consists:

1. In a diet suitable to the patient's condition.
2. In exercise suitable to the patient's condition (Terraincur).

* "Verhandlungen des Congresses für innere Medicin," Bd. vii (1888), S. 67.

† "Krankheiten der Brustorgane," Bd. iv der Vorlesungen, Leipzig, F. C. W. Vogel, 1891, S. 425.

‡ Oertel, "Therapie der Kreislaufstörungen," v. Ziemssen's "Handbuch der allgemeinen Therapie," Bd. iv, 1.–4. Auflage, 1884–1891. "Therapeutische Monatshefte," 1887 and 1888. "Verhandlungen des Congresses für innere Medicin," Bd. vii (1888). v. Leyden's "Handbuch der Ernahrungstherapie und Diätetik," Bd. ii, 1, S. 55 ff. J. Bauer, "Allgemeine Behandlung der Kreislaufstörungen," Pentzold-Stintzing's "Handbuch," Bd. iii.

In regard to the diet of heart patients, Oertel starts out with a theory which has only been partly substantiated. He assumes that the blood is diluted and its total mass is increased.

The latter has never been proved (compare p. 98), and the degree of dilution of the blood in low grades of cardiac insufficiency is a question that must be decided in each individual case. By far the greater number of investigations were made on hospital patients in an advanced stage of heart disease, whose nutrition probably was not good in the first place.

Oertel's object is to diminish the quantity of blood and at the same time to increase its concentration.

In addition to this, he seeks not only to reduce the hydremia, but also to diminish the percentage of fat and to raise the percentage of albumin in the body.

Since obesity occurs only in a certain proportion, and, when all is said, a small proportion of heart patients, Oertel's treatment must be limited to that small proportion.

This was clearly stated by Oertel himself from the very beginning; but the point is frequently overlooked, and the criticism brought to bear on his teachings has, therefore, not always been quite fair.

For the purpose of defining the indications for interference in an individual case Oertel employs the procedure which he has called "difference-determination" (Differenzbestimmung).

The patient continues to live exactly as he had been in the habit of living for two days, during which time the fluid ingested and the urine excreted are accurately measured. During the succeeding two days the quantity of fluid ingested is considerably diminished, while the amount of solid food remains the same; the ingested fluid and that excreted by the kidneys is, of course, measured as before.

The calculation is made by subtracting the smaller number from the larger, and the result is indicated by prefixing the sign plus or minus. For example:

First and second day:

Fluid ingested, 8000 grams; urine excreted, 5000 grams. Result, — 3000 grams.

Third and fourth day:

Fluid ingested, 3000 grams; urine excreted, 4000 grams. Result, + 1000 grams.

The question is whether the difference-determination is a sufficiently accurate indication of the amount of water contained in the human body?

First in regard to the quantity ingested?

Oertel uses the figures contained in Pettenkofer and Voit's work on metabolism.* To this there is no objection. With one exception the experiments are performed on the same individual, and I shall, therefore, confine myself to the figures obtained from that individual.

First we must inquire whether it is justifiable, in calculating the amount of water ingested, to neglect the water contained in the solid food. The answer to this question will be found in the adjoining table, which I have constructed from the numbers contained in the original.

It appears from this table that the figures which indicate the percentage of water in the solid food are so nearly constant that, for purely practical purposes, such as Oertel had in mind, they may be excluded; for the total exclusion of nitrogen from the diet, as in experiment XII, need not be considered in the present discussion:

* "Untersuchungen über den Stoffverbrauch des normalen Menschen," "Zeitschrift für Biologie," Bd. ii (1866), S. 459 ff.

	Number of Experiment.								
	V	VI	VII	VIII	IX	X	XI	XII	XIV
Total quantity of fluid ingested...	2066	2386	1852	2692	2267	3705	3683	1475	1770
Proportion of the above contained in the solid food.	333	359	374	332	352	676	656	68	318
Amount of water contained in the solid food expressed in percentage of the whole	16.1	15.0	20.2	12.3	15.5	18.3	17.8	4.6	17.7
	Ordinary Diet, Resting.			Ordinary Diet, Working.		Albuminous Diet, Resting.		Non-Nitrogenous Diet, Resting.	Ordinary Diet taken in two meals, Resting.

The same thing applies to the quantity of water contained in the feces when the output is calculated.

But what about the amount of water eliminated through the skin and lungs as compared with the water excreted through the kidneys?

I give an analysis of the experiments performed by Pettenkofer-Voit. In this table the amount excreted through the kidneys is taken as the standard (100), and the water eliminated through other organs of the body, with the exception of that which is lost in the feces, is compared with this standard. The table includes the starving experiments, which were omitted from the preceding:

	Number of Experiment.											
	I	III	IV	V	VI	VII	VIII	IX	X	XI	XII	XIV
Quantity of urine	100	100	100	100	100	100	100	100	100	100	100	100
Quantity of water eliminated through the skin and lungs.	116	94.3	283	64.8	95.6	73.5	183	118	59	55	111	97
	Starvation, Resting.		Starvation, Working.	Ordinary Diet, Resting.			Ordinary Diet, Working.		Albuminous Diet, Resting.		Non-Nitrogenous Diet, Resting.	Ordinary Diet taken in two meals, Resting.

Here we find considerable differences in the figures. The greatest difference is observed on comparing the resting with the working body. This is only natural, since the evaporation of water through the lungs and skin is much more active when the individual is working than when he is at rest.

The character of the diet is not without considerable influence. On an albuminous diet much more water is excreted by the kidneys than by the skin and lungs. It is possible that in these experiments the large quantity of beer, 2.76 liters per day, is chiefly responsible for the increased amount of water excreted through the kidneys.

But aside from these fluctuations, which are due to recognizable causes, we observe other differences too great to be neglected and for which no cause can be discovered (for example, experiments V and VI).

Oertel* gives a table showing his difference-determinations on the man who was the subject of the Pettenkofer-Voit experiments. I have calculated the difference-determinations from the figures given in a somewhat different form, taking as a basis the actual percentage of water in the liquid ingested and of the urine. In experiment IV Oertel made a mistake in calculation, but otherwise the differences are very small, as may be taken for granted.

Number of Experiment.	Quantity of Water Ingested	Quantity of Urine	Difference between the Two.	Difference Expressed in Percentage.	Diet and Occupation.
	in twenty-four hours.				
I.....	1028	1147	+ 119	+ 11.2	Starvation, resting.
III.....	987	863	— 124	— 12.6	
IV.....	1978	746	— 1232	— 62.3	Starvation, working.
V.....	1683	1279	— 404	— 24.0	Ordinary diet, resting.
VI.....	2025	1056	— 969	— 47.9	
VII.....	1479	1303	— 176	— 11.9	
VIII.....	2360	1116	— 1244	— 52.7	Ordinary diet, working.
IX.....	1905	1194	— 711	— 37.3	
X.....	3129	1882	— 1247	— 39.6	Albuminous diet, resting.
XI.....	3027	2205	— 822	— 27.2	
XII.....	1407	832	— 575	— 40.9	Non-nitrogenous diet, resting.
XIV.....	1351	1103	— 248	— 18.4	Ordinary diet taken in two meals, resting.

The environment of the subject was practically uniform as long as each experiment lasted. On the individual days of the experimental period the diet was practically the same; changes in the quantity of fluid ingested are indicated in the table.

In spite of this, enormous differences are observed in the results of the individual days of the same experimental period.

Take, for example, experiments V-VII. Here the difference-determinations, completed after Oertel, show a variation of more than 400 per cent, while the difference in the quantity of water ingested is only 25 per cent. I believe any one who will examine the figures impartially will be convinced that:

In the case of a healthy individual Oertel's difference-determinations are not applicable; they are absolutely valueless for determining the quantity of water contained in the body. Hence the standard [mean—Ed.] relation laid down by Oertel between the urine evacuated and the fluid ingested, namely from 20 to 30 per cent. less urine than ingested fluid, which he deduced from numerous experiments on healthy individuals, is of no value either.

* "Handbuch," S. 65 der 4. Auflage.

How do the figures apply in cases of cardiac insufficiency?

Oertel* divides them into three groups:

1. Cases in which he assumes the existence of serous plethora. This he asserts is associated with the accumulation of large quantities of water in the tissues, even in the absence of true cutaneous edema. "The patients present a spongy, bloated appearance; the complexion is pale and sallow; the visible mucous membranes are pale or present a faintly livid redness." The condition is due to the copious ingestion of solid and liquid food, especially the latter (including even water). "If there is no actual disease of the kidneys and the strength of the heart is not too greatly impaired, restriction of the fluid is followed by a considerable increase in the urinary secretion."

The table (see below) shows that this was true in four cases reported by Oertel. Unfortunately, the patient's body-weight is not given; for it would indicate whether he was in the habit of drinking large quantities of fluid.

Number of Experiment, Age and Sex of the Patient, Diagnosis.	I. Before Restricting Ingestion of Fluid.			II. During the Restriction of Fluid Ingested.			Number of Days under Observation.	
	Daily Ingestion of Fluid.	Daily Excretion of Urine.	More or Less Urine than Fluid Ingested.	Daily Ingestion of Fluid.	Daily Excretion of Urine.	More or Less Urine than Fluid Ingested.		
	Cubic Centimeters.			Cubic Centimeters.			I.	II.
1. Female, aged fifty-two. Marked scoliosis and cardiac hypertrophy, failure of compensation.	1449	1075	— 374 Maximum fluctuations. — 70 — 767	657	809	+ 152 Maximum fluctuations. + 60 + 285	6	15
2. Male, aged fifty. Insufficiency of the aortic valves; aneurysmal dilatation of the arch.	1468	1220	— 248 Maximum fluctuations. — 150 — 370	750	956	+ 206 Maximum fluctuations. + 70 + 400	5	27
3. Male, aged sixty. Emphysema, cardiac hypertrophy, pronounced stasis.	1840	1530	— 310 Maximum fluctuations. — 60 — 560	850	1188	+ 338 Maximum fluctuations. + 200 + 600	2	4
4. Female, aged sixty-eight. Obesity; fatty heart, fatty degeneration of the heart muscle; stasis; edema.	1568	1264	— 304 Maximum fluctuations. — 230 — 280	846	1088	+ 242 Maximum fluctuations. + 49 + 539	2	8

In fact, this does not appear to have been the case. It would also enable us to determine the effect on the body-weight of restricting the quantity of fluid ingested.

II. Cases with considerable damage to the circulatory apparatus —valvular lesions and imperfect compensation—in which, however, the functional condition of the kidneys is fairly good and there has been

* Compare "Handbuch," p. 71 *et seq.*

no excessive ingestion of fluid. This group also includes cases of valvular lesions with beginning disturbance of compensation, in which the venous apparatus is decidedly overloaded, as indicated by the stasis, but in which there is no increase of the quantity of water in the body.

"Restricting the quantity of fluid ingested is not followed by any increase in the excretion of urine; on the contrary, the *minus* of the urine is not infrequently relatively large."

I subjoin a short review of the facts in the case reported by Oertel (No. 3). It is a case of mitral insufficiency "with rather advanced disturbance of compensation," in a man forty-eight years of age.

I. March 1–4.			II. April 6–10.			III. May 11–14.			IV. June 6–8.			V. June 23–25.		
Fluid.	Urine.	More or Less Urine.	Fluid.	Urine.	More or Less Urine.	Fluid.	Urine.	More or Less Urine.	Fluid.	Urine.	More or Less Urine.	Fluid.	Urine.	More or Less Urine.
Cubic Centimeters.			Cubic Centimeters.			Cubic Centimeters.			Cubic Centimeters.			Cubic Centimeters.		
1000	928	— 72	1080	1004	— 76	995	1007	+ 12	1300	873	—427	1250	983	—267
Maximum fluctuations on the individual days of the series.		+ 50 —290			+ 90 —520			+ 130 — 60			—160 —580			+ 130 —600

The difference between these cases and those of the first group is that the urine column constantly shows a deficit when the individual portions of the experimental period are viewed as a whole. The diminution is greater during the period when more fluid is ingested, but does not appear to bear any direct relation to the ingestion of fluid.

As the body-weight is not given, it is useless to speculate on the cause. The ingestion of fluid was by no means large, the average for the entire series being only 1125 c.c. a day; nor is it possible to estimate the effect of the ingestion of fluid on the patient because the body-weight is not known.

III. "Owing to severe circulatory disturbances, the result of various diseases of the heart muscle and their complications with valvular lesions, disturbances of compensation and diseases of the kidneys with progressive diminution of the urinary secretion, there has been a copious effusion of serous fluid.

"Depending on the condition of the kidneys and the strength of the heart, restriction of the quantity of fluid ingested in these cases is followed either by an increase in the amount of urine excreted, which may be quite marked, or by a proportionate reduction, the quantity of urine indicating more or less actual deficit."

The following table (p. 154) shows the calculation based on Oertel's figures. It shows considerable differences in individual cases. Nevertheless it is doubtful whether an individual who evacuates more than

two liters of urine, as in case 1, can be said to be suffering from cardiac failure in the ordinary sense of the word, even though there be considerable edema. It is probable that in this case we have to deal with the special form of cardiac disturbance which has been described on another page (p. 69 *et seq.*). This is further shown by the fact that the patient, about five weeks after treatment had been begun, "climbed a fairly high mountain in an hour without fatigue," as Oertel* reports. The two other cases, in spite of the fact that the fluid was restricted, constantly show a certain diminution in the quantity of urine excreted.

I cannot see any advantage in adhering to this classification. None of the groups is susceptible of being sharply defined, so that the classification can be of no advantage in actual practice.

All things considered, it must be conceded that restricting the ingestion of fluid may have a favorable effect on cardiac weakness, but the question must be tested in every individual case. It is shown by the marked fluctuations in the quantity of urine excreted on different days—I have indicated the maximum amounts in the tables—that certain other factors of which we know nothing whatever are also operative. It is well known that such variations in the excretion of the urine occur in cardiac weakness even in the absence of any kind of treatment.

Number of Experiment; Age and Sex of the Patient; Diagnosis.	I. Before the Restriction of Fluid.			II. During the Restriction of Fluid.			Number of Days under Observation.	
	Amount of Fluid Ingested per Day.	Urinary Excretion per Day.	Excess or Deficit of Urine.	Amount of Fluid Ingested per Day.	Urinary Excretion per Day.	Excess or Deficit of Urine.		
	Cubic Centimeters.			Cubic Centimeters.			I.	II.
1. Male, aged sixty-four years. Insufficiency; hypertrophy and dilatation; fatty degeneration of the heart muscle; moderate obesity; marked edema of the lower extremities and of the scrotum.	2265	2125	— 140 Maximum fluctuations on the individual days of the period.	737	1587	+ 850 Maximum fluctuations on the individual days of the period. + 460 + 1250	1	31
2. Male, aged fifty-four years. Chronic myocarditis; hypostatic albuminuria (Stauungsalbuminurie); edema.	2000	1315	— 685 Maximum fluctuations on the individual days of the period. — 540 — 830	893	470	— 423 Maximum fluctuations on the individual days of the period. — 200 — 350	2	6
3. Male, aged thirty-six years. Insufficiency of the mitral valve; complete rupture of compensation; albuminuria, edema.	1200	420	— 780 Maximum fluctuations on the individual days of the period. + 0 — 0	871	394	— 477 Maximum fluctuations on the individual days of the period. — 310 — 450	2	5

* *Loc. cit.*, p. 83.

There is no doubt that Oertel deserves great credit for having pointed out the importance of controlling the ingestion of fluid in the treatment of those suffering from heart weakness. But his theories may be allowed to rest on their own foundations.

It is probable that we shall soon gain some fresh insight into the question of utilizing the introduction of water into the body for therapeutic purposes.

Working with the methods of the present day, Dennig* published a series of experiments which show what a marked effect can be produced by withdrawing considerable quantities of water from the body. A noteworthy feature is the marked difference observed in this respect in different individuals.

So far as one may judge by these recent experiments and by older ones of mine† carried out with inadequate methods, it is absolutely necessary to exercise the most careful control in withdrawing large quantities of fluid. Unless plenty of water is stored up in the tissues, one cannot be sure that the procedure will not be followed by injurious results. If there is a deficiency, there is some danger of causing more or less tissue loss and, not least of all, loss of organized albumin.

Oertel also insists that, if the heart patient is overburdened with fat, he should be relieved of a corresponding amount. Such a procedure is undoubtedly perfectly justifiable, as an excess of body fat overloads the heart; but it is impossible to say how much fat is removed from the surface or the tissue of the heart during a denutrition cure. Nevertheless, while the removal of fat from the heart itself would be a very great advantage, reduction of the adipose tissue of the entire body in itself is also most beneficial.

It is unnecessary to enter in detail into Oertel's explanations of his method. Our knowledge of metabolism and the manner in which it may be influenced for therapeutic purposes has made considerable progress since his day. The stereotyped methods of Banting, Ebstein, and the like can no longer be followed in ordering a denutrition cure: every individual case must be approached with a full understanding of the entire question and the realization that he represents a special problem.

This problem is the more difficult in heart patients because, while the body-fat must be reduced, the nitrogen-balance must be preserved and, indeed, the percentage of albumin increased, if necessary. That this is possible has been shown by investigations.‡

We now come to the other part of Oertel's method: exercises suited to the conditions of the heart patient—"Terraincur."

The data necessary for judging an individual case are obtained by noting the mediate and immediate influence on the work of the heart of muscular exercise.

It is not necessary to mention each detail of the investigation; it will be enough to refer to the result as a whole. We know that muscular exercise causes an acceleration of the heart-beat and usually also an increase of the arterial pressure, which is accompanied by strengthening of the respiration. The combined effect of these factors on the heart

*"Die Bedeutung der Wasserzufuhr für den Stoffwechsel und die Ernährung des Menschen," "Zeitschrift für diätetische und physikalische Therapie," Bd. i (1898), S. 282 ff. und Bd. ii (1899), S. 292 ff.

†"Ueber das Schroth'sche Heilverfahren," "Deutsches Archiv für klinische Medicin," Bd. i (1865), S. 196 ff.

‡Compare Dapper, "Ueber den Stoffwechsel bei Entfettungscuren," in "Beiträge zur Lehre vom Stoffwechsel des gesunden und kranken Menschen," by Carl v. Noorden, Berlin, Hirschwald, 1894, Heft 2, S. 65 ff. An excellent review of the present state of our knowledge is found in Dr. P. S. Richter's article: "Ueber Entfettungscuren," "Zeitschrift für diätetische und physikalische Therapie," Bd. i, S. 300 ff., 1898.

muscle may be favorable or unfavorable. It is favorable if the demands on the heart are not great enough to cause fatigue; unfavorable, if the demands exceed that limit. Exercise enables the heart muscle in a great measure to adapt itself to an increased demand; by gradual training an individual who has been suitably prepared is enabled to do hard muscular work, as, for example, mountain-climbing, with ease. The training affects the skeletal muscles as well as the heart and the respiration. An individual also learns to adapt his breathing to the muscular demands. Experience has shown that even in highly trained athletes the heart may give out if it is overtaxed. The experiences of Allbutt and his guide have already been described.

These conditions apply quite as much to a heart which is naturally strong as to one that is weak. In the case of the latter it is obvious that a much smaller demand suffices to bring on fatigue, and cardiac weakness often becomes so marked that the organ is unable to meet the extra demands of even a very slight degree of muscular exertion.*

From all this it appears that the principles underlying Oertel's teaching are unassailable; but when they are applied to the treatment of heart patients, medical supervision is absolutely indispensable.

The arrangement of Terraincur resorts after Oertel's suggestions, and his Terraincur charts, which contain all the conditions to be considered, are in themselves most excellent; unfortunately, human nature was not sufficiently taken into account in these arrangements. The heart patient is apt to get the impression from the colors and figures placed along the paths that the treatment is purely mechanic, a kind of ready-made apparatus that may be used by any one with equally good results,—and so he consults the chart more frequently than the physician. It must also be remembered that Oertel originally recommended his procedure only for a definite form of heart weakness. The effects of excessive beer-drinking, which are so wide-spread in Munich, the so-called "beer-heart," represent the foundation of this form of heart disease. There can be no doubt that in this condition a suitable amount of bodily exercise may be most useful, especially if the quantity of beer is at the same time greatly reduced. Many physicians are of the opinion that a heart which has been damaged by excessive drinking of beer and wine and excessive ingestion of food quite frequently recovers its functional power if it is protected against these injurious factors during a course of Terraincur. Heart weakness due to other causes should not be treated by the Oertel method. A Terraincur is a very questionable procedure, which, unless properly supervised, always involves the danger of heart fatigue; and how is it possible to exercise adequate supervision over the patient? In proportion as the heart weakness is more pronounced, greater caution is required in attempting to improve the condition by increased muscular exercise, especially mountain-climbing.

The so-called **Nauheim† treatment,** elaborated in the main by Augustus and Theodore Schott, must be regarded as a special method.

*In regard to this entire question, see pp. 20, 21, and 40 *et seq.*

†There is no exhaustive treatise on the subject. The most important papers dealing with the principles of the method are: Augustus Schott, "Die Wirkungen der Bäder auf das Herz," "Berliner klinische Wochenschrift," 1880, No. 25. "Beiträge zur physikalischen Diagnostik des Herzens," "Centralblatt für die medicinischen Wissenschaften," 1881, No. 23–26. "Die Bedeutung der Gymnastik für Diagnose, Prognose und Therapie der Herzkrankheiten," "Zeitschrift für Therapie" (Josef Weiss), 1885, No. 20, 21. "Zur Therapie der chronischen Herzkrankheiten," "Berliner klinische Wochenschrift," 1883, No. 33. Augustus and Theodore Schott, "Die Nauheimer Sprudel- und Sprudelstrombäder," *ibid.*, 1884, No. 19. Theodore Schott, "Die Behandlung der chronischen Herzkrankheiten," Berlin, Eug. Grosser, 1887. "Ueber Herzneurosen," Eulenburg's "Realencyklopädie," 2. Auflage, 1892. [See also, besides, other recent articles: Baldwin, Wm. W., "Some Personal Observations and Experiences of the Schott Treatment of Heart Disease," "Medical Record," February 13, 1904. James M. Anders, "The Schott Method of Treating Diseases of the Heart

The Schott method of treatment consists in the use of brine baths, with or without free carbonic dioxid, combined with special muscular exercises. The effect of the two procedures is the same. How they act is not definitely known, and the attempts to explain the rationale of the procedure have, in my opinion, not proved successful. I therefore regard the method as a purely empirical one and present it in that light.

The object of the procedure is to enable a dilated heart that is unable to expel all its contents to empty itself completely. At first this object is only partially attained; but as the treatment goes on, the ventricle empties itself more and more completely. The nutrition of the heart muscle itself is improved as the dilatation subsides and the circulation becomes more active.

The crux of the question is whether this assumed effect on the heart can be positively proved. If that is the case, then the therapeutic results are quite comprehensible.

I can personally testify that the Schott method of gymnastics is capable in a short time of considerably diminishing the cardiac dulness and at the same time strengthening the pulse. These results practically admit of no other interpretation but that given by Augustus Schott, the originator of the method; namely, that the overdistended heart is relieved of its burden.

I will give a short account of a case which convinced me that the facts are correct. *À priori* it did not seem to me probable that a well-marked cardiac dilatation could in a short time undergo noticeable diminution, and I wished to have ocular proof. While I was in Nauheim Dr. Theodore Schott very kindly gave me an opportunity to examine certain patients for myself before and after the resistance exercises. Among these patients was the following:

A man, thirty-five years of age, a patient of my own, with a high degree of mitral insufficiency and all the sequelæ of that condition. At the time of examination there was marked disturbance of compensation and pronounced cardiac weakness.

According to the results of my percussion the transverse diameter of the cardiac dulness at the level of the nipple was 27.5 centimeters, while the longitudinal diameter (in the median line) from the upper limit to the xiphoid was 16 centimeters.

After the resistance exercises, which lasted half an hour including the rests, these two diameters diminished; the transverse was only 18 centimeters and the longitudinal, 13 centimeters. In other words, the reduction amounted to 9.5 centimeters in the transverse, and 3 centimeters in the longitudinal, direction.

The apex-beat had moved 4 centimeters toward the median line. The pulmonary-hepatic border was, of course, determined also and was found to be higher after the resistance exercises.

Fig. 24 on p. 158 is taken from a reduced photograph of my drawing (which was a tracing of the lines on the patient's chest).

I have had no opportunity of observing the immediate effect of the baths on the heart; but there is no reason to doubt the Schott brothers' statements, and, besides, they have been confirmed by prominent physicians. Grainger Stewart and Dr. Holman, also Saundby,* observed

and Blood-vessels," "Journal of the American Medical Association," January 14, 1905, pp. 116–124. Thorne, Bezly, "The Schott Methods of the Treatment of Chronic Diseases of the Heart, with an Account of the Therapeutic Exercises," Fifth Edition, 1906. Blakiston's Son & Co.—Ed.]

*Compare Robert Saundby, "The Nauheim (Schott) Treatment of Heart Disease," "British Medical Journal," November 2, 1895, with tracings of the heart dulness before and after the bath and the exercises, and sphygmograms. H. Newton Heineman (New York), "Die physikalische Behandlung der chronischen Herzkrankheiten (nach Schott)," "Deutsche medicinische Wochenschrift," Jahrgang 1896, S. 525 ff. The author studied the procedure in Nauheim and afterward observed the effect of the exercises in v. Leyden's clinic.

improvement of all diameters; a reduction of one English inch, equal to 2.5 centimeters.

I shall not describe the conditions at the Nauheim baths and the procedure as it is practised there, but I shall give a detailed description of the treatment as it may be employed anywhere with artificially prepared baths instead of the natural springs.

It must be remembered that Augustus Schott, from the very beginning, took it for granted that artificial baths could be substituted for the natural springs and even formulated the necessary instructions.

The treatment is to be begun with brine baths containing 1 per cent. of sodium chlorid and 0.2 per cent. of calcium chlorid. Gradually these percentages are increased, with due regard to individual conditions, up to a maximum of 3 per cent. sodium chlorid and 0.25 per cent. of calcium chlorid.

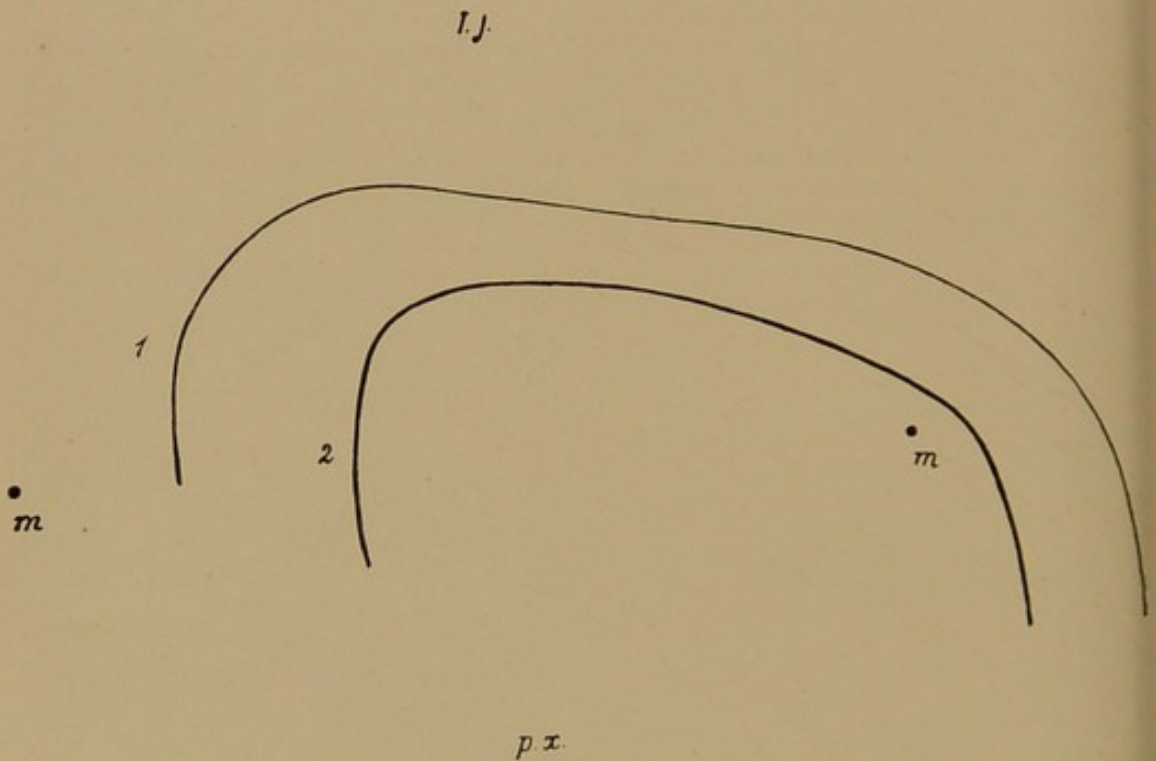


FIG. 24.

A. Schott* gives the following rules in regard to temperature, duration of the bath, etc.

At the beginning of the treatment the duration should never be more than ten minutes; if the patient is weak, even five minutes may be sufficient. Rheumatic and anemic patients and those who cannot stand much cold should begin with baths at a temperature of 27° R. (92.7° F.). When the heart is weak it cannot be depended upon to neutralize the peripheral loss of heat by increasing the force of the circulation. When the pulse is weak, the skin cold, and the patient is in a reduced state of health from malnutrition, the reduction of only 1° R. (2.2° F.) in the temperature of the bath is enough to cause chilliness, which may be harmful to the patient. After a bath that is too cold one may find that all the signs of cardiac disturbance and dilatation, rapidity, irregularity, and reduction in the size of the pulse, dyspnea, etc., have increased. Rheumatic patients, even with fairly strong hearts, should not begin the course of treatment with baths at a lower temperature than the above, because the tendency to peripheral loss of heat is marked in such patients and there is always danger that the blood may be forced into the already damaged vessels and tissues, the *partes minoris resistentiæ*. For this reason the transition to colder temperatures

*"Zur Therapie der chronischen Herzkrankheiten," "Berliner klinische Wochenschrift," 1885, No. 33.

should be made, on the average, somewhat later in rheumatic than in heart patients. On the other hand, temperatures above 27° R. (92.7° F.) are practically never, and temperatures above 28° R. (95° F.) in my experience never indicated except in those cases in which the pulse and arterial tension are abnormally high from the beginning. Even in such cases, while higher temperatures are well borne, they unfortunately do not act as a heart tonic.

Instead of increasing the temperature of the bath it is better to shorten the duration. The patient is advised to lie perfectly quiet during the first half minute after entering the bath of the prescribed temperature, even if he finds the water somewhat cool, especially when it accidentally splashes against him, until a feeling of perfect comfort gradually comes over him. But if this feeling of perfect comfort is not experienced after half a minute and the slight feeling of chilliness persists, the bath water should be warmed until the unpleasant sensations disappear.

The temperature of the bath should never be reduced more than ½ R. (1.1° F.) at a time.

A second chill must be avoided as much as possible.

If the patient is warm when entering or rapidly warms up in the bath, he must not be allowed to get chilly again; if he does become chilly, it is a sign that the duration is too great and must be reduced in the subsequent baths.

As the favorable effects of the baths gradually increase, their strength must be increased both by reducing the temperature of the water and by increasing the concentration of the salts. When this stage has been reached and the weakened heart has gained considerably in strength, free carbon dioxid is added to the bath.

In the artificial baths this is done as follows: Commercial bicarbonate of sodium and crude hydrochloric acid (42 per cent.) are added in equal quantities by weight to the bath water. This leaves a slight excess of the alkali, which is useful for protecting the metal bath-tub and at the same time the patient's skin.

In the beginning 100 grams of each should be added to a bath of about 250 liters of water, and this quantity may be gradually increased until 1500 grams of each ingredient are added (250 liters or quarts are equal to about 62 gallons; 100 grams are equal to about one-fifth of a pound; 1500 grams, about 3 pounds).

The bicarbonate of soda is first dissolved and poured into the bath water, while the hydrochloric acid is not added until everything else is ready. The acid should be poured out under the water, holding the mouth of the bottle over the bottom of the tub and gently moving it about in all directions as the acid escapes. When the bath is to be prepared in a hurry, the mouth of the bottle is held immediately below the level of the water and moved rapidly to and fro without splashing.

The layer of carbon dioxid which forms above the surface of the water must be removed by fanning, the window being open.

The minimum temperature of any bath is 22° R. (81.5° F.). The maximum duration is twenty minutes.

"At the beginning of the treatment most patients require an occasional day of rest on which the bath is omitted, sometimes after the first, but usually not until after the second, bath. After that the number of baths given in succession without an interval of rest can soon be increased."

As a matter of precaution the bath should always be omitted on one day of the week.

The patient's general condition and the condition of the heart must be kept constantly under accurate supervision; the effect of the bath determines the temperature and duration of the next one. Schott lays great stress on the subjective sensations of patients who are receiving the bath treatment: "If a patient feels tired for one or two hours after the first bath and then recovers completely, he may be given the same bath on the following day; but on no account may the strength of the bath or its duration be increased. If the fatigue lasts longer than two hours, the bath should be omitted on the following day. This principle should be observed during the entire course of treatment. When a series of baths of increasing strengths have been prescribed, each bath must be regarded as a task which the patient must be able to accomplish without any subsequent fatigue before he is allowed to take up the next. If he does become unduly fatigued, the course is begun over again after a day's rest with a bath of slightly diminished strength."

The strength of the bath should be increased as rapidly as possible until a distinct effect is obtained, carefully avoiding any excess. "Unless the invigorating effect on the heart is noted immediately after the bath during the beginning of the treatment, the bath is not sufficiently strong." The pulse ought to become slower and stronger, and a distinct reduction in the size of the cardiac dulness ought to be demonstrated immediately after the bath. This reduction in the size of the dulness should always be the object aimed at, even if it does not persist the entire day.

Schott quite rightly insists that a course of Nauheim treatment requires a considerable length of time.

I have described this part of the procedure in detail because it can always be carried out with patients in ordinarily comfortable circumstances. Unfortunately this cannot be said of the second part of the procedure, the resistance exercises. With regard to these I shall confine myself to fundamental principles.

A. Schott says the main point about the resistance exercises is that the movements must be performed very slowly, although with some force. In order to produce this simultaneous slowing and strengthening, resistance is necessary, which may be supplied either by the patient himself inhibiting the movements by exerting the antagonistic muscles or by another person—a so-called gymnast,—after the manner of the Swedish movements. Definite groups of muscles, namely, such as are coördinated for the movements of the extremities, are brought into action in a certain order.

The most important rule for the patient to observe is to overcome the resistance slowly and uniformly without in the least hurrying the respiration, so that he always has enough wind to speak without difficulty. The faintest embarrassment of the breathing should be a signal for resting, and in the same way, if the heart-beats increase in frequency, the patient must cease the exercise at once.

A hot-water bag is used by the Schott brothers to produce a temporary effect on a greatly dilated and feebly contracting heart. Rubber bags are filled with hot water ranging in temperature up to 60° R. (169° F.), and the entire chest wall, especially the precordial region, and sometimes also the back, are subjected to a gentle tapping with the hot-water bag until the patient becomes conscious of a distinct sensation of heat. This application of heat is said to be particularly efficacious in cases of sudden heart weakness.

These are the main points. It ought to be mentioned that mountain-climbing, after Oertel, properly belongs to the mechanic methods of treating heart disease. After the heart has become considerably strengthened, good results are obtained by cautiously prescribing a certain amount of mountain-climbing.

No special diet is prescribed, as it must be regulated according to individual conditions.

From all I have seen, Schott's method of treatment represents a distinct advance in the treatment of cardiac weakness and deserves to be more generally known among physicians than it is at the present time.

Instead of the resistance being supplied by a gymnast or the patient's own inhibition, which requires a good deal of training and is not always easy to learn, Zander's apparatus may be used for the purpose.* An obvious advantage of these machines is that the degree of resistance to be overcome can be accurately gauged. It is hardly necessary to say that careful medical guidance and supervision are indispensable. Of course, the use of Zander's machines is out of the question anywhere but in large cities or at popular bathing resorts.

The therapeutic value of muscle massage is not rated very high. It should be remembered, however, that since the skin and the subcutaneous connective tissue are also acted upon, massage tends to set the stagnating mass of blood with its accumulated red blood-cells in motion

* See v. Reyher, "Ueber Herzmassage und Herzgymnastik," "Zeitschrift für diätetische und physikalische Therapie," Bd. i (1898), S. 197 ff.

and forces them at least temporarily to take more part in the general circulation.

Popischil* describes a carefully systematized hydrotherapeutic method of treating the heart, but it does not seem to me that the method has been sufficiently elaborated to meet the demands of general practice.

Drugs.—What is the position of pharmacotherapy in the treatment of cardiac weakness? That it does not play a subordinate part we owe to *digitalis*. Powerful as the remedy is, however, it also has its limitations. He who would use digitalis must have an accurate knowledge of the drug and all its limitations and must never forget that the friend may become a foe. I know of but one indication that vouches with almost absolute certainty for the therapeutic action of digitalis. This indication is given when the following conditions are present:

1. The diseased heart contains fibers of unequal functional capacity and is, therefore, somewhat wasteful of its energy. This is the form of heart weakness that I have described in detail elsewhere.†

2. The heart must not be too greatly fatigued; it must still retain a certain degree of strength, indicated by the fact that, although most of the beats are feeble, an occasional vigorous contraction is present.

Under these circumstances digitalis acts chiefly on the pneumogastric nerve. Inhibition of the pneumogastric regulates the wasteful action of the heart, which exhausts individual heart-fibers without increasing the efficiency of the muscle as a whole; for the weaker fibers are inhibited and allowed to rest, while the stronger fibers alone are called into activity and at the same time are given sufficient time to recuperate. Thus the vicious circle which I have attempted to describe above is interrupted and the heart gains fresh strength through its own exertions because it is provided with sufficient blood for its proper nutrition.

It is possible that the stimulating action on the muscle-fibers of the heart, which is attributed to digitalis, is also in part responsible for the result. In that case we must assume that the inhibiting effect does not influence the weaker fibers, but for the stronger fibers digitalis becomes an additional spur.

Let it not be thought that I take exception to the employment of digitalis in other forms of cardiac weakness. Good results may be obtained in these also. So conscientious an observer as Penzoldt‡ reports, "I have seen cases with a pulse of 40 or even 30 to the minute which showed a general improvement, not only without a decrease, but indeed with an increase, of the pulse frequency." I merely wish to say that in these cases one cannot rely on the remedy's proving efficacious.

My experiences at the bedside have led me to desist from the remedy altogether in cases of protracted heart weakness whenever the pulse becomes small, regular, exceedingly feeble, and frequent. It seems to me this is an indication that the heart is merely capable of performing the amount of work necessary for the continuance of life. In this condition its equilibrium is so unstable that the slightest, let us say, alteration suffices to disturb it and to bring on serious, if not fatal, consequences.

* "Hydrotherapie bei organischen Herzkrankheiten," "Deutsche Medicinalzeitung," 1895, S. 581 ff.; also contains the discussion which followed his paper in the Balneologische Gesellschaft.

† See pages 69 and 73 and observations 4 and 7.

‡ "Lehrbuch der klinischen Arzneibehandlung," Fischer, Jena (1897), 4. Auflage, S. 193.

One is naturally inclined to try to bring down the pulse with digitalis; I at least have never succeeded in doing so.

It is difficult to give a satisfactory explanation of the manner in which the effect of digitalis on the blood-vessels fits in with the action of the remedy as a whole in cases of heart weakness. If a general constriction of the arterial pathway occurs, the resistance to the left ventricle must increase in proportion to the stenosis. This interferes with evacuation of the left heart. Judging from animal experiments, all that digitalis can do is to call out reserve strength and, since the exhausted heart has none at its disposal, it cannot be rendered capable of doing any more work. It may be suggested that obstruction to the blood in the distal arteries causes an increase of pressure at the beginning of the aorta, forcing more blood into the coronary arteries. But this is hardly an explanation, as it is doubtful whether the heart can be strengthened in this way. The dilatation and distention of the ventricle which must necessarily ensue if it cannot evacuate its contents properly on account of obstruction to the flow of blood, is probably a much more powerful factor than the problematic increase of the amount of blood in the coronary arteries.

Digitalis, when it is effective at all, is quite similar in its action to the mechanic methods of treatment. The indication is always the same: to stimulate the heart temporarily to perform more work for its own good. The advantage of digitalis is that it develops its full activity in a comparatively shorter time. As an offset it has the disadvantage that it can only be employed for a comparatively short period. It is not possible to keep up the use of the drug and thus maintain the curative effect indefinitely; for the therapeutic effect is soon followed by the toxic effect, which is destructive to the heart. Digitalis belongs to the substances which have a cumulative action—drugs which, even when given in small doses, accumulate within the body because elimination is difficult, and, after a certain limit has been exceeded, cause intoxication. Owing to this peculiarity the greatest caution is necessary in the administration of digitalis, the more so as we usually prescribe the crude drug, the leaves, which vary greatly with respect to the percentage of curative or noxious glucosids which they contain. The habitat of the plant, the year when the leaves were grown, the length of time they have been kept—all have an important bearing on the character of the drug. These variations probably explain the great difference of opinion in regard to the size of the dose. In Tübingen we rarely have to use more than a total quantity of 3 grams (45 grains), which we find quite sufficient to produce the full effect of the drug.

My custom is to give at most 2 grams (30 grains) during two or three successive days, followed by an additional gram (15 grains) on the succeeding day. I order the simple infusion without any adjuvant of any kind. At least two weeks and sometimes more are allowed to elapse before the drug is given again.

Occasionally digitalis is also given in pill form. A smaller quantity, a total of 1 to 1½ grams (15 to 22 grains), distributed uniformly over a period of three to five days, should be given. It is probable that more of the glucosids are dissolved and rendered absorbable by the digestive juices than by the hot water of the infusion. It is also possible that some of the digitoxin, which is not present in the infusion, is also absorbed in this way. The latter is a powerful tissue irritant and may be responsible for

the fact that leaves taken in substance are more apt to produce vomiting than the infusion.

It has been advised to give digitalis by the bowel in order to avoid as much as possible any irritation of the stomach; my experience of the method is not sufficient to enable me to give any opinion. Penzoldt says (*loc. cit.*, p. 197): "After a cleansing enema, about 30 c.c. of a strong infusion of digitalis, equivalent to 0.4 gm. (6 grains), should be injected at body temperature into the bowel, twice a day, the patient being instructed to retain it as long as possible. While this does not altogether prevent gastric symptoms, it does diminish their severity and delays their appearance." It would be better to add a little starch and a few drops of laudanum to the enema in order to moderate the effect of digitalis on the rectum. An infusion of 0.3 gm. (4½ grains) of the leaves to 10 c.c. (2½ drams) of percolate has also been tried by hypodermic injection, one Pravaz syringeful (1 c.c.) two or three times a day. Whether this can be kept up without causing abscesses and whether the method has any special advantage still remains to be determined.

The *acetum digitalis* has been dropped from our pharmacopeia. On the other hand, the formula for the preparation of *tincture of digitalis* has been retained. It is prepared by taking five parts of the freshly crushed herb and six parts of alcohol. [In the U. S. P., 1900, 10 per cent., one-third weaker than according to U. S. P. 1890. British Pharmacopœia, 1 part to 8. So eminent a pharmacologist as Cushny, and many experienced clinicians, consider the tincture the best preparation of digitalis in most cases. The belief that this preparation is inferior to some others, especially the infusion, seems to be due chiefly to the fact that the other preparations are usually prescribed in doses that contain a much larger amount of the drug than does the tincture; for example, the physician often prescribes the infusion because the tincture fails, and gives a tablespoonful at a dose. The corresponding dose of the tincture would be about 45 minims, usually very much more than the dose that failed to give satisfaction.—Ed.] Neither of these preparations is very reliable and should never be used except when the quality of the plant itself cannot be counted on.

The German pharmacopœia does not mention any of the substances that are regarded as the active agents of the mother plant,* nor has it been possible to induce the profession to adopt any one of these substances, great as the need is for an agent of uniform efficacy. The following have been and are no doubt still being tried:

1. *Digitoxinum crystallisatum Merck*, which is dispensed in tablets of ¼ mg. ($\frac{1}{250}$ grain). The dose is ½ mg. ($\frac{1}{125}$ grain) up to 2 mg. ($\frac{1}{30}$ grain) per day, when taken internally. The drug can also be given by enema in the following formula by Penzoldt:

℞. Digitoxini crystallisat. (Merck) ..... 0.01 (⅛ grain)
Alcohol ..... 10.00 (2½ drams)
Aq. dest ..... 200.00 (6 ounces).
M. S.—15 c.c. (½ ounce) of this solution in 100 c.c. (3 ounces) of water by enema, one to three times a day.

2. *Digitalinum verum Kiliani.*—Internally, 0.002 to 0.006 ($\frac{1}{30}$ to $\frac{1}{10}$ grain) up to 0.02 (⅓ grain) as a maximum per diem. The formula is:

℞. Digitalini ver. (Kiliani) ..... 0.02 (⅓ grain)
Alcohol ..... 10.00 (2½ drams)
Aq. dest ..... 70.00 (2 ounces)
Syrup. simplic ..... 20.00 (5 drams).
M. S.—One tablespoonful every one to three hours.

Both substances are said to be suitable for hypodermic administration, but the puncture is very apt to become the seat of inflammation, especially when digitoxin is used. [The editor has used this preparation in a series of cases, without finding any advantage over the tincture of digitalis.—Ed.]

Digitalis usually requires about thirty-six hours for the full development of its favorable effect. The pulse becomes slower, fuller, and more regular. If the urine was diminished in quantity, an increase takes place, sometimes amounting to a veritable flooding. Edema, if present, diminishes, especially edema of the skin and subcutaneous connective

* Compare Kobert, "Lehrbuch der Intoxicationen," Enke, Stuttgart, 1893, S. 678 ff.

tissue. Effusions into the peritoneal and pleural cavities also diminish, although more slowly than the cutaneous edema. The swelling of the liver becomes less, the appetite is not infrequently improved, and the dyspeptic symptoms subside. As a result of the improvement in the action of the heart, the accumulation of blood in the systemic veins disappears and the marked cyanosis of the skin and mucous membrane ceases.

If the heart is still in fairly good condition, a single administration of digitalis may be followed by improvement lasting for some time, provided the subsequent demands on the heart do not exceed its limit. In other cases the improvement lasts only a short time; a few weeks of improvement may, however, be counted upon in most cases. Again, the drug may fail almost altogether, in which case the prospect of recovery is very doubtful.

In regard to the cause for this failure of the drug to act, all that can be said in a general way is that it must be sought in the condition of the heart muscle. The muscle may be practically used up to begin with; or some injurious influence to which it is constantly exposed may permanently destroy its structure and thus gradually destroy its power. This is usually the case in the form of insufficiency following an infection, particularly septic infection. The point will have to be referred to again at length in connection with endocarditis. Another factor in the prognosis is the presence of bronchial catarrh, which is apt to develop when the heart is weak and is slightly improved when the organ is strengthened by the administration of digitalis. The bronchial catarrh does not subside altogether; it continues as an independent disease and thus greatly interferes with the recovery of the heart.

As the administration of digitalis must be limited in point of time, it is most important, if the case is to be treated intelligently, to take full advantage of the favorable period when the force of the heart has been improved by the use of the remedy.

Now is the time to resort to auxiliary methods, such as baths and exercises. Oertel and the Schott brothers have always insisted that the procedures which they recommend sometimes cannot be instituted until after digitalis has done its work.

The toxic symptoms of digitalis should be briefly mentioned. As a rule, the first symptoms point to gastric disturbance, disinclination to take food, pressure, nausea. This is accompanied by flashes before the eyes. The cardiac symptoms which manifest themselves later are ushered in by an indistinct feeling of anxiety and a slight irritability of the heart. Later the heart-beat is either greatly retarded or the action becomes frequent and irregular. This irregularity is a most important symptom, as the inexperienced is in danger of mistaking it for an indication for the continued use of digitalis. The caution, of course, applies to those cases also in which digitalis has been taken without the physician's knowledge and before his orders had been given.

The symptoms in the case of grave intoxication are unmistakable: vomiting, diarrhea, vague cerebral symptoms, headache, vertigo, auditory and visual disturbances, dilatation of the pupils. This is followed by profound lassitude and, if the condition is protracted, a marked impairment of the nutrition. Sudden death from heart failure may occur on the slightest provocation, such as assuming the erect posture.*

* See also Binz, "Vorlesungen über Pharmakologie," Hirschwald, Berlin, 1891, 2. Auflage, S. 227 ff.

Tincture of strophanthus, prepared from the seeds of the plant, may be mentioned as a remedy which is used somewhat in the same way as digitalis. The usual statement that it is a succedaneum of digitalis is not quite accurate.

Unfortunately, the preparations of strophanthus, like those of digitalis, are far from uniform in their action.

The effect of the drug is to increase the action of the heart as a whole; but how the effect is produced is not known. Strophanthus has no cumulative action and can, therefore, be given for a long period. In the cases in which the drug acts favorably the effect, including the improvement in the heart action, is the same as after the use of digitalis, except that the frequency of the heart-beat is not, as a rule, diminished, and that, in my opinion, is the chief reason why the drug is inferior to digitalis. It is well to begin with a small dose—say five drops three times a day—and increase it gradually.

It is possible that some individuals may possess an idiosyncrasy for strophanthus. It is, however, uncommon, and possibly it is a purely imaginary idiosyncrasy. During recent years the current gossip among the bathers in Nauheim was against tincture of strophanthus, and some patients suffering from severe heart disease, who had taken the remedy for some time, attributed the change for the worse in their condition to the much-used drops. A general impression of this kind often becomes an article of faith, particularly as the scapegoat—the physician—also gets his share of the blame.

The tincture, either alone or with a certain quantity of compound tincture of cinchona, is the best preparation; the cinchona partially disguises the taste, which is unpleasant to some people.

Heart stimulants are indicated when the heart is to be temporarily stimulated to greater exertion. Under certain circumstances, as in the presence of pulmonary edema, this is absolutely necessary. In other conditions the indication is less urgent. It must always be borne in mind that the heart regulates its nutrition by its own activity, and that its power depends on the state of its nutrition. A far-sighted physician will often order a stimulant as soon as the first signs of cardiac weakness make their appearance. It is impossible to give absolute rules. It is to be remembered that cardiac stimulation is justifiable only for the purpose of improving the nutrition of the heart. It must never be carried to the point of fatiguing the organ, but the precise limit must be determined in each individual case. A heart which is suffering from the effects of a febrile infectious disease, such as croupous pneumonia, for example, cannot be judged in the same way as one which has become permanently weakened from muscular disease. Whatever the condition may be, the question how far stimulation should be carried must never be neglected; it must determine the choice of the remedies to be employed.

For the purpose of rapidly bringing on an improvement in the strength of the heart *hypodermic injections of ether* are most suitable. The dose should not be too small. Five Pravaz syringefuls—corresponding to about 4 grams [by weight—Ed.] (one fluidram)—of ether represent the normal dose for the adult. The action of the drug is sometimes defeated by giving too little at a time; for it is not a matter of indifference whether a certain quantity of ether is given at one dose or in the course of an hour. Ether is rapidly broken up in the body and excreted; and if it is administered too slowly, the concentration in the blood and tissues necessary to enable the remedy to exert its effect cannot be attained.

The injection may be repeated at intervals of one to one and one-half hours. But from the nature of the case—since, as a rule, the heart is quite feeble when ether is used—one cannot hope to derive much benefit from these repeated injections.

Camphor is slower in its action, but its effects are more lasting. It is best administered hypodermically in the form of the [German—Ed.] officinal camphorated oil, one part of camphor in ten of olive oil. [The U. S. P. Linimentum Camphoræ is made of 1 part camphor to 4 (1 in 5) cotton-seed oil.—Ed.] For the adult I prescribe 5 grams (say 1 dram) of the oil. A point on the trunk should be selected for the injection because the circulation is more active in the trunk in cases of heart weakness than in the extremities. In cases of heart weakness due to slowly developing disease of the muscle-fibers I usually allow one to one and one-half days to elapse before again administering the remedy.

For the purpose of combining the rapid action of ether with the more lasting action of camphor some physicians use the solution of the drug in ether. Personally, I prefer to inject the two drugs, one immediately after the other. An ethereal solution of camphor causes violent pain and is perhaps also more apt to produce abscesses.

There is a somewhat wider choice of heart stimulants that are given by the mouth. Strong, hot infusions of *coffee* and *tea* stimulate the heart action, the effect developing within a short time, particularly when they are mixed with large quantities of alcoholic beverages, such as rum, cognac, and the like. They are not inferior to ether. But a heart patient is not always in a condition to take enough by the mouth, besides which the effect is not lasting and it is hardly safe to repeat the dose very often.

The case is different with *wine*. In the case of those who are not used to alcoholic beverages great care must be exercised. The effect on the brain, the excitement and secondary depression ("der Rausch and der Jammer") occur in heart patients as in others, except when there is fever. Then the effect of alcohol on the brain, whether the patient is accustomed to taking it or not, is so slight that it requires large quantities to produce any noticeable effect.

By taking into consideration the heart patient's mode of life and the strength of the heart at the time, the administration of wine—which in such a case is to be regarded simply as a drug—can be suitably regulated. Not even those who forbid the use of wine in general in heart disease will take an exception to its use under these circumstances.

The Schott brothers (see p. 160) recommend a peculiar method of stimulating the heart by the application of heat. I have also used this method, but I allow the heat to act for a longer time. A tin receptacle covered with thin flannel and curved so as to adapt itself to the shape of the thorax* is filled with water as hot as the patient can bear and applied to the precordia. The application is kept up for days or weeks, the high temperature being continued. The part of the chest with which the fomentor comes in contact should be covered with a thick pad of woolen material and a similar covering should be placed around the fomentor.

In some cases of heart disease, particularly of acute origin, the good effects of this method were unmistakable; also in cases of chronic muscular weakness when, for any reason, there is grave disturbance of the general condition associated with palpitation and anxiety. But I have never been able to demonstrate any permanent effect on the action of the heart.

* An article of this kind is sold in the stores under the name of "fomentor."

The application of cold to the heart is a common practice in cases of apparently unnecessary cardiac excitement—palpitation, anxiety, and marked increase in the frequency of the pulse. There is no objection to this practice. According to my experience it is impossible to determine beforehand whether a higher or a lower temperature than that of the body will benefit the patient, so that I always allow him to find out for himself which is more acceptable.

When every attempt to restore the strength of the heart proves unsuccessful and the cardiac weakness attains an extreme degree, certain special indications must be met.

Dropsy.—The best way to combat dropsy is to stimulate the action of the kidneys. Digitalis, by virtue of its action on the heart, indirectly stimulates the kidneys and causes them to excrete more urine. This has already been mentioned. The number of true diuretics which can claim a direct action on the glandular tissue of the kidneys is very limited. Not one of them is absolutely reliable, not even when reinforced by other therapeutic measures, be they mechanic or medicinal, for stimulating the action of the heart, although an attempt to do this should always be made. The value of combining heart stimulants with diuretics is most frequently exemplified when digitalis is combined with calomel.

Calomel is one of the best diuretic remedies that can be given in heart weakness. It may be ordered in the dose of 0.2 gm. (3 gr.) three times a day. I do not like to give calomel more than three days at a time, because I have never been able to satisfy myself that any good is accomplished by doing so. Others, however, set the limit at twelve days. If the drug produces much diarrhea, 0.01 gm. ($\frac{1}{6}$ gr.) of opium may be added to each powder. The constitutional effect of mercury must always be borne in mind and the patient carefully watched accordingly. If the excretion of urine is increased at all, it usually takes several days for the drug to develop its full effect.

J. Bauer* advises against the use of calomel "when there is some disease of the kidneys in addition to the general disturbance of the circulation, in cases of relatively rapid paralysis of the heart after a debauch and, finally, in cases of extensive degeneration of the heart muscle." Grave disease of the kidneys, in the opinion of most authorities, contraindicates the use of the remedy, and this view is well founded. In the other two conditions mentioned by Bauer it would seem to be difficult to define the indications accurately.

Among other remedies should be mentioned:

Caffein, which has a specific action on the kidneys besides being a cardiac stimulant. Thanks to Riegel,† who lays down rules for its proper employment, the drug has come into favor again. His observations show that it may act very favorably. It is, however, a very uncertain remedy and cannot be relied upon.

Owing to its great solubility in water and the permanency of the solutions, Riegel recommends combinations resulting from the action of an organic acid, salicylic, cinnamic or benzoic on sodium and caffein as a base. He orders 0.2 gm. (3 gr.) at a single dose; at the most 1.8 gm. (27 grains), usually not more than 1.2 gm. (18 gr.), in twenty-four hours. A newer preparation is caffein sulphuric acid (coffeïnsulfosäure) in combination with sodium, lithium, and strontium; *symphorol* is the *nom de guerre* for the sulfocombination, to which the designation of the inorganic substance desired is added. Dose, 4 to 5 gm. (60–75 gr.) per diem.

* "Allgemeine Behandlung der Kreislaufstörungen," Penzoldt-Stintzing, "Handbuch der Therapie innerer Krankheiten," Jena, Fischer, S. 597 der 2. Auflage, 1898.

† "Ueber die therapeutische Verwendung der Coffeïnsalze bei Herzkrankheiten," "Verhandlungen des Congresses für innere Med.," 1884, iii, S. 292, ff.

Theobromin, which is closely related to caffein, is to be mentioned as an almost pure diuretic. *Diuretin* (theobrominum natrio-salicylicum) is the preparation generally used. The maximum dose is 1 gm. (15 gr.); 8 gm. (2 drams) in twenty-four hours. It is best prescribed in a watery solution, say in peppermint water. It is not absolutely certain in its effects, but has a number of successes to its credit.

I still occasionally use some of the older diuretic remedies. Necessity is the mother of prayer, and the necessities of a patient with dropsy are great, but I cannot say that my prayers have often been heard. On one occasion only I witnessed a veritable flood of urine after the exhibition of the followng mixture, which contains such a multiplicity of the vaunted medicines that it would do the heart of any old-time apothecary good to read it:

℞. Acet. scill. ..... 20.0 (5 drams)
Liquor potass. carb. q. s. ad perfect. saturation
Aq. petroselin. ..... 120.0 (4 ounces)
Succ. juniper. inspissat. ..... 30.0 (1 ounce)
Spirit. æther. nitros. ..... 20.0 (5 drams)
M. Sig.—One tablespoonful every two hours.

That was in 1870, and never since then have I had any success with the mixture, which is not exactly palatable.

Large quantities of water can be extracted from the body by means of diaphoresis. *Pilocarpin* as a diaphoretic is by no means a harmless drug, and ought not to be used too freely. The best preparation is a solution of the chlorid, and it should be given hypodermically, beginning with the dose of 0.005 gm. ($\frac{1}{12}$ gr.), and under no circumstances exceeding 0.02 gm. ($\frac{1}{3}$ gr.).

Penzoldt * says: "The remedy is not only permissible, but even indicated in desperate cases which resist other methods of treatment; but the physician must always remember that he is using a two-edged sword. The danger of collapse must always be kept in mind."

In addition, pilocarpin stimulates the secretion of the bronchial mucous membrane, and its action in this respect is so marked that even Oertel,† who advocates its use, expresses his misgivings as follows: "It seemed to me in some cases that the stimulant effect of pilocarpin on the secretion of mucus in the air-passages which is accompanied by considerable diminution of the vital capacity of the lungs, the occurrence of spastic neurosis (singultus), insufficient breathing, and attacks of asphyxia, is even more dangerous than the action of the drug on the heart muscle. This effect on the bronchial mucous membrane, to be sure, is only occasional. Under these circumstances the increased excretion of fluid into the bronchi may give rise to a series of unpleasant accidents which may endanger the patient's life. On the other hand, when the lungs are free and the heart is not too weak, the injections are always well borne."

It is my impression that pilocarpin has never become popular in general practice.

The method formerly in vogue of combating dropsy in heart patients by inducing watery evacuations from the bowel is practically never resorted to at the present time.

We must resort chiefly to physical methods of treatment, and it is obvious that the effect they produce on the skin is of primary importance. Various methods are employed to induce profuse sweating. However they may be applied, the fundamental principle laid down by Liebermeister‡ always holds good: "Sweat secretion is brought about by all

* *Loc. cit.*, p. 256. † "Handbuch," S. 100.

‡ "Anwendung der Diaphorese bei chronischem Morbus Brightii," "Gesammelte Abhandlungen," Leipzig, F. C. W. Vogel, 1889, S. 148.

those conditions which bring the temperature of the external skin approximately to the level of the temperature of the interior of the body."

The indication can be met by applying heat from the outside, which may be effected in the following ways:

1. By means of baths of a temperature higher than that of the body. The heat is taken up by the skin until it has been raised to a state of equilibrium with the interior of the body, after which the skin gives off heat to the body.

Liebermeister employed baths of gradually increasing temperatures up to 42° C. (107.6° F.) and over. After the bath the patient is wrapped in blankets for several hours. The secretion of sweat begins while he is in the pack and may be very profuse; in the case of kidney patients a loss of over two liters by weight has been observed after a sweat-bath.

2. By the so-called Russian steam-baths, which were probably first employed on a large scale in Bartels' clinic. The patient is exposed to a temperature of about 50° to 55° C. (122° to 131° F.) and becomes saturated with aqueous vapor. After the patient leaves the steam-bath he is also wrapped in blankets.

There is no fundamental difference between these two procedures. They both produce a rise in the temperature of the body, which in a given case may be quite considerable. Thus Liebermeister found that after a protracted bath, when the temperature was gradually raised to 42.2° C. (108° F.), a temperature of 39.6° C. (103.2° F.) was recorded in the mouth. After a steam-bath of 56° to 60° C. (132.8° to 140° F.) I* observed a rectal temperature of 40.7° C. (105.2° F.). The steam-bath causes a much more rapid rise of the body temperature than a gradually heated ordinary tub-bath, and this may be productive of very grave symptoms which, in my opinion, constitute an absolute contraindication to the use of Russian baths in all cases of cardiac weakness.

The contraindication to the use of gradually heated tub-baths is less positive; nevertheless, too great an increase of the patient's temperature, which is always followed by an increase in the heart's action, must be avoided in administering these baths. Another difficulty is that the dropsical condition of the skin in heart patients interferes with the copious secretion of sweat,† and that is probably the reason why the procedure, which is so useful in kidney patients, is so rarely employed in cases of heart weakness.

3. In the *hot-air* or so-called *Irish-Roman baths* the patient is exposed to heated air of the temperature of about 60° C. (122° F.), containing but little aqueous vapor. The skin becomes greatly heated and excretes sweat in abundance, the evaporation of which renders sufficient heat latent to keep the patient's temperature normal, or at least to permit only a very moderate rise. To guard against an undue rise in the temperature, the patient may be allowed to breathe ordinary air while his body is exposed to the superheated air of the apartment.

A number of contrivances have recently been devised for administering a hot-air bath in the sitting or lying posture and allowing the patient to breathe the outside air. Either aqueous vapor or heated air may be used. Bauer (*loc. cit.*, p. 591) describes such an apparatus briefly as follows: A wire frame corresponding in length to the length of the patient's body from the shoulders to the feet is covered with blankets, leaving only the head free. Steam or hot air is then conducted into the closed space by means of a pipe.

* "Deutsches Archiv für klinische Medicin," 1868, Bd. iv, S. 357.

† See Liebermeister, "Abhandlungen," S. 147.

Unfortunately, there are times when all these measures fail and the physician is obliged to remove the effusions by external drainage. This he is always reluctant to do, for in most cases it brings only temporary relief and may be productive of harm.

Hydrothorax (hydrops pleuræ) is not a frequent occurrence. Large accumulations of fluid in both pleural cavities are not well borne by heart patients, and their appearance is really a sign that the end is near; for the kidneys under such conditions are rarely free from grave disease. As a rule, the accumulation is small and causes only slightly insufficient expansion of the lungs.

In cases of ascites removal of the fluid may give the patient great relief and is occasionally followed by considerable increase in the excretion of urine.

One cannot, of course, count on the latter result, and the evacuation of the fluid is not always followed by an improvement in the patient's appetite, digestion, and assimilation. But these advantages ought perhaps to be taken into account and may counterbalance the disadvantage incident to the loss of body material, especially albumin, which necessarily accompanies evacuation of the fluid. Personally, I do not believe this loss is very great. On the other hand, I quite agree with those physicians who defer the first paracentesis as long as possible and repeat it rarely. But when the ascites is causing the patient great suffering, I never hestitate to operate.

Anasarca.—The technic of procedures for the removal of cutaneous dropsy has not reached a high stage of perfection. The over-stretched skin must not be allowed to rupture and must, therefore, be punctured or incised, after which precautions must be taken to guard the parts adjacent to the punctured or incised wound from the injurious effects of constant maceration with the fluid, which softens the protecting covering—the epidermis. Inflammatory and destructive processes, from simple eczema to septic infection or gangrene, are then apt to develop. Even when great care is exercised, the dressings sometimes will not take up all the fluid, which may spread over other parts of the body and give rise to great discomfort or even become a source of great danger.

Cleanliness and disinfection are absolutely necessary.

Various procedures are available. Puncture with the trocar, after Southey (Southey's trocars), is frequently employed [and is infinitely safer—Ed.]. The trocar and cannula, the latter about 4 cm. in length and 1.5 to 2 mm. in width, are introduced into the subcutaneous connective tissue; the trocar is then withdrawn, leaving the cannula in place, and rubber tubes are attached which take up the escaping fluid and convey it into a vessel. [Southey's trocars as sold are smaller, but even with them one can take away enormous quantities of fluid without discomfort.—Ed.]

Another procedure, which does not seem to me to have obtained the recognition that it deserves when carefully carried out, is drainage by means of a funnel, as suggested by Straub.* The funnel should be small, with smooth, well-rounded edges, the upper opening about 4 to 5 cm. in diameter. A rubber tube about 4 mm. in diameter having been attached to the lower end of the funnel, the latter is firmly applied to the point from which the fluid is to be removed, after the skin has been punctured

* "Beitrag zur operativen Behandlung der Oedeme der Haut," Tübingen dissertation, under v. Liebermeister. 1882.

in two or three places. The funnel is held firmly in place until the escaping fluid has filled the rubber tube, the end of which is immersed in a vessel of water by the side of the bed. In this way the action of gravity is utilized and can be increased or diminished at will by changing the distance between the punctured area in the skin and the opening of the rubber drainage-tube.

The following method was recently recommended by P. Fürbringer:* "A cannula with a lumen of 5 to 6 mm. is inserted into the tissue as nearly flat as possible and parallel to the surface of the skin, that is, close to the corium, and brought out again through the skin at the opposite end—in other words, the subcutaneous space is tunneled. Next a rubber tube, about two meters in length, and just large enough to fit inside the cannula, and provided at its middle with three openings for drainage a few centimeters apart, is introduced so that the section of the tube containing the openings corresponds to the channel made by the cannula, after which the latter is removed by slipping it over the drainage-tube. The two extremities of the latter are placed in a vessel of water, and the entire contrivance is held in place much in the same way as a hair-pin on a woman's head, and, being practically in equilibrium, requires no other support. An ordinary loose antiseptic dressing is all that is needed. The living tissues around the tube soon close up, so that the escape of fluid from the two wound openings ceases altogether." The method deserves to be carefully tried; Fürbringer has used it successfully in six cases.

Simple puncture and incision remain to be mentioned. Fürbringer† recommended scarification by means of long deep incisions in the dependent portions of the body, which should be supported on pads of peat.

Gumprecht suggests‡ moss pads as suitable for taking up the escaping fluid. He refers to Fürbringer's procedure and also suggests that the scarified limbs be suspended with gauze bandages so as to hang clear of the pads.

C. Gerhardt§ employs puncture and observes all the precautions customary in major operations. The skin having been cleansed with scrupulous care, from four to eight punctures are made on the anterolateral surface of each thigh. The patient during the operation is placed in an arm-chair with the legs supported on a board, which is placed over a wooden vessel. As soon as the punctures have been made, a heavy dressing, consisting first of gauze next to the skin, and over that thick layers of sterilized cotton, is applied to the thighs, with a few turns of a gauze bandage. During the day the patient remains in the arm-chair as long as he can, and when he is in bed, the legs are supported on water-proof pads. As soon as the cotton has become saturated, usually once or twice a day, the pads are rapidly changed, the attendant first carefully cleansing his hands. If the cotton or gauze has stuck to the puncture wounds, it must be loosened with a 3 per cent. solution of carbolic acid or a 0.5 per cent. solution of bichlorid of mercury.

* "Zur mechanischen Behandlung des Hautödems (subcutane Schlauchdrainage)," "Deutsche medicinische Wochenschrift," 1899, S. 6 ff.

† "Ueber die Behandlung des Hydrops," "Deutsche medicinische Wochenschrift," 1890, S. 233 ff.

‡ "Die Technik der speciellen Therapie," Fischer, Jena, 1898. The section devoted to the mechanic treatment of anasarca, which is very carefully done, begins on p. 298.

§ "Ueber Einstiche in das Unterhautbindegewebe," "Deutsche medicinische Wochenschrift," 1892, S. 137.

Large quantities of fluid can be withdrawn from the skin by means of these procedures.

Gerhardt has seen as much as 10.3 liters (quarts) drawn off in a single day. The systemic effect of removing the serum from a dropsical skin may either manifest itself by an immediate increase in the excretion of urine, or diuretic remedies, which until then had had no effect, will now become efficacious. On the other hand, fatal accidents have been observed when the fluid was withdrawn too rapidly, death taking place with the symptoms of autointoxication, cerebral anemia, or paralysis of the heart.

[*Dechloridration*, or the treatment by limitation of sodium chlorid, has a place in the treatment of cardiac dropsy, though its chief importance is in nephritis. A complicating nephritis, of course, often coëxists with a heart lesion, and then furnishes its own indication for "salt-free" diet, but even in cases of cardiac anasarca without evidences of nephritis the treatment is often useful. It consists, in the first place, in leaving out all the salt that is usually added in the preparation of food, as from bread, meats, and butter, for the amount of salt naturally in most foods is so small as to be negligible. Milk diet is salt-free in the sense used; bouillon is not. In strict salt-free treatment the diet must be specified with reference to the sodium chlorid content, as well as to the calories. Some, but not all, patients object seriously to the withdrawal of salt, but almost all of them soon become accustomed to the change.*—Ed.]

Many heart patients suffer from insomnia and one naturally wishes to give them a good night's sleep from time to time. The question is what remedies may be allowed and which are to be avoided under such circumstances?

Chloral hydrate I personally never use under any circumstances. Shortly after the introduction of this otherwise excellent remedy in the year 1870 I observed such disastrous results from the administration of 1 gm. (15 gr.) in a patient of mine whose heart was not even seriously affected that I have never since that time ordered chloral in any case of chronic cardiac weakness. This has since become a general rule.

Chloralformamid is less dangerous than chloral hydrate and has even been recommended by some authorities; but this drug also has been known to produce unpleasant symptoms in heart patients. It should not be given in a larger dose than 1 gm. (15 gr.) at first.

Sulfonal, *trional*, and *tetronal* do not act very well in advanced cases of heart weakness, as they reduce the blood pressure and injure the red blood-corpuscles.

This leaves practically nothing but *morphin*, which, however, must be used with the greatest caution.

I prefer hypodermic injections because of the certainty that the dose will be taken up in a short time when that method of administration is used; while absorption through the gastro-intestinal tract may be delayed and we do not know the possible extent of this delay, so that the remedy is uncertain as a hypnotic when given by the mouth. The influence of morphin on the respiratory center must never be neglected. In bronchial catarrh attended by the secretion of large quantities of tenacious mucus requiring cough for its removal, morphin must not be given.

I am in the habit of giving 0.005 gm. ($\frac{1}{12}$ gr.) at a single dose—never more than 0.01 gm. ($\frac{1}{6}$ gr.).

The question of *venesection* finally remains to be discussed. Personally, I have not had enough experience with the method. The few cases

* See "La Cure de Déchloruration," by Widal and Javal, Paris, 1906. "The Dechloridration Treatment in Diseases of the Heart," Ernest Barié, "International Clinics," vol. i, sixteenth series, p. 26.

of rapidly developing failure with pulmonary edema in the course of a pneumonia in which I have had an opportunity of noting the effects of venesection have not made me an enthusiastic supporter of the procedure. How it acts in cases of slowly developing cardiac weakness I cannot say from my own observation. Liebermeister* expresses himself as follows on this point:

"Cases are frequently seen, particularly in heart disease, in which copious bleeding would be useful and life could be saved by a timely venesection. In our clinic here in Leipzig venesection is not infrequently resorted to in the case of patients with severe disturbance of compensation, and we have often succeeded, by means of the procedure, in keeping patients alive for a time when they were on the point of death from extreme heart weakness. Indeed, in a few cases the procedure was so successful that compensation was gradually restored for a considerable length of time."

The indications mentioned by Liebermeister, aside from the usual ones,—threatening pulmonary edema and threatening cerebral hemorrhage, although he does not consider the last of any practical value because he says it is rarely possible to foresee a cerebral hemorrhage,—are the following:

"1. The stagnation of blood in the cerebral veins when sufficient, either directly or through the resulting cerebral edema, to interfere markedly with the mental functions.

"2. When digitalis and other remedies have failed and the patient is in danger of dying from excessive diminution of the force of the circulation."

By way of explanation he adds: "Even the older physicians believed that venesection was indicated in heart disease by a small, intermittent pulse and coldness of the extremities; we may add that the more marked the signs of venous plethora, distention of the visible veins, and excessive cyanosis, the more urgently is blood-letting indicated. Whoever has made an autopsy in cases of death from failure of compensation and has seen the enormous quantities of blood accumulated in the veins and cavities of the heart must have entertained the thought that copious blood-letting would have been the thing to do in such a case."

"3. In cases in which, when the patient is first seen, the diminution of the circulation is so great that there is no time for digitalis or any other drug to act and no remedy except stimulants can be employed because death appears to be imminent. In such cases venesection may afford a respite and the time thus gained may enable the physician to obtain a good therapeutic result."

I shall confine myself to a few general remarks:

Venesection is a mechanic procedure. The abstraction of blood from the vein diminishes the amount of blood that enters the right auricle and thus lessens distention of that chamber and of the right ventricle. This enables the right side of the heart to contract more completely and to force more blood into the left heart. The larger the column of blood that enters the aorta, the greater will be the supply of blood to the heart, thus improving its nutrition and increasing its functional power. Another important effect of venesection is the absorption, by the general circulation, of the fluid from the edematous lungs and consequent liberation of pulmonary surface for the interchange of gases.

* "Vorlesungen über specielle Pathologie und Therapie," Bd. iv, "Krankheiten der Brustorgane," F. C. W. Vogel, Leipzig, 1891, S. 431 ff.

Granting that all the indications are present in a given case, it then becomes necessary to perform venesection in as short a time as possible; the blood must be allowed to escape from a widely opened vein if the pressure on the right heart is to be adequately relieved.

Liebermeister refers to a technical difficulty: "Sometimes, when the veins are greatly distended, the blood escapes very freely at first on opening the vein; but after a time, as very little blood enters the veins from the capillaries, the blood only escapes drop by drop." In such a case the patient should be made to work the muscles of the part where venesection has been performed as much and vigorously as possible, and the part should also be massaged in the direction from the capillaries toward the venous trunks, a procedure which was tried and found to be effective by the older physicians.

That the actual loss of blood is not desirable is pretty generally admitted. The rule is to remove the smallest possible quantity of blood in as short a space of time as possible. This is positive. Another rule is that the procedure must not be repeated at frequent intervals. By means of venesection we gain time either for recovery to take place by natural processes, as by crisis in a croupous pneumonia, for example, or to enable a therapeutic procedure to develop its effect. It may be worth mentioning that the employment of heart stimulants in combination with venesection performed in the presence of the above indications was warmly recommended by the older physicians.

I do not feel called upon to enter any further into the doctrine of venesection. The opinion which I gave long ago I have had no reason to change in the course of time.* But facts are more important than theoretic considerations, and observations such as Liebermeister reports must receive due consideration.

* Compare Jürgensen, "Blutentziehungen," in v. Ziemssen's "Handbuch der allgemeinen Therapie," Bd. i, 2, 1880.

ENDOCARDITIS.

BY

THEODOR VON JÜRGENSEN, M.D.

ENDOCARDITIS.

INTRODUCTION.

The description of endocarditis which I present varies in some respects from that ordinarily given in text-books. The reason of this is that in my material I have been able to observe most cases from the very outset and to trace them through many years. In this manner I have gained views that differ somewhat from those ordinarily accepted.

I need not dwell upon the fact that in this clinical study of endocarditis I depend chiefly upon my own experience. I set myself the task of presenting, in the most thorough manner possible, the facts as I have seen them, and each reader may judge from these what material I had at my disposal, and what I lacked.

I do not think it is correct to regard endocarditis as a separate entity. "Pancarditis"—this will be the diagnosis of the future. But he who makes this diagnosis must know how to distinguish the various parts which compose the whole, and to weigh the relative importance of each. The object should be, of course, in each individual case to analyze the composite picture of pancarditis into its constituents—endocarditis, myocarditis, and pericarditis. We must remember, however, that these elements are only parts of the same disease. One hundred years from now the nerves of the heart will also receive the attention which they merit.

It is difficult to determine whether the various etiologic factors which affect the heart exercise any special influences upon the various tissues of this organ; for example, whether streptococci affect the myocardium more frequently than staphylococci, etc. In my opinion, we are, in this respect, still in darkness, which is scarcely relieved by a ray of light.

The observations which I have made myself are possibly applicable only at the place and the time at which they were made, and, therefore, my description is possibly only of limited value. Yet, it is also possible that it may call attention to apparently insignificant details which may facilitate the early recognition of heart disease. This is, indeed, of great importance for the patient.

An exposition of our knowledge of endocarditis which coincides with observations at the bedside must be based upon the following principles:

1. That endocarditis is always part of a general infection the severity of which determines the clinical course.

2. That the endocardium is never affected alone, but also the entire heart. The work of the heart may be interfered with by insufficient closure of the diseased valves, or as the result of the narrowness of their orifices, but also in consequence of the loss of power in the heart muscle,

following lesions which affect this structure directly, and which arise from either inflammatory or degenerative processes.

We cannot at present decide the extent to which the nervous structures contained within the heart take part in the disease.

HISTORIC NOTES.

The fundamental work on endocarditis we owe to Bouillaud.* He states correctly that a very insufficient knowledge of endocarditis was exhibited by writers who preceded him, and even Laennec speaks of endocarditis as a "very rare" disease. Bouillaud asserts that endocarditis occurs at least with the same frequency as pericarditis, and adds that the time is not distant when his opinion will be generally accepted. This prophecy has been fulfilled.

The anatomic portion of Bouillaud's book, although excellent for the time when it was written, of necessity had to succumb to the changes which were wrought during the great advances in pathology subsequently made. Yet his recognition of the connection between endocarditis and acute articular rheumatism, sepsis, and pneumonia, remains to this day. Bouillaud also observed correctly the relation of the endocarditis to the valvular lesion, and its recurrence in cases in which such lesions have once developed.

It is interesting to note the position which Bouillaud takes upon the question as to whether other portions of the heart are involved in the inflammation of the endocardium and, if so, which portions of this organ are so affected. Pericarditis is by no means a rare accompaniment of endocarditis, and his Case 13 presents an excellent example of this kind. In speaking of "carditis" (myocarditis, according to our nomenclature), he says†: "I have never met a case of carditis which was not complicated with endocarditis or pericarditis, and I must admit that the symptoms of these last two inflammatory conditions took my entire attention."

This utterance enables us to understand how the myocardial lesions which are not made evident by striking physical signs have been treated like Cinderella from the start.

Even the voice of a Stokes was not heeded when it gave the muscle that which this structure merited.

It is characteristic how little importance was attributed to the fundamental principles expressed by Stokes, even by such a prominent physician as Bamberger. I may quote only one place to show how clearly Stokes expressed his opinion: "It is, then, in the vital and anatomic conditions of the *muscular fibers* that we find the key of cardiac pathology; for, no matter what the affection may be, its symptoms mainly depend on the strength or the weakness, the irritability or the paralysis, the anatomic health or the disease of the cardiac muscle."‡

Bamberger§ criticizes several passages in Stokes' treatise, and continues as follows: "Every impartial reader who is acquainted with the subject must say that we have learned nothing new from Stokes'

* "Traité clinique des maladies du coeur," Paris, J. B. Baillière, 1835, vol. ii, pp. 1–253. German translation by A. F. Becker, Leipzig, Gustav Wuttig, 1836, vol. ii, pp. 1–150.

† *Loc. cit.*, p. 302.

‡ "Handbuch der Krankheiten des Herzens und der Aorta," by Dr. William Stokes, translated by Dr. J. Lindwurm, Würzburg, Stahel, 1854, p. 110.

§ "Beiträge zur Physiologie und Pathologie des Herzens," "Virchow's Archiv," Bd. ix (1856), p. 540.

book in Germany; that many of his opinions are decidedly wrong, and that Stokes does not know many things which we have known for a long time in Germany."*

The description of the symptoms of endocarditis was given by Bouillaud in an excellent manner, and was based upon numerous cases. The man was able to see—that is the impression, at any rate, which one gains from his descriptions. He was not so good, however, as regards treatment. He used to order an almost incredible amount of blood-letting. Thus, in a young man aged seventeen years, Bouillaud removed 3180 gm. of blood in ten days. But it is true that this was his worst case. (Observation No. 84.) The work of Virchow† brought a great deal of knowledge on the subject of endocarditis into prominence. Autopsies performed with a thoroughness which had not been known until then; careful observations at the bedside; conclusions which an astute creative mind drew from these, and which were tested by the simple method of experiment; great and small things both weighed in their relative importance—all these showed the master.

In the course of time questions arose as to the real etiologic factor of endocarditis. Virchow, in 1856, observed, in a case of septic endocarditis,‡ certain rounded or oval masses of small granules embedded in a small amount of hyaline material which acted in a peculiar manner toward reagents: "If it were possible to connect these bodies found in the blood with endocarditis, then the connection (between the general disturbances and the organic lesions in the case under discussion) could be more easily established. Yet I must admit that it is advisable to await the results of further investigations in this direction."

The beginning of the investigations as to the cause of endocarditis may be traced to the observations of the Norwegian authors, Winge and Hjalmar Heiberg (1869). Robert Koch then created methods which permitted the isolation and study of the pathogenic microbes. These are but milestones upon the way thus far traversed.§

ETIOLOGY.

The pathogenic microbes which are known to bear some relation to endocarditis are the following: The staphylococci, especially Staphylococcus pyogenes aureus; Streptococcus pyogenes, in its various subtypes; the diplococcus of pneumonia. [Wells found endocarditis noted in 3 per cent. of all cases, or 4 per cent. of all fatal cases in his large statistics. But in 517 postmortem reports, endocarditis was noted in 22 per cent., a proportion probably more correct than the smaller one. Lesieur produced endocarditis in rabbits by subcutaneous injection of pneumococci. Pneumococcus endocarditis may occur, of course, without present or past pneumonia, sometimes without other organisms,

*[The criticism was directed chiefly against Stokes' views regarding mitral murmurs. See Stokes for his side of the polemic.—Ed.]

† "Thrombose und Embolie, Gefässentzündung und septische Infection," "Gesammelte Abhandlungen zur wissenschaftlichen Medicin," Hamm, G. Grote, 1862, S. 219–732.

‡ *Loc. cit.*, p. 711 *et seq.*, case II.

§ The Bacteriologic Literature in Baumgarten's "Jahresberichten." Refer also to the extensive works of Weichselbaum, "Beiträge zur Aetiologie und pathologischen Anatomie der Endocarditis," "Ziegler's Beiträge," Bd. iv, S. 127 ff. (1889). Francis Harbitz, "Om Endokardit e. c.," "Norsk Magazin for Laegevidenskaben" (1897).

sometimes with pyogenic cocci.—Ed.] These are by far the most important and most frequent germs found in endocarditis. Then comes the gonococcus, the importance of which has been recognized more and more within the last few years.

[Gonorrhea is an important starting-point for endocarditis, and the gonococcus alone is often the cause, though perhaps in the majority of cases there is an association with pus-germs—streptococci and staphylococci. The pyogenic organisms are often the only ones found in sections or cultures from cases with a gonorrheal history. Negative cultures may be due to faulty technic, but in some cases the gonococci are probably killed by the high temperatures, the other organisms remaining. It is especially in chronic cases that gonococci are not demonstrable. Several observers have cultivated the germs from the blood during life.* Gonorrheal endocarditis often affects old valvular lesions. Symptoms of mild attacks of endocarditis often occur during or after gonorrhea, but as recovery always follows, the cause and pathologic anatomy are not known. The endocardial change is only discovered by accident. There is little or no fever. The lesions seem to be either mitral or aortic. Hypertrophy sometimes occurs, but usually subsides.

Endocarditis following gonorrhea, due either to gonococci alone or to a mixed infection, often has a severe and malignant course. The endocarditis may be the only internal lesion, or there may be that and arthritis or septicemia. Fever, chills, and other septic symptoms may be severe, and subjective symptoms, especially dyspnea, may also be very severe. Murmurs are wide-spread and the lesions difficult to localize. Hypertrophy is often marked. The vegetations are often unusually large and fleshy. Ulceration and perforation of valves are frequent. The myocardium may be involved. As regards the valves affected, von Hofmann found the aortic valve affected in 21 cases out of 32—the mitral in 5, pulmonary in 5, tricuspid in 1. Sometimes several valves are involved. Embolism is rare, but phlebitis sometimes occurs. Recovery from the acute symptoms is rare, and is usually followed by severe lesions.†—Ed.]

Weichselbaum also found in isolated cases the following germs: Bacillus endocarditidis griseus; Micrococcus endocarditidis rugatus; Micrococcus endocarditidis capsulatus, and, in addition, he mentions as "credible" Bacillus pyogenes fœtidus (Passet), and a non-motile fetid bacillus (Fraenkel and Saenger).

[Other occasional forms are: Bacillus pyocyaneus,‡ "Micrococcus (diplococcus) zymogenes,"§ "Micrococcus tetragenus citreus."‖ The influenza bacillus has probably been found a few times.** The meningococcus was found by Gwyn, Salomon, and Warfield and Walker.††—Ed.]

* See Harris and Johnston, "Johns Hopkins Hospital Bulletin," vol. xiii, October, 1902, p. 236.

† Lartigau, "Study of a Case of Gonorrheal Ulcerative Endocarditis with Cultivation of the Gonococcus," "The American Journal of the Medical Sciences," January, 1901; Karl Ritter von Hofmann, "Gonorrhoische Allgemeininfection und Metastasen," review of the literature since 1890, "Centralblatt für die Grenzgebiete der Medizin und Chirurgie," Bd. vi, 1903, S. 308; Thayer, W. S., "On Gonorrheal Septicemia and Endocarditis," "Transactions of the Association of American Physicians," vol. xx, 1905, p. 391.

‡ Blum, "Centralblatt für Bakteriologie," Bd. xxv, 1899, S. 113.

§ MacCallum and Hastings, "Johns Hopkins Hospital Bulletin," vol. x, p. 41.

‖ Sterling, "Centralblatt für Bakteriologie," Bd. xix, 1896, S. 141.

** Mabel F. Austin, "The Johns Hopkins Hospital Bulletin," vol. x, 1899, p. 194.

†† "Bulletin of the Ayer Clinical Laboratory of the Pennsylvania Hospital," No. 1, 1903.

There is still to be added the Diplococcus tenuis of G. Klemperer,* which has been seen in some cases in von Leyden's clinic. In addition, a number of microbes have been seen at times which have not been cultivable, and, therefore, could not be studied in detail. Opinions are still divided as regards the occurrence of the typhoid bacillus, the Bacterium coli, the tubercle bacillus, and of the germ of syphilis, as causes of endocarditis, but possibly a greater importance should be attributed to the bacterium coli.

[As regards the typhoid bacillus, it seems to act often with other organisms, especially pyogenic cocci, but the specific germs can probably set up lesions. Di Vecchi produced experimental endocarditis with typhoid toxin. The position of Bacillus coli is still unsettled, but it has also been used to produce experimental lesions. Endocarditis is not rare in tuberculous patients (5 per cent., Marshall), but most of the cases are due to secondary infections. In some other cases there are tubercle bacilli in non-specific histologic processes, and the bacilli then are probably only accidental deposits. Marshall groups cases resulting either directly or indirectly from the action of tubercle bacilli under five heads: 1, Miliary tuberculosis; 2, true tuberculous endocarditis; 3, tuberculous cardiac thrombi; 4, tuberculous endocarditis from extension of a myocardial tuberculosis; 5, toxic endocarditis of tuberculous origin. Many so-called endocardial miliary tubercles are really subendocardial, lying beneath the elastic coat of the endocardium. True tuberculous endocarditis has been observed in several cases since Tripier reported the first one (1890). In this the nodule on the valve looked like a tubercle and sections showed giant- and embryonic cells. Michaelis and Blum produced verrucose endocarditis with tubercle bacilli in rabbits. Marshall describes fully the criteria of endocardial tuberculosis and gives summaries of cases observed in the Johns Hopkins Hospital.† Braillon, who made a diagnosis of tuberculous endocarditis during life, found no specific changes. He thinks tuberculous endocarditis is especially likely to occur in infections of low virulence, as in serous membrane tuberculosis.‡ Since Howard (1893) first reported finding diphtheria bacilli in endocardial lesions, but few cases have been recorded. The process is likely to be rare on account of the slight tendency of the organisms to enter the blood-stream. In Wright's case§ the germ was not very virulent. It affected the aortic and tricuspid valves, and was associated with pneumococci, colon bacilli, and staphylococci. In the case of Roosen-Runge‖ also, the tricuspid and aortic valves were affected. The germ in that case, as in the original one of Howard, was not virulent. F. Meyer was able to cause endocarditis in animals by intravenous and intracardial injections of diphtheria bacilli. Subcutaneous injections were negative. Di Vecchi was also successful with toxins.**—Ed.]

* "Deutsche medicinische Wochenschrift," 1894, p. 919 *et seq.*, and Society Supplement of same year, p. 123.

† Marshall, Henry T., "Endocarditis in Tuberculosis," "The Johns Hopkins Hospital Bulletin," vol. xvi, September, 1905, p. 303.

‡ "Revue de Tuberculosis," 1904. See, also, Sorgo and Suess, "Wiener klinische Wochenschrift," 1906, Bd. xxix, S. 176. They believe tubercle bacilli may cause non-specific histologic changes in the valves.

§ "Boston Medical and Surgical Journal," 1894, vol. ii, p. 359.

‖ "Münchener medicinische Wochenschrift," Bd. l, 1903, S. 1252.

** "Centralblatt für Bakteriologie und Parasitologie," Ref. Bd. xxxvi, 1905, S. 550.

The requirements which Robert Koch* considers necessary in order to bring indisputable proof that an organism is the true cause of a disease cannot be fulfilled to their full extent in all cases in all the microbes which have been suspected. When these germs do not grow upon artificial soil, it is impossible to determine the conditions under which they live, and it is impracticable to inoculate them into animals. In such cases we must content ourselves with examinations of the tissues which are now rendered possible by the improved technic of staining. "The facts which are gained by this method may possibly show so much evidence that only the extremest skepticism would object that the organisms discovered are not the cause, but merely an accompanying manifestation of the disease. It is true that this objection often has a certain logical basis" (R. Koch).

The question as to what conclusions are possible without cultures and without inoculation into animals must be decided in each individual case. It is noteworthy that the majority of investigations show that "the streptococci are very inconstant in their behavior in animal experiments, independently of the pathologic process in man from which they are derived, and of the manner in which they are inoculated."†

It is unquestionable that the vitality of the germs which cause sepsis in its broad sense—including the pneumococci—is limited. Very often the germs themselves cannot be demonstrated and only the tissue changes which they produce are evidence of the fact that they had been there. These changes and clinical observations must then serve as a basis for conclusions. Naturally, we cannot always be sure in such cases, and we are entitled to entertain more or less strong doubts.

Endocarditis is most frequently seen in connection with acute articular rheumatism. This is universally recognized, but the question, What is acute articular rheumatism? is becoming more and more urgent. Here we encounter great difficulties, and I am unable to express an opinion which can be substantiated by definite scientific evidence.

There is a typical clinical picture which has been fully admitted into pathology. Senator‡ has given a very clear description of it. We older physicians are well acquainted with this picture in all its features, even to the smallest details, for we have seen it often enough. Here and there a case appears in the practice of a busy man, which does not seem to fit into the typical scheme (Kussmaul, 1852), but, as a rule, it is the same scene with a tiresome repetition. With the discovery of the curative properties of salicylic acid and its preparations—an action which may be almost characterized as specific—the unity of the disease seems to be just as well established as that of malaria.

And yet warranted doubts have arisen. I shall permit the facts to speak, and confine myself at first to my own observations.

Until the year 1872, there had been 100 cases of acute articular rheumatism in its ordinary form among a total of 15,242 patients admitted to the Poliklinic in Tübingen—that is, 6.6 per 1000.§ Then came a change.

* "Die Aetiologie der Tuberculose," "Mittheilungen aus dem Kaiserlichen Gesundheitsamte," edited by Dr. Struck, Bd. ii, S. 3, 4, Berlin, Hirschwald, 1884.

† Harbitz, *loc. cit.*, pp. 118, 119.

‡ Von Ziemssen's "Handbuch der speciellen Pathologie und Therapie," Bd. xiii, S. 13 *et seq.*, second edition, 1879.

§ Further data in Quenstedt, "Zur Aetiologie der Rheumarthritis acuta," in "Mittheilungen aus der Tübinger Poliklinik," Bd. i, S. 218, Stuttgart, Schweizerbart, 1886.

Cases of septic disease became more frequent, especially those with unknown portals of entrance, and many terminated fatally,* especially at the beginning of this period. After a while these cases became less severe, although severe affections still did occur and do occur. But in the great majority of cases we see but few dangerous manifestations nowadays. During this time there developed a form of disease which in many respects bears resemblance to acute articular rheumatism, but still is quite different from the latter. I think the following peculiarities can be distinguished:

1. The bones are involved almost without exception; they are painful to pressure—often extremely so. Usually the shafts of the long bones are involved; less frequently, the ribs, the pelvis, the cranial bones. As a rule, an extensive continuous area in the same diaphysis is affected, or there may be separate foci, but usually more than one bone is involved.

2. The joints are, it is true, somewhat sensitive. They show slight swellings and manifest spontaneous dull pain, but piercing or tearing pain is not always absent. All these manifestations are far less marked than in typical rheumatism.

3. Sweats and miliaria do not appear to the extent which is common in acute rheumatic arthritis.

4. Salicylic acid and its preparations do relieve the pains at times, it is true, and do lower the temperature to some extent, but this does not occur always, in spite of large doses. In no case have we seen that success which entitles these remedies to the rank of specifics. Another fact which I consider very important is that a patient will at first react fairly well to salicylic acid; but the remedy proves almost without effect during recurrences which come on later, perhaps years afterward—for example, case III.

I need only add that these cases are often also accompanied by perihepatitis, perisplenitis,—friction sounds over the spleen and liver coincident with respiration, sensitiveness to pressure, and pain over the enlarged organs are the clinical signs,—as well as by polymorphous or nodular erythema.

If there had been only isolated cases of this kind, I would not have expressed any opinion, but I am speaking of a change in the clinical picture which grows more and more marked from year to year; so much so that lately I have scarcely seen the old form. At present I make the diagnosis of sepsis, and in this respect agree with Singer,† although I do not consider myself entitled, as he does, to include acute articular rheumatism in the etiologic group of diseases produced by the germs of suppuration. It is possible that this is so, but as yet we have no proof. This subject has been exhaustively treated in the large treatise of Pribram,‡ as well as in Singer's book, and in these volumes a detailed index to the literature of this subject may be found.

Whatever may be the final decision in this question, heart disease is frequent, whether or not we regard acute rheumatism as an etiologic factor distinct from septic infection.

Endocarditis at times develops in the course of those diseases which lead to severe disturbances of nutrition, such as pulmonary tuberculosis, cancer, various forms of chronic nephritis, etc.

* Cf. Dennig, "Ueber septische Erkrankungen," Leipzig, F. C. W. Vogel, 1891.

† "Aetiologie und Klinik des acuten Gelenkrheumatismus," Vienna, Braumüller, 1898.

‡ Nothnagel, "Specielle Path. u. Therap.," vol. vi, Sect. T.

We must also remember that any acute severe disease may cause similar damage to the heart. If it has resulted from the invasion of microbes, and if these have not yet disappeared from the body of the patient, then the germs which cause endocarditis and which enter and settle in the endocardium may give rise to a mixed infection. In other cases, when the primary disease has disappeared, we speak of a secondary affection. The latter is not difficult to interpret, but it is not always easy—in fact, it is sometimes impossible—to determine the condition of things when a mixed infection is present. If the germs belonging to the first infection are still present in the circulating blood, they may be found in the heart, together with those which produce the endocarditis. The first-mentioned germs did not cause endocardial inflammation, but have only settled in places which were favorable for their growth. Whether or not they influence the local changes must be left undecided.

It is not to be forgotten that atheromatous changes in the endocardium are apt to favor the development of endocarditis in a high degree.

Given these numerous possibilities for the development of endocarditis, it is scarcely necessary to give statistics as to its distribution according to age, sex, etc.

Starting from the premises that endocarditis is due to an infection by germs, we must admit as self-evident the proposition that the germs circulate in the blood and are borne to the heart by the circulation. We cannot deny the possibility that germs may reach the heart through a wound or through neighboring structures, but this occurs very rarely.

The question as to how and whence the germs reach the blood may be answered as follows: An injury of the protective structures which cover the skin, the mucous membranes, and the serous membranes must precede the invasion of the germs. In some cases the portals of entry which thus open may be easily recognized. This is the case in infections of the puerperal period and in wound infections. At other times this recognition is impossible, and we then speak of cryptogenetic sepsis. This particular class of cases is of great importance in the clinical study of endocarditis.

The second question is no less important: What conditions favor the localization of bacteria on and in endocardium? Experience teaches that a fatal termination of the general disease may take place without an involvement of the endocardium. Myocarditis and pericarditis may occur, and yet the endocardium may escape gross changes. The latter event is less frequent.

A case in point is that reported by Dennig* in my Poliklinic (Case No. XVI), in which there was an infection with Staphylococcus pyogenes aureus. Death occurred at the beginning of the sixth day of the disease. The experiments of Ribbert† show similar cases.

Therefore it is not sufficient for the development of endocarditis that the bacteria which cause it circulate in the blood, even if their number is considerable. What else is necessary? Experiments showed that, if an injury of the valve had preceded the invasion of the germs, it was easily possible to bring about the localization of these organisms when they were introduced through the blood-vessels.

* *Loc. cit.*, p. 85.

† "Ueber experimentelle Myo- und Endocarditis," "Fortschritte der Medicin," Bd. iv, S. 1, 1886.

Ottomar Rosenbach* was the first to employ the method of Cohnheim—the mechanic influence of a sound introduced through the right carotid upon the valves of the aorta. As he did not employ in all instances sounds free from bacteria, he succeeded in performing a direct inoculation in two of his experiments. Later on, others purposely injured the valves and afterward injected pathogenic microbes into the veins. It was found that even "a very slight injury of the endocardium (by a mere passage of the sound over it) or a cauterization (Prudden; silver nitrate) sufficed."†

"The number of bacteria which were introduced did not have to be necessarily large, and I was successful in the use of the diplococcus of pneumonia even with relatively small numbers, while in one case, in using the Streptococcus pyogenes, I was able to induce an endocarditis by merely contaminating the sound with these germs" (Weichselbaum).

Wyssokowitch, Orth, and others employed the same methods, and Netter did the same in his experiments on pneumococcus endocarditis.‡

Ribbert succeeded in localizing the Staphylococcus aureus upon the endocardium, without previous injuries, but he was able to produce endocardial changes only upon the bicuspid and the tricuspid valves. The germ was cultivated upon potatoes, and in order to obtain trustworthy results, an emulsion which was not too fine was prepared by triturating the upper layers. The particles had to be as large as could pass through the cannula of a hypodermic syringe. In addition, the experiments failed if the amount of culture was very small.

Ribbert§ remarks concerning the mode of origin of the local disease: "We must imagine the process to be as follows: Either the particles of potato had remained adherent in the angles and lines of insertion of the tendinous cords, or else had passed over the surface of the valves and had become adherent to these during the closure. In any event, it is easily explained how the adherent bacteria could have been pressed into the endothelium and there had gone on in their further development."

Ottomar Rosenbach‖ pointed out that a mechanic injury to the valve is followed by more severe tissue changes than take place in endocarditis in man. No objection can be made to this statement, but the experiment could and should bring an explanation only as regards the question whether or not an endothelial injury favors the localization of bacteria. The relation of the severity of the injury to the subsequent events is a question of subordinate importance, and the methods of experiments pursued by Ribbert are sufficiently close to the actual conditions which obtain in a patient with endocarditis. Such small soft particles of potato could not have produced severe injuries to the tissues.

Another possible mode of origin of endocarditis has been demonstrated by Köster.** He showed that bacteria penetrate from and with the blood into the vessels supplying the endocardium, and that the germs multiply

* "Ueber artificielle Herzklappenfehles (Aus dem pathologischen Institute zu Breslau)," "Archiv für experimentelle Pathologie und Pharmakologie," Bd. ix, S. 1, 1878.

† Weichselbaum, *loc. cit.*, p. 218.

‡ Cf. the critical review of this subject by Baumgarten in his Jahresberichte (1885–1887).

§ *Loc. cit.*, p. 11.

‖ "Bemerkungen zur Lehre von der Endocarditis, etc.," "Deutsche medicinische Wochenschrift," 1887, S. 730; "Die Krankheiten des Herzens," Vienna and Leipzig, Urban and Schwarzenberg, 1897, S. 149.

** "Die embolische Endocarditis," "Virchow's Archiv," Bd. lxxii, 1878, S. 257.

and spread from there in many directions. If the valves are healthy, then this can only happen in the bicuspid and tricuspid, which contain vessels, while the aortic and pulmonary valves do not. Therefore, the entrance of microbes directly from the blood is only possible in the two valves first mentioned.

But the endocardium, which lines the cavities of the heart,—the parietal endocardium,—may be involved in the bacterial invasion. This does not occur very often, although possibly but little attention has been paid to this possibility, inasmuch as I have quite frequently seen lesions in the parietal endocardium in my autopsies. Nauwerck* is probably the first who paid adequate attention to this variety of localization. In such cases the invasion may take place directly from the blood of the heart, or else from valves already affected, by direct extension by continuity, or by infection from another source, such as an affected portion of a valve that has either been torn off or has come into contact with the wall through the influence of a strong blood-current. Finally, the endocardium may be affected by extension from the myocardium, which may be the seat of the primary disease. These possibilities have been summarized by Heim,† who worked under Ribbert.

The theory as to the development of endocarditis which has been very generally adopted is as follows: The endocardium is injured by pathogenic bacteria which enter into the body and produce an infection. The poisons which develop as the result of the life-processes of these bacteria are the essential factors. Examination shows that the first effect of the germs which enter the tissues is a local necrosis. The latter can only be attributed to the effect of the poisons generated by the bacteria themselves.

It is possible that enough poison may be absorbed into the blood from the original site of the bacterial invasion, so that the endothelial cells of the endocardium are injured. The same may take place by the absorption of a secondary bacterial focus. It may be added that an elevation of the body temperature is often present, which in itself is unfavorable; also that irregularities in the heart's action may develop which favor the longer sojourn of the bacteria which are present in the blood, and which gain entrance into the heart. A further circumstance which may be mentioned is the fact that at times the nutrition of the tissues in general is injured through an insufficient compensation of the loss.

The chief point to be noted is that the power of resistance of the endothelial cells must be diminished to the needful extent in order to allow the bacteria to settle and to multiply upon the site of the disease.

An embolism in the vessels of the valves is a rare occurrence, according to the many investigations which have been made on this question. As a rule, there is a direct invasion from the blood of the heart. The question may be asked, therefore, whether other contributing causes may be demonstrated in addition to the changes in the endothelium. The answer to this is, that anything which mechanically favors the adherence of microbes, also facilitates their penetration. When the endothelium is functionally in the full possession of its powers it prevents the coagulation of the blood. If the latter occurs, and if a thrombus has formed

* "Ueber Wandendocarditis und ihr Verhältniss zur Lehre von der Spontanen Herzermattung," "Deutsches Archiv für klinische Medicin," Bd. xxxiii, 1883, S. 210.

† "Ueber zwei Fälle von Endocarditis parietalis," dissertation, Zürich, 1897.

over the injured spot, then the localization of bacteria at that place is almost certain. This coagulation does take place.*

It is to be further noted that anything which presses the bacteria further into the tissues favors their localization. We have seen above the explanation which Ribbert gives for his experiment. These conditions are not always so simple in man, but Ribbert says correctly that they may at times be perfectly identical. "Large particles of tissues that contain bacteria, such as, for example, detached portions of thrombi, may be thrown against the valves in the same manner as in the experiments reported, and may give up some of the bacteria which adhere to them." In most cases more complicated conditions are present which simultaneously favor the adherence of, as well as the penetration of, germs. This viewpoint facilitates the understanding of a number of the peculiar features of endocarditis.

Virchow† says: "If these observations (greater tension of the pulsating blood within the vessels, especially within the veins, with its influence upon the walls) be applied to endocarditis, then we gain a considerable insight into the history of this disease. For it is the narrow portions of the heart which are subject most frequently to disease, and among them especially those which are exposed the most to friction and tension.

"The valves are the parts of the endocardium most frequently affected first, and in these, again, most often the lines of closure of the valves —in other words, those portions which lie opposite each other when the valve closes, and therefore are exposed especially to friction. This explains why the chief site of the disease is always found upon the side of the valve which lies toward the blood-stream, and not upon the free border, but, in the case of arterial valves, underneath it, in the case of the auriculoventricular valves, above this border. The Arantian bodies are usually affected along with the earlier changes, for they are exposed to friction more than the other structures. Among the valvular structures it is chiefly the anterior flap of the mitral which suffers first and most, inasmuch as it is subjected to the greatest tension in virtue of its attachment at the aortic orifice.

"Next to the valvular flaps it is the tendinous cords which are affected, and they suffer to the greatest extent in those places where they divide into thicker or thinner strands. The inner walls of the heart are third in the order of involvement, and among them first the left auricle, then the right auricle, then the inner surface of the left ventricle, especially the septum, under the aortic orifice, and, last of all, the right ventricle, at times in the cone of the pulmonary artery. The auricular appendages are more frequently affected than the auricles."

Virchow also attributes the greater frequency of endocarditis on the left side of the heart in extra-uterine life to the greater thickness of the muscles of the left ventricle, while the opposite conditions obtain in intra-uterine life.‡

Ottomar Rosenbach§ lays a great deal of emphasis upon the larger amount of oxygen contained in the blood on the left side of the heart.

* See Section on Pathologic Anatomy.

† "Gesammelte Abhandlungen," p. 508.

‡ "Ueber die Chlorose und die damit zusammenhängenden Anomalien im Gefässapparate, insbesondere über Endocarditis puerperalis," Berlin, Hirschwald, 1872, S. 19.

§ "Herzkrankheiten," S. 154.

This, he claims, offers a more favorable culture-medium for the bacteria. This theory has found but few adherents.

Virchow found a hypoplasia of the vessels, especially of the aorta, in a number of cases of endocarditis occurring in the puerperium which had come to autopsy. This change in the vessels also increases the vulnerability of the endocardium on the left side, and the amount of pressure and tension are increased through the greater resistance of the narrow arteries.

No other conditions, aside from these, are known to favor the development of endocarditis. It is possible that a certain weakness of the heart as a whole may be transmitted by heredity, but nothing can be said with certainty as to this.

[Robinson* has made a valuable study of the relation between congenital malformations of the heart and acute endocarditis. He found in the literature seventeen fully reported cases, not taking cases in which only the valves were deformed. The small number he ascribed, no doubt with reason, to the fact that many cases die too early for endocarditis to develop. He finds that the malformation most frequently attacked is the one in which life is most prolonged—viz., obstruction to the pulmonary outflow—and that the right side is especially affected.—Ed.]

It is a universally acknowledged fact that a heart which has once been attacked with endocarditis is very frequently attacked again. It is difficult, however, to give definite data on this subject. Weichselbaum found twelve cases of primary endocarditis against seventeen in which the disease had occurred several times—in other words, a proportion of three to four. It is impossible to say, however, how many of those who died would have had another attack of endocarditis if they had not succumbed to the first. This fact alone renders the comparative figures gained at autopsy doubtful from the start, and diminishes their significance in respect to secondary invasions.

Even clinically it is impossible to give more than rough estimates, and only those cases must be considered which have been observed from the beginning up to the fatal termination, and in which the recurrence or non-recurrence of endocarditis could have been recognized without fail. This is a condition which I regard as impossible of fulfilment. Even if we could fulfil it, a long time would have to elapse in order to secure a sufficiently large collection of such observations.

It has been stated that about three-quarters of all cases show a recurrence of the disease sooner or later. I do not believe that this estimate is excessive.

How should we explain the tendency to a recurrence of the disease? One thing is certainly of importance: the tissue changes which remain after an endocarditis has healed may favor the localization of bacteria in a place which has formerly been affected. This may take place directly, in virtue of the fact that the affected tissues offer an uneven surface which gives a better hold for the bacteria that circulate in the blood, or possibly this influence may be indirect. These places suffer a loss of endothelium over a considerable extent. This favors the coagulation of the blood, which can easily take place upon the destroyed or weakened tissues. Upon these thrombi, the bacteria find good opportunities for settlement. Another circumstance which favors bacterial localization

* "Bulletin of the Ayer Clinical Laboratory of the Penna. Hospital," No. 2, January, 1905, p. 45.

is that, as Köster notes,* the vessels within the thickened valves favor the accumulation of bacteria from the circulating blood. How do the microbes reach their place of activity in cases of recurrent endocarditis? Various possibilities are open: (1) The portal of entrance is somewhere in the body; there may be a fresh invasion from this focus. This is probable in those cases when the same bacteria are found in the focus of entrance, as well as in the newly affected endocardium, and when they circulate in the blood, which is their means of communication, or are found in those organs which favor the accumulation of fine particles from the blood by the arrangement of their vessels (the spleen, the liver, and the kidney). Weichselbaum reports three cases illustrating this point: In his "Case III" the patient was a man aged thirty-six years, who died with signs of severe sepsis. He had had open ulcers in the sole of the foot for some time. Weichselbaum found at autopsy a recurrent ulcerating endocarditis at the mitral and the aortic valves; infarcts in the spleen and in the kidneys; and a double pneumonia. The same bacillus was demonstrated under the microscope and by cultures in the mitral vegetations, in the spleen, and in the granulation tissue of the ulcers in the sole of the foot. Four experiments with rabbits by means of injections of cultures of these germs into the veins of the ear after a mechanic injury to the aortic valves succeeded in three instances in bringing about an endocarditis. The same bacillus was obtained by cultures from the blood and from the several foci in the animals experimented upon. This germ Weichselbaum designated as the Bacillus endocarditidis griseus.

Weichselbaum thought that in this case the bacillus penetrated into the system through the ulcers and produced then a fresh endocarditis, but he did not venture to say whether this germ had also produced the ulcers.

It has not been shown with absolute certainty that the ulcers were caused by the same germ. It is probable that the germ in question caused the first affection of the heart, and that it remained in the endocardium for a considerable time without producing any effects. The ulcers upon the foot of this man had existed for three years before the outbreak of the fatal infection, but I should not draw any conclusions from this fact either for or against the view taken by Weichselbaum. The fact that a rare germ was found in the ulcers and also in the endocardial lesions is greatly in favor of his view. The infarcts in the spleen are also probably to be referred to the invasion of the germ from the diseased endocardium through the circulating blood.

Weichselbaum also published two further observations in which he interpreted the endocardial lesion as due to reinfection. In one of these cases (XIV) the Streptococcus pyogenes was found; in the other (XVIII), the Staphylococcus aureus. Another interpretation is probably correct, especially in the latter case.

We must always remember, in considering these questions, that germs may enter the body without leaving distinct traces at the portals of entrance in the form of tissue changes. Therefore, recurrent endocarditis may also occur "cryptogenetically." Of late a great deal of stress has been laid upon angina which is followed by general sepsis. I have also observed these cases and they are emphasized in Dennig's monograph.† Within the last few years I have observed in several instances that a

* *Loc. cit.*, pp. 274, 275.

† *Loc. cit.*, p. 41. A case in point is case XXII, see below.

recurrence of sepsis, and in one case a recurrence of endocarditis, was preceded by angina.

2. Pathogenic organisms remain at the site of the original infection, either within the primary focus, or in its vicinity, and for a time they do not produce any disturbances, but become capable of infection once more when conditions favorable to this develop. This sequence of events has been demonstrated with certainty in the case of the tubercle bacillus, and it may be affirmed also in the case of the pyogenic cocci.* The following case, at least, speaks in favor of this probability:† The patient was a man aged sixty-three years, who had had an osteomyelitis in the lower diaphysis of the right femur fifty years ago. For a long time there had been a discharge of thick pus, and the knee-joint had become permanently ankylosed. No other disease had occurred during all these years. During the past year the patient had been suffering from "rheumatism," which recurred at frequent intervals, and was accompanied at times by excruciating pains in the right leg. Müller made a diagnosis of recurring osteomyelitis. At the operation, a large cavity was found in the markedly thickened and very dense femur, this cavity being filled with greenish-yellow pus, and, in addition, there were numerous small abscesses in the tibia. Cultures showed the presence of the Staphylococcus pyogenes aureus, which was discovered by Garrè as the cause of the bone abscesses following osteomyelitis. Inoculations into rabbits gave positive results and Müller himself served as an involuntary object of experiment, inasmuch as he developed a large number of small furuncles on his hands after the operation. Garrè,‡ who agrees with Kraske that in late recurrences of osteomyelitis there is a new invasion of bacteria from the blood, supports his contention in the following way: "Let us consider what striking changes in the capillary structures of a bone are produced by severe purulent inflammations, and let us remember that capillaries of narrow caliber or with irregular dilatations (capillary aneurysms) may be present permanently in the cicatrix. Then it will be easily understood that such mechanic obstructions and such local anomalies of circulation do favor most markedly the deposition and localization of micro-organisms from the blood."

In opposition to this view Müller remarks that, in his case, the abscess was surrounded on all sides by newly formed and very dense bony tissue. Therefore the penetration of bacteria from the blood had been rendered impossible. [?]

We do not know a great deal as to the behavior of bacteria in the healed foci of the endocardium. Birch-Hirschfeld§ demonstrated the presence of staphylococci in five cases "in very old and highly thickened and calcareous valves." Ziegler‖ agrees, adding that "under certain conditions bacteria may be found in cases of endocarditis which have existed for a long time." Birch-Hirschfeld does not attribute any special significance to his observations on "calcified cocci," but believes that conditions in old diseased valves are favorable for the localization of

* *Cf.* Graetzer, Hugo, "Dissertation" (Tübingen, 1899), in which the bibliography of this subject has been thoroughly reviewed.

† Müller, Kurt, "Ueber Knochenabscesse," "Archiv für klinische Chirurgie," Bd. lv, 1897, S. 782.

‡ "Ueber besondere Formen und Folgezustände der acuten infectiösen Myelitis," "Beiträge zur klinischen Chirurgie," edited by Bruns, Bd. x, S. 279.

§ "Verhandlungen des Congresses für innere Medicin," Bd. vii (1888), S. 346, 347.

‖ *Ibid.*, p. 348.

bacteria. It is impossible, for the present, to decide whether one or the other possibility is to be considered alone, or whether both must be dealt with. Whoever believes in a fresh invasion must not forget that such an event is by no means ordinarily seen in sepsis. How many patients who had been infected once, no matter how severely, remain free from recurrences, provided their hearts remain unaffected and their lives have been spared! How few, on the other hand, are spared recurrences, whose hearts have been involved! But even this is no proof, and the only way to solve this question is by experiment.

PATHOLOGIC ANATOMY.

The changes *which are seen with the naked eye* are as follows: Transparent, gelatinous, white, yellow, or reddish nodules, slightly elevated above the surface, varying in size from a pin-head to a bean, and adhering to the edges of the valves or irregularly scattered over both surfaces, either isolated or coalescing. Similar nodules are often seen upon the tendinous cords of the papillary muscles. The free surface is often warty, like the comb of a cock. This form has long been called verrucous endocarditis; Corvisart compared it to condylomata. When the portion which lies toward the free heart cavity is covered with larger vegetations, it is styled "endocarditis polyposa" or "villosa."

When ulceration occurs, we find ulcers of considerable size, covered with adherent reddish or yellowish masses, with a sticky, soft surface, which often overlaps the edges. This is ulcerating endocarditis. "When purulent foci become visible in the tissues, the process may be spoken of as pustular endocarditis" (Ziegler). The tissue changes which occur in endocarditis are more important than these old naked-eye classifications.

Ziegler briefly summarizes the appearances found in the tissues:* "The influence of the germs at the site of their localization is probably in all cases a more or less deep-seated degeneration of the affected structures. If the bacteria penetrate deeply into the endocardial tissue (Plate I, Fig. 2 b), beginning at the surface, then there may be in some cases a more or less extensive necrosis, so that the tissues which are occupied by bacteria lose their nuclei (Plate I, Fig. 2 c). As the result of changes in the chemic and physical constitution of the tissues in which the bacteria settle and multiply, thrombi very soon are deposited upon the surface of the infected portions, and in most instances they are finely granular blood-platelet thrombi (Plate I, Fig. 2 d) without cellular elements. At times one finds leukocytes and red blood-cells adherent to these (Plate I, Fig. 2 f), and also threads of fibrin (Plate I, Fig. 2 e), so that mixed thrombi develop.

"The tissues of the aortic and pulmonary valve which do not contain any vessels, as a rule show inflammatory infiltration only at a late stage, because the leukocytes necessarily must emigrate from the base of the valve. If the bacterial foci, however, are situated in the mitral or tri-

* "Lehrbuch der speciellen pathologischen Anatomie," 9. Auflage, Jena, Fischer, 1898, S. 31. "Ausführlichere Mittheilungen über endocarditische Efflorescenzen," "Verhandlungen des Congresses für innere Medicin," Bd. vii, 1888, S. 339.

My friend Ziegler was kind enough to give me two original drawings, which are reproduced herewith (Plate I).

cuspid valves which contain vessels (Plate I, Fig. 1), then inflammatory exudation soon appears, in virtue of which the affected valvular tissue is infiltrated more or less densely with cells (Plate I, Fig. 1, e, f, g). To the exudation from the vessels are added, sooner or later, proliferative processes which produce a more or less abundant embryonic or granulation tissue. This new tissue may grow into the thrombi covering the superficial lesions. Gradually the new tissue takes the place of the thrombi and exudates, so that an endocardial excrescence or efflorescence is formed.

"If the bacteria penetrate more deeply into the valvular tissue without producing an extensive necrosis, we may find a diffuse proliferation of tissue and a formation of granulation which combine to cause a thickening of the valve.

"If tissue-destruction and ulceration have supervened, then granulations form at the edge and at the bottom of the ulcer, and these granulations may penetrate into the thrombi when such thrombi cover the ulcerated surface, and thus the granulations may rise over the surface of the valve. Thrombotic deposits of small size are usually replaced entirely by connective tissue, but many of the larger thrombi, such as occur in ulcerative endocarditis, often remain, shrunken and calcified, provided the thrombus is not detached and washed away earlier, so that the valves may be found covered with hard, calcified, chalky deposits."

It is generally admitted that the first effect of the bacterial invasion is a necrosis of the tissues. Even the experiments of Ribbert show this. This necrosis is followed by other changes, but we must emphasize the fact that a marked change takes place when the necrotic or inflammatory processes are replaced, sooner or later, by proliferative lesions. Köster* describes this sequence with great clearness: "The first effect of the micrococci upon the valves of the heart is a more or less extensive necrosis, while ulceration, purulent and exudative inflammation, and granulating inflammation are only reactive processes which in turn produce the various forms of endocarditis." This view is now generally adopted. Anatomically, we may distinguish between warty and ulcerating endocarditis, provided we admit that we are speaking of things which are not essentially distinct.

Still another question must be considered. Do we find any tissue changes in the endocardium which are not referable to the original action of the bacteria, and which yet resemble in their outward form those changes which are directly due to these germs? Ziegler† answers this question in the affirmative, and remarks that "the warty valvular thrombi which are seen, sometimes upon apparently normal valves, or sometimes upon thickened atheromatous or calcareous valves, are usually situated upon an endocardium in which exudative processes are either entirely absent or at least are present to a very limited extent. The connective tissue upon which they are situated is either unchanged or is degenerated in some way (there is often hyaline degeneration), or else is in a state of proliferation. Regressive as well as progressive changes often occur in the same valvular region, and the latter alterations lead to the formation of connective tissue in the thrombus, and so to thickening of the valve.

I am of the opinion that these processes are incorrectly classified with the bacterial changes under the heading of endocarditis. The process is not characterized by inflammation, but by thrombosis and by an

* *Loc. cit.*, p. 265. † *Loc. cit.*, (*b*), p. 342.

PLATE I.

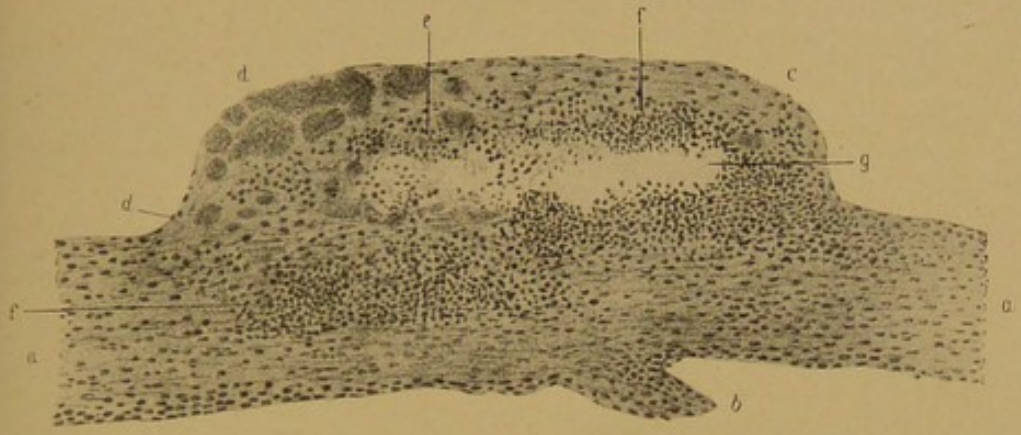


Fig. 1.—Endocarditis mycotica pustulosa of the tricuspid valve following infection from a wound of the left foot (together with hemorrhagic septic pneumonia) (after Ziegler). *a*, Tissue of the posterior flap of the mitral valve; *b*, chorda tendinea; *c*, pustular excrescence on the mitral valve; *d*, staphylococcus pyogenes aureus; *e*, streptococci with pus cells; *f*, pus cells without cocci; *g*, small abscess. From a specimen hardened in alcohol and stained with gentian-violet, iodin, and vesuvin. × 40.

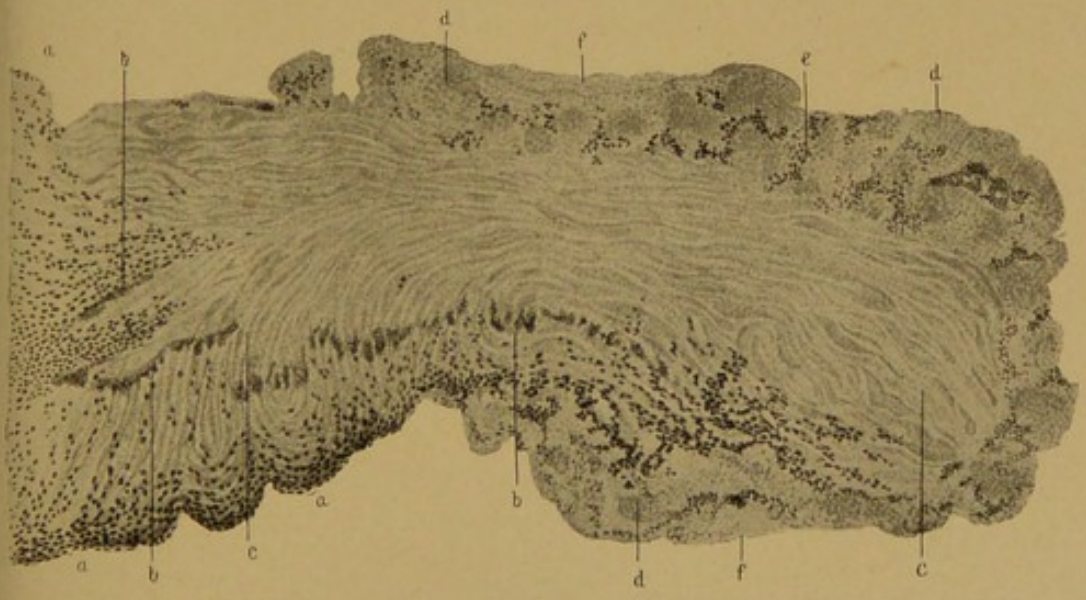


Fig. 2.—Endocarditis mycotica of the aortic valve (probably staphylococcus pyogenes aureus (after Ziegler). *a*, Normal valve tissue; *b*, masses of cocci; *c*, necrotic non-nucleated tissue; *d*, granular blood-platelet thrombi; *e*, fibrin threads with leukocytes; *f*, red blood-corpuscles. From a preparation hardened in alcohol and celloidin, stained with gentian-violet and vesuvin, and mounted in Canada balsam. × 40.

endocardial proliferative growth connected with thrombosis, which is analogous to the proliferative changes in the intima of the arteries and veins accompanying thrombosis and embolism. If we do not wish to speak of warty thrombosis and thrombotic proliferation of the endocardium, but wish to include the process in the great group of inflammatory affections, we can speak of it as a non-mycotic thrombo-endocarditis.

It remains to discuss the origin of these thrombi. Judging by their structures, they consist of a granular substance which is formed of agglutinated blood-platelets. To this may be added a more or less marked amount of fibrin, in threads or thicker strands, which incloses leukocytes and red blood-cells. The thrombi, therefore, consist of elements which are also found in similar deposits in the heart or in the arteries, which develop in the circulating blood and which are also found in artificially produced thrombi. They must also depend for their origin on the same or similar causes as artificial thrombi, namely, changes in the walls, slowing of the current, or the formation of eddies which favor the attachment and agglutination of the blood-platelets on the surface of the endocardium.

Ziegler does not invoke chemic influences as the causes of the non-bacterial proliferations of the endocardium. He considers that the mechanic causes which are present as the result of cardiac weakness are fully sufficient to produce these changes. On the other hand, Harbitz* believes that auto-intoxication, which occurs in the diseases that most frequently accompany these formations, play some part, especially in cancer, tuberculosis, and chronic nephritis. But this explanation is only a hypothesis.

The immediate results of the inflammation of the endocardium on the valves and the papillary muscles are as follows: In the higher degrees of inflammation, which pursue a rapid course, we find perforations in the semilunar valves, detachments from the walls of the aorta (severe endocarditis is very rare in the pulmonary valve), and diverticula toward the side of greatest pressure (valvular aneurysms). In addition, we find in the case of the bicuspid and the tricuspid valves the detachment of the tendinous cords which are fastened to their edges, and which may be destroyed by the process.

Less severe and more slowly running inflammatory or non-inflammatory processes, accompanied by proliferation of connective tissue, lead to adhesions, thickenings with or without calcareous deposits, and to the shortening of the valves. The tendinous cords may also become shorter, thicker, adherent to one another when opposed surfaces are inflamed, and remain in immediate contact for a considerable time. In the end, as always, a shrinking of these tissues takes place, and the result is an insufficiency of the valves and a narrowing of their orifices.

Acute endocarditis may occur at the beginning of the aorta, but usually it is only the ascending portion which is involved, and of this, most frequently that part nearest the heart. The exudative pericarditis which accompanies a severe endocarditis produces such distinct clinical manifestations that they have been long since given full recognition. Bouillaud's work is a proof of this. The change in the pericardium is still more easily demonstrated at autopsy, and thus Bouillaud lays great stress upon the frequent coexistence of endocarditis and pericarditis.

This does not obtain in the myocardium. Severe changes—large abscesses especially—are by no means frequently demonstrable. It is

* *Loc. cit.*, pp. 31, 32.

true that Bouillaud does report such cases from the observation of Corvisart, Laennec, and from his own experience. He says, in this connection:* "Let us note, to begin with, that until now general carditis (myocarditis) has not been observed alone, but always was accompanied by endocarditis or pericarditis."

The present state of our knowledge leads us to the statement that the involvement of the heart muscle is the general rule in endocarditis.

The anatomic forms of this muscular involvement are manifold. There may be true inflammation, with the formation of abscesses, as the result of the reaction of the living tissue against the bacteria which have entered and have produced necrosis. Ribbert has described the process in detail. There may be also foci of softening in the heart-wall which may lead to dilatation—cardiac aneurysm—or even to perforation. They occur with preference on the left side of the heart, in the papillary muscles, or even in the septum. Finally, there may be a variety of degenerations. The detailed description of all these changes belongs elsewhere.

It remains only to be mentioned that the involvement of the myocardium and the pericardium may result from the invasion of germs from the blood, but a direct extension of the inflammation by continuity may also occur. This is the explanation given by Nauwerck in his case, and Ziegler agrees with him.

Affections of other organs are found in the great majority of cases in the bodies of persons who have died of endocarditis. These affections are partly the results of the general infection and partly the effect of the endocarditis. The cavity of the heart in these cases is filled with blood which may easily be mixed with bacteria from its walls, just as the bacteria may be associated with thrombi upon the walls. The constant movements of the heart muscles favor this admixture in a high degree. These masses circulate, then, with the blood which has been driven into the aorta, and bring tissue changes into all places where they can remain for a longer time. The manner in which these changes develop depends upon the nature of the emboli brought to the tissues. When bacteria play a rôle in the process, the principle, which is now everywhere recognized, that the character of the lesion depends upon the number and the virulence of the germs, holds good.

It is to be noted that the species of bacteria is of importance in determining the character of the metastases. Weichselbaum,* in discussing the observations of Wyssokowitsch and Birch-Hirschfeld, agrees with them that an endocarditis due to streptococci usually produces an anemic necrosis in its secondary foci, while an endocarditis produced by staphylococci usually results in pus-formation.

In addition to this, the "bland" emboli produce chiefly a mechanic effect, which may be combined with the pathologic changes mentioned above, or which may in itself lead to severe consequences, especially when large arterial branches are closed by occluding thrombi.† The spleen, the kidneys, and the brain are especially exposed to embolisms, and most frequently show the corresponding changes—infarcts and abscesses with their sequels.

A question arises here: Could not the marked participation of the entire heart in endocarditis be brought about by the fact that the blood

* *Loc. cit.*, p. 292. † *Loc. cit.*, pp. 214, 215.

‡ Virchow, "Gesammelte Abhandlungen," Cases 8 and 9, pp. 426–433.

which has been infected with bacteria is pressed directly out of the heart cavities into the coronary arteries which arise at the beginning of the aorta? Mechanically, this is possible, but the conditions are too complicated to permit a well-founded hypothesis.

Other tissue changes which accompany endocarditis are best considered in the chapters on the diseases of the respective organs. We need only add here that the bodies of persons who die of endocarditis present the appearances indicating a severe general infection or an insufficient heart's action, and often a combination of these two factors. [An important addition to the study of endocarditis has been made by H. Königer, "Histologische Untersuchungen über Endocarditis," "Arbeiten aus dem path. Institute zu Leipzig," 1903, H. 2.—Ed.]

CLINICAL FEATURES.

The clinical picture of endocarditis is extremely variable. It is composed of: (1) The participation of the body as a whole (infection); (2) the disturbances in the heart; (3) the affection of one or another organ which takes place at the start, or at a later period.

Each of these series of phenomena may be more or less prominent; one or the other may be seen at the beginning as the chief feature of the clinical picture, and may be so prominent that but little, or at times nothing, else is demonstrable besides this one factor. If the patient dies within a short time, nothing further is observed, but if he lives for a longer time, it is rare to find that the further development of the clinical picture in one or the other direction is lacking.

A classification of endocarditis which is practically useful to the physician becomes more difficult, in proportion to our knowledge of the disease. The classification cannot be based upon the variety of germ causing the disease; for no germ produces such marked differences in the clinical picture that these furnish data for a classification. Neither do the various species of germs give rise to essential differences in the course or the termination of the disease.

Anatomic studies have taught us that the distinction long observed between ulcerative and warty endocarditis is impossible. The anatomic changes in the endocardium are, in general, not the sole criteria, because even in cases which rapidly lead to death only the beginning of tissue change may be found, and no ulceration whatever. The classification, therefore, must be clinical, and cannot be expected to be based upon sharp and distinct characteristics. We must be content to distinguish a severe form from a mild one, and be satisfied with the scantiest characterization. We must remember, also, that a mild infection may become severe, and that one which appears severe may grow milder. If we admit the expressions "benign" and "malignant," they must be applied only to a definite period of time, for that which applies to-day is no longer valid to-morrow. We must always bear in mind the possibility of a rapid change in the clinical condition. From all this, it appears that there is no "typical" clinical picture of endocarditis. Some very acute cases begin suddenly, with chills, rapidly rising high temperature, extreme loss of strength, and almost complete clouding of the senses. These conditions may remain to the end, which may occur within a few days. The following is an example:

Case 1.—A man aged fifty-eight years; previous history unknown. At midnight between the twenty-seventh and twenty-eighth of February, 1890, he was suddenly taken with a severe chill. In the morning he tried to get up, but could not stand on his feet and was obliged to return to his bed. As there was an epidemic of influenza at that time, it was thought that he had this disease. His friends, therefore, waited until the twenty-first of March, at noon, before they applied to the polyclinic. "The patient coughs a great deal, and does not seem to be well."

On examination, the very robust man, semiconscious, murmured to himself and did not take any notice of his surroundings. When called, he answered and spoke a few intelligent words, but immediately began to wander—asked to go to the field, as he had a great deal of work to do, etc. Both feces and urine were passed involuntarily. He attempted to help when he was undressed for the examination, but after a brief effort collapsed. The respirations were difficult—39 per minute; the pulse was 110, irregular, the peripheral arteries atheromatous. On percussion of the lungs a slight dulness over the right lower lobe behind. On auscultation in the same area there was soft bronchial breathing below the angle of the scapula and fine moist râles, both on expiration and inspiration. Over the rest of the chest there was sharp vesicular breathing with a few dry râles. The heart dulness was normal. The heart tones seemed clear, but could not be heard very well on account of the loud breathing. Palpation of the body (muscles, bones) elicited no evidences of pain.

7 p. m.: Complete coma. The pupils, which had reacted at noon, were very narrow and did not dilate to any extent when they were shaded. The patient received a cold bath and it was reported that he had slightly revived after that. The nurse noted that the limbs of the patient were very stiff and could scarcely be bent during the bath.

11 p. m.: The condition of the patient remained the same, but the pupils were widely dilated. He died at 3 a. m. March 31st without remission of the coma. The temperature, which was taken every two hours, took the course indicated in the chart (Fig. 25).

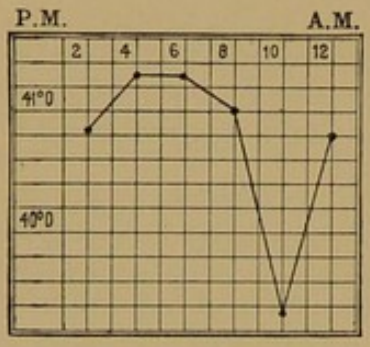


Fig. 25.

Autopsy.—Twelve hours after death, by Professor von Baumgarten; Extract from the record.—"The rigor mortis is very marked. There are numerous small petechiæ in the skin. The heart is moderately enlarged; the muscles of the right ventricle show a slight degree of hypertrophy. There is slight insufficiency and stenosis of the mitral valve, produced by the shrinking of the connective tissue of the valve in the longitudinal and transverse diameter, as well as by an adhesion of both flaps with a calcified bridge of connective tissue. A few gray, fibrin-like excrescences are found at the free lower border of the aortic flap of the mitral. The tendinous cords of the flap are markedly thickened. A soft, gray, slightly adherent deposit is found over a very limited area, at the boundary between the posterior and the left aortic valve. The heart-muscle shows no hemorrhages and no embolic foci.

"The right lung shows, about the middle of the lower lobe, two hemorrhagic foci, about one square centimeter each. The left lung shows a hemorrhage at about the middle of the lower lobe. The spleen is somewhat enlarged (14 cm. long), dark-red, somewhat toughened, but still softer than in stasis, and shows no hemorrhages or infarcts. The kidneys show numerous hemorrhages, up to the size of a pin-head, which lie chiefly in the outermost cortical zone, and which are characterized frequently by yellow centers. Here and there there are also minute yellowish foci, which are surrounded only by a very narrow hemorrhagic area. Numerous small hemorrhages are found upon the mucosa of the stomach, with correspondingly large erosions. The brain is congested and edematous. The liver is congested."

Report of the bacteriologic examination: "Cover-glass preparations were made during the autopsy from the endocardial deposits, and on staining they showed numerous streptococci in pure culture.

"Plates of agar-agar which were inoculated with the same material and were kept in a thermostat at 37° C. showed a large number of colonies of streptococcus, of which some were inoculated into gelatin broth, and produced an absolutely pure culture of streptococcus. Sections were made from various portions of the lesions in the endocardium which had been hardened in alcohol, as well as from the foci in the kidneys which had been noted on naked-eye inspection. These were stained according to the method of Gram-Günther. The endocardial preparations showed

the presence of fibrinous deposits which were studded with numerous masses of streptococci, while the tissues which lay beneath them were in places markedly infiltrated with small round-cells. A necrosis of the valvular tissue was not found.

"The sections of the kidneys showed numerous emboli of streptococci in the smaller arteries, particularly in the region of the cortex, around which there were hemorrhages of various sizes, and in some places extensive epithelial necrosis of the convoluted tubules, while the glomeruli of these portions as well as the straight tubules showed a distinct nuclear stain throughout. In the neighborhood of the emboli composed of streptococci there was only a slight inflammatory reaction." Great masses of streptococci were also found in the hemorrhagic foci in the right lung.

This is a clear case of most severe endocarditis, which terminated fatally within seventy-five hours. It began undoubtedly in the endocardium of the valves on the left side of the heart. The mitral had been affected previously to a slight degree, but there had been complete compensation. The patient continued to do the exhausting work of a grape-gatherer until the last, and only a slight hypertrophy of the muscular structures of the right ventricle was needed to enable him to go on with his work. And yet, it was the valve which had been injured many years previously which became death's portal of entrance.

The general symptoms did not differ from those which occur in every severe infection. A positive diagnosis was, therefore, impossible. One was obliged to think, at first, of a pneumonia which had not yet fully developed: a sudden onset with chills, high fever, a ratio between the respiration and the pulse as 1 is to 2.8, and the physical signs in the lower lobe of the right lung which possibly pointed to a beginning localization.

A *slow onset* is more frequently noted; lassitude, indefinite, often wandering, "tearing" pains, slight chills or chilly sensations, aversion to mental labor, still more markedly present than disinclination to physical activity, depression, and loss of appetite. There are also sleeplessness and distinct emaciation. The patients continue in this manner for days, and sometimes for a week or more, without complaining of any other symptoms than the above-mentioned signs of general malaise.

The physician who is called at this time always finds, I believe, some changes in the course or in the elevation of the temperature; often sensitiveness on pressure over the long bones; sometimes a slight swelling of the joints, and pains in the legs, which, however, do not interfere with walking. The spleen may possibly be slightly enlarged. The signs on the part of the heart can only be brought out by very careful examination, and consist usually in a slight irregularity in the rhythm and tension of the pulse, or still more frequently in an insufficient circulation, a cold nose, cold ears, slight cyanosis of the skin, especially on dependent fingers and toes. There may be no other developments, and gradually, but only in the course of weeks, the patient improves. But the valvular disease which fully develops in time—months afterward—teaches us what the real cause of the trouble had been.

It is true such a seemingly favorable course is not always observed. After a time the temperature rises suddenly, with more or less marked chilly sensations, up to a distinct chill, or else it rises slowly from day to day. The general condition of the patient, his nutrition, and his strength suffer markedly. The dulness over the spleen becomes larger.

By this time there are generally some signs in one or another of the organs, due to the development of the bacterial infection, and the earliest of these are seen in the joints, the bones, and the muscles. But friction-

sounds may also be heard over the liver, the spleen, and the pleura, although they are usually coincident with the other manifestations mentioned.

Hemorrhages into the skin, usually petechiæ of small size, may also occur, and in some instances there may be various kinds of exanthemata (erythema multiforme). The heart-muscle grows weaker, and as this goes on the cyanosis of the skin, the coldness of the hands and feet, and the imperfect filling of the arteries grow worse. The irregularity of the rhythm which has been present from the beginning becomes more distinct. The heart-sounds which were at first somewhat low and muffled, but still pure, become changed, grow less well defined, become reduplicated, or are accompanied by murmurs which are usually variable in their location and their intensity. At times there may also be true or false pericardial friction-sounds. The heart increases in size, as is shown not only by the enlargement of its area of dulness, but also by the position of the apex-beat. Secondary affections may also develop from the heart through the detachment of larger fragments, or the propagation of bacteria which multiply abundantly on the endocardium. In this manner new foci develop here and there, and the picture grows more and more similar to that of sepsis. The individual case is characterized by the peculiarity that the affection of the embolically involved organ is marked enough to dominate the entire scene. This occurs, for example, when an embolism occurs in the brain or when an acute nephritis develops—these two being examples of a coarser and finer secondary involvement. As evidence of this I present the following case:

Case II.—A mason, aged eighteen years, was admitted on November 6, 1890. Eight days before he had slight sore throat and general malaise. After a few days of improvement the symptoms grew more severe, so that he was obliged to stop working. The tonsillar abscess burst on the eighth, and although the slight fever disappeared, the general condition remained poor. The patient did not complain of anything special, but was apathetic and very clumsy in his movements. On the tenth he developed a peculiar affection of the striped muscles, which will not be described in detail for the present.

Let us confine ourselves to the behavior of the heart. Repeated examinations, the last of which was made on the eleventh, showed the absence of any disturbances. It was not until the thirteenth, with a temperature of 37° C. (98.6° F.), that the pulse rose to 84 and became slightly irregular. On the following day there was pain on pressure at various points over the long bones, and a slight elevation of temperature. On the fifteenth, there was some cyanosis and slight undulation of the veins in the neck, as well as an extension in the area of dulness over the heart (probably due to the retraction of the lungs), but the irregular pulse varied within a few minutes between 84 and 107. The heart tones were pure. The cyanosis improved considerably until the seventeenth of November, while the venous pulse had disappeared entirely. The pulse now grew more quiet and more regular, but on the first of December irregularity was again noted and gradually increased. On the sixth the pulse became interrupted at every tenth beat. The use of digitalis was now thought to be indicated, and a good result was obtained by the administration of two grams in infusion.

The extension of the area of dulness to the right, a finger's-breadth beyond the right sternal border, was noted for the first time on December 15th, but the apex-beat was still within the mammary line in the fifth intercostal space. The heart tones, which had been pure until then, now became somewhat muffled. The heart's action remained abnormal, and became distinctly less vigorous and less regular. Thus, on December 24th, the pulse showed, in as many consecutive minutes, 120, 108, 105, 96, and 102 beats. It is not necessary to give in detail the very accurate observations which were later on made in this case: There were frequent variations, but no more severe disturbances of the heart than those already related. It was noted, however, that the frequency of the pulse gradually increased, and that it began to decrease only at the end of the eleventh week of the disease. The following table, which was

made up from the regular morning counts of the pulse, shows the behavior of these variations:

TABLE OF PULSE-RATE IN CASE II.

Time.	Average.	Maximum.	Minimum.
First week	73	102	70
Second "	74	84	63
Third "	77	90	69
Fourth "	89	99	84
Fifth "	107	120	90
Sixth "	116	120	108
Seventh "	111	124	93
Eighth "	106	141	87
Ninth "	118	138	96
Tenth "	116	132	108
Eleventh "	124	132	118
February 1 to 7	100	—	—
February 8 to 11	85	—	—

The greatest danger in this case lay in the nephritis (see p. 245), which became evident as early as the fourteenth of November through complete anuria, which continued for thirty hours and was accompanied by severe cerebral disturbances —uremic coma. The heart had kept up during this time,—the third to the fifth of January,—and only grew weaker again in the following days. It was probable, however, from all appearances, that the insufficient work of the heart was an important factor in the distribution of the dropsy which followed the nephritis. The patient was discharged in April, 1891, and in May of the same year he resumed his work. He even believed that he had gained strength as compared with his former condition, and considered himself in good general condition. He died on February 4, 1892, from acute yellow atrophy of the liver. There had been no diminution in the heart's action during this short fatal illness.

The autopsy (Professor von Baumgarten) showed the following old lesions: The heart was somewhat larger than the fist. The mitral valve showed small fibrous nodules on and above the line of closure, and the free border of the aortic valve was somewhat thickened and rounded. This shows that the first disease, which was a septic infection from the tonsils, was accompanied by an endocarditis, the presence of which had been suspected. The recovery was complete, as the function of the valves had been restored. The sepsis proper at that time had not been a very severe one, and the general disease was of comparatively milder character. The maximum temperature was 39.3° C. Life was threatened by the nephritis, but even the kidneys recovered, so that nothing was left of the older changes except here and there narrow bands of increased interstitial tissue. Bacteria which could be connected with either disease were not found.

The disease may remain *stationary*. If it tends to a fatal termination, the outcome depends either upon the general disease, which is manifested by fever in the last stage, or by failure of nutrition and cachexia. In these circumstances the cause of death is not the affection of one definite organ, but a fatal ending may take place more or less rapidly as the result of the involvement of the heart, of the brain, or, most frequently, of the kidney. These cases constitute what may be called the septic form of endocarditis.

An *insidious course* is usually noted when endocarditis follows acute articular rheumatism. No definite time can be given for the onset of this endocarditis. Possibly it begins with a feeling of palpitation of the heart, or even merely with the sensation that a heart is present, but if the patient who complains of these sensations be examined, we rarely find anything.

The most positive sign, in my opinion, is the disturbance of the heart's action as a whole, which will not be easily missed by those who examine for it daily, and who carefully note all the signs even at their very onset. We must always pay attention to respiration in these cases; for very painful joints which feel any marked jolt or even the unavoidable change of position of the body during deep inspiration, will force the patient to very slight expansion of the lungs. The results are cyanosis, an increased frequency of the pulse, and a visible movement of the blood in the veins of the neck. The enlargement of the heart area induced through the retraction of the lungs, which are but slightly distended by the respiratory muscles, and the increased intensity of the diastolic tones over the pulmonary artery, all contribute to the diagnosis of an endocarditis, localized in the mitral, when systolic murmurs can be heard. Nothing can be determined from the behavior of the temperature, so long as the swellings of the joints continue to be present. When these have disappeared, then, indeed, any rise or any abnormal distribution of the temperature during the day will point to the involvement of the heart. As a rule, the course of these cases is not a very acute one and the clinical picture is limited to the changes in the heart with their effects upon the circulation. But even here we find exceptions: severe general illness and widely distributed metastatic local affection.

We must emphasize once more how difficult it is to determine the boundary which separates septic infection from acute articular rheumatism. Does such a boundary exist, anyway? I have already expressed my opinion (see p. 182).

It is only when the heart shows changes which permanently diminish its functional powers that we can judge from the clinical picture of endocarditis whether a primary or a recurrent affection is present. All these changes, whether they affect the muscular mass, the valves and orifices, or whether they involve adhesions with the pericardium, exercise their own effects upon the work of the heart.

These old lesions may produce such a marked effect in a very short time that a cardiac failure results in the course of a rather slight recurrent affection, and rapidly leads to a fatal end. If it were possible to follow more closely the various attacks or a recurrent endocarditis, the steadily increasing succession of lesions would be shown clearly enough. An example for this very frequent type of the disease is the following:

Case III.—The patient was a man thirty-four years old (at the time of his death) who was first attacked with acute rheumatism when he was eight years of age, and this attack obliged him to remain in bed for almost six months. Repeated relapses occurred, but were less severe, and nothing could be discovered about their exact number. The affected joints remained painful and there was palpitation of the heart for some time afterward, together with a distinct lowering of nutrition. In his ninth year he had chorea.

First admission to the policlinic, June, 1876.—He was then a boy of thirteen years, with large bones but somewhat flabby muscles, and but little fat. His heart was dilated considerably in both directions, and measured 17 cm. transversely and 11 cm. vertically. The area of dulness extended on the left side from the upper margin of the second rib outward to the anterior axillary line, and downward to the fifth rib. On the right side it extended from the same level as on the left, and outward to the extent of 5 cm. across the median line. The apex-beat was markedly diffuse, prominent, and situated in the fifth intercostal space, being visible out to the anterior axillary line, but most distinct at 3 cm. below the nipple.

The lungs were not retracted. The liver dulness began at the upper margin of the sixth rib. On auscultation, a loud systolic murmur was heard over the apex; no diastolic tone was made out. The aorta showed, on auscultation, at first an im-

pure systolic tone, which in the course of a few days became a distinct murmur that was transmitted into the subclavian and the aorta. The pulmonary valve showed a markedly increased second sound. The pulse was 128 as a maximum, regular, and its frequency was independent of the temperature. It was 124 at 40° C. (104° F.); 128 at 38.5° C. (101.3° F.); and 120 at 37° C. (98.6° F.). There was no swelling of the joints, but there were muscular pains and a stabbing pain in the region of the heart. These symptoms rapidly disappeared. Salicylic acid was given, beginning on the fourteenth of June.

Second admission—from December 7, 1893, to March 3, 1894. It was impossible to determine with certainty whether a relapse of the disease had taken place in the interval, as the patient could not state definitely the years when he had had attacks. He was then thirty years of age, had selected the trade of a carpenter, and had been able to pursue the hard labor connected with this trade in spite of the fact that he had been troubled at times with shortness of breath and with palpitation of the heart. His body developed according to his conditions of living. He was a "sturdy man of middle height with very strong muscles," we find stated in the history taken on December 7th.

For two weeks before admission he had been feeling indisposed and very tired, and during the night between the sixth and the seventh of December he developed fever and acute pains in the joints of the feet and hands. There was swelling, redness, and tenderness on pressure in both ankles and wrists, and to a lesser degree in the elbows and knees. These symptoms disappeared completely after the use of small amounts of sodium salicylate for two days, and they did not return.

The heart was dilated in both directions. On the left side the cardiac dulness began at the lower border of the second rib, and extended two finger-breadths across the mammary line. On the right side it extended to the right sternal border, beginning at the second rib. The apex was in the sixth intercostal space, outside of the

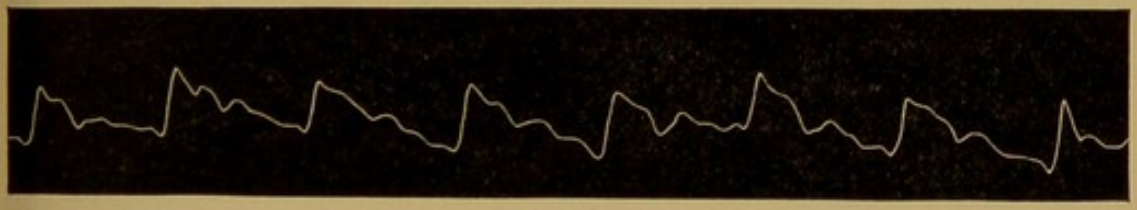


Fig. 26.

mammary line. On auscultation there was a loud systolic murmur at the apex. Over the aortic area the signs at first were ill-defined, but after a few days a distinct diastolic and a less distinct systolic murmur appeared. The pulmonary second sound was markedly accentuated. In addition to these signs of aortic insufficiency there was a rapid pulse, a double diastolic sound over the crural artery, and a capillary pulse at the finger-nails.

On admission, the pulse was still regular and vigorous, but eight days later it was found to be irregular (Fig. 26). This irregularity continued to the end of the observation, and at times was actually intermittent. The different days of the disease offered considerable variations in this respect. When the patient remained perfectly quiet, the pulse was different from that observed when he exerted himself even slightly. The temperature was measured three times daily until February 9, 1894, but during the last week it was taken only twice daily.

During all this time, there were only two slight elevations recorded (maximum 38.2° C. (100.7° F.)—December 24, 1893, to January 23, 1894), not counting the rise of temperature at the beginning (maximum 39° C. (102.2° F.)). On the whole, there had been rather subnormal temperatures. Before the patient was discharged the heart was carefully examined once more. The dulness had become somewhat less extensive, and was diminished by one intercostal space above, while the apex-beat had become elevated to the extent of one intercostal space. It was especially interesting to note that the respiration had a marked influence upon the pulse, and that during inspiration the pulse-wave was weaker. The murmurs were distinct as before, and a systolic bruit was heard over the carotid. The general condition and the strength of the patient were satisfactory, so that he could pursue his work fairly well.

Third admission—from November 6, 1894, to March 7, 1895. Since his last sojourn in the hospital the man could go on with his trade without marked difficulties

on the part of the heart. It was only for the preceding fourteen days that he had felt some pain in one or another of the joints. During the night from the fifth to the sixth of November he had been very feverish, with slight chills and severe pains in the joints. On the left side, the ankle, the knee, the wrists, and the shoulder were involved, and in the morning the right arm and leg also began to trouble him. Medical attendance was immediately requested.

On admission he showed a marked diffuse erythema of the skin on the forehead and the face, and a more circumscribed erythema upon both palms, while the legs showed scattered spots of the size of a lentil. Almost all the joints became swollen after a few days, reddened and painful on motion and on pressure, and even the articulation of the jaw on the right side took part. There was, in addition, at first a slight, later an increasing, sensitiveness to pressure over the shaft of the humerus, the radius, and ulna, over the thigh on both sides, and the muscles of the arm. Salicylic acid, which had been used before against these symptoms with quick success, produced this time a less distinct improvement.

In the further course of the disease pleurisy developed on the right side on the fourth day, the exudate being rather small. On the sixth day a pericarditis with a moderate exudate was discovered. On the sixteenth to the twentieth day there was a severe diarrhea. On the fifth day for the first time, and then repeatedly at frequent intervals, there were attacks of cardiac asthma, which increased to a veritable angina pectoris.

The heart did not show any great difference as compared to its condition noted previously, except the signs of pericarditis. The pulse, which was frequently irregular, to a slight extent became weaker, and there was a distinct diminution in the working capacity of the heart.

The temperature was taken three times daily from November 6th to March 6th. I shall speak again on this point later on, but shall mention here only that the maximum was only 39.1° C. (102.4° F.) even when pleurisy and pericarditis developed. The symptoms lingered a long time, and again and again the pains in the joints returned. The last elevation of temperature was noted on February 21st (38.4° C.—191.1° F.). The patient was discharged with considerably diminished capacity for work.

Fourth admission—from September 22d to October 14, 1897. He had been getting along fairly well for the preceding two years, and although he could not work as well as formerly, he had not been seriously ill. He continued at his trade in spite of temporary discomfort through palpitation of the heart and shortness of breath.

The local symptoms over the heart were scarcely changed, but the pulse had become notably irregular in both strength and rhythm. There was slight cyanosis, but no edema nor swelling of the joints and no fever. Rest in bed and an ice-bag over the heart sufficed to relieve the patient of his complaints within a few days, and to regulate the heart's action. He was, therefore, discharged with a warning to take care of himself.

Fifth admission—from the twenty-fifth of October until death on the tenth of November, 1897. The patient had been so well in the interval that he had been doing light work in the field and about the house. But in the last few days he had been suffering from great fatigue, loss of appetite, and a feeling of fulness in the region of the stomach, together with palpitation of the heart.

On examination he showed cyanosis, venous pulse in the neck, enlarged liver. There was no edema, but the pulse was less full and was irregular. Everything pointed to failure of the heart's action. These signs increased, and as early as the fourth of November there was an attack of cardiac asthma which recurred with increasing intensity on the fifth, the seventh, and the ninth.

These attacks were accompanied by a tendency to syncope. The secretion of urine was diminished, and on the sixth amounted to 300 c.c. The specific gravity was 1026. For the first time there was a slight amount of albumin. There was some edema of the legs.

The cardiac dulness began to spread, from the fifth to the eighth of November, to the extent of 4 cm. in each direction, while the apex-beat moved 2 cm. outward, the pulse became smaller, and there was a continuous marked dyspnea. The amount of urine secreted during the last day was 200 c.c. In the morning of the fatal day there was great excitability; the patient jumped out of bed, ran about the room, and was subdued with difficulty. Coma followed, and death came easily. The temperature on the sixth was 38.4° C. (101.1° F.) and on the ninth 38° C. (100.4° F.), —that is, slightly elevated,—but on other days it had been slightly subnormal.

Autopsy (Pathologic Institute; Privatdocent Dr. Henke; abstract).—"The cadaver of a strong man, well nourished, with well-developed muscles. Edema of both

lower extremities, especially in the region of the ankles. The pericardium lay exposed to the extent of an outspread hand. When I attempted to open it, a loose but extensive adhesion between both pericardial layers was revealed. The heart was enlarged to the extent of two and one-half times its normal size. Two fingers could be passed through the mitral valve, and three fingers easily through the tricuspid. The right ventricle was strongly hypertrophied, the walls averaging 5 mm. in diameter and the trabeculæ being thickened. The left ventricle was markedly dilated and hypertrophied (12 mm.). The aortic valves did not close when water was poured into the aorta. They were shortened, their borders were markedly thickened and rounded, while at several points there were delicate fresh warty formations. There were also thickenings at the line of closure of the mitral, in some places chalky deposits, and, here and there, very delicate warty formations.

"The lungs and the brain were edematous. The kidneys were normal, except that there was a fresh hemorrhage about the size of a dollar on the left side between the capsule and cortex. The mucous membrane of the stomach and of the small intestine was congested, covered with tenacious mucus, and showed numerous hemorrhages. The liver was of the cyanotic 'nutmeg' type. The spleen showed no changes and was not enlarged."

My colleague, von Baumgarten, gave me the following data concerning a more detailed study of the heart in this case: "There were no fresh lesions anywhere. The small excrescences on the endocardium consisted of connective tissue at their base, while the superficial fibrinous layers were perfectly free from cocci or other bacteria. The myocardium, when examined in the fresh state, showed only a slight fatty degeneration of the muscle-fibers. No foci of myocarditis could be discovered with the naked eye at any point. On the other hand, small patches of new connective tissue were found on microscopic examination at various points in the left



Fig. 27.

ventricle, patches in which the muscular fibers had been completely destroyed—in other words, foci of so-called fibrous myocarditis. But in none of these was cellular infiltration noted, and in none were there any cocci or other bacteria to be found. It is very possible that these fibrous foci were residues of previous acute (infectious) myocarditis, but we have no proof of this in the histologic picture, which may be exactly identical in a chronic myocarditis due to arteriosclerosis."

In this case I had the patient under observation for over twenty-one years, and his first attack was of even earlier occurrence. In spite of his severe valvular lesions, this man had been able to pursue his trade, which certainly was not an easy one. He had lived through many good years, but afterward there came a second and a third serious relapse. He, fortunately, recovered from both, and there followed a period of rest for two and a half years, during which his capacity for work was diminished somewhat, but still remained fairly good.

During the last months of his life a much more distinct failure of the heart's action came on—whether with or without a renewal of the infection we cannot say. Finally, death occurred with symptoms of subacute heart failure. The sphygmogram (Fig. 27) shows diagrammatically the condition of the heart on the day before death.

It is noteworthy that the clinical picture which during the first two attacks resembled that characterizing acute rheumatism accompanied by endocarditis deviated considerably from this type, beginning with

the onset of severe relapse (1894–1895). From that time on, salicylic acid proved of no avail.

We shall find abundant opportunity to discuss the variable nature of the clinical picture in this case in speaking of the individual symptoms. Let us, therefore, turn to the consideration of these.

INDIVIDUAL SYMPTOMS.

FEVER.

The temperature is so variable in endocarditis that, in order to include every possibility, one must say that there is no type of fever which may not occur in this disease. A somewhat better understanding on this question may be gained by turning from the variable form of temperature-curves to the conditions under which they occur. It is true, however, that such a conception must be superficial; the essential nature of the phenomena remains unknown.

An essential factor in the study of fevers is a recognition of the constancy of the normal body-heat of a healthy man.* This normal temperature in the average person shows but slight variations from 37.2° C. (98.6° F.) in the twenty-four hours, taken by the rectum. As a rule, the temperature rises from morning to evening, and sinks from evening to morning, and usually temperatures exceeding 38° C. (100.4° F.) do not occur in healthy persons, provided they do not do excessive muscular work. But while this is the fundamental law, there are exceptions, in which both the distribution of the temperature over the various parts of the day, as well as the absolute values of the individual measurements, are altered for a short time. [Herbert French points out the important fact that in subjects of old valvular disease the temperature is often low. If with such a case infectious endocarditis is associated, a rise of several degrees will produce, seemingly, only a normal temperature or very slight fever.—Ed.]

The next thing to be considered is the action of disease-producing causes upon this normal temperature. In general, it may be said that these causes produce a rise of temperature, the extent of which depends, in the first place, upon the character and severity of the pathogenic factor. The greatest variability may be noted in this respect, seeing that living organisms are the causes of infections.

The proof of the connection between the variations of temperature and the cause of a disease is distinctly furnished in the case of the plasmodia of malaria, the appearance and disappearance of which produce rises and falls of temperature in the affected subject. The examination of the curve of a simple tertian fever teaches that low subnormal temperatures appear for a time after the termination of an attack—manifestations of the tendency to reëstablish the normal daily average.

It is probable that the same condition obtains in septic infections.

* My investigations at Kiel showed to what degree this obtains. These researches were published in the monograph, "Die Körperwärme des gesunden Menschen," Leipzig, F. C. W. Vogel, 1873. The observations made in healthy soldiers in Ludwigsburg by Dr. Heinrich Jaeger ("Ueber die Körperwärme des gesunden Menschen," "Deutsches Archiv für klinische Medicin," Bd. xxix (1881), S. 516) agree with this in all respects. The fundamental rules are also adhered to in children from one to three years of age. *Cf.* P. Steffens, "Beiträge zur Pathologie der Masern" (aus der Tübinger Poliklinik, "Deutsches Archiv für klinische Medicin," Bd. lxii (1899), S. 330).

The invasion of the bacteria of sepsis is followed by an increase in body temperature, sometimes to such an extent that the temperature runs a course similar to that noted in intermittent fever or with the continuous high fever which obtains in the severe forms of malaria. But there may also be only slight and transient fever, just as is seen in chronic insidious cases of malaria, and sometimes these rises of temperature may only be recognized by the most careful examinations.

The duration of life of pus-cocci which have entered the body is not a definite one, nor is their power of reproduction and the increase or decrease of their virulence regulated in any manner known to us. If we add, that the resistance of the patient is variable to begin with and is diminished to a very variable extent by infection, we may easily understand why the course of fever in these cases is so extremely variable.

I have gained very important indications for the treatment of localizations of septic processes in the heart by the careful study of the temperature, and these criteria may be gained especially from slight rises of body-heat. Therefore a detailed consideration of this subject is necessary, not only from the viewpoint of clinical investigation, but also from the aspect of practical medicine.

There are cases of endocarditis which are due to sepsis in which the development of valvular disease is not accompanied by rises of temperature at any time. Here I must admit that this is true only so far as may be determined by measurements in the rectum three times daily—in the morning, at noon, and in the evening. These measurements are sufficient in the great majority of cases. [But they do not indicate the actual course of the temperature. In obscure cases the temperature should be taken every two hours and a graphic chart should be made.—Ed.] It is very improbable that even occasional transient rises of temperature, say to 39° C. (102.2° F.), may occur at other hours of the day, and this is especially improbable when nothing in the curve indicates such rises for many months. The patients remained in bed during the entire period of observation, which at times lasted for months. The following is a short summary of a case of this kind:

Case IV.—The patient was a boy aged fourteen years, who was admitted on October 7, 1898, and discharged on June 21, 1899. The previous history showed the occurrence of measles and of pleurisy on the right side. During the latter part of August he had been complaining at times of palpitation of the heart and of a stabbing pain on the left side, after bathing in the Neckar. In the early part of October these symptoms grew worse, and continued even during rest. There were, in addition, headache, sleeplessness, and possibly some loss of flesh and strength.

On admission there was moderate chorea. The heart was not enlarged, but its action tumultuous, irregular, and the systolic sound over the aorta was muffled. On the left side the bones of the arm and of the leg were painful to pressure. Four days later marked symptoms developed on the part of the nervous system—catalepsy, peculiar disturbances of the reflexes, etc. I shall return to the discussion of this group of symptoms further on.

During the first week the signs of heart failure developed: Venous pulse, cyanosis, cold limbs, nose, and ears, in the well-heated room. Then, during the second week, there were circumscribed hemorrhages into the skin and sensitiveness on pressure over a greater number of bones. The pulse became still more irregular and numbered, on one day, for example, 90, 76, and 60 beats respectively, in three consecutive minutes, although the patient was perfectly at rest and had not moved for some time before. The respiration became irregular, and a slight increase in the cardiac dulness slowly developed.

On December 20th the dulness was found to have spread upward to the extent of one intercostal space, and to the right to the extent of 2 cm., while the apex-beat had moved over the same distance outward. The systolic bruit over the aorta was

always present—at times more distinct, at times less marked. There was never any albumin in the urine, and the amount of the latter, observed for six weeks, varied somewhat on different days, but did not deviate a great deal from one liter. The specific gravity varied more extensively, as, for example, within five days it was 1018, 1024, 1013, 1021, and 1029, respectively. The amount of fluid ingested corresponded approximately to the amount of urine secreted.

It would be tiresome to continue to quote further data from a long, carefully kept clinical history. In the course of time all the symptoms disappeared, the last being the signs of cardiac weakness. The patient was allowed to rise on June, 5, 1899, and was discharged on June 21, 1899.

The examination on that day showed the following physical signs:

The area of cardiac dulness extended on the right side somewhat beyond the left sternal border, and upward up to the third rib. The apex-beat was in the fifth intercostal space, one finger's breadth outside the nipple. A systolic murmur was heard over the aorta, and transmitted into the carotid and the subclavian.

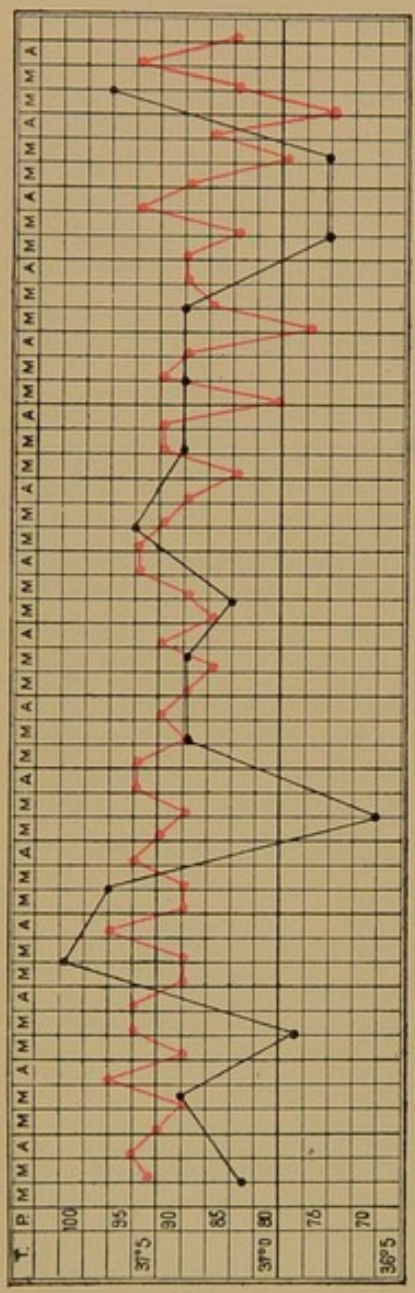


Fig. 28.

In this case there had been, therefore, a septic infection which had produced but slight general, but considerable local, effects, and which had involved the nervous system in addition to the heart. The temperature was taken for over thirty-two weeks and it was found that it never reached 38° C. (100.4° F.). The maximum was 37.7° C. (99.8° F.). Fig. 28 shows the behavior of the temperature from November 1st to November 16th. The chart also shows the behavior of the pulse, which was counted every morning by the assistant physician before the patient had been moved. The great variations which occurred independently of the fever are distinctly shown in the pulse-curve of this chart (Fig. 28).

But another peculiarity of this case should be noted: The distribution of the temperature in the course of the day differed from the normal, as may be seen by examining the curve, but was still more distinct when one took the averages. These daily averages for the period from the first to the sixteenth of November were as follows: Morning temperature, 37.36°; noon temperature, 37.54°; and evening temperature, 37.51° C. If the individual days be studied more in detail, we find that the highest figures were recorded sometimes in the morning and sometimes at noon, while lower temperatures, even subnormal ones, were usually noted in the evening and, at most, the evening figures equaled those recorded at noon. This peculiarity gradually disappeared. Thus, we find the following averages in the period from the twenty-seventh of April to the tenth of May (twenty-sixth and twenty-seventh weeks): Morning temperatures, 37.09°; noon temperatures, 37.12°; and evening temperatures, 37.09° C. Only toward the last—May 25th to

June 7th (thirty-first and thirty-second week)—the usual relations were reëstablished: Mornings, 36.93°; noons, 37.14°; and evenings, 37.21° C.

Such disturbances in the course of the temperature occur so frequently that I consider them of great importance. A sign which occurs not infrequently during a pericarditis is that a rise of temperature may appear without any externally discoverable cause, after the temperature had not exceeded the normal for a long period of time. In mild cases these rises of temperature appear only for a day or two, and extend but little over 38° C. (100.4° F.).

An example of this is furnished by the patient whose history is recorded under case III (second admission, December 18 to 25, 1893). At that time the curve had not shown any rises of temperature, nor any signs of other localizations for the nine days preceding. Fig. 29 shows the moderate rise and the slight depression of temperature which followed.

When the disease is more severe, the intercurrent rises are higher and continue for a longer time. The same patient showed this repeatedly during the third admission. I shall take the days from December 24th to January 14, 1895, but must note that pains in the arms coincided in time with the rise in temperature. This was, however, the only symptom which could be connected with the fever. Whether the rise in temperature in this instance was induced through the invasion of cocci directly from the heart or through a reawakening of the local process cannot be stated with certainty.

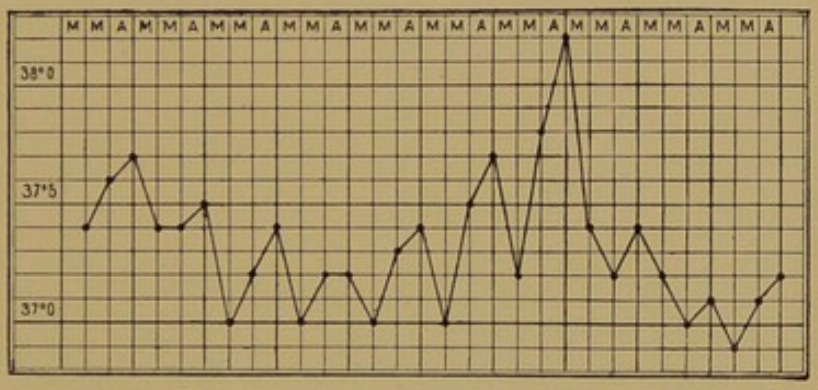


Fig. 29.

When septic infection in general is complicated by endocarditis, I think it is proper to lay stress upon such occurrences. The cocci are not necessarily limited in their activity to the heart or to the endocardium alone, and the variations should not be charged to the endocarditis, but to the sepsis, and only prove that the infection has not ceased to exist.

The clinical notes from January sixth show a less tumultuous heart's action, and this may be interpreted by assuming that a large number of bacteria were driven from the heart into the bones, with the result that the heart's action was rendered easier. At all events we cannot conclude from this occurrence that such signs developing in the course of endocarditis have nothing to do with the heart disease itself.

Fig. 30 shows, even in its first portion, before the fever had occurred, that there was a certain unsteadiness in the temperature, which fluctuated up and down with an irregular distribution over the different parts of the day. This continued also during the second portion of this period, and with the rise of temperature came the reaction, which lasted during the following days. The daily averages behaved at this time in the way which I have just described, and the same was true for the days during which there was fever, as well as for the preceding days. If we divide this period, consisting of twenty-two days, into two equal portions of

eleven days each, corresponding to a certain extent to the course of the temperature-curve, the averages will be as follows:

	Mornings.	Noons.	Evenings.
I. From December 24, 1894, to January 3, 1895	37.46°	37.45°	37.24°
II. From January 4 to January 14, 1895	38.04°	37.86°	37.80°
Averages	37.75°	37.66°	37.52°

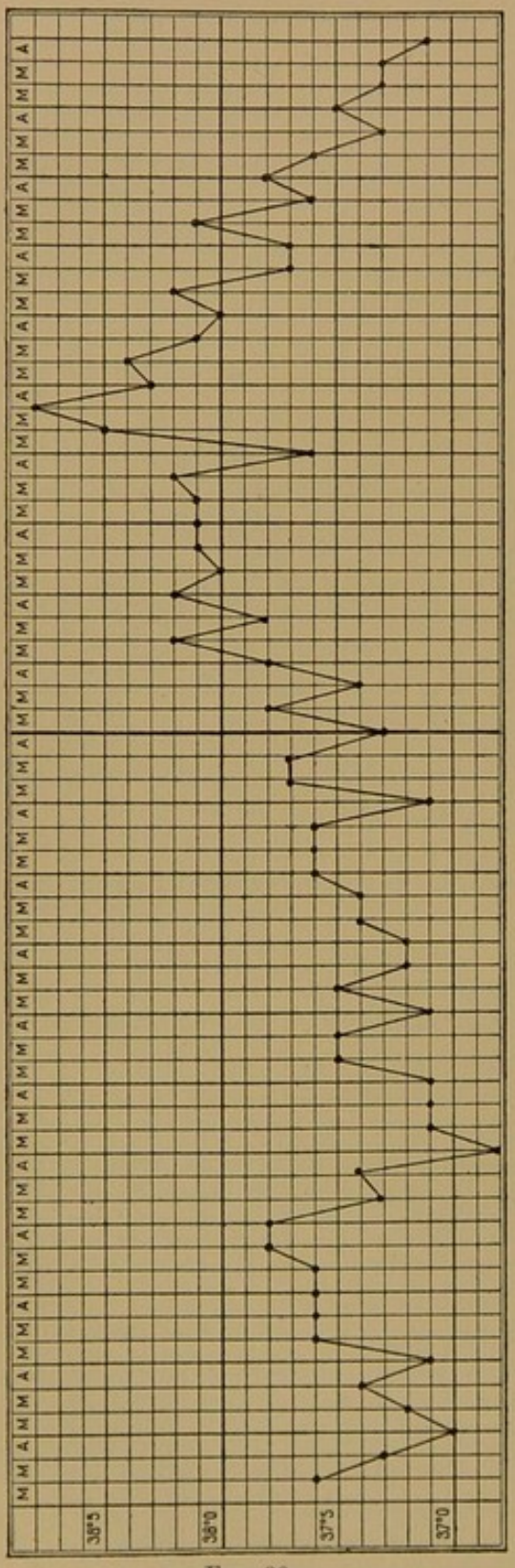


Fig. 30.

This capriciousness in the course of the temperature is really the only rule which can be laid down, and occurs even in the severe cases of endocarditis. An example of this is the following case, which is clean-cut in the sense that no metastases were found in any other organ at autopsy in addition to the severe lesions found in the heart, although the course of the disease had shown distinct symptoms in both bones and joints.

Case V.—A girl, ten years of age, admitted August 21, 1896. Died on November 14th of the same year. There was no previous history of any importance. Ten or twelve days before she began to complain of headache and sore throat. A few hours afterward a general eruption appeared, together with pain in both ankles and high fever, and toward evening slight cerebral symptoms. There was at first an improvement, so that the parents thought that she would recover. Three days before admission, however, she grew worse again, with high fever; pain in the ankles and in the right knee; a general eruption, as before; great weakness and prostration. In the course of a few hours her condition seemed to have become so serious that a physician was called.

On admission, erythema multiforme was found upon the chest, the back, and both thighs. The wrists and ankles on both sides and the right knee-joint were red and swollen. Active movements were impossible, and passive movements caused great pain. The mucous membrane of the pharynx was congested, the lungs extended downward behind to the tenth vertebra. The right lung in front reached the level of the sixth rib. The cardiac dulness extended on the left side from the third rib; on the right side, to the left margin of the sternum. The apex-beat was in the fifth intercostal space and the heart-sounds were faint but clear.

After a short time, however, the heart was found to be more severely involved. On August 22d the heart-sounds were muffled, while on the twenty-third the heart became slightly irregular. On the twenty-eighth severe palpitation developed. On September 15th, the area of dulness spread to the right sternal border. On November 16th the systolic sound at the apex was no longer quite pure. On September 19th a distinct systolic murmur was heard for the first time at the apex, and did not disappear to the end. The apex-beat became more diffuse and moved 1 cm. outward from its original position. The cardiac dulness extended to the right somewhat beyond the right sternal border. The diastolic sound over the pulmonary area was accentuated (October 5th).

Personal Examination, November 2d and 3d.—There was some prominence of the cardiac area; the apex-beat was very diffuse, and the heart-beat was visible on the left side to the anterior axillary line. At that point a distinct systolic thrill could be felt. The upper limit of the cardiac dulness was at the upper border of the third rib. On the right side it extended from two to three centimeters beyond the right sternal border. The cardiac area measured 10 cm. vertically and 15 cm. horizontally.

On auscultation, a loud systolic murmur was heard at the apex of the heart, transmitted up to the pulmonary area. The diastolic sound over the pulmonary area was remarkably loud. Over the aorta the systolic tone was indistinct, but the diastolic seemed to be pure. The same conditions were observed over the heart to the end.

The affections of the joints caused the greatest trouble to the little patient. As early as August 26th there was, in addition, sensitiveness to pressure over the long bones. These pains, to which were added muscular aches, were so severe that the patient could not change her position when it was necessary to take the temperature by rectum. The temperatures in the rectum were used only occasionally beginning with September 25th, and beginning with October 5th to the twenty-fifth of that month only axillary temperatures were taken.

Swellings of the joints no longer occurred after the end of October, and the sensitiveness on pressure over the bones disappeared. In its stead there appeared a widely distributed hyperesthesia of the skin, which was sometimes painful at the slightest touch.

Symptoms of consolidation were noted at the same time in the lower lobe of the right lung, with an increase of the respiration to 54 per minute, but without any appreciable influence upon the temperature. There was no expectoration in spite of the marked cough. In the course of the next few weeks various foci developed at different portions of both lungs and the respiration remained very frequent. The urine showed no greater quantity of albumin than is seen in ordinary cardiac weakness. Emaciation was noticed at the very beginning of the disease, and increased constantly. The extremely exhausted child died, finally, under the manifestation of cardiac failure. The dyspnea became very marked toward the end.

The following are the essential features of the report on the autopsy, made by Prof. von Baumgarten:

The diaphragm on the right side was at the level of the fourth, on the left side at the level of the sixth, intercostal space. "The heart could not be covered by the wholly outspread hand. The pericardium was completely adherent everywhere. The apex extended to the upper border of the seventh rib. On the left side, the pericardium was adherent to the lung, and extended to the axillary line; on the right side almost to the nipple line. When I tried to open the pericardial sac I found both layers intimately adherent, but not so closely that they could not be separated. This adhesion extended over the entire pericardium, less markedly behind than in front."

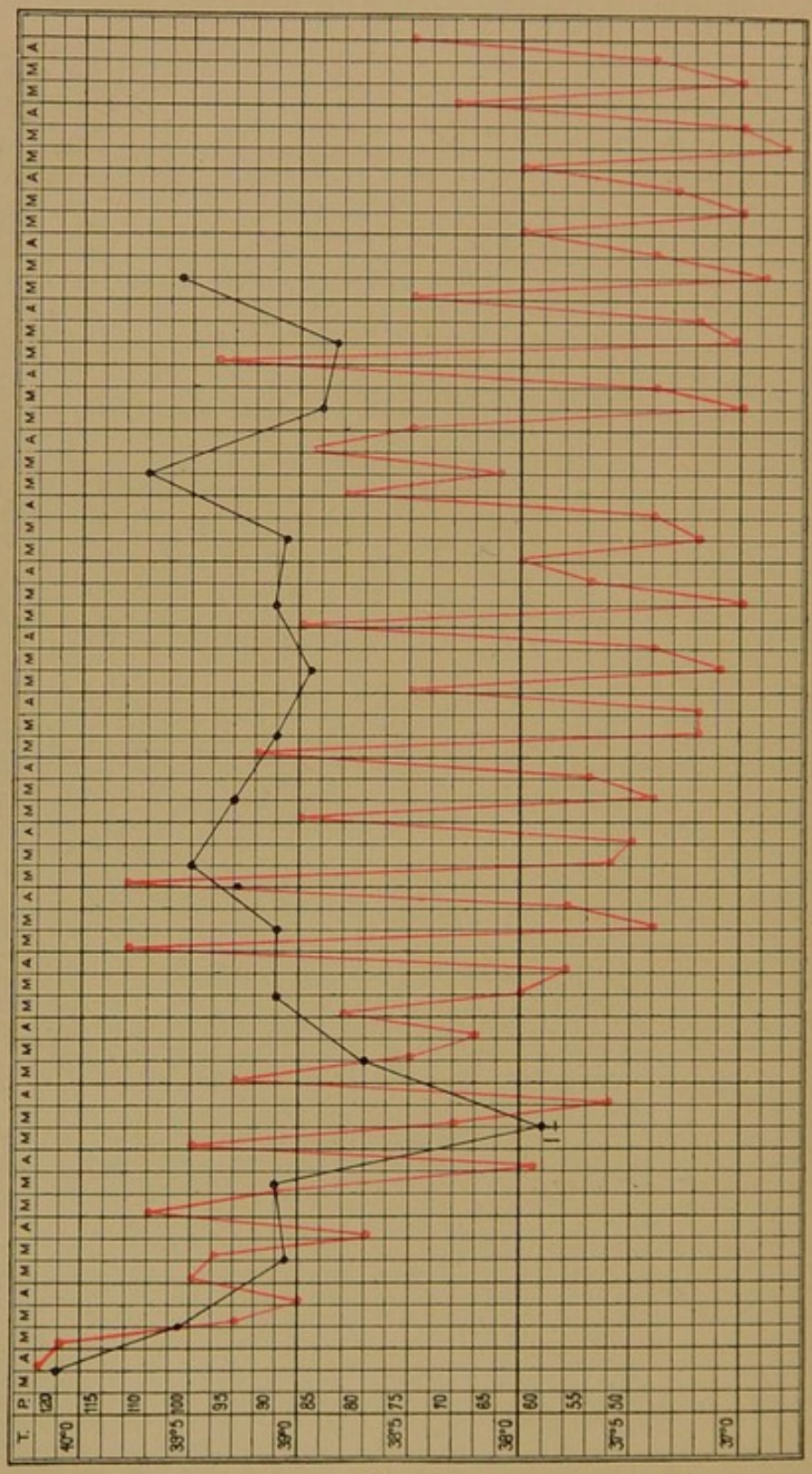
On removal, the heart was found to be twice as large as the fist of the cadaver, and was about the size of an adult heart. Its apex was rounded. Its longitudinal groove was also rounded, and the apex was essentially formed from the left ventricle.

The pericardium showed an irregular, ragged membrane corresponding to the adhesions, with a dull, glistening surface and in some places a velvety look. There were no tubercles.

Both sides of the heart were enlarged, the right somewhat more than the left. The right side was dilated, with trabeculæ and papillary muscles markedly hypertrophic. The tricuspid and the pulmonary valves were normal. The left auricle was dilated and the pectinate muscles were considerably hypertrophied. The left ventricle was markedly dilated and its walls greatly hypertrophied. Fine warty formations connected with each other in the form of a grayish-red ring were seen upon the line of closure of the aortic valves. These vegetations were so small that they could be found only on careful examination. A similar ring of excrescences which, however, were larger, was found at the line of closure of the mitral valves. The

valvular tissue was slightly thickened at the site of these lesions. The tendinous cords were very thin and tender. The myocardium was of a good color and did not exhibit anything abnormal.

The lungs showed in several places foci of lobular pneumonia, which on section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Unfortunately, no notes were taken concerning the microscopic and bacteriologic examinations.

The case was observed during a period of eighty-five days. In the beginning, the observation was not as satisfactory as might be wished, as it was during the long vacation, when I was absent. Under other circumstances it is scarcely possible that the pericarditis could have been overlooked. The temperature was taken regularly by the nurses. It shows the point that I wish to demonstrate, namely, the irregularity of the temperature-curve. The part of the chart which is reproduced herewith covers the period from the beginning of the observation to September 24th—the first five weeks. The morning records of the pulse which were taken for a short time show how absolutely independent the pulse was from the temperature.

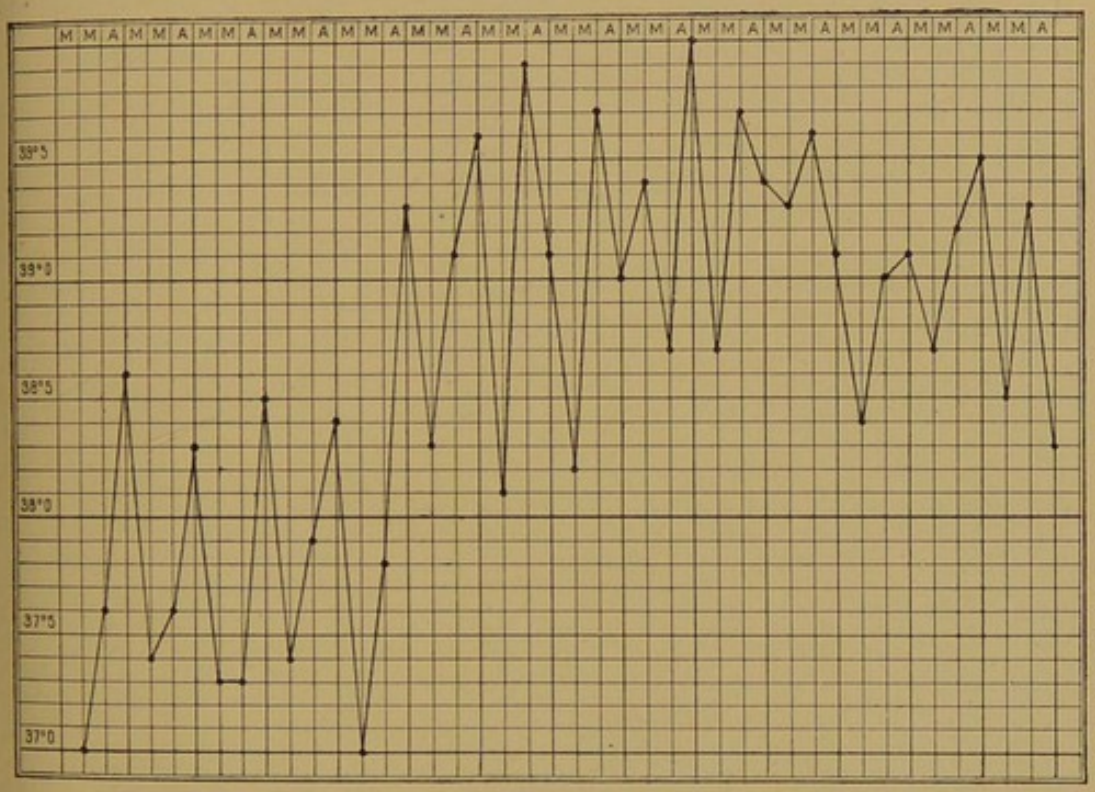


FIG. 32.

The fever in this case remained at the same moderate height until two days before death (it was below 38° C. (100.4° F.) then). Neither chills nor marked rises were noted.

Fig. 25 shows a picture of what occurs when the disease develops acutely. It may also happen, however, that an insidious course may take a sudden turn in another direction. This fact is always announced by the temperature. An example of this kind is as follows:

CASE VI.—A woman, aged forty-one years, was admitted to the polyclinic at Kiel on December 31, 1871, and died on February 10, 1872. Six years previously she had had pains in various joints, but these were not very much swollen. She said that she had not had any fever with these symptoms. The duration of that attack was seven weeks, of which three were spent in bed. At the end of November, 1891, she had a similar attack.

On admission she showed the typical symptoms of a sciatica on the left side, but these signs soon lost their characteristic features. There followed pains in the ankle

and wrist on the right side and in the elbows and shoulders on both sides, with a slight febrile movement, but without swelling of the affected joints. There came next vague pains in the head, the chest, the back, etc. Later, in the first week of January, there was a widely diffused catarrh of the finer bronchi, which remained for a considerable time, especially in the apices.

Both on admission and at a subsequent careful examination on December 31st no signs of any affection were found over the heart. It was only on January 26, 1872, that a slight blowing systolic murmur was heard over the aorta, together with a cardiac dulness which was found to extend beyond the normal toward the left. On the thirty-first, dilatation of the left ventricle was noted. The dulness extended from the upper border of the fourth rib, 2.5 cm. outside the nipple, down to the apex-beat, which was indistinct and displaced downward. There was no extension of dulness to the right. All the sounds were faint, and those of the pulmonary and over the right ventricle were muffled, but pure. At the apex, however, there was a murmur superimposed on the systolic sound, while the diastolic sound was pure. At the aorta there was a scraping systolic bruit, but no systolic sound. The diastolic sound was muffled, but not a murmur. These phenomena were heard also over the arch of the aorta and over the carotid and subclavian. A distinct diastolic murmur over the aorta which was transmitted to the other great vessels was heard a few days before death (February 7th), but the sound was also present. The pulse showed aortic insufficiency much earlier than the symptoms over the heart. It was a marked "pulsus celer."

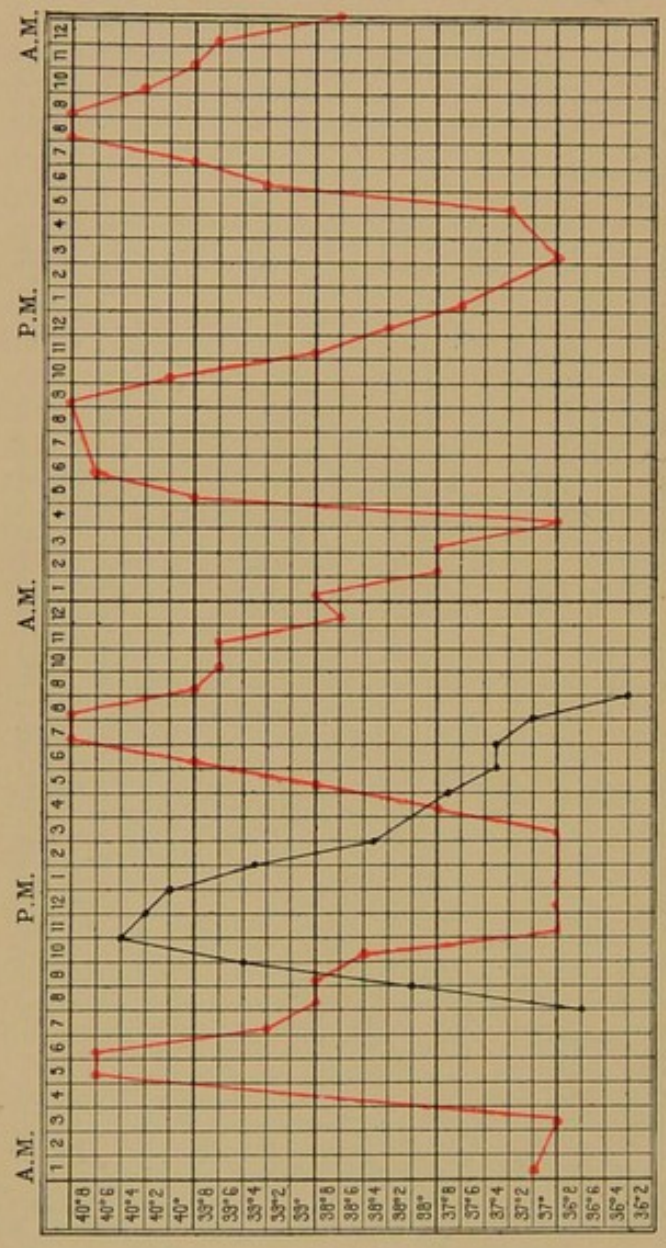


Fig. 33.

The fever was slight during the first month of the disease, but this changed with the beginning of January. There were at first rises which, to a certain extent, resembled the type of subcontinuous fever, but subsequently they were accompanied daily by one or two attacks of chills and reached 40° C. (104° F.). About the middle of the month the temperature elevations reached 41° C. (105.8° F.) and over, and there were more frequent chills. There was not one day like the other, and sometimes the elevation reached only 39° C. (102.2° F.) and even less.

Frequent almost hourly measurements were undertaken during the last four days of life, as is shown by the chart (Figs. 33 and 34). It seems that the behavior of the temperature during each succeeding attack exhibits a

characteristic difference between this case and those of malaria. The observations of Sidney Ringer,* which have been marked on the chart (Fig. 33, first day), exhibit the temperature in quotidian intermittent fever, and show this difference distinctly. But other differences were noted in other directions. Thus the relation of the chills to the rise of temperature was noteworthy. In this case several hours passed during which there were more or less marked chills, before the temperature began to rise. It happened even that the thermometer remained at 37° C. (98.6° F.) for almost four hours, although there was a very severe chill. At other times the chill began to appear only when the temperature had reached 39.4° C. (102.9° F.) or thereabouts.

We know that in malaria the temperature rises even before the chill itself begins and, at any rate, immediately after the chill; that the temperature at once reaches its highest point when the chill is ended, and that it rapidly sinks again from that time on. There is, therefore, a marked difference between the chill and the temperature as observed in malaria, on the one hand, and as seen in this case, on the other. Whether this difference holds true in tropical fevers is another question. Another point worthy of note is that quinin had but little influence upon the temperature-curve and that it never succeeded in aborting an attack.

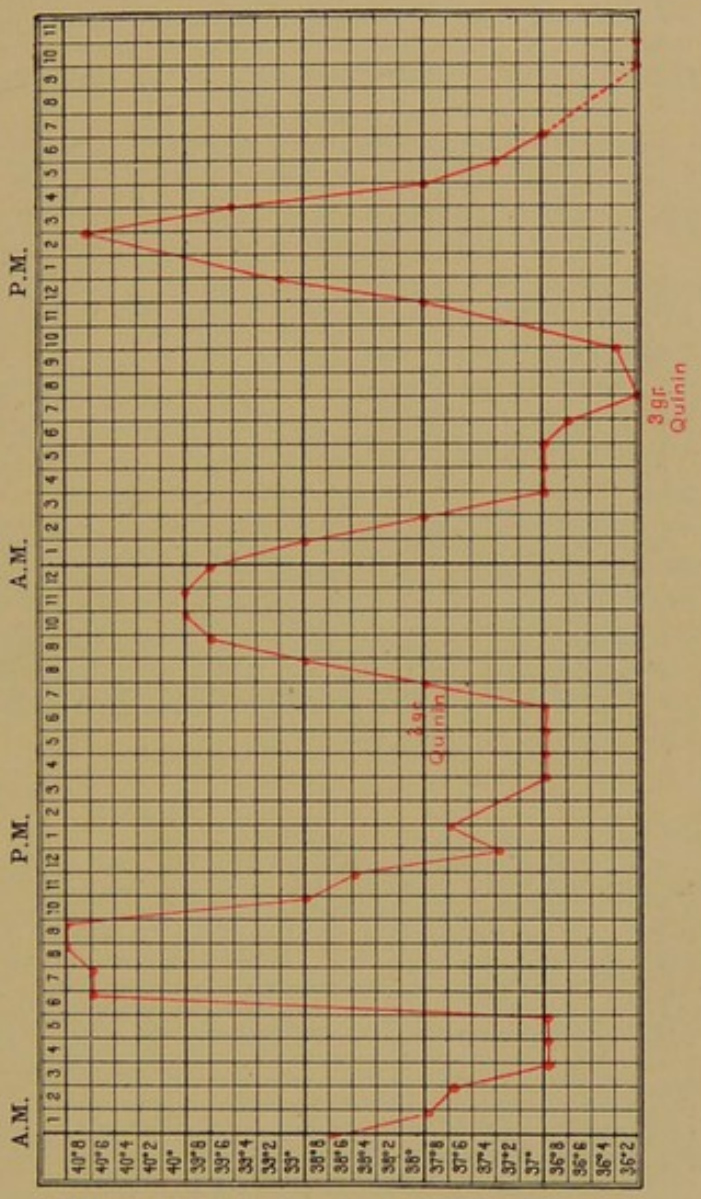


FIG. 34.

Death occurred under the manifestations of heart failure. The autopsy showed the following conditions:

"The heart, which was sent to me, showed a moderate dilatation of the left ventricle without any demonstrable hypertrophy. The heart muscle was of a pale, grayish-violet color, but on microscopic examination did not show any fatty changes. Of the valves, those on the right side and the bicuspid were delicate and completely intact. On the other hand, the aortic valves showed remarkable lesions.

"These were found only upon the right valve, while the other two were completely

* "Medico-Chirurgical Transactions," vol. xlii, 1859.

intact. The right cusp, however, was totally transformed into a soft, reddish tumor of irregular thickness, measuring at some places 8 mm., at others only from 2 to 4 mm., in thickness. No thrombi adhered to its soft and sticky surface. This mass was as if it were corroded near the attached border on both sides, and in the middle of each of these corroded surfaces there was an aperture through which a fairly thick probe could be easily introduced from the sinus of the valve into the ventricle; the ascending aorta was perfectly normal" (Cohnheim).

I was obliged to perform the autopsy myself under difficult conditions. The spleen was enlarged, 15.7 cm. long, 10.1 cm. wide and 4.6 cm. in thickness. The liver was swollen, the lungs edematous, hypostatically congested; but there were no emboli which could be seen with the naked eye. The brain, unfortunately, could not be examined, but the patient had remained free from signs of cerebral affections until the end. We, therefore, were dealing with a case of endocarditis in which the course was uncomplicated by any unusual conditions.

In connection with the subject of fever, I may mention, finally, that subnormal temperatures (36° C. (96.8° F.) and below) may be observed for a longer or shorter time during the course of the disease.

After all that has been said here, it must be admitted that there is no advantage is trying to establish types for the fever of an endocarditis. I may add only one more fact: the fluctuations in temperature may extend anywhere from 34° C. (93.2° F.) to almost 43° C. (109.4° F.).

[Much time is lost in the pseudomalarial cases of endocarditis, which seem more frequent than the foregoing account would indicate. The alleged differences in the details of the paroxysms are not present when most needed. Any type of malaria may be simulated. The much-abused therapeutic test often intensifies the error. Those who have not an exact knowledge of malaria, and who give quinin in such cases, often look upon accidental or temporary falls of temperature as evidences of malaria. Before quinin is used search should be made for malarial parasites, and the drug not given at all unless they are actually present. In doubt the case should be treated as one of sepsis or typhoid fever rather than malaria.*—Ed.]

HEART.

The only general rule which may be established as regards the heart in this disease is as follows: A disturbance in the heart's action is never absent, and it always occurs in the form of a diminished functional efficiency.

At the beginning of the disease there is a peculiar change during systole which is difficult to describe. The only appropriate expression for it is "cardiac uneasiness." If the lungs are not in the way, the more extensively exposed heart does not show the uniform regular beat observed in normal persons. Its action gives the impression of a certain irregularity, and it seems as though one group of muscle-fibers contracts more vigorously than the neighboring group, as though this group contracts a little more rapidly than the rest of the heart. With this, there seems to be a somewhat more marked concussion of the thoracic wall, but when the hand is placed upon the chest, we find that this is not so in

* See Dock, "Endocarditis and Intermittent Fever," "Boston Medical and Surgical Journal," 1895, vol. cxxxiii, p. 457; *ibid.*, "Double Quotidian Fever with Endocarditis," "New York Medical Journal," 1897, January 30, p. 143; W. Coleman, "The American Journal of the Medical Sciences," 1905, March, p. 381. Regarding fever in endocarditis in general see French, Herbert, "The Practitioner," 1904, vol. lxxiii, p. 753; and Thacher, "The American Journal of the Medical Sciences," 1906, vol. cxxxi, p. 29.

reality. And yet, somehow, the tactile sense gains the impression that the movement of the contracting heart is irregular. The systolic sounds are somewhat less sharp and rounded and are muffled, but we cannot say that there are murmurs at this early stage. As a rule, the diastolic valvular sounds are altered in the same manner.

[Teissier* calls attention to an early symptom that may at times be of assistance. This is a pulsation in the second left intercostal space and a double apex-beat. Teissier explains these as due to the difficult contraction of the left auricle, which causes the pulsation and the first apex-beat, the second being the systolic impulse, which may be weaker than the first one.—Ed.]

The sequence of the cardiac movements is very often no longer quite regular, even at this stage, but in order to detect this we must count the pulse for about a minute or even longer. In making this observation we will also find that the vigor of the individual beats is not quite uniform. All these things may be detected also by auscultation, as well as by feeling the pulse (compare Fig. 26).

Very frequently the arteries are not as full as normally, and the tension of their walls is lessened, the pulse being easily compressed. Along with this there is cyanosis, though this is but slight in the beginning.

Subjective symptoms, such as palpitation, stabbing pains in the cardiac region, and a slight feeling of anxiety, may be present, although they



Fig. 35.

do not occur necessarily. All these phenomena grow worse after the slightest exertion. The patient need only sit upright in order to make especially evident the accumulation of blood in the capillaries of the dependent portions of the body which are furthest removed from the heart: The hands and feet grow blue and cold, and a slight pressure leaves a white mark upon the skin of the trunk. The nose and the ears change their color and their temperature in a short time.

These are signs of heart weakness. They increase gradually from the phenomena just described. The irregularity in the work of the heart becomes more and more prominent, and there may be a true interruption in its action, or an alternation of vigorous contractions with weaker pulsations. The pulse shows the presence of cardiac insufficiency very clearly, but as yet only for a short time and transiently. Fig. 35, which shows the tracing of the carotid pulse in case IV at the end of the third week (November 16th), may serve as evidence.

At this stage the serious consequences of insufficient heart action, such as edemas, are still absent and the urinary secretion shows as yet no diminution in quantity: the average daily elimination in case IV was 1152 c.c. during the period from October 30th to November 13th, and

* "Bulletin médical," 1903, No. 17.

1181 c.c. from November 16th to December 6th. There were no marked differences in the daily amounts during these two periods. This is because the cardiac weakness at this stage is only transient. It may even be said that this weakness occurs only in paroxysms. The following sphygmogram (Fig. 36) of the radial, taken three days later (November 19th), demonstrates this fact.

The subjective symptoms, however, increase, and in addition to the disturbances which are felt over the heart there are at times veritable attacks of cardiac asthma.

The frequency of the pulse is next subjected to greater variations, even when the patient is at rest. No relation is found between this frequency and the temperature, or only a very slight correspondence of the two phenomena. On the other hand, muscular movements and changes in posture are of great importance in their effect upon the pulse-rate. The following will serve as an example.

CASE VII.—A boy aged fourteen years. Admitted September 18, 1891. A year before admission he had a nephritis which lasted for a short time and from which he recovered completely.

On admission, he was found to be a muscular boy who stated that he had not been perfectly well for the past few days, that he had suffered from headaches, especially at nights, and had easily grown tired. On the day of admission he complained of chilly sensations, more severe headache, and more marked prostration.



FIG. 36.

There was slight edema over the eyelids; the urine was free from albumin. There was high fever, but local manifestations were completely absent.

On September 19th he had several fluid stools, and this symptom continued on the two following days. On the twenty-second, the stools became solid again, and continued so to the rest of the observation. On September 24th the subjective symptoms were improved to such an extent that the boy could get up from bed. The temperature had sunken markedly from its high level at the beginning of the attack, and only on that day it reached 37.9° C. (100.2° F.) in the evening, while, as a rule, subnormal temperatures had prevailed before. The chart (Fig. 37) shows how great and prolonged the disturbances in temperature may be even after a general infection which in itself seems of no importance. The long duration of the subnormal temperatures is especially noteworthy.

The heart, which had previously appeared within normal limits, giving dulness up to the right sternal border, but perfectly clear sounds on auscultation, showed slightly extended boundaries on October 11th and 12th. On the right side the dulness extended to the parasternal line; on the left, to the third intercostal space, while the apex-beat was found in the nipple line, but still in the fifth intercostal space. There was, in addition, a faint systolic murmur at the apex, and a diastolic sound over the aorta, the latter not quite pure in quality. Neither of these murmurs could always be heard. The following observations were recorded on the pulse: On September 18th it was 112 in the recumbent position and 130 when sitting up. On the nineteenth it was 100 and 120 respectively in these positions; on the twenty-fourth it was 69 and 111, and on the twenty-fifth it was 70 and 108.

Irregularities in the pulse-rhythm were noted as early as the twentieth of September; on the twenty-fourth, we find the note: "The extremely irregular pulse is noteworthy today. The pulsations are sometimes strong, sometimes weak, and at times there is an actual interruption of the pulse." This continued at least

throughout the entire month of October, and could be still demonstrated in a less marked degree in the middle of December, as we see in the tracing in Fig. 38.

The frequency of the pulse had diminished (at rest) to 57 beats in the first part of

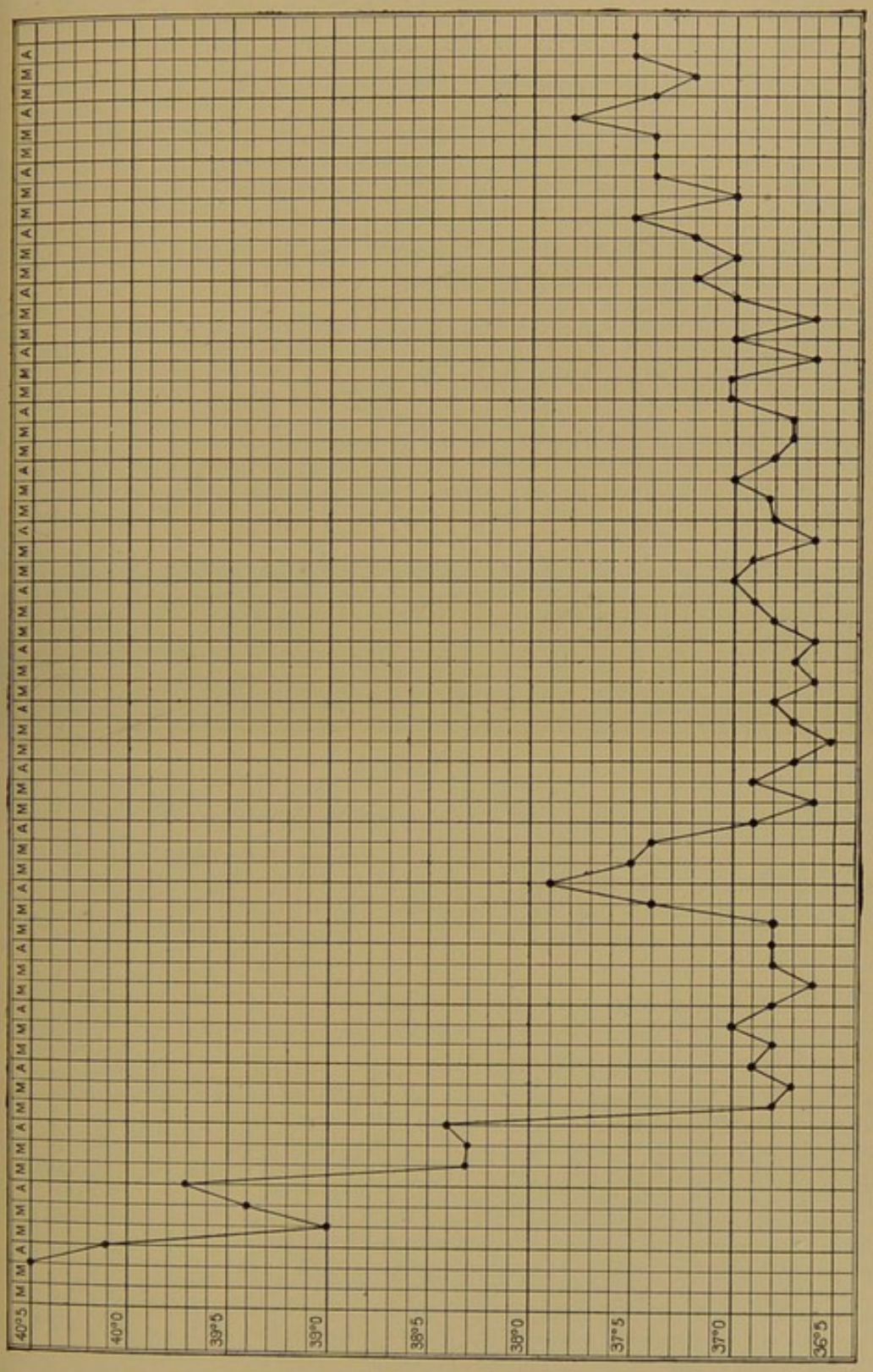


Fig. 37.

October, but beginning with October 11th it rose to 100 and even to 120. It continued in this manner during the entire month of November.

The relation of the temperature to the frequency of the pulse was as follows on some of the days:

	Temperature.	Pulse.
September 18th,	40.5° C. (104.9° F.)	112
October 11th,	37.2° C. (98.96° F.)	108
October 16th,	37.1° C. (98.78° F.)	117
October 19th,	37.0° C. (98.6° F.)	112
October 24th,	37.0° C. (98.6° F.)	120

No dependent relation, therefore, existed between these two phenomena.

We have seen that in case II, the pulse with the patient at rest showed in ten consecutive seconds 17, 15, 14, and 13 beats. In case IV, the pulse during three consecutive minutes showed 90, 76, and 60 beats, respectively.

In the following case, which I report briefly, the functional disturbances of the heart were very prominent:

Case VIII.—A strong muscular man, aged twenty-seven years, was admitted on April 29, 1890. There was at first constipation, with the appearances of peritoneal irritation, which quickly disappeared. On May 21st the patient felt very ill, the temperature reached 39.4° C. (102.9° F.), there were severe pains in the joints, with swellings of these parts and their surrounding tissues and redness of the skin. The same phenomena were noted over the glabella, the left cheek, and the eyelids. A widely diffused septic eruption; inflammatory transient changes in various muscles. A marked swelling of the spleen, the maximum measurements being 15 by 5 cm. There were repeated attacks of excruciating pains in the abdomen and

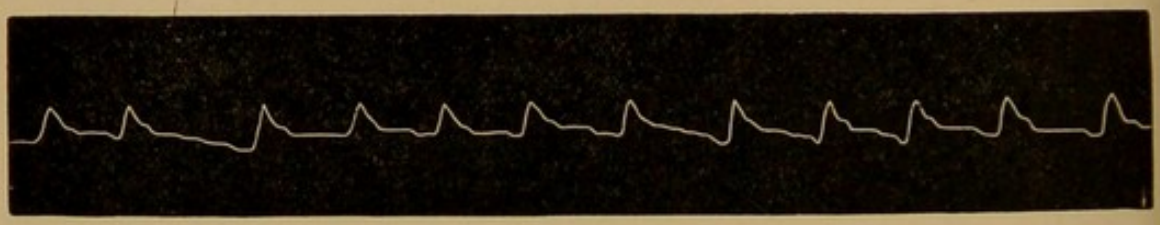


Fig. 38.

of sensitiveness on pressure over the belly. Marked emaciation. Moderate irregular fever during the first four weeks.

The table on page 219 shows the interdependence of the heart's action and the movements of the body, although the latter increased the demand upon the heart but slightly. It also shows the great change which occurred within a few days. Mental excitement certainly did not play any rôle in this patient during the examinations.

The extremely low pulse-rates which are sometimes observed, even at the early stages in endocarditis, deserve attention and are exemplified in the two cases which follow:

Case IX.—A girl aged seventeen years. Admitted February 24, 1886. She complained at first of fatigue in the extremities, pains in the legs, pressure in the region of the stomach, and chilly sensations (February 14th). All these symptoms continued unchanged until the twenty-third. On that day there were pains in various parts of the body and swelling of the right wrist. On admission, the temperature was 39.2° C. (102.5° F.) and the heart was found to be already involved. During the further course of the disease pains and swellings were noted over various joints, together with swellings at the epiphyses of some of the long bones. There was, in addition, a widely distributed pleural friction-sound, but no effusion. A slight nephritis was present with prolonged diminution in the amount of urine secreted. There was enlargement of the spleen, the maximum measurement being 12 by 8 cm., and there were numerous disturbances of the central nervous system, which are to be mentioned again in another connection. The duration of the observation was twenty-two weeks. There were seven attacks of fever of shorter or longer duration, some of which appeared without any particular local signs. During the entire time the highest temperature was 39.2° C. (102.5° F.), while the lowest, 36.8° C. (98.2° F.), was only recorded once.

CASE VIII.

Date.	Temperature.	Pulse. Resting	Pulse. Moving	Remarks.
May 6....	38.0° C. (100.4° F.)	72 Fairly regular	135 Very irregular	Marked cyanosis of face, hands, and feet; severe irregularity of heart on rising; palpitation in which the formerly barely visible cardiac movements become excited, visible over a wide area.
May 21....	38.0° C. (100.4° F.)	90	90	Systolic murmur at the apex; rubbing over the pulmonary, synchronous with the cardiac movements, independent of respiration.
May 27....	37.3° C. (99.1° F.)	80 Pretty regular	152 Very irregular; intermittent	Pretty regular pulse while quiet in bed; on leaving the bed irregular pulsations and movements visible in the cardiac area; severe subjective palpitation; both last during the few moments out of bed, quickly subsiding on lying down.
May 28....	37.1° C. (98.7° F.)	70 Very irregular	90 Very irregular	Cyanosis of face less.
May 30....	37.0° C. (98.6° F.)	79 Intermittent	80 Intermittent	Distinct systolic murmur at the apex; pericardial friction disappeared since the twenty-fifth.
June 6....	37.0° C. (98.6° F.)	66 Regular	144 Very irregular	No visible cardiac movement while lying down; on rising, movement becomes evident, visible over a wide area.
June 11....	36.0° C. (96.8° F.)	78	87	Patient remains in bed.
June 15....	No longer taken; no alteration since May 25th	76 Regular	85 Very irregular	After rising; apex systolic, murmur continues.

Fig. 39 shows the behavior of the pulse during the first five weeks of the disease, when the heart was most markedly affected. The morning temperatures are also noted upon this chart, as they were measured at the same time as the pulse was counted. It follows—(1) That with a temperature which never had sunk below 37° C. (98.6° F.), the pulse from March 5th to March 19th had remained under 50, showing a minimum of 38 (March 15th). Low figures were also found when the pulse was counted repeatedly in the evening. These coincided with the higher temperatures which prevailed at that time of the day. (2) That there was from the beginning an irregularity of the pulse and a diminished fulness of the arteries. Repeatedly we find the note "the pulse can hardly be felt." (3) That although a certain parallelism existed between the pulse and the temperature, this was only of the most general character. There was no indication of a dependence, such as is observed normally. During the subsequent weeks of the disease these phenomena continued, although in a lessened degree.

Case X.—A man aged thirty-seven years, admitted on March 2, 1888. During the past eleven years he had repeated attacks of "pain in the limbs." During the night from the first to the second of March he felt slight pains in the right leg, and on admission showed signs of sciatica on the right side. There was, however, an elevation of temperature which reached 39.5° C. (103.1° F.) even on the first day of the disease. On March 4th—the third day of the disease—there were widely distributed pains in the limbs, including bones and joints, and swellings of the joints. The spleen was enlarged from the first, measuring 10–11 cm., and showing fine friction-sounds which could be heard over it as well as over the liver. Friction-sounds were also heard over both pleuræ, beginning with the fifth day of the disease. An excited and irregular heart's action was present from the fourth day. The cardiac area, which was at first normal, gradually increased in size. This could be clearly demonstrated on the twentieth day. At the same time there were phenomena on auscultation which did not disappear again. The duration of the observation was ten weeks. The first period of fever lasted from March 2d to March 6th; the second lasted from March 24th to April 1st.

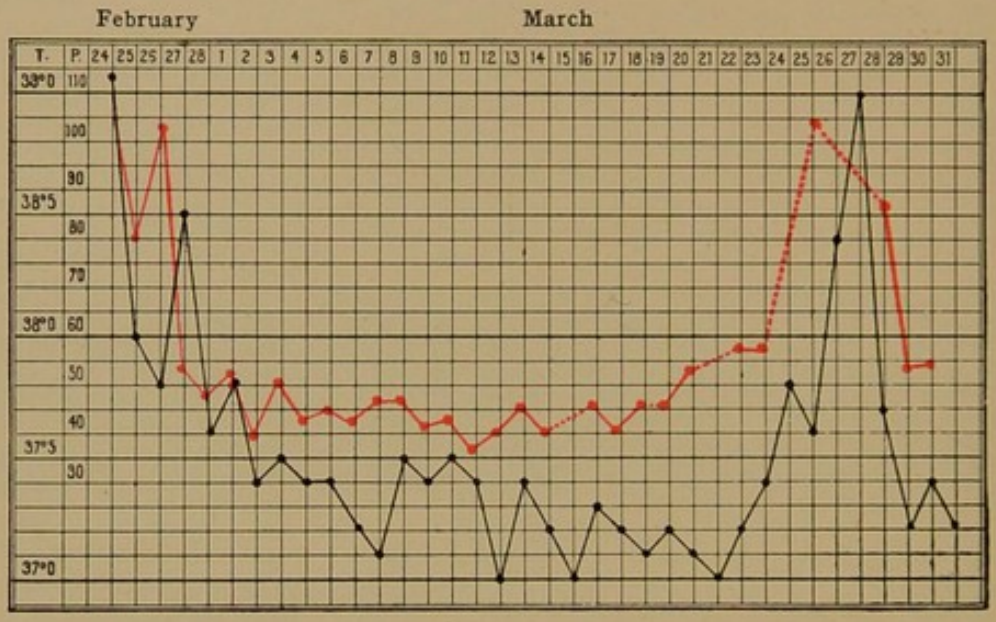


Fig. 39.

Fig. 40, which was drawn in the same manner as the preceding chart, shows the following:

(1) The frequency of the pulse was less than 50 only for a short time and reached its minimum of 38 beats on March 17th. Neither on that day nor on the preceding day did any appreciable changes occur in the condition of the patient—in fact, he felt somewhat better.

(2) There was no more true correspondence in the course of the temperature and of the pulse than was noted in case IX.

(3) The disturbances over the heart, on the whole, were much more marked in this case, but it does not appear from the observations that they were worse when the pulse was least frequent.

Changes in physical signs are almost always noted only after the disturbances in the heart's action have been evident a variable period of time. As a rule, the auscultatory signs appear appreciably earlier than the extension of the cardiac dulness in any direction.

Thus, in the history of the case last mentioned (case X), we find the following notes: March 5th: "Heart-sounds pure." March 11th and 13th: "A loud systolic murmur over the apex of the heart and over the aorta; diastolic pulmonary sound accentuated." March 21st: "The systolic murmur is especially loud over the apex. The systolic sound over the ascending aorta is very faint, reduplicated, and somewhat

fluttering. Heart boundaries: The cardiac dulness is only slightly extended; the apex-beat in the fifth intercostal space outside the nipple line."

"The autopsy on August 20, 1890, showed considerable changes in the mitral and aortic valves, which indicated old endocarditis. There was, in addition, a marked hypertrophy and dilatation of the heart."

Dilatation of the heart may coincide with the acoustic phenomena, and both may occur very early in the disease, although this is less frequent, as was seen in the following case:

Case XI.—A girl, aged sixteen years; admitted on November 22, 1893. Her illness began on October 31st with headache and prolonged chilly sensations. On the following day the symptoms grew worse, and a peculiar phlegmonous swelling appeared on the face. The temperature was very high (40.9° C.—105.6° F.). The heart was found to be involved considerably, even on admission. The pulse was small, easily compressed and irregular.

November 3d: Heart-sounds muffled; a systolic blowing sound, variable in intensity at the apex; in addition, pericardial friction. Heart boundaries: the upper

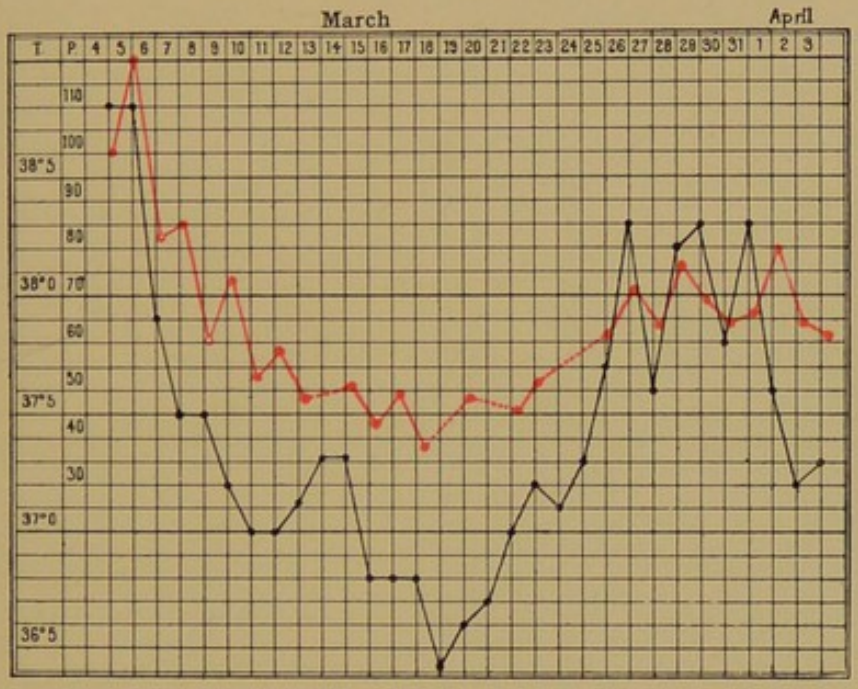


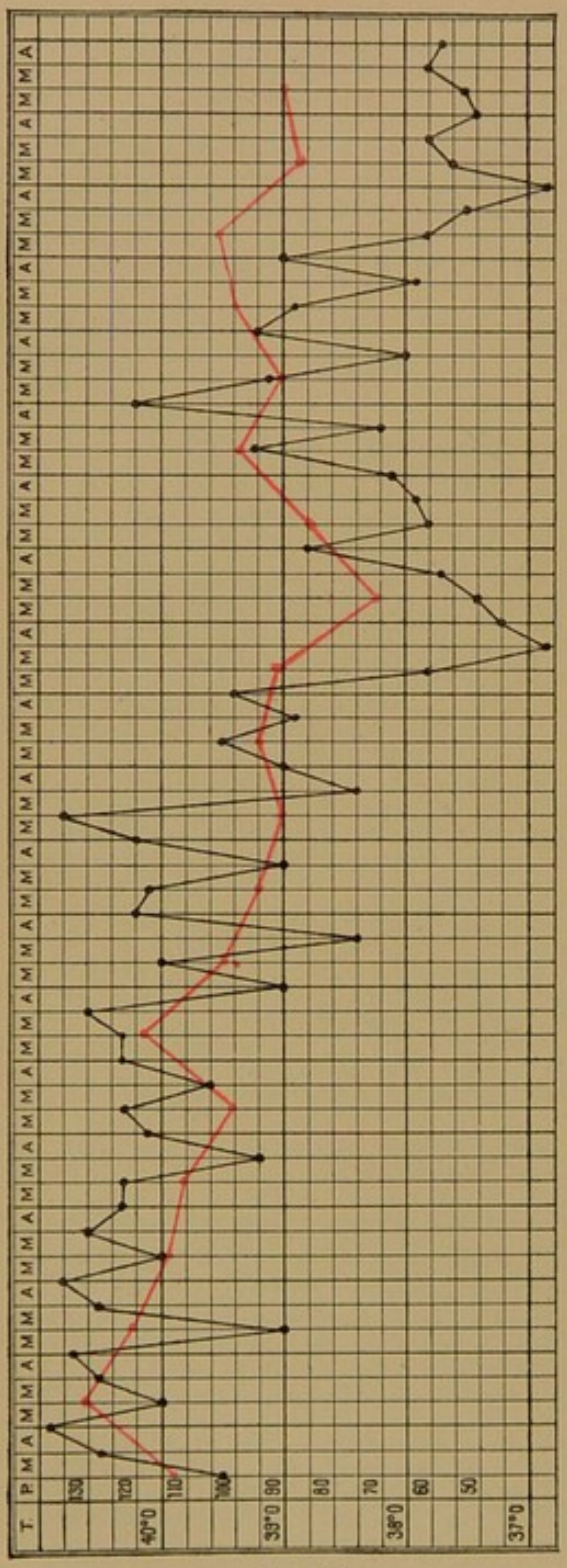
Fig. 40.

border of the fourth rib; the middle of the sternum. The apex still in the fifth intercostal space, but somewhat outside of the nipple line. November 10th: Heart-sounds pure; the dulness just to the left sternal border. December 18th: Heart-sounds: A loud systolic murmur over the aorta and the carotid, a double sound over the crural; dulness has extended to the left and upward. January 9, 1894, systolic murmurs over the apex and the aorta. Dulness one finger's-breadth beyond the right sternal border, and upward to the third intercostal space. Apex-beat outside the mammary line.

When the patient was discharged on May 8, 1894, she had all the signs of a complicated valvular affection involving both the aortic and the mitral. Cultures from the blood showed the presence of Staphylococcus pyogenes albus, and the septic infection involved, in addition to the heart, the serous membranes, including the pleura, the pericardium, and the coverings of the spleen and the liver; also in a less marked degree the central nervous system and the muscles. In addition, the kidneys were involved; albumin, blood, and casts were present in the urine from the beginning of the disease to November 27th. The spleen reached a maximum size of 9 by 14 cm. Profuse diarrhea was present from the eighth to the fifteenth of November. The temperature reached 40° C. (104° F.) and over daily during the first

two weeks, and after an intermission of a few days there was again a rise to the same height. Then followed a fairly sudden fall. The temperature did not exceed 38° C. (100.4° F.) from November 28th to January 11th, but there were irregularities in the course throughout the day.

Figure 41 shows that, even with high fever and a severe involvement of the heart, there was no appreciable relation between the frequency of the pulse and the temperature. In this case this was even more distinctly demonstrated than in those cited previously. No relation could be shown between the temperature and the gradually developing anatomic changes in the heart.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fact that this organ had again been attacked by a renewed infection. The following table shows the course of the disease:

TABLE OF CASE XII.

Date.	Maximum Temperature.	Pulse-rate.	Quality of Pulse.	Boundaries of the Heart.	Heart-sounds.	Other Symptoms.
Aug. 6..	38.3° C.	86	Slightly irregular.	As on admission.	A faint diastolic murmur also heard over the aorta.	During the night, severe pains over the heart. Slight effusion in left pleura. Friction sound. Heart's action excited.
Aug. 16..	39.5° C.	96	Irregular dicrotic.	As on admission.	Impure systolic sound over aorta. Diastolic murmur.	Heart's action excited. Cyanosis of face more marked. Pleurisy disappeared since the eleventh.
Aug. 20..	38.2° C.	99	Irregular in force and rhythm.	Unchanged.	....	....
Aug. 22..	39.0° C.	86	Interrupted once a minute.	Unchanged.	....	Patient found unconscious in the morning, and had fallen out of bed. Left hemiplegia (embolic).
Aug. 28..	38.1° C.	94	Fairly regular.	Unchanged.	A distinct systolic murmur over the aorta. The diastolic murmur less marked.	Marked edema of the eyelids since the twenty-sixth. Urine perfectly normal (kidneys remain free).
Sept. 16..	38.9° C.	96	Irregular dicrotic.	Unchanged.	An impure systolic tone over aorta, and a diastolic murmur. Signs unchanged over apex and pulmonary, since admission.	Paralytic symptoms slightly improved during past few weeks. Pain and swelling of various joints now and in second half of September. Spleen dulness slightly enlarged, eighth to ninth. Though fever only moderate (never over 39° in evening) patient much emaciated.
Sept. 28..	38.5° C.	94	Fairly regular.	Unchanged.	Distinct aortic systolic, weaker diastolic murmur.	Pains in joints have disappeared. Patient feels pretty well.
Oct. 18..	38.5° C.	104	Very irregular.	Dulness extends two finger-breadths to right of sternum.	Systolic and diastolic murmurs over aorta as well as over apex.	Occasionally slight pains in the joints and muscular pains.
Oct. 29..	37.5° C.	87	Markedly dicrotic.	Upper border of third rib, right sternal border Apex; 2 cm. outside of nipple line, fifth intercostal space.	Systolic and diastolic murmur over aorta; the systolic also heard over ascending aorta and the carotid.	Severe pains in the fingers of the left and in the entire right hand. Joints swollen, red, and wrists painful on pressure. Petechiæ over both palms since the twenty-third. Increasing emaciation.

TABLE OF CASE XII.—(*Continued.*)

DATE.	MAXIMUM TEMPERATURE.	PULSE-RATE.	QUALITY OF PULSE.	BOUNDARIES OF THE HEART.	HEART-SOUNDS.	OTHER SYMPTOMS.
Nov. 7..	38.0° C.	100	Regular.	Not changed since October 29th.	Systolic murmur over aorta much louder than the diastolic. The latter is audible over a larger part of the sternum, on the right side. Over the ascending aorta and carotid, a systolic blowing sound; a systolic thrill over the latter.	Patient feels fairly well during the day, gets worse at night. Very severe cerebral symptoms. First delirium, then coma. On the ninth delivered of a seven months' fetus, stillborn. Patient did not notice that she had given birth. (Compare below, p. 260.)
Nov. 11..	40.0° C.	138	Irregular, interrupted dicrotic.	Slightly increased to the right. Small area of dulness 3 cm. high and 2.5 cm. wide in first intercostal space, right side, near sternum.	Murmurs over mitral very indistinct. Very loud over the aorta, especially the systolic. Also over the dulness on right side of sternum (aorta ascendens).	On November 10, complete paralysis of the right half of the body. On the left side repeated contractions. Death, November 11th, 4.45 P.M., the patient remaining unconscious to the end.

Acoustic changes which indicated the participation of the aorta and its valves were noted, in addition to the functional disturbances and severe subjective symptoms, after a short time—about two weeks. These changes consisted in systolic and diastolic murmurs which occurred in irregular alternations, at first transiently, sometimes louder, sometimes more faintly, but toward the end permanently. It took more than twelve weeks, however, before increased cardiac dulness could be demonstrated. At first only the right side of the heart was involved, but after about a week the left side followed. Before the end there was again an enlargement in which the ascending aorta took part.

On the whole, the behavior of the temperature was the same as has been described above in several instances. The elevation fluctuated within moderate degrees, and during the course of fifteen weeks 39° C. (102.2° F.) were reached or slightly exceeded (maximum, 39.5° C.—103.1° F.) only eight times. The minimum temperatures were as low as 36.3° C. (97.3° F.). The irregular distribution over the twenty-four hours was very noteworthy, but this changed during the last few days of life. There was a rapid rise, which was interrupted by unexplainable fluctuations, but shortly before death 40.9° C. (105.6° F.) was registered (Fig. 42). Finally, a postmortem rise to 41.5° C. (106.7° F.) was noted (Fig. 43).

I shall give only the essentials of the very detailed anatomic report of von Baumgarten:

"The heart, on the whole, is moderately enlarged. On the right side it projects beyond the right sternal border to the extent of 1½ finger-breadths. Its apex is in the sixth intercostal space, one finger-breadth outside of the nipple line. The heart, as a whole, is of a marked conic shape, the apex being made up exclusively of the left ventricle and the anterior longitudinal sulcus deviates strongly to the right from the median line.

"The epicardium contains a moderate amount of fat. The aorta shows a dilatation the size of a walnut projecting to the right from its first portion. The heart measures 12 cm. in length; the wall of the right ventricle is, without epicardium and trabeculæ, from 4 to 5 mm. thick. The left ventricle measures, in the same way, 2 cm. Resistance is encountered when the finger attempts to pass through the left articulo-ventricular orifice. On the right side three fingers are easily passed through the opening. On opening the heart the mitral is found markedly thickened

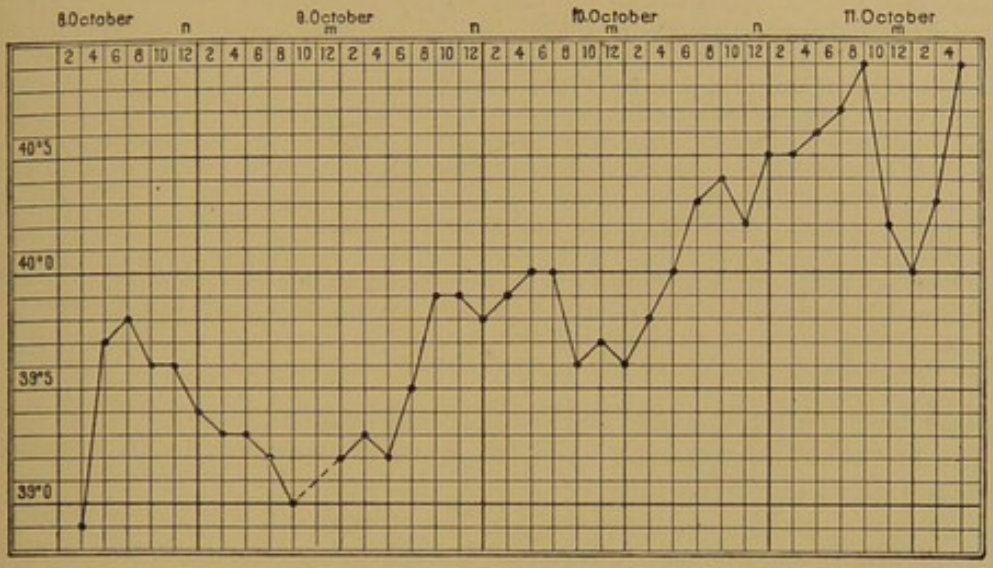


FIG. 42.

and its lower free border considerably shrunken. There are several grayish-red thrombotic deposits the size of a pea along the line of closure, firmly adherent to the tissue. All the flaps of the aorta are markedly thickened and their upper free border considerably shrunken. There are thick, grayish-red thrombi, especially along the lines of closure, but also beneath these, which cannot be removed without violence. Similar warty and nodular deposits cover almost the entire surface of the aortic flap of the mitral on the side toward the left ventricle, as well as the endocardium of the left ventricle near the membranous septum.

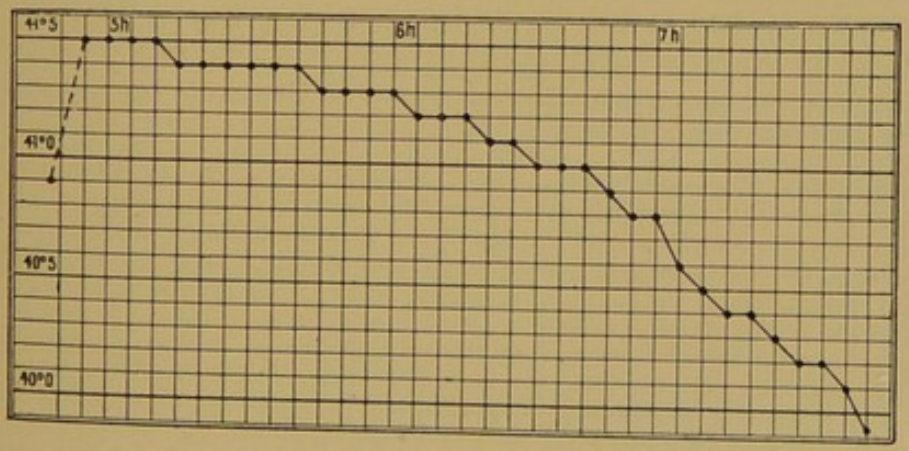


FIG. 43.

"The papillary muscles, as well as the trabeculæ, are markedly hypertrophied. The cavity of the left ventricle was only slightly dilated.

"The tricuspid is 10 cm. in diameter. The cavity of the right ventricle was somewhat dilated, as well as that of the right auricle. The papillary muscles, the trabeculæ, and the pectinate muscles are moderately hypertrophied. The aorta shows no changes even in the dilatation described above, excepting a slight thinning of its

wall corresponding to this projection. The heart muscle, as a whole, has a good grayish-red color and there are no inflammatory foci anywhere."

In the further account of the autopsy we find: "The meninges at the base of the brain show a slight milky opacity. A thrombus measuring about ¾ cm. in length is found in the left internal carotid where this artery passes into the beginning of the artery of the Sylvian fissure and the anterior communicating branch. This thrombus adheres closely to the wall of the vessel, distends the latter, and passes into the artery of the Sylvian fissure as well as into the anterior communicating branch. The arteries which lie beyond it are markedly collapsed and flattened. There is a second thrombus, about ½ cm. in length, in the left artery of the Sylvian fissure, of greenish color; it distends the wall and is adherent to it, and extends into the neighboring arterial branches. On the right side there is a thrombus completely filling the walls of the artery of the Sylvian fissure at the point where this vessel passes over the island, and this thrombus also extends into the neighboring branches. On section through the hemispheres the right side shows a fairly extensive softening of the internal capsule and of the greater portion of the lenticular nucleus. On the left side a fresh reddish area of softening is found in the middle segment of the lenticular nucleus.

"Furthermore, there are older infarcts in the spleen and kidneys. There is a grayish-red embolus, with puriform softening in its center, in the hepatic artery, astride of the angle of division of the two main branches. There are purulent bronchitis and splenization in the lower lobes of both lungs."

The bacteriologic examination showed large masses of staphylococci in the endocardial deposits. There were also considerable numbers of these germs in the fresh suppurating embolus in the left artery of the Sylvian fissure. In the older infarcts in the spleen and the kidney there were also masses of cocci which, however, were less numerous. No cocci could be found in the meninges.

We cannot determine with certainty, either here or in any other case, whether the new attack arose from the previously involved tissues or whether these structures had only offered favorable opportunities for the localization of staphylococci from elsewhere. It may be said with certainty, however, that the clinical picture does not offer in these cases any appreciable differences from those seen in the primary affection.

In the *recurrent forms, which pursue a slower course*, the heart muscle is, as a rule, involved more and more, in addition to the lesions at the valves and orifices. The final result is the clinical picture of heart failure, in which the changes in the endocardium occupy the background. Case III offered an example for this type of chronically recurrent heart disease. I shall relate another case which I was able to follow for more than six years to its fatal termination.

CASE XIII.—*Recurrent endocarditis; insufficiency of the mitral and the aortic; stenosis of the mitral orifice, and in a lesser degree also of the tricuspid; aortic stenosis; dilatation of the ascending aorta. The heart muscle relatively competent; the lungs also. Cachexia due to frequent pregnancies and excessive loss of blood during labor; amyloid degeneration of the kidneys, intestines, and spleen. Thrombosis in the internal carotid and in the left Sylvian artery. Death from croupous pneumonia.*

Mrs. H., aged thirty-six years, at the time of death; had erysipelas of the face several times when she was young. She also had chlorosis, and her menstruation began only when she was twenty years old, but had continued regularly since then. She was married at twenty-seven, and had two children. Everything was normal during the first pregnancy, but the child was delivered with forceps. The puerperium was perfectly normal, and she was able to continue doing very exhausting work without exertion, as before. In December, 1885, she became pregnant for the second time. At first everything went well, but as the work of the field became more and more difficult during the spring and summer, she suffered much from stabbing pains in the side, shortness of breath, and "pressure over the heart." She was obliged often to sit down and "get her wind"; was always very tired, and finally became incapable of doing any hard work.

First admission. September 19, 1886. Discharged March 18, 1887. The patient

was delivered on the day of admission. The placenta had to be separated manually and there was considerable hemorrhage, accompanied by slight collapse. The heart was markedly enlarged; the apex-beat in the sixth intercostal space. There was a systolic thrill at the apex and a diastolic thrill over the aorta. The sounds were not pure at any point; indistinct, without definite rhythm, and the second pulmonary tone could not be distinguished.

On the following days there were slight elevations of temperature without local symptoms. The pulse was accelerated, 110 to 130 per minute; the respiration between 24 and 30. The patient recovered slowly, and during the fifth week she was able to get up for several days. The behavior of the heart and of the circulation during the subsequent months was as shown in Table I, p. 228.

During the time of observation, which covered half a year, the highest frequency of the pulse was 135, the lowest 80, and the average of the morning observations was 101. The pulse was always more or less irregular. The temperature was elevated only for a short time. On one occasion, on January 28, 1887, it rose to 39.2° C. (102.5° F.) at noon, but after four hours it sank to 38.4° C. (101.1° F.). The lowest temperature was observed on March 13 at 8 o'clock in the morning and was 36° C. (96.8° F.). However, greater variations in temperature than occur normally were noted later, and the regular distribution over the day, as is generally observed in health, was absent.

The patient left the hospital at the end of March to visit relatives. At the second admission, from June 14th to September 24, 1887, she showed, in addition to the changes in the heart and the aorta, that she was succumbing more and more to the disease. Her voice especially had become so weak that one had to listen carefully in order to understand her. There was a slight catarrh of the true vocal cords, which continued for some time. A report from the surgical clinic, dated August 1st, was as follows:

"No paralysis of any particular muscle can be detected. The larynx, as a whole, however, moves somewhat less actively than normally. The glottis closes completely when the patient exerts herself, but during ordinary phonation both the cartilaginous as well as the ligamentous glottis shows a gap which is very slightly wider than normal. The true vocal cords are very slightly thickened, light gray in color, and covered with a little mucus. The rest of the larynx is normal. The cause of the muffled voice may, therefore, be sought in the deficient nutrition of the laryngeal muscles, especially, however, in a weakness of the air current."

The pulse was again about 100. There was no rise of temperature. In addition to the dilatation of the ascending aorta (see Table II, p. 229) the bounding pulse [p. celer.—Ed.] was noteworthy.

A more careful observation for the third time began on February 15, 1888. In the meanwhile the patient had only been seen from time to time during office hours. The weakness had greatly increased, and during the last few months the patient would leave her bed reluctantly and with a great deal of exertion. She had once more become pregnant. The behavior of the arterial system and the heart for the period from June 14, 1887, to February 25, 1888, is shown in Table II, p. 229. A slight diminution in the size of the cardiac dulness occurred up to the first of April, but the dilatation of the ascending aorta had in the meanwhile become more extensive (by about 0.5 cm.). The frequency of the pulse was at first 100, or thereabouts, but diminished somewhat later on. The lowest average for a week was 70, and the lowest single count was 60. Of the new manifestations, we may mention dry pleurisy on the right side and slight nodular erythema. No elevation of temperature was noted. The woman remained under treatment with unimportant interruptions until her death.

In addition to bronchial catarrh, which, however, did not become very severe at any time, she complained from time to time of pains in the long bones, in the joints, and in the muscles. Table III, p. 230, contains data concerning such an attack.

On the whole, there were but slight variations in the cardiac signs during the last years of life. The severe disturbances at the valves of the left side of the heart and the dilatations of the aorta remained practically the same. Transient enlargements of the area of cardiac dulness were noted from time to time, especially when bronchial catarrh was present. On the other hand, there was a change in the temperature during the last three months. Formerly the temperature had been perfectly normal or even subnormal with a few exceptions, but during the last period there were more or less pronounced rises which occurred not infrequently. It is scarcely necessary to demonstrate this by means of tables which I possess, and which comprise the record of fifteen weeks with usually four daily measurements.

TABLE I.

	Sternal Line.	Nipple Line.	Apex-beat.	Venous Orifice; Mitral or Tricuspid.	Arterial Orifice; Aortic or Pulmonary.	Other Features.
	Dulness.					
Left.....	October 29, 1886: Upper border of third rib.	From upper border of third rib to apex in curved line with convexity upward.	Sixth intercostal space; anterior axillary line.	A rough, rasping prolonged murmur at apex, systolic; also a distant diastolic murmur, and a faint tone heard besides. Two prolonged rasping murmurs in the fifth intercostal space, near the sternum.	A systolic tone also heard over the aorta, but impure and accompanied by a blowing murmur. A long, rough, hissing diastolic murmur; no second sound.	Marked concussion of the chest-wall. The carotids and subclavians pulsate markedly. A dull heart-sound may be heard without any aid at a distance of one meter from the patient. Systolic thrill felt over apex. Diastolic over aorta. Epigastric pulsation.
Right.....	A finger-breadth beyond this.			No distinct tones to be heard.	No distinct tones can be heard.	No venous pulsation at the neck.
Left.....	November 3d: Lower border of second rib.		November 3d: Sixth intercostal space, and projects slightly beyond the anterior axillary line.	November 3–6: Diastolic murmur is more marked at the apex, while the systolic is still the more prominent.	November 3–6: Nothing but a double murmur transmitted to the carotid artery.	November 3–6: A systolic thrill over aorta now also felt. The diastolic is now more marked and extends about three finger-breadths to the left.
Right.....	Nov. 3d: Extends over this by three finger-breadths.					November 6: For the first time, a weak pulsation of the veins of the neck.
Left.....				January 26, 1887: Much fainter systolic murmur at the apex. March 1st: murmur is again louder.	January 26, 1887: The systolic aortic murmur is much louder than the diastolic.	
Right.....						March 1, 1887: Hepatic-venous pulse.

TABLE II.

	Sternal Line.	Nipple Line.	Apex-beat.	Venous Orifice, Mitral or Tricuspid.	Arterial Orifice, Aortic or Pulmonary.	Remarks.
	Dulness.					
June 14,1887.						
Left.....	Third rib.		Fifth intercostal space a little outside of the nipple.	Diastolic murmur over the apex.	Systolic murmur over the aorta, transmitted into the carotids.	The left side of the chest is somewhat prominent, the ascending aorta enlarged, as shown by percussion in the second right intercostal space. Width, 4 cm. Height, up to the sternoclavicular articulation, where there is a marked systolic thrill. Pulsus celer.
Right.....	One finger-breadth beyond the same.			No distinct tones.	No distinct tones.	No venous pulse.
Feb. 25,1888.						
Left.....	At the second rib.	Reaches nearly up to the second rib.	Sixth intercostal space a little beyond the nipple line.	At the apex a systolic and a diastolic murmur.	Over the aorta a systolic and a diastolic murmur. The systolic is longer.	Extensive violent concussion of chest-wall. Marked thrill over apex, longer and more marked over aorta. Ascending portion gives dulness from second right intercostal space to the sternoclavicular articulation. Width, 5 cm. Heart-sound very dull; audible at 1 meter distinctly.
Right.....	6 cm. beyond the sternal border.			No distinct tones.	No distinct tones.	Veins of neck pulsate at expiration. No systolic pulse.

TABLE III.

	Dulness.		Apex-beat.	Venous Orifice, Mitral or Tricuspid.	Arterial Orifice, Aortic or Pulmonary.	Remarks.
	Sternal Line.	Nipple Line.				
December 17, 1888.						
Left.....	Second intercostal space.		Sixth intercostal space, two finger-breadths beyond the nipple line.	Blowing systolic murmur over the apex.	Loud systolic and faint diastolic murmur over the aorta.	The movements of the heart visible over a considerable area on the chest-wall. Thrill, chiefly systolic, over the region of the apex, and in second right intercostal space. Loud muscular tones of heart audible at distance. Venous pulse at neck.
Right.....	Four finger-breadths from the sternal border.					

Table IV (p. 231) gives a review of the period from July 15th to September 13th, during which the frequency of the pulse, the amount of urine secreted, and the temperature were noted in groups of ten days each.

It will be seen how little change there was in the frequency of the pulse and how independent the pulse-rate was of the temperature. The amount of urine secreted is in this case no criterion of the work of the heart, for the autopsy showed amyloid degeneration of the kidneys. It must be emphasized, however, that during the last few weeks the amount of urine secreted increased up to two liters and still showed that a considerable amount of functional capacity existed in the heart muscle. It seemed as though the patient would recover once more, but a pneumonia appeared on September 27, 1892, after an attack of right-sided hemiplegia with aphasia, and produced the fatal end.

The following brief notes may be given from the very accurate report on the autopsy (Professor von Baumgarten):

"There was a slight edema of the ankles and of the dorsum of the right hand. A small amount of clear fluid was found in the peritoneal cavity; in the left pleural space there was no effusion, while in the right there were a few tablespoonfuls of a cloudy purulent fluid. In the lower lobe of the right lung there was fresh red hepatization. There were old adhesions below and behind on the right side. Both lungs were moderately emphysematous. There was a fresh edema.

"*The Heart and Blood-vessels.*—The heart was more than twice as large as the fist, and was quite extensively covered with fat. The apex was formed of the left ventricle alone. The right auricle and ventricle

were dilated, and contained a considerable amount of blood, most of which was clotted. The base of the ventricular cone *in situ* measured 10 cm., and its length was 12.5 cm. The arch of the aorta projected markedly forward within the pericardium. The left auricle contained a considerable amount of coagulum. The mitral orifice barely admitted the tip of one finger. Water introduced into the aorta drained off immediately. The right auricle contained a large yellow clot. The tricuspid was large enough to admit two fingers easily. The pulmonary artery and its valve were normal. Both ventricles and both auricles were considerably dilated. The right auricle, measured without the epicardium and without the trabeculæ, was 4 mm. thick, on the left side 14 mm. The papillary muscles of the left ventricle were markedly thickened. The tricuspid was thickened evenly, but not very markedly at its edge; its flaps and its tendinous cords were not shortened. The mitral showed a markedly diffuse thickening; its tendinous cords were short and much thickened. A reddish, uneven plaque, in part incrusted with lime, in part composed of fresh soft masses, was found upon the auricular surface of the aortic flap. The plaque was of the size of a dime and several millimeters thick. There were several fibrinous clots upon the surface. The mitral orifice was 3.5 cm. wide, the valves stiff, slightly movable. The aortic aperture was 5 cm. wide. The semilunar valves were markedly thickened, shortened, but slightly movable, and adherent to one another at their ends. There were no chalky deposits or thrombi on these valves. The aorta widened up to 8 cm. between the aortic valve and the origin of the great vessels.

"The heart muscle was quite flabby in consistence and somewhat brownish in color. A focus of the size of a wheat-grain, brownish-red in color and somewhat depressed, was found in the wall of the left ventricle. The endocardium and the intima of the aorta did not show anything abnormal, save the changes in the valve. No thrombi were noted anywhere in the heart cavity.

"In the brain there were

TABLE IV.

	Pulse.			Urine Secreted in Cc.			Temperature in °C.			Remarks.
	Aver.	Maxim.	Minim.	Aver.	Maxim.	Minim.	Aver.	Maxim.	Minim.	
July 15 to 24	81	99	68	423	600	220	38.7	39.5	37.7	Average urine only determined for nine days.
July 25 to August 3	82	96	70	372	560	150	38.2	39.4	37.6	
August 4 to 13	79	90	70	692	1100	480	38.2	39.3	37.4	
August 15 to 24	80	84	72	?	840?	400?	38.0	39.6	37.3	
August 25 to September 30	76	84	72	?	950	420	37.4	38.0	37.0	Owing to frequent involuntary urination no exact measures could be obtained.
September 4 to 13	85	96	72	1297	2000	810	37.4	37.7	37.0	Average urine only for nine days.

thrombi in the trunk of the left carotid, and in the left Sylvian artery. There was also softening in the left temporal lobe, the central and the lower frontal convolutions. There was an old infarct in the spleen and the liver presented the picture of a cyanotic 'nutmeg' liver. A number of veins in the right parametrium were filled with loose mixed thrombi. Microscopic examination showed marked amyloid degeneration in the kidney, spleen, and intestines."

Further microscopic examinations revealed: "Deposits on the mitral valve consisted chiefly of connective tissue with a little fibrin, and on staining did not show cocci. A slight thickening was found in the intima of the artery of the Sylvian fossa, resembling that of obliterating endarteritis or the earlier stages of arteriosclerosis."

We were dealing, therefore, with a disease which could not be proved to have originated through rheumatic or septic infection. Possibly the erysipelas which occurred four or five times during the patient's youth may be considered in the etiology. The beginning of the cardiac affection could not be traced more accurately, for, even at the first admission, there had been a completely developed change on the endocardium of the valves, with all its consequences. The valvular symptoms varied, especially during the first period of observation, and a great deal of variation was also noted in the extent of the cardiac dulness, which would increase and then again decrease in length, as well as in breadth. The position of the apex-beat also changed accordingly. The dilatation of the ascending aorta was discovered during the course of the first year of observation.

The narrowing of the tricuspid valve, which was slight, may be said to have had rather a beneficial effect. A venous pulse of marked character was seen at the neck only for a short time. The heart muscle stood its ground for a comparatively long time, and this possibly was the reason why the frequency of the pulse, on the whole, had remained so low—naturally with a few exceptions. Therefore, the heart had been granted a sufficient period of rest, even during the more advanced stages of the disease. Even when the terminal pneumonia developed, the pulse rose only to 108, although the temperature reached 41° C. (105.8° F.).

The patient, naturally, was not spared the bronchial catarrh which is common in these cases, though it was comparatively benign. The lungs of the young woman were not extensively changed, and they, therefore, aided the circulation considerably. Another factor in this connection was the impossibility of doing any kind of hard work and the consequent necessity for rest. The patient remained in bed a great deal because she could not do otherwise, and during the bad periods of her disease this rest continued for months. Our nurses then took care of her household and it was possible in this manner to secure appropriate diet for her.

Her loss of nutrition may be blamed largely upon the frequent pregnancies and the abundant hemorrhages during the labors. Her children were by no means delicate and developed well. The amyloid degeneration of the intestines, the kidneys, and the spleen which was found in the cadaver was probably referable to the continuous insufficiency of nutrition which was intimately connected with the losses of blood (compare p. 259).

Another noteworthy feature of this case was the fact that effusions into the subcutaneous tissues and into the cavities were only slight, and were scarcely worth mentioning during the last period of life.

It may be easily understood that changes in the valves and valvular orifices which were the result of previous endocarditis, with their sequels,

—hypertrophies and dilatations,—showed their effects in a recurrence of the disease. It is difficult to say what influence this may have upon the composite picture of the disease, but it may be stated, in general, that the balance which is established through acquired compensation can be disturbed more easily than when the heart has not been previously affected by endocarditis.

Thus the results of simple observation at the bedside show that after every endocarditis there is a diminution in the functional capacity of the heart. The heart muscle itself is usually injured, but we cannot say as yet exactly in what manner, for the reason that we lack sufficient anatomic investigations conducted during the early stages of the disease.

Ernst Romberg* reported two cases in which he examined the condition of the heart during the early stage. He found inflammatory and degenerative processes, hyaline thrombi in the smaller branches of the coronary arteries, and isolated perineuritic processes. Nothing special was found in the ganglia. One of his cases developed after acute articular rheumatism, and the heart on examination was found free from old lesions, but showed a verrucous endocarditis which had its principal seat at the mitral, although it affected to a lesser degree the aorta and the tricuspid. There was, in addition, a total synechia of the pericardium—therefore practically a pancarditis. In the second case there were slight ulcerative changes on some of the warty excrescences. In this instance also there had been acute articular rheumatism, combined with chorea, immediately before the endocarditis. The lesions found were almost identical with those of the preceding cases, except that the pericardium was less markedly affected and was not obliterated. Changes in other organs which could be referred to sepsis were completely absent in both cases.

Romberg points out that the possibility of a purely functional change in the myocardium cannot always be excluded. What rôle such a change may play must be decided by further anatomic investigations. We do not know whether a complete recovery may not occur when the tissue changes are present but less marked. It is possible that in spite of the rich capillary anastomoses, the thrombi in the finer divisions of the coronary arteries may become even transiently disturbers of the circulation in the heart. Nor have we a clear insight into the connection of the nerves with the disturbances of the heart in endocarditis. May not bradycardia possibly be connected with changes in the nerves? All questions which relate to these subjects have been discussed by Romberg. On the whole, I agree with his opinions, and cannot here enter into minor differences, as the discussion of the subject belongs to myocarditis. [In some cases of bradycardia with degeneration involving His's bundle there has been endocarditis continuous with the former process. As bradycardia of the heart-block type occurs in some cases of sepsis, it is probable the cause lies in an extension of the endocarditic process; the conducting fibers, and not the nerves, being affected.—Ed.]

RESPIRATION AND RESPIRATORY ORGANS.

The respiratory organs are subject in endocarditis to numerous changes of multiform character. These lesions extend over the entire respiratory

* "Ueber die Bedeutung des Herzmuskels für die Symptome und der Verlauf der acuten Endocarditis und der chronischen Klappenfehler," "Deutsches Archiv für klinische Medicin," Bd. liii (1894), S. 141.

tract, beginning from the larger bronchi and ending with the respiratory tissues proper. All forms of inflammation may occur—the superficial variety, which affects only the epithelium, may be present, or the deep inflammation, which penetrates into the framework of the lungs. There may be a serous, a fibrinous, a hemorrhagic, or a purulent exudate, the latter destroying the surrounding tissues. The lesions vary widely in extent. In most instances, at least in the cases in which pus cocci come into play, there are lobular foci which affect larger areas only when they coalesce. The invasion of pneumococci, on the other hand, tends to produce lobar inflammations from the start, but there are exceptions to this rule.

The pulmonary affections are often partial manifestations of the general disease, and there may not be any local focus in the endocardium. If such a local focus is present, then large numbers of bacteria which are lodged therein as in an incubator may pass into the circulation and may settle in the lungs. This occurs when, as usually happens, the left side of the heart is affected. If the right side of the heart is affected, then an invasion of the lungs can take place directly from there. During these invasions fragments of thrombi may be swept along with the bacteria into the blood current, after having grown upon the heart-wall, and having been torn off by the circulatory movement. Simple hemorrhagic infarcts or larger abscesses may arise, according to the nature of the embolus. The same lesions may occur, although less frequently on the whole, as the result of thrombosed veins of the systemic circulation.

When the bronchi have been primarily involved (this is by no means uncommon in the more severe forms of sepsis) and when the catarrh involves the finer tubes, as it does often enough, then bronchopneumonia develops, such as we are accustomed to see in connection with severe bronchitis.

This general review of the pulmonary lesions of endocarditis must suffice here. The attempt to differentiate the various forms in the different cases is not an easy task and belongs to the bedside. In this connection we must speak, however, of a disturbance of the respiration which depends upon other causes and is so frequent that it is necessary to consider it in detail. I shall also mention a few observations on pulmonary affections in discussing these cases.

The fact that not everything is as it should be with the respiration is shown by the pulse-respiration ratio, which deviates from the normal standard of 4.5 : 1. In endocarditis, in both the severe and the mild form, there is frequently a change in the quotient $P \div R$ in the sense that it becomes smaller than normal. Let us quote a few cases. Some of them show clearly the cause of the diminution of the quotient, while others do not.

A man, aged thirty years, was admitted on November 29, 1894, with the symptoms of a diffuse myelomeningitis. During the further course of the disease an aortic endocarditis developed, together with signs of a general sepsis (see case XVIII).

The relation between the pulse and the respiration was abnormal, even before distinct symptoms developed on the part of the heart, although the rather subnormal temperature had already come to be irregularly distributed during the day, so that an average for the fifteen days of observation was as follows: Mornings, 36.9° C. (98.4° F.); afternoons,

37.23° C. (99.4° F.); evenings, 37° C. (98.6° F.). On some days the evening temperatures were as low as 36.6° C. (97.8° F.). The relation between the pulse and the respiration appears in the following table:

Date, December, 1894.	Frequency of Respiration.	Frequency of Pulse.	Ratio $\frac{P}{R}$.
10	22	80	3.6
11	20	72	3.6
12	18	70	3.9
13	21	87	4.1
14	15	80	5.3
15	16	78	4.9
16	18	82	3.5
17	18	75	4.2
18	24	84	4.5
19	24	75	3.1
20	21	72	3.4
21	21	78	3.7
22	21	78	3.7
23	24	84	3.7
24	27	96	3.6

It will be seen how variable the quotient in question was in this case. At first it was smaller than normal, then it exceeded the normal standard to a certain extent, but only for a short time, and finally again grew smaller. There was no irregular fluctuation from day to day, but the change always extended several days in succession.

Neither from the behavior of the temperature nor from any other symptom can we gain an insight into the effect of the cardiac changes from this case. After an interval of a year the patient was again admitted, on February 15, 1899. A considerable amount of disturbance in the function of the spinal cord and a lesion at the aortic valve had remained. In addition to the renewed local disease there had been three attacks of a prolonged and diffused septic exanthem. A rise of temperature was observed only on the day of admission (38.4° C.—101.1° F.), but otherwise there were, on the whole, subnormal temperatures, with a rise to 37.4° C. (99.3° F.) on one occasion only. The distribution was again irregular. The average for three daily measurements between February 16th and March 19th was: Mornings, 36.84°; noons, 36.96°; and evenings, 36.96°. The relation between the pulse and the respiration is shown in the table on p. 236.

As this table demonstrates, the ratio is here again below the normal—almost always. Very small variations on the individual days were noted, especially during the second part of the observation. No direct connection between this ratio and the other symptoms could be traced.

Both observations teach that an increase in the frequency of the respiration is the cause of the altered ratio. The frequency of breathing is certainly abnormal, in consideration of the fact that the patient remained at rest in bed and had a low temperature. During the first period of the observation there were 20.6 respirations, and during the second period there were 19.5, to the minute. What the cause of this acceleration was could not be determined.

Date, 1899.	Frequency of Respiration.	Frequency of Pulse.	Ratio $\frac{P}{R}$
February 16	21	81	3.9
17	18	78	4.3
18	19	72	3.8
19	18	75	4.2
20	18	81	4.5
21	19	84	4.4
22	18	81	4.5
23	18	75	4.2
24	19	72	3.8
25	20	84	4.2
26	19	75	4.0
27	20	75	3.8
28	19	69	3.6
March 1	20	75	3.8
2	19	72	3.8
3	20	75	3.8
4	21	75	3.6
5	21	78	3.7
6	20	78	3.9
7	20	72	3.6
8	20	69	3.5
9	19	78	4.1
10	20	69	3.5
11	20	75	3.8
12	21	75	3.6
13	20	75	3.8
14	20	75	3.8
15	20	72	3.6

On the other hand, the increase in respiratory frequency which was observed for a time in case IV may be referred to disturbances in the muscular activity. There was in this case a distinct although mild chorea.

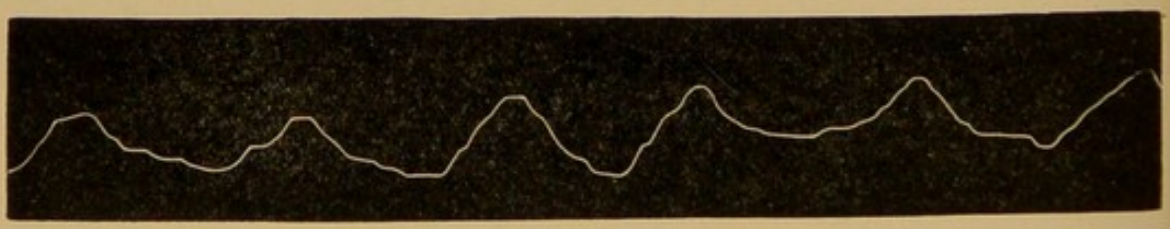


Fig. 44.

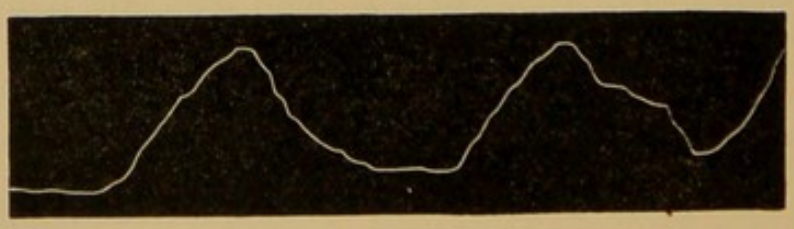


Fig. 45.

The involuntary muscular contractions which may be distinctly seen in the respiratory curve (Figs. 44 and 45) are referable to this. That it affected muscular activity is certain.

The number of respirations was always high with the temperature.

low; during the period which includes the observations recorded above it was 20 a minute, but fluctuated from 18 to 26.

The respiratory curve was recorded at the same time as the curve taken in Fig. 35; in other words, during periods of very irregular heart's action. Fig. 44 reproduces a number of respirations in their natural size. Fig. 45 shows the irregularities in the curve during ascent and descent still more clearly in a somewhat enlarged photographic print. The tambour was placed under the ensiform process.

Date, 1890.	Frequency of Respiration.	Frequency of Pulse.	Ratio $\frac{P}{R}$.	Temperature.	
				Minimum.	Maximum.
November 13	27	84	3.1	37.0°	38.0°
14	28	81	2.5	37.4°	38.4°
15	30	84	2.8	37.7°	39.0°
16	33	100	3.0	37.8°	39.0°
17	36	96	2.7	37.8°	38.2°
18	30	60	2.0	36.6°	37.6°
19	20	70	3.5	36.8°	37.3°
20	18	63	3.5	36.6°	37.2°
21	20	66	3.3	36.7°	37.3°
22	22	75	3.4	37.3°	37.9°
23	27	82	3.0	37.5°	38.5°
24	34	81	2.4	37.6°	38.2°
25	30	84	2.8	37.0°	37.4°
26	24	66	2.8	36.6°	37.0°
27	24	81	3.4	36.8°	37.1°
28	24	69	2.9	36.8°	37.8°
29	23	69	3.0	37.4°	38.5°
30	34	84	2.5	37.4°	38.4°
December 1	24	90	3.8	37.0°	37.6°
2	21	74	3.5	37.0°	37.8°
3	24	75	3.1	36.9°	37.8°

The respiration was different in case II. The table shows that the frequency of respiration became very high from November 13th to November 18th, that it diminished on the 19th, and so continued to the 22d, and that it again increased from the 24th. The cause was the extremely severe muscular pain, which became almost unbearable at any motion. The patient was actually afraid to breathe, and the respiration became superficial and very cautious. The number of respirations diminished at once when an improvement set in on the 19th, while it again increased when the patient had a turn for the worse which reached its acme on the 24th.

The table again shows that the diminution in the quotient P÷R is essentially caused by the increase in the frequency of respiration. The temperature, which was not very high, did not have any appreciable effect upon this ratio, as it affected both factors in the same way. I may mention only that, during the further course of this case, there was no other disturbance in the ratio than was seen during the first three weeks, except in single days, in spite of the fact that there was a complication of nephritis which led to a severe uremia. The frequency of the pulse had increased permanently, the respiration varied on individual days, but, on the whole, it was increased. This is shown in the following table:

Week of Disease.	Frequency of Respiration.			Ratio $\frac{P}{R}$.	Remarks.
	Average.	Maximum.	Minimum.		
1.....	29	36	20	2.9	The different days were noted in greater detail in the table on p. 237.
2.....	25	30	18	3.0	
3.....	25	34	21	3.1	
4.....	22	28	18	4.1	
5.....	26	28	21	4.2	
6.....	27	28	24	4.2	
7.....	24	26	22	4.6	
8.....	26	30	22	4.1	
9.....	25	34	16	4.6	
10.....	26	30	22	4.5	
11.....	25	27	24	5.0	

When the frequency of the pulse is markedly diminished (bradycardia), the quotient P÷R is naturally diminished, unless the number of respirations simultaneously decreases. This was not so in case X, as is shown by the following table:

Date, March, 1888.	Respiration.	Pulse.	Ratio $\frac{P}{R}$.	Temperature.		Remarks.
				Minimum.	Maximum.	
9.....	20	72	3.6	37.0°	37.2°	No special features noted in this case during the last part of the disease.
11.....	21	57	2.7	37.0°	37.2°	
12.....	21	48	2.3	36.7°	37.1°	
15.....	21	42	2.0	36.8°	37.2°	
16.....	21	48	2.3	36.8°	37.3°	
17.....	20	38	1.9	36.8°	37.4°	

The number of respirations remained almost the same, while the pulse sank from 72 to 38. The temperature continued almost at the same height.

There were much greater variations in case IX. The pulse, which diminished in frequency from February 27th (about the thirteenth day of the disease), was at first not followed by an appreciable change in the frequency of the respiration, which was quite high for the low temperature, and varied between 21 and 24. Then, suddenly, on the evening of March 9th (corresponding approximately to the twenty-third day of the disease), the respiration rose to 34 and 36. In the morning no changes were noted, and the pulse varied from 42 in the morning to 58 in the evening at rest, while it reached 70 when the patient got up. The cyanosis, which had grown more marked during the preceding week, increased still further.

In this case there seemed to have been disturbances in the respiratory movements which were produced as the result of some central nervous changes, for pauses were noted at the acme of inspiration. There were also other cerebral symptoms: slight delirium, which did not correspond to rises or sudden changes in temperature. The bradycardia continued for a long time, but the increased frequency of respiration lasted still longer. Subjective respiratory difficulties were added to these from time to time.

From the history of case V, I have constructed three tables which are inserted here. Table A includes the first ten days after admission. The signs of a heart affection begin to develop, the lungs seem to be perfectly free, and yet there is already an absolute increase in the respiratory frequency and a distinct diminution of the quotient P÷R. Table B begins in the middle of the fourth week of observation and lasts to the middle of the fifth week. It shows that the frequency of respiration is still considerable (average, 24.6), but is lessened in comparison with the first period (average, 27.6), in spite of the fact that the affection of the heart had developed further by this time. The quotient P÷R averaged 4.6 and was, therefore, greater than normal. On some days this figure was exceeded considerably.

TABLE A.

Date, August, 1896.	Frequency of Respiration.	Frequency of Pulse.	Ratio $\frac{P}{R}$.	Temperature.	
				Minimum.	Maximum.
21	37	120	3.2	40.1°	40.2°
22	37	102	2.8	39.0°	39.5°
23	30	88	2.9	38.7°	39.7°
24	28	90	3.2	38.0°	39.5°
25	20	60	3.0	37.6°	39.3°
26	24	80	3,3	37.8°	38.8°
27	26	90	3.5	37.4°	39.8°
28	24	89	3.3	37.4°	39.8°
29	26	100	3.9	37.4°	39.0°
30	24	94	3.9	37.4°	39.2°

Symptoms observed during this period: Multiform exudative erythema; swelling and painfulness of the joints, signs of endocarditis beginning with the twenty-fifth; pain on pressure of the thighs beginning with the twenty-sixth.

TABLE B.

Date, September, 1896.	Frequency of Respiration.	Frequency of Pulse.	Ratio $\frac{P}{R}$.	Temperature.	
				Minimum.	Maximum.
15	22	95	4.3	37.0°	39.3°
16	24	112	4.7	38.1°	39.6°
17	28	120	4.3	38.1°	39.9°
18	28	125	4.5	38.2°	39.7°
19	25	120	4.8	38.7°	40.0°
20	23	118	5.1	38.7°	39.7°
21	22	115	5.2	39.1°	39.6°
22	25	102	4.1	38.4°	39.2°

Symptoms observed during this period: The affection of the heart was more prominent; the emaciation increased. Here and there new joints and bones become involved.

A glance at the figures shows that the increased quotient P÷R is due to the increased frequency of the pulse. Why did this rise? Certainly not on account of the temperature, which was much higher than it had been during the first period. At that time again, from some undiscoverable

cause, the frequency of the pulse was comparatively low, at least at the beginning.

TABLE C.

Date, October, 1896.	Frequency of Respiration.	Frequency of Pulse.	Ratio $\frac{P}{R}$.	Temperature.	
				Minimum.	Maximum.
23	44	140	3.2	37.0°	38.5°
24	42	134	3.2	37.1°	38.6°
25	44	136	3.1	37.0°	38.6°
26	42	134	3.2	37.5°	38.8°
27	48	132	2.8	37.9°	38.7°
28	46	132	2.9	37.6°	38.8°
29	48	135	2.8	37.9°	39.0°
30	54	132	2.4	37.3°	39.0°
31	48	140	2.9	37.9°	38.6°

Symptoms observed at that time: On the twenty-fifth cough; on the thirtieth, foci in various parts of the lungs; dulness, crepitant râles, and finally bronchial breathing.

Table C, from the beginning of the second month of the observation, shows once more a marked increase in the frequency of respiration, together with the distinct diminution of the quotient $P \div R$, although the frequency of the pulse also had become increased. The reason is easily discerned: Foci were developing in the lungs, which steadily increased the frequency of the breathing. I may note here a case in which there was also a pulmonary affection which assumed the type which is very often seen in the course of severe protracted cases of endocarditis, and which often enough leads to death.

Case XIV.—A girl, aged twenty-two years, died on July 17, 1894. She had complained of pains in the limbs since 1882, when first seen in the dispensary. At that time the heart had already become affected. Relapses occurred as follows: 1883: Mitral disease, chiefly insufficiency, was distinctly marked. The attack lasted for a considerable time, but was not very severe. 1888: A mild attack. 1889: From May 16th to July 12th there was a severe attack in which the heart was severely involved. There was pericarditis with a considerable effusion of fluid. The base of the triangle measured 15 cm., its height, 11 cm. The first attack in that year, beginning with May 20th, was followed by a second, which was less severe, but was accompanied with a renewed effusion into the pericardium on June 24th. The aorta was also involved. On the left side there was a considerable effusion in the pleura. Later, there were ill-circumscribed signs of consolidation in the right lower lobe and a small pleuritic exudate. The heart muscle was markedly damaged, especially in the later phases of the attack. The temperature repeatedly rose to over 40° C. (104° F.). The last rise of temperature occurred at the beginning of the seventh week of the disease.

1890: A milder attack, lasting from December 16th to January 20th. Insufficiency of the aortic valve was clearly made out on admission, presenting, among other signs, a characteristic "*pulsus celer.*" In the course of the attack pericarditis occurred without any demonstrable effusion. There were temporary attacks of heart failure. The temperature rose only to 39.8° C. (103.6° F.), but after December 31st, there was no more fever. Slight relapses occurred in July, 1890, and in July, 1893.

Last admission, from July 2, 1894, until death, July 17, 1894: The nutrition of the patient was very much impaired as compared to her former condition. The affection of the heart had an evident effect upon the distribution of the blood.

Heart Outline.—Above, the heart dulness was at the lower border of the second rib. On the right side, it was 0.5 cm. outside the right sternal border.

The apex-beat was in the sixth intercostal space, most distinct slightly outside the nipple line, but very diffuse. The dulness extended in the shape of an arc, convex outward from the second rib to the region of the apex. Strong pulsations

of the thorax with each movement of the heart, the impulse extending throughout the area of dulness and its neighborhood. The closure of both aortic and pulmonary valves was palpable.

Auscultation.—The region of the apex: a loud systolic and a somewhat weaker diastolic murmur, with corresponding thrills. Aortic: two tones, together with a diastolic and a somewhat weaker systolic murmur. Pulmonary valve: a systolic murmur resembling that heard at the apex, also a loud diastolic tone. A double tone was heard over the crural and brachial arteries. A systolic jugular pulsation. The venous pulse of the liver was so marked that a sphygmogram was easily recorded from the considerably enlarged organ (Fig. 46). Pulsus celer irregularis.

On admission, there were many fine and large moist râles; expectoration profuse, watery, foamy, slightly red, and contained a few red blood-corpuscles; there was a severe hacking cough which lasted four days. There was an effusion in the left pleural cavity, extending to the angle of the scapula. Over this area there was diminished respiration, which extended forward to Traube's space, which was filled with fluid. This effusion could not be demonstrated at the end of about a week.

On the right side there was slight diffuse dulness and diminished breathing, which appeared at first on July 13th in the region of the scapula. On the following day this dulness became more marked, extended somewhat more upward, and could not be defined with accuracy. A slight dulness was also noted on the posterior aspect of the upper left lobe. Over these places, on both sides, there was diminished vesicular breathing, the râles had diminished, and the expectoration had become very slight. The local manifestations could not be determined with the same accuracy during the next few days which preceded death. The feeling of cardiac anxiety and difficulty in breathing varied in severity. The heart's action became somewhat calmer, but the edemas increased. Death occurred without any special symptoms.

The following were the chief points noted at autopsy (Pathologic Institute: Dr. Roloff): "The pericardium was exposed to an extent exceeding the size of the palm of a hand. It was firmly adherent to the adjoining parts of the lungs. The apex of the heart extended to the sixth rib, outside the mammary line. The two layers of the pericardium were closely adherent to each other by dense adhesions, which in some places were loose and edematous.

"The heart was more than twice as large as the fist of the cadaver. The ventricles were dilated the left one more than the right. The mitral valve admitted two fingers, and the flaps could be felt adherent to each other and projecting into the lumen. Water introduced into the aorta flowed out very rapidly. On opening the heart the mitral valve was found contracted in both flaps, and the line of closure studded with very delicate warty structures. The tendinous cords were not markedly thickened. A circumscribed patch with a somewhat roughened surface, but without any real excrescences, was found upon the posterior aspect of the aortic flap, near the origin of the aorta.

"The aortic valves were considerably shortened, their borders thickened and studded with verrucosities. There was no narrowing of the aortic lumen. Pulmonary and tricuspid valves negative.

"The wall of the right ventricle was 5 mm. thick, its musculature, on the whole, pale, and studded with intensely yellow spots which were especially numerous in the papillary muscles. The left ventricle was 11 mm. thick, and its muscular structure flabby, yellowish-red, and in many places showed dull yellow spots with indistinct outlines. The coronary arteries were dilated and thin walled. A number of small, rounded, white thrombi were found in the right auricle, among the pectinate muscles.

"The lungs were distended, and adherent to the chest-walls, especially in their lower and lateral aspect. The left lung contained air, but was slightly increased in consistence. Its lingula was bluish-red and somewhat tough. The pulmonary tissue was dark-red, very rich in blood, and on pressure exuded an abundant amount of foamy fluid. The bronchial mucous membrane was swollen, reddened, and covered with bloody mucus. The branches of the pulmonary artery contained fluid blood; including the branches leading to the lingula. The airless tissue of the lingula was partly dark-red, partly grayish-red, in color, and appeared distinctly granular on section.

"The right lung was very similar to the left, and showed a marked hyperemia, but contained air. There was increased consistence and edema. The anterior half of the middle lobe showed the same appearance as the lingula on the left side, and its vessels also contained nothing but fluid blood.

"There was, in addition, hyperplasia of the spleen; fatty degeneration of the kidneys; a cyanotic and jaundiced 'nutmeg' liver; congestive catarrh of the stomach; thrush in the esophagus; and anemia of the brain."

The bacteriologic examination: "The microscopic investigation of smears from the warty structures on the endocardium showed a few diplococci and short chains consisting of four links. No bacteria were found in the juice of the spleen nor in the infarcted portion of the lung."

On admission, there had been edema of the lungs, with which there could have been infarcts in these organs. An effusion into the pleural cavity which was not inflammatory, but a transudate, was the next phenomenon noted. It disappeared rapidly, however. Finally, about four days before death, a consolidation in a portion of the right lung appeared, with more distinct clinical signs. In heart disease the infarct formation which was present here, in addition to the process which plugged the branches of the pulmonary artery, is by no means rare. The fact that this infarct formation arises first through the permeability of the capillaries for red blood-cells seems to point to nutritive changes in the walls of the vessels, changes which, as we know, may be induced by a slowing of the blood current. This sluggishness of the blood-stream necessarily follows upon heart weakness, and the latter here became distinct enough toward the end of the patient's life.

The marked slowing of the pulse which then appeared may probably be referred to central causes, but we know from experience that a slowing of the pulse is more frequent when there is fatty degeneration of the heart muscle. The reason for this, however, still lacks the proper explanation.

An important feature of this case was the fact that, while the patient's condition had become fairly bearable at the beginning of the last attack, yet the last relapse, which in itself was mild, became extremely severe in a very short time (compare case III). In the notes of the history which were made on admission we find the following:

"The patient had been feeling quite well since last winter, but during the last few days she complained of palpitation, shortness of breath, and very persistent cough." Fourteen days later she died, and her death was certainly due chiefly to heart failure.

The following table shows the course of the respiration and of the pulse:

Date, July, 1894.	Frequency of Respiration.	Frequency of Pulse.	Ratio $\frac{P}{R}$.	Temperature.	
				Minimum.	Maximum.
3	42	120	2.8	37.9°	39.0°
4	45	120	2.7	37.8°	38.6°
5	44	120	2.7	37.2°	38.4°
6	39	122	3.1	37.4°	38.3°
7	36	120	3.3	37.8°	38.3°
8	39	128	3.3	37.4°	38.3°
9	38	111	3.0	37.5°	37.9°
10	42	117	2.8	37.3°	38.4°
11	44	117	2.7	37.0°	37.9°
12	44	120	2.7	37.1°	37.9°
13	45	120	2.7	37.8°	38.4°
14	50	102	2.0	37.3°	38.2°
15				Temperature	
Morning	56	104	1.7	37.2°	
Evening	62	92	1.5	36.2°	
16					
Morning	60	75	1.3	36.0°	
Evening	42	60	1.4	35.7°	
17					
Morning	42	60	1.4	36.2°	

At that time the symptoms were: A profuse, foamy, slightly bloody expectoration, almost purely serous in character; no foci in the lungs, but circumscribed catarrh. On July 6th there was an effusion into the left pleura, which disappeared on the 14th. Beginning with July 13th an indistinctly circumscribed dulness appeared in the right lower lobe, accompanied by diminished breathing. This area of dulness varied in extent, but was never sharply circumscribed until death. The heart weakness increased gradually, and the same may be said of the failure of nutrition.

The tracing of the venous pulse of the liver, taken on August 7th, shows how long the heart's action continued to be comparatively vigorous.

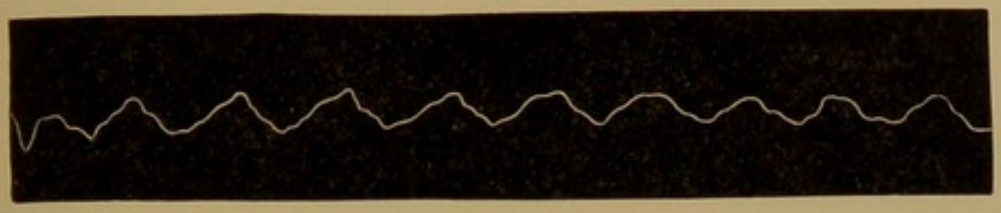


FIG. 46.

It is no easy work to transmit regular pulsations to a considerably swollen liver in the manner shown in Fig. 46, when there is such a marked disturbance of the mitral valve, and such an extensive insufficiency of the tricuspid. The sphygmogram of the radial artery taken five days later (Fig. 47) does not indicate a marked heart weakness, although it shows great irregularities. And yet, on the following day, the onset of consolidation of the lungs marked the beginning of the end.

The cases in which there is a development of foci in the lungs as the result of a direct localization of the bacteria of sepsis,—those in which infarcts form in the lung as the result of emboli or autochthonous thrombi occurring with markedly weakened hearts, and those cases in which pleu-



FIG. 47.

risy gives rise to pain or effusion,—are accompanied by disturbances in respiration which are far more easily understood. In these cases the cause of the respiratory disturbances is the diminution in the available area of the lungs, or their diminished expansion consequent upon pain.

In its ultimate analysis this phenomenon, however, is dependent upon the central control of respiration. It is possible that, in the cases in which such mechanic interference cannot be demonstrated, some substance generated by the pus cocci themselves—let us say, a metabolic product of these germs—may directly influence the respiratory center. For, whether the heart be regarded as the chief site of the infection or not, the respiratory difficulty is present.

Dennig* regards all our cases as instances of septic infection, arriving at this conclusion as the result of a careful analysis of our histories: "As regards the respiratory apparatus, we must note, in the first place, that the breathing is, as a rule, considerably increased in frequency, and that this increase takes place independently of fever, inasmuch as we find such increased respiration very often during defervescence or in the presence of very slight rises of temperature. We find between 30 and 40 respirations per minute, or even more, without being able to account for this frequency by percussion or by auscultation. In other cases the foci we are able to demonstrate are so small that they alone could not possibly be the causes of the increased frequency of breathing. Nor does the autopsy give a clue to the cause of the dyspnea. Perhaps we may find here and there small hemorrhages or an increased amount of blood in the lungs. In other cases there may be miliary abscesses. To this last class, perhaps, the hypothesis advanced by Rühle may be applied. This author thinks that the increase of frequency of the respiration, without any dyspnea in the strictest sense, is probably derived from a stimulus carried by centripetal nerves. This stimulus is probably exercised by the numerous tuberculous eruptions upon the peripheral fibers of the vagus in the lung. We know that a certain degree of stimulation, when applied to the central end of the severed vagus, increases the frequency of the diaphragmatic contractions (Traube). In septicopyemia the miliary abscesses play the same rôle as the tuberculous eruptions just mentioned, and thus may produce an irritation of the peripheral fibers of the vagus. In those cases in which a negative result is obtained both during life and at the autopsy we may fall back upon the action of ptomains, and may assume that the products of metabolism of the bacteria so influence the respiratory center that the frequency of respiration is increased."

Litten† touches briefly upon the subject. "The phenomena on the part of the respiratory organs were not very prominent," but, he continues further on, "the bronchial catarrh is a very frequent, almost characteristic, accompaniment of septicemia." He considers, however, the anatomic, rather than the clinical, conditions.

Ernst Romberg‡ expresses himself as follows in the discussion of "acute malignant endocarditis": "The respiration is usually markedly increased in frequency, probably as the result of central influences. Subjective dyspnea, however, is not usually noted. The marked acceleration of the breathing is but comparatively rarely due to extensive infarcts, pneumonias, pleurisies, or to a pneumothorax which develops as the result of the perforation of a suppurated infarct."

Therefore, even the large material of the Leipzig clinic has not given any further clue to the solution of this question.

The conclusion, therefore, must be: When a disturbance of the respiration occurs, as it often does, in an endocarditis, we must first seek a reason for it which may explain its origin. If we do not succeed in finding such a reason, then we are permitted, in the present state of our knowledge, to think of the direct influence of the germs causing the disease—the products of their metabolism—upon the respiratory center. At any rate, this must remain the emergency diagnosis until experiments conducted under proper and perfectly accurate conditions demonstrate the proof for this possibility.

KIDNEYS.

Disturbances of the renal function occur frequently in all forms of endocarditis. They are caused: (1) By septic infection. (2) By substances which are carried into the kidneys from the left side of the heart, and which act more or less strongly as local causes of disease. (3) By circulatory changes caused by the heart weakness.

Although this is the general synopsis of this matter, it is not easy, in fact, impossible, to classify everything that occurs within these three groups. He who does not care to be schematic, will be embarrassed if

* "Septische Erkrankungen," S. 122. † *Loc. cit.*, p. 450.

‡ "Handbuch d. praktischen Medicin," Ebstein und Schwalbe, Bd. i, S. 978, Stuttgart, Enke, 1899.

he attempts to apply this classification to all cases. It is better, therefore, to bring out the facts learned by observation, and to place them in the foreground.

A little albumin, and possibly a small number of casts, often occurs in the urine temporarily. This seems to be an occurrence common to all acute infectious diseases. We assume that the excretion of smaller or larger quantities of substances which are in themselves but slightly effective produces morbid changes when it takes place through the kidneys. High fever may also be the cause of these very slight and temporary changes. In case IX the affection of the kidney was of somewhat longer duration. The urine contained small amounts of albumin from the second of March (end of the first week of treatment, approximately corresponding with the first week of the disease) until May 17th

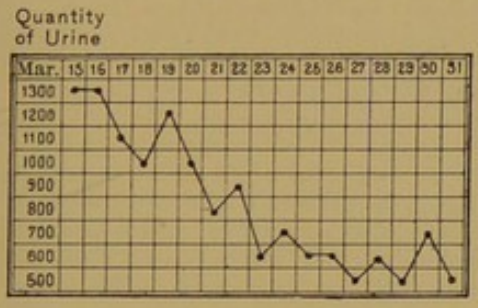


FIG. 48.

(about ten weeks). There was, in addition, an abundant sediment consisting of desquamated epithelia, together with a few granular casts. A noteworthy feature was the fact that the daily amounts excreted, which at first were normal, soon began to diminish and continued to be low, even after the albumin had completely disappeared.

Fig. 48 shows this diminution in the amount of urine at its beginning. During the first six days the average excretion was 1150 c.c. During the following eleven days it sank to 636.

The specific gravity varied greatly at first, and fluctuated between 1004 and 1020. In the daily amounts collected separately for each day it fluctuated between 1025 and 1031, although no cause could be assigned for this. Until May 8th, the amounts were not measured daily, but from this day on until the end of the observation (July 18th) the amount of urine excreted daily was regularly measured.

Fig. 49 shows how the normal height was reached gradually, with considerable fluctuations. The following periods, each embracing ten days, gave the steadily increasing averages shown in the table:

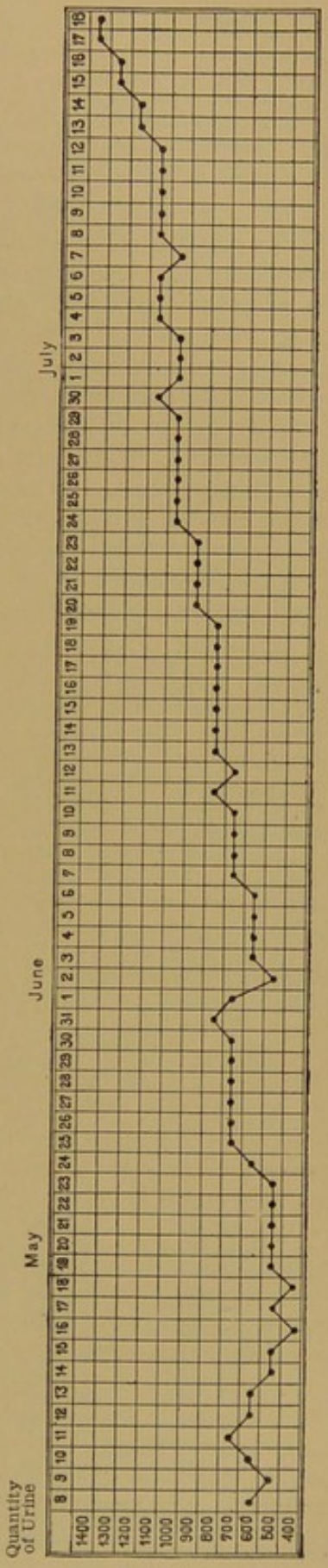


FIG. 49.

	Amount.
May 8-17th	539 c.c.
May 18-27th	550 c.c.
May 28-June 6th	610 c.c.
June 7-16th	730 c.c.
June 17-26th	897 c.c.
June 27-July 6th	1030 c.c.
July 7-16th	1123 c.c.

It is probable, therefore, that there was a renal affection here. The prolonged albuminuria pointed to this, in spite of the fact that it was so slight. It is impossible to say, however, how it came to pass that the secretory power of the kidneys was limited—was less than normal for so many weeks. We must remember that even very extensive tissue changes may occur in the kidneys without producing any effect in the urine. The case reported by Litten* demonstrates this.

Edemas (or any other signs which might point to a renal affection) were absent. When the patient was discharged, there were no signs of renal disturbances. The cardiac lesion gave just as few clues to its origin as the general infection. There were some slight cerebral disturbances, it is true, but these could not have been of great importance.

Case II teaches us (see p. 198) to what degree recovery can take place in a functionally gravely injured kidney.

Invasion had taken place from the tonsils, in which there had been phlegmonous angina. The infection took place slowly. Signs of nephritis appeared almost at the same time with the peculiar affection of the muscles of which we shall speak further on. (See p. 275.) At first only small amounts of albumin were found, which, however, increased rapidly and, together with the character of the sediment, showed the involvement of the kidney. A few days after the first appearance of the albumin there appeared formed elements in addition to a large mass of disintegrated tissue (detritus), viz., a considerable number of white, and a small number of red, blood-cells; renal epithelia, either free or packed into casts, together with many white and a few red blood-cells. On that day the amount of urine was 1800 c.c., and the reaction was strongly acid; the specific gravity, 1012. The urine was cloudy, yellowish, with a slight reddish tinge. The amount of albumin was 11 grams per liter (according to Esbach). A very careful record of the daily amounts of the urine, of its specific gravity, and its albumin content (according to Esbach) was kept, which I present here in tabular form:

Date.	Daily Amount.			Specific Gravity.			Albumin in Per Cent.		
	Maximum.	Minimum.	Average.	Maximum.	Minimum.	Average.	Maximum.	Minimum.	Average.
November 14–30	2200	1100	1680†	1014	1010	1012‡	2.0	0.2	0.9
December 1–10	1600	1130	1320§	1015	1013	1013	1.2	0.8	0.9
11–20	1240	600	800	1017	1013	1015	2.5	1.3	1.8
21–31	1020	430	660	1019	1017	1018	3.4	2.2	2.6
January 1–10	700	230	600	1020	1017	1018	5.2	2.4	3.6
11–20	1300	600	1000	1019	1016	1017	5.4	1.9	3.8
21–31	2400	1080	1700	1016	1015	1015	2.1	0.8	1.3
February 1–20	2480	1950	2200	1017	1015	1016	1.0	0.2	0.6

* "Charité Annalen," 1880, vol. vii, p. 162. Reported more in detail in my work on "Scarlet Fever" (see this series, vol. iv, 2, p. 176, etc.).

† Only twelve days with accurate data. ‡ Determined only for fourteen days.

§ Only seven days with accurate data.

This table shows that the amount of urine was not diminished at first. It never was less than 1000 c.c. per day until December 12th. The specific gravity varied about 1012, and was, therefore, quite low, a fact which, however, may be attributed to the lowered nutrition of the patient. From December 13th on, the amount of urine diminished considerably, and at the same time the specific gravity rose somewhat, although not very much. During the period between the first and the tenth of January it reached the minimum, and on the fourth and fifth of January there was almost complete renal anuria, which is not expressed in the table, because the figures in the latter apply to the forty-eight hours of these two days.

Fig. 50 shows the fall and subsequent rise of the amount of urine.

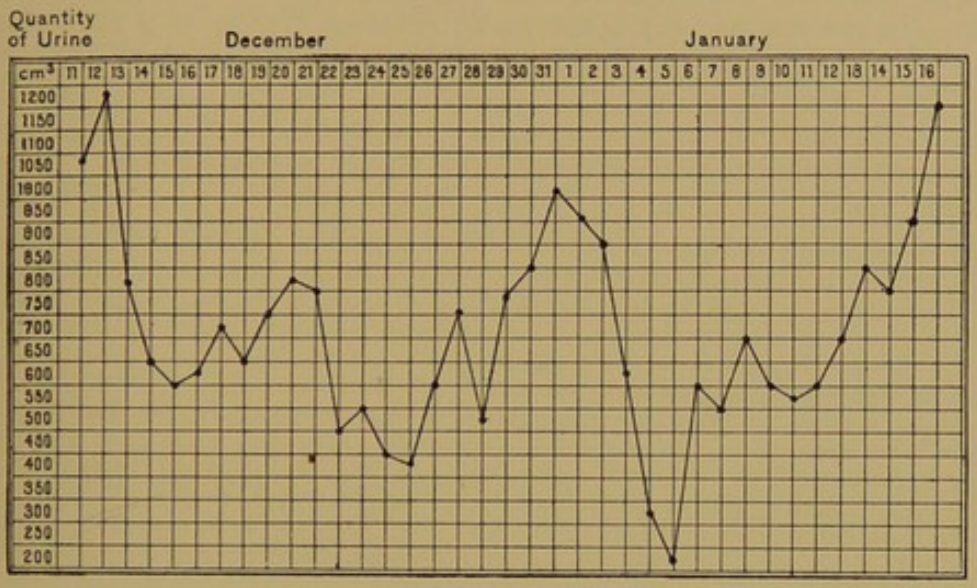


FIG. 50.

The specific gravity, which was measured daily, varied but little, and even on those days which preceded and followed the anuria it did not rise above 1020.

The numbers which expressed the amount of albumin, naturally, do not give more than a faint clue as to the actual amount excreted. This is all that Esbach's method can be expected to do; a method characterized by Carl von Noorden* as: "Esbach's albuminometer is merely a toy." On one of the days when the amount of albumin was very high the percentage obtained upon testing accurately, according to the directions, was 4.6 per cent. by Esbach's method. H. Dreser, who at that time was privatdocent in Tübingen, found but 2.4 per cent., by means of a gravimetric analysis, which he kindly made for me.

The only undoubted fact which I wish to point out is the high percentage of albumin in the urine in this case; for at times the entire urine in the test-tube coagulated in the ordinary heat-test. It must be noted, also, that the amount of blood, which increased at first very rapidly in the urine passed at night, always was smaller than that excreted during the day. In order to compare the blood in the night urine and the blood in the day urine, these were collected from noon to midnight and from midnight again to noon. This difference in the two urines disappeared after the eighth of December. The opposite ratio between the night urine and the day urine was observed in the amount of albumin. The

* "Lehrbuch der Pathologie des Stoffwechsels," S. 374.

amount of urine excreted did not differ between the day urine and the night urine, nor did the specific gravity.

The disturbances in the kidney manifested themselves as follows: On November 27th there was a slight puffiness of the face. On December 21st there was an edema of the skin, which extended over the entire body. On January 6th there was a hydrothorax on the right side, the patient always lying on that side. Uremic symptoms appeared on December 17th and increased slowly in severity. At first there was only severe headache and vomiting; then a severe attack on January 4th.

The patient, who had fallen into a deep sleep after a period of excitement at midnight, accompanied by severe headache, was found in a state of deep coma in the morning. His head was bent backward; his mouth half open; his eyes firmly closed. The pupils were small and reacted but little. Severe cutaneous irritation and loud shouting did not produce any impression. The limbs were in a perfect state of relaxation. The plantar reflexes practically absent. The temperature on the preceding day had been no higher than 37.5° C. (99.5° F.), but rose in the morning to 38.8° C. (101.8° F.), and in the afternoon at 4 o'clock to 39.3° C. (102.9° F.). In the evening the same condition continued, but seemed more threatening on account of Cheyne-Stokes' breathing. The pulse also was smaller, weaker, and had increased to 150 beats. The end seemed near.

On the morning of January 5th, however, the patient awoke, asked immediately to pass urine, and passed over 200 c.c. The temperature had also fallen to 37.4° C. (99.3° F.) and did not rise in the course of the day. The patient did not remember anything that had occurred, but complained only of pressure in the head and was very apathetic. He improved in every way toward evening.

Beginning with January 7th the patient began to suffer from disturbances of vision. An ophthalmoscopic examination by an expert (Dr. Souchay, ophthalmologist) showed the following: "In the right eye, the neighborhood of the papilla is quite markedly clouded. The papilla itself is indistinct. On the right side and below the latter there is a small white spot from which smaller white dots pass upward. There are no hemorrhages. On the left side there is a similar picture. The papilla and its neighborhood are clouded and indistinct. The diagnosis is albuminuric retinitis."

The amount of urine now increased rapidly once more. The headache and vomiting soon ceased; the edema disappeared entirely on February 2d, and no traces of the retinitis could be found on March 3d. The convalescence had progressed so far at the beginning of April that the patient could leave his bed in order to perform light housework. In the beginning of May he again began to do the work of a mason. The albumin finally disappeared from the urine only at the beginning of July.

The patient died, as I have mentioned before, from an acute atrophy of the liver, on February 4, 1892. The autopsy showed nothing macroscopically that would point to the presence of an old nephritis. On the other hand, on microscopic examination, there were "here and there distinct narrow streaks of increased interstitial tissue, residues of a nephritis from which the patient had recovered some time ago." There was no evidence of a "contracted kidney" in the proper sense. There remained, therefore, but very insignificant traces of the severe nephritis through which the patient had passed.

This is not always so, and in case X the patient developed a mottled, contracted kidney in the course of about two and a half years. The following may be noted from the history of the first attack in this case, lasting from February 2d to March 10, 1888:

A few days after admission there were already traces of albumin in the urine. Three weeks later the albuminuria increased and numerous red cells, a few white cells, some granular casts, and some isolated renal

epithelia appeared. The organized sediment was present only for a few weeks in considerable amounts, and on the fifteenth day, after the albumin could be chemically demonstrated (Heller), only a few red and a few white cells appeared microscopically. The albumin continued to be present in traces until May 25th (the eighty-second day from the beginning of this symptom), and from that time on did not disappear until the end of the observation (June 2d).

The great variations in the specific gravity on the individual days in this case were noteworthy. The specific gravity varied quite irregularly between 1030 and 1005, the reason not being apparent. This case was accompanied by a marked bradycardia, but no connection could be traced between the specific gravity and this symptom on the part of the heart. The endocarditis in this instance had left a very severe complicated valvular defect, and the patient suffered a relapse in the first week of January, 1889. At the same time nephritis appeared once more.

Soon (January 12th) there appeared in the abundant sediment a considerable number of red and white blood-cells, and of blood as well as epithelial casts. The amount of blood began to diminish on January 23d, and there was no blood on February 12th. The casts diminished in number beginning with March 3d, but hyaline and epithelial casts were still present at the close of the observation (May 8th). The specific gravity varied between 1011 and 1015. The amount of urine varied between a maximum of 2600 and a minimum of 1000 c.c. The albumin (Esbach) fluctuated between 1.5 per cent. as a maximum and 0.4 per cent. minimum.

Beginning with July, 1890, the man became permanently bedridden and was treated until his death, which occurred on August 20th. Hyaline and granular casts occurred in large numbers in his urine, together with white blood-corpuscles, but without any red blood-cells. The amount of urine varied between 1500 and 1800 c.c.; the amount of albumin between 0.5 and 1 per cent. I do not give more accurate figures, as we were not sure whether the entire amount of urine was collected and delivered by the somewhat negligent wife. The patient died of "dropsy," which seemed to have been caused both by the heart and the kidneys, as was also shown afterward at autopsy.

The autopsy showed (Pathologic Institute, Dr. Troje): Extensive pericardial adhesions; a marked hypertrophy and dilatation of both ventricles. Both flaps of the mitral were thickened fairly uniformly and partly adherent. A large number of hard nodules could be felt within the flaps. The aortic valves were thickened, calcified in small areas, and the right flap was adherent to the middle flap for a short distance. The left flap on section was found to be thickened to the extent of over 4 mm. The heart muscle was not fatty degenerated and was light-brown in color.

The capsule of the left kidney was thickened and could not be easily stripped. The surface was finely granular, mottled, due to many yellowish-white islands of tissue, of the size of a pin-head, surrounded by a network of contracted portions of dark-red color. The kidney measured 11 cm. in length, 5.8 in its widest part, and 2 cm. in thickness. On section the cortex above was 4 mm., and below, only 2 mm., in thickness. A speckled appearance was seen in the cortex and in the region of Bertini's columns, owing to the fact that there were also yellowish-white islands surrounded by reddish areas. In the region of Bertini's columns these yellowish-white foci were especially numerous and close to one another, so that the columns assumed a clay-yellow, opaque appearance, in contrast with the rest of the cortex, which in general appeared to be grayish-red.

The right kidney was 11 cm. long, from 2.5 to 4.5 cm. wide, and 2 cm. thick. On section the cortex was fairly evenly 6 mm. in width, and approximately the same picture was found on the left side. The yellowish-white foci were so numerous and

so confluent at one place that a strip of infarct-like appearance was formed about 7 mm. long and from 2 to 3 mm. wide.

Microscopic examination (some sections were examined in the fresh state; others were hardened in alcohol and stained with picrocarmin and hematoxylin, Gram-Günther's and Weigert's method. They were taken from various parts of the kidneys): In the first sections it was found that the portions of the cortex which had appeared whitish-yellow in color to the naked eye, and which were especially marked in the columns of Bertini, always contained urinary canals in a state of preservation, but the epithelia showed a marked fatty degeneration.

On the other hand, in those portions of the sections which had appeared red to the naked eye there was, for the most part, a complete destruction of the kidney parenchyma proper.

The same was found in the stained section: the columns of Bertini and the insular portions of the rest of the cortex showed urinary canals lined with markedly granular and swollen but still stainable epithelia, with nuclei that still could take up dye, and which contained hyaline casts and in most cases were found to be dilated, especially in the superficial cortical layers. The remainder of the cortex was transformed into connective tissue rich in cells and in vessels, in which the more or less atrophic shrunken and hyaline glomeruli were often arranged closely packed in groups, while no constituents of the parenchyma proper could be recognized in them. The pyramids seemed to be altered but little. Only here and there they showed in the straight tubules a marked fatty degeneration of their epithelia. At the same time these tubules contained a large number of hyaline casts. No micro-organisms were found in any part of the renal tissue.

There is no doubt that an acute nephritis in this case had terminated in contracted kidneys. The possibility that an interstitial nephritis had existed from the onset of the septic infection must be rejected, on account of the high specific gravity of the urine (1030), which continued for a long time during the first part of the disease, and was present at a time when there was no evidence of a marked cardiac weakness. Nor was there a hypertrophy of the left ventricle present at that time.

We cannot find any other cause than septic infection for the disease which affected the heart and the kidneys permanently, involved the joints, the bones, the pleura, and the peritoneum for a time, and even produced foci of the disease in the peripheral nerves. The absence of the microbes which originally produced the disease in the kidneys of the cadaver may be easily understood. A long time had elapsed after the germs had entered before death occurred as the result, not of a fresh infection, but of disease in the already damaged organs—the heart and the kidneys.

Experience teaches that a renal affection may occur as the result of septic infection and may become dangerous to life without involving the heart, or at least the endocardium, in any manner that can be demonstrated.

Septic nephritis, which morphologically belongs to the polymorphous anatomic group of acute nephritides, is clinically distinguished at times from that form of renal disease which we regard as the type of acute nephritis—namely, the nephritis following scarlet fever. And we must emphasize especially the fact that this type of nephritis may run its course almost without any symptoms. The warning signs which we are accustomed to see, the edemas, the symptoms belonging to the kidneys proper, such as sensitiveness on pressure over the renal region, may all be absent. Only the daily examination of the urine, which should never be neglected in any case of fresh endocarditis, even when there are no signs of a general disease, will give clues as to the condition of the kidney. It may occur, however, that no urine can be obtained, and then everything may remain in the dark until the autopsy. This occurred in the following case:

Case XV.—A girl, aged seven and one-half years. Treatment from February 6th to 17, 1890. Death on the latter date. After a few days of vague symptoms of illness the child was admitted, complaining of pain on swallowing and on moving her head. "The neck is also stiff." The head was held inclined to the right, and every movement was avoided with anxiety. Both active and passive motion in all directions was accompanied with severe pain. There was a considerable reddening and a slight swelling of the soft palate, but no membranes; moderate swelling of the cervical glands.

Massage of the muscles of the neck was given, together with a gargle of solution of potassium chlorate, for five days, when the head was again freely movable, and the redness and swelling had disappeared. The child was discharged.

She was re-admitted on February 12th with a bullous erysipelas of the face, diarrhea, renewed pains in the neck, but the movements of the head were free and swallowing was painless. There was a diminution in the amount of the urine secreted and only a few drops were passed with the stools. The temperature was below 37° C. (98.6° F.). During the night of February 14th she became delirious, but in the morning had a little stupor but was not unconscious. She complained of headache, and since the thirteenth had been vomiting repeatedly, with severe retching. The diarrhea continued. There was severe dysuria and only a few drops of urine were passed with a great deal of pain. The erysipelas slowly extended. The temperature on the evening of the thirteenth was 37.2° C. (98.9° F.). Otherwise it was below 37° C. (98.6° F.).

I saw the child for the first time at noon on February 15th, and found that the erysipelas had begun to recede in the portions first affected, but had spread over the right ear to the posterior aspect of the neck, showing still very distinctly the zigzag margins. The child was placed upon the table for examination, and it was noticed that she held her back and neck rigidly. Her hands groped for a firm support. The legs were crossed over each other, rotated inward; the feet were in plantar flexion. The two lower lumbar vertebræ, and these alone, were sensitive to touch. Pressure on these, either from the back or from the abdomen, produced pain. The reflex irritability was diminished on both sides, especially on the right. The patellar reflexes were completely absent on the right side, and diminished on the left. Tickling the soles of the feet had to be repeated frequently before a reflex could be obtained. Dorsal clonus was indistinct and more pronounced on the left than on the right. *Taches cérébrales* were found only on the thighs.

The pupils were dilated without giving a distinct reflex in the rather dim daylight. The respiration was irregular, sometimes deep, sometimes shallow, and interrupted by pauses of ten seconds each. The pulse was 63 per minute, irregular in both strength and rhythm. The abdomen was uniformly soft, retracted, and allowed the bodies of the vertebræ to be felt distinctly. There was nowhere a trace of edema.

The child complained of severe headaches and vomited repeatedly after retching. There had been no stool since the afternoon of the fourteenth. The retention of urine continued. In the evening a catheter was introduced, but only a few drops of clear urine was obtained. On this occasion it was noted that the entrance to the vagina, especially the mouth of the urethra, was markedly reddened and very painful.

On the sixteenth she had a movement of the bowels with which a little urine was mixed. The stupor increased, the vomiting and the irregularity of the pulse and respiration continued; the temperature did not reach 36° C. (96.8° F.). In the evening it was only 35.2° C. (95.3° F.). Death occurred at 4 o'clock in the morning on February 17th without further manifestations.

The autopsy (von Baumgarten) showed: "A retropharyngeal abscess on the right lateral wall of the pharynx, beginning near the posterior wall, but not quite reaching to the median line, and corresponding to the levels of the second and third cervical vertebræ. The left tonsil was normal; the right was somewhat enlarged, but without any abscess formation.

"The left kidney was markedly enlarged and its capsule easily stripped off. Its surface showed marked injection of the stellulæ of Verheyn, in addition to small effusions of blood here and there. On section, the kidneys showed a great contrast between the dark-red pyramids and the grayish-yellow cortex, in which there were a number of minute points of intensely yellow color less than a pinhead in size in addition to a diffuse cloudy swelling. These had their chief seat in the region of the labyrinths. There were also seen on section several small hemorrhages in the cortex. The cortex was distinctly swollen and infiltrated with edema. The same appearances were seen in the right kidney.

"Microscopically the following appearances were found (preparations stained with picrocarmin): The epithelia of the convoluted tubules were very markedly swollen, cloudy, and in some of them necrosis had already appeared. In addition to the inflammatory edema, there were also numerous cellular infiltrations in the interstitial tissue. A large number of casts were found in the tubules, showing that there had been albuminuria. Another feature was a very marked thickening of Bowman's capsules by concentric layers of spindle cells and a fairly well-marked swelling of the epithelium of the glomeruli. There was, therefore, an advanced parenchymatous and interstitial nephritis, with considerable involvement of the glomeruli."

Other features found were anemia of the brain and of the spinal cord, edema of the brain, ecchymoses on the outer surface of the dura which actually formed a path along the longitudinal sinus. The heart muscle was strikingly pale and flabby in the right ventricle, which was somewhat dilated. The valves were normal.

The bronchial glands were almost totally filled with old caseous foci. The lungs were edematous, but showed no foci. There were hemorrhages in the somewhat large spleen, in the small intestine, and more prominently in the colon. In the latter there were also two abscesses, a little larger than peas, near the ileocecal valve. Microscopically, numerous chains of cocci were found in the fresh pus from the retropharyngeal abscess. Cultures proved that these, as well as the bacteria obtained from the erysipelatous skin and the blood from the kidney, were true pus cocci, corresponding to the type of Streptococcus pyogenes.

The clinical picture in this case was not properly interpreted. I had thought of tuberculous meningitis at the base, combined with erysipelas, but could not make a definite diagnosis. The retropharyngeal abscess did not manifest its presence on account of its position. The fact that a severe nephritis was probably present was recognized, but no decision was reached in the absence of an examination of the urine, which could not be made, inasmuch as no urine was obtainable. The signs of involvment of the central organs may be attributed without difficulty to uremia, or else may be simply interpreted as effects of septic infection, which at that time were but imperfectly known to me. (See below, p. 259.)

The urine in these cases resembles, in general, that of acute nephritis arising from other causes; blood, albumin, sometimes organized elements, such as casts and epithelia from the tubules, etc., are present. Even the course of the nephritis is similar, so far as it may be determined from examining the urine. At first there is a diminished amount with increased specific gravity. Earlier or later, according to the severity of the infection, but in all cases at some stage, the amount gradually increases after one or more weeks, while the specific gravity again diminishes. The blood disappears earlier than the albumin and the casts.

At what stage of the disease does nephritis supervene? This does not seem to be subject to a definite rule, but seems to depend upon the severity of the infection or, more correctly, perhaps, upon the amount and character of the substances which pass through the kidneys. In case XI the signs of nephritis had fully developed as early as the third day of the disease, and were found on urinary analysis. Beginning with the twenty-seventh day, however, nothing was to be found in the urine, which was examined for a period of seven weeks afterward.

An even course ending in recovery is most frequently seen. In such cases the kidneys heal completely, or are left with such slight disturbances as do not essentially affect their function (example, case II). No definite statement can be made as to the frequency of these mild cases, for the number of observations is not sufficient, but it seems to me that the frequency of nephritis varies at different times. Further experience is necessary to establish this.

I should like to note, however, that a nephritis which has by no means passed away may become worse, even after the renal affection had reached its acme. This was the case in the following instance:

Case XVI.—A girl, nineteen years old. Admitted February 12, 1893. In December, 1891, she had had an attack of migrating erysipelas, to which was added an ulcerating stomatitis, and later a nephritis. She was treated in the medical clinic for almost seven weeks. She did not seem to be quite as well as formerly after that time. On February 5, 1893, she suffered from general malaise immediately preceding her menses, which were accompanied by severe pains in the abdomen. There were, in addition, severe pains in the limbs, which impelled the patient to ask admission to the hospital.

On admission, she showed an extensive and exquisite sensitiveness to pressure in the bones of the upper, as well as in those of the lower, limbs, the ribs, and the cervical vertebræ. In the latter she also suffered spontaneous pain. There was a slight swelling in the ankles. The muscles of both extremities and over the cervical vertebræ, as well as the skin over the chest and the entire abdomen, were very painful to pressure.

The sensorium was free. There were clonic contractions of the whole body every two or three minutes. The area of cardiac dulness extended from the fourth rib above, and was limited on the right side by the median line of the sternum. The apex-beat was in the fifth intercostal space at the mammary line. The heart's action was excited; the tones very faint, but clear. The urine was reddish-black in color, contained about 0.2 per cent. of albumin, numerous red blood-cells, epithelia, and casts. The temperature was 40.4° C. (104.7° F.); the pulse, 100; the respiration, 30.

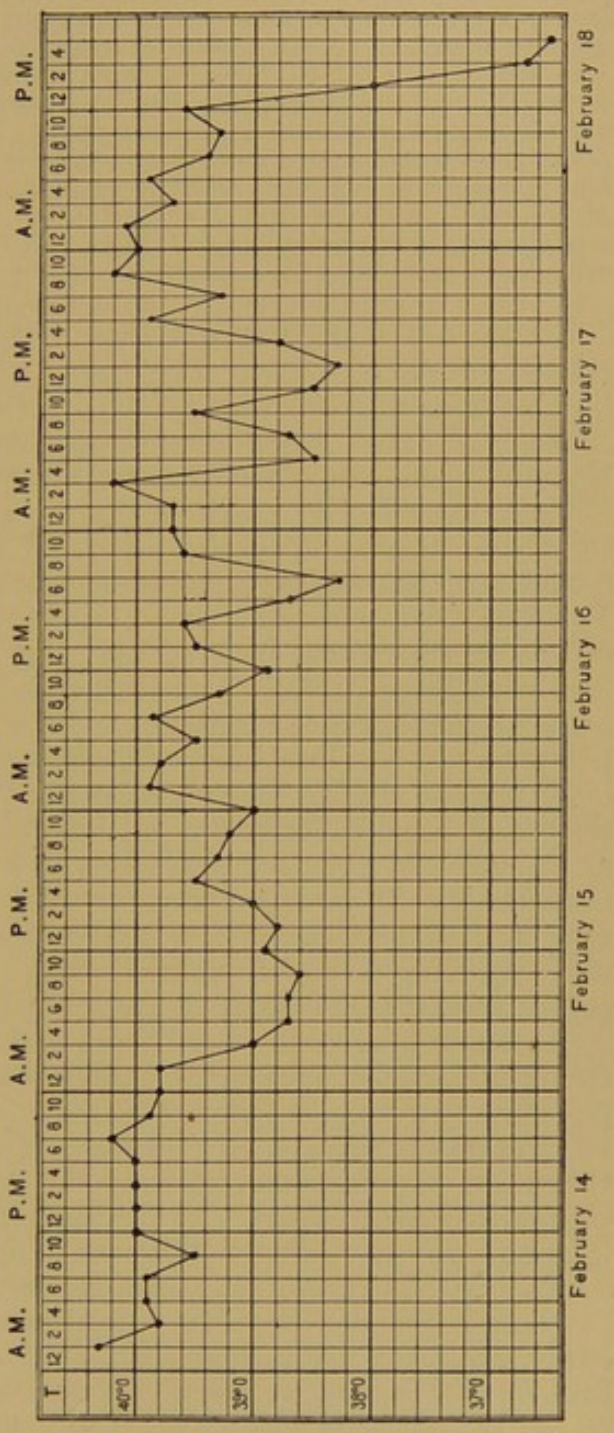


Fig. 51.

Fig. 51 shows the temperature during the first few days, the measurements being taken every two hours. This chart demonstrates how irregular the distribution of the temperature was over the twenty-four hours, and how the temperature suddenly fell to subnormal on February 18th. This subnormal temperature, however, did not last long. Four hours later (at 8 o'clock in the evening) the fever had again risen to 39.5° C. (103.1° F.). From that time on there was, on the whole, a fall of temperature, although we still find 40.2° C.° (104.3° F.) on February 25th. Between February 28th and March 5th the fever no longer

reached 39° C. (102.2° F.). It remained low in spite of the increased intensity of the cardiac symptoms; on February 26th there was an attack of oppression, a feeling of fear, and a fine pericardial friction sound rapidly extended over the cardiac area. The urine also showed some improvement during that time.

The patient was tempted to get out of bed for a time on account of her apparent improvement. She claimed to have gotten up on March 7th, but probably it was the day before. At all events, on the eighth she was found out of bed by her sister, who had returned home unexpectedly.

Her indiscretion did not remain unpunished: The pains in the bones and joints increased; the urine showed a considerably increased amount of blood and a larger number of casts. Its quantity sank to below 500 c.c. (minimum, 410). The pulse-rate increased to 132 (the maximum during the two first weeks of observation had been 112), and the temperature rose to 40.8° C. (105.4° F.). It was only beginning with March 19th that less than 38° C. (100.4° F.) was recorded. The exacerbation of nephritis was of shorter duration. As early as the fourteenth the amount of urine had increased to 1270 c.c., and the amount of blood and organized sediment had grown less. At the beginning of April, when the girl considered herself cured and was discharged from the hospital, there was still some albumin and the amount of urine had increased to over two liters.

There are still other diseases of the kidneys which are intimately related to endocarditis. The forms of nephritis which have hitherto been discussed may all be referred to the general effects of the microbes of sepsis. These forms are probably essentially the result of infection by substances which circulate in the blood and are eliminated by the kidneys—substances which are not organized, but are poisons derived from the metabolism of the infecting microbes. We must distinguish from these the forms in which there are demonstrable foci in the kidneys. The obvious conclusion is that in these forms the disease is not the result of soluble substances in the blood, but of bodies carried to the kidneys and localized in them. The further course of these affections depends, as in all embolic processes, upon the size of the mass of foreign bodies present and upon the ability of these to produce tissue changes.

From the clinical viewpoint, it must be said that these intruders, on the whole, produce far less severe manifestations than do absorbed poisons, and that the affections of the kidney to which they give rise are even often overlooked. There is a reason for this, namely: the fact that they enter only limited portions of the kidney. Even when these are totally destroyed, there always remains enough unaffected tissue in the gland to continue its function without great impairment.

In such fulminant cases as case I, it is hardly possible to suspect bacterial emboli in the kidneys, still less to recognize them. (See autopsy, p. 196. In this case there were streptococci.) A small amount of albumin and a few red blood-cells may appear in the urine and may be found therein, if any urine can be secured. Yet we cannot conclude anything definite from this alone. As the patient in case I passed urine and feces involuntarily, we were not tempted to render a decision.

In case XII the autopsy-findings in the kidneys were as follows:

"Both kidneys are rather smaller than normal. The capsules are easily stripped. On the surface there are numerous depressed, yellowish-brown patches, which on section prove to be wedge-shaped, dense, yellow or yellowish-brown foci which stand out from the surrounding regions but are not surrounded by an appreciable zone of reaction. Their size varies from that of a cherry-pit to that of a cherry." The bacteriologic examination in this case showed that the staphylococci, which were abundantly present in the endocardial foci, also occurred in the renal infarcts, although in these they were found in sparse groups. In this

case no changes could be shown in the kidneys during life. Although there had been frequent edemas, especially of the eyelids, which kept up the suspicion of a renal affection, no albumin was found on repeated examinations. The opposite was true in the following instance:

Case XVII.—A woman, aged twenty-seven years. First admitted August 6, 1889. Death January 30, 1890. She had had pains in the limbs since 1877, with frequent repetitions. In 1878 changes in the heart were found at the Policlinic, viz.—mitral insufficiency with slight stenosis. Later on there were again pains in the limbs and joints, and the heart continued to trouble the patient. (Statement of the patient, who lived out of town after she was married.)

On admission she complained of palpitation of the heart. There was no noteworthy increase in the area of dulness. There was a loud systolic murmur over the apex. The diastolic pulmonary tone did not seem to be accentuated. The joints were swollen and there was pain in the abdomen; nausea after eating, and continued moderate fever.

Toward the middle of October the symptoms increased in severity. There was a septic eruption, and extremely severe pains in the right arm, the thigh, and the leg, in the diaphyses as well as in the epiphyses, and the joints were slightly swollen. The heart was also more markedly affected at that time. I shall quote the facts which interest us here, from the clinical history:

On November 8th, at 10 o'clock in the morning, the patient was suddenly seized with the most excruciating pain in the abdomen. She was very much excited, very restless in bed, and could not bear to remain in any one position. Soon afterward she vomited three times, the stomach contents colored red by wine. Then followed signs of collapse. The hands, feet, nose, and ears grew cold; the skin was covered with cold perspiration, the pulse was small, easily compressed, difficult to count, numbering about 120 to 130 beats; the respiration rapid and shallow.

The pains were localized especially in the ileocecal region and in the adjoining portions of the abdomen, but they radiated from there toward the back. The pains diminished after an injection of morphin (0.02 gm.), and the patient fell asleep until evening. On November 9th there was great lassitude, but no further attacks of pain. Every change of posture, however, was avoided on account of the fear of pain. The abdomen was slightly swollen, and on palpation the ileocecal region was especially sensitive, although there was also pain on pressure on the right side toward the back. The pains in various other places diminished from that time on to the twelfth of November, but the region of the right kidney was painful on pressure. The patient had the first movement of the bowels on November 14th. She had been treated with opium.

On October 18th repeated examinations had not shown albumin in the urine. On November 9th albumin was found, and was also present at later examinations. Regular examinations of the urine were not made after that time, as this happened during an epidemic of influenza.

On November 16th there was marked strangury. Urine was passed in copious amounts only after warm poultices had been applied. A specimen examined contained a sediment of urates, in addition to moderate amounts of albumin, but nothing else.

The condition grew worse during the following weeks, although no special complications arose. On January 3, 1890, the patient again felt sudden severe pains in the abdomen, as during the first attack. The abdomen was swollen and sensitive to pressure, this time especially on the left side, although the right side was not perfectly free. The attack was shorter in duration than the first.

As regards the urine, it was noticed again on January 26th that it contained moderate amounts of albumin. No blood was ever found. During the last period of life the emaciation increased more and more, and toward the end there were abundant hemorrhages into the skin. There was always fever whenever the temperature was taken. During the last weeks this was not done, on account of the epidemic of influenza which had attacked all the nurses. The fever was moderate and its maximum was 39.7° C. (103.4° F.).

The autopsy (von Baumgarten) showed, in the first place, a severe old lesion of the mitral valve, with a diffuse fresh endocarditis. I give the findings of the heart in full: "On opening the left auricle, this cavity was somewhat dilated, the epicardium clouded all over, and the auricular appendage empty. On opening the mitral valve there appeared a diffuse fibrous thickening of the flaps. Their free borders were thickened. In addition, the aortic flap of the mitral was adherent to

the posterior flap. At the line of closure of the valve there were a large number of yellowish nodular excrescences, partly coalescent, firmly adherent to the valve tissue, and organically connected with it.

"The auricular wall of the posterior flap was covered with flat, cauliflower vegetations which extended upward over the line of closure. The adjacent endocardium was covered with fibrin-like deposits of pin-head size, or larger, and of a grayish-red color. These deposits, however, could be detached from their underlying tissues without visible loss of substance. Larger masses of vegetations arose also from the inner wall of the auricle. The papillary muscles were moderately hypertrophic; the tendinous cords were markedly thickened and shortened. Hemorrhages in the endocardium and in the muscle were not so prominent on the left side as on the right. They showed in many instances small whitish spots or striations at their centers, more clearly on the left side than on the right. Longitudinal sections through the muscle also showed numerous large and small hemorrhages, without visible white centers."

The enlarged spleen contained numerous yellow or yellowish-red infarcts, some of which were dry, while others were transformed into a brownish-yellow mass of pus. The following were the findings in the kidneys:

"The left kidney was quite large and weighed 230 gm. Its capsule was easily stripped; its surface smooth. The latter showed at various points yellow spots of various size, with square or rhombic outlines, which on section were found to be wedges penetrating into the depth of the kidney. The foci were whitish-yellow throughout, and, as in the spleen, were without visible zones of demarcation.

"Miliary and submiliary hemorrhages without whitish or yellowish centers were also found in the cortex. The trunk of the renal artery was open.

"The right kidney was scarcely one-quarter as large as normal, and weighed only 70 gm. It showed distinct embryonal lobulation and was, for the most part, transformed into a yellow necrotic substance. The renal artery, which appeared large in proportion to the organ as a whole, was completely plugged by some grayish-red tissue. On transverse section the obliterated vessel had a flattened shape.

"The bacteriologic examination showed the presence of Staphylococcus albus. This was the only germ found, and it had cultural peculiarities which corresponded perfectly with the tissue changes that appeared on microscopic examination.

"Streak cultures from the endocardial vegetations grown upon agar plates were transferred to gelatin, and it was found that the staphylococcus possessed a slight degree of peptonization which resulted in a very slow melting of the nutrient gelatin.

"The markedly thickened valvular edges showed an extensive necrosis, which involved the adjoining portions of a tendinous cord that happened to be included in the section. This necrosis was manifested by a total absence of nuclear staining. Large masses of staphylococci were found upon these necrotic areas, which stained brownish-red with picrocarmin, and a beautiful blue with Gram-Günther's stain. Fresh inflammatory reactive phenomena on the part of the chronically inflamed and indurated valvular tissue were completely absent.

"Even the infarcts of the kidney and spleen showed only a slight cellular infiltration, a slight chronic productive inflammation, and no trace of a demarcating suppuration at the boundary of their necrotic areas, which were characterized by the absence of nuclear staining. A somewhat more marked small-cell infiltration could be demonstrated in the immediate neighborhood of the larger arteries of the infarcted regions, which were filled for considerable distances with enormous masses of staphylococci. The biologic phenomenon, the slight peptonizing property in gelatin cultures which the staphylococcus manifested, was, therefore, completely in correspondence with the slight pathogenic irritant action of the emboli of this coccus, which were found present on microscopic examination."

In this case the acute symptoms which developed suddenly on November 8, 1889, and on January 3, 1890, were in all probability referable to emboli in the kidneys, the first of which occurred in the right organ, the second in the left. Both attacks were accompanied by the signs of a visceral neuralgia, together with marked irritation of the peritoneum. It is true that during the first attack the sensitiveness on pressure was chiefly localized in the ileocecal region, and was only clearly present in the right kidney later on. But then it was more marked there than at any other point. The possibility that there may have also been a coprostasis cannot be denied. Yet it is very rare in coprostasis to find such

sudden attacks of pain of such exquisite character and accompanied with such marked symptoms of collapse.

The fact that so extensive an embolus as was found in the kidney developed irritative effects upon the peritoneum of this region is probably dependent, to a certain extent, upon the anatomic relations between the right kidney and the peritoneum (the right kidney is covered by peritoneum at its upper two-thirds), as well as upon the contact of the kidney with the right flexure of the colon. The peculiar property of the staphylococcus which was the infecting agent in our case, and which did not possess the quality of producing severe inflammatory changes, is probably responsible for the fact that a suppuration of the kidney did not occur, but that only a dry necrosis was produced. This interpretation is supported by the events during the second attack. The same symptoms, but less severe in character, occurred, and the tissue changes in the left kidney were less marked.

On account of the insufficiently accurate observations which were made in this case, for reasons that have been cited, it was uncertain whether albumin first appeared one day after the attack of November 8th. It is noteworthy, at all events, that edemas were absent to the last, in spite of the extensive diminution of the kidney tissues, and in spite of the fact that the cardiac activity was certainly not at its normal level.

So far as the temperature is concerned, we cannot draw any conclusions as to the time when the kidney emboli developed.

In addition to these facts, gleaned from my own observations, I shall cite here some data given by some prominent investigators, who have thoroughly studied the subject.

Litten, in his large work on septic diseases, accurately describes the changes which occur in the kidneys.

It must be clearly understood that his material consisted of cases of acute septic infection, the majority of which had developed in the puerperium. Among his 35 cases there were 23 women, some of whom had had abortions, while the others had labors at term. Only in 2 cases was the cause of the disease unknown. Litten found endocarditis in 22 autopsies. Only in 5 cases were the remains of an older endocarditis present. This also characterizes his material as unique.

Litten has with perfect clearness described the essential changes which sepsis produces in the kidney. Of these, I shall note as especially interesting: (1) All the changes that go on in the kidneys are the same whether an acute endocarditis accompanies the septic infection or not. Metastatic abscesses in the kidney occur especially also in the latter class of cases. Thus, for example, in cases VIII and XIII of Litten's table large metastatic abscesses were noted. Bacteria may also be swept into the kidneys and be found therein without producing any changes in the surrounding tissues. "It is often astonishing to find on microscopic examination that large capillary areas, especially in the glomeruli, are packed with bacteria, while there is not a trace of necrosis or inflammation in the tissues involved."*

(2) "Interstitial sclerotic processes are found very early in addition to the diffuse small-cell infiltration."

Weichselbaum also gives accurate data concerning the anatomic and bacteriologic features of the kidneys (in endocarditis). He found parenchymatous nephritis in six cases out of twenty-nine fresh suppura-

* *Loc. cit.*, p. 452.

tive and non-suppurative infarcts, as well as old ones, and in one instance also purulent cystitis and pyelitis. On the whole, his findings were the same as those of Litten in that form of endocarditis which was not characterized by a very acute invasion.

Weichselbaum records no less than 18 cases of recurrent endocarditis, 11 of which were anatomically characterized as "ulcerative." But the kidneys were involved also in the 7 cases of verrucous recurrent endocarditis. Possibly it was not an accident that parenchymatous nephritis was present in 4 cases. Suppurative foci or bacterial emboli were absent in all cases, with the exception of that which was accompanied with cystitis and pyelitis.

The following case reported by Ebstein* deserves consideration: A man, aged forty-two years, showed symptoms of a hemorrhagic nephritis during the thirteenth week of his disease. These symptoms increased within the weeks following. Four days before death it was still found that "the urine showed the typical changes of a hemorrhagic nephritis." About fourteen days previously there had been a condition which pointed to uremic intoxication.

At the autopsy there was no sign of a nephritis. The changes which were observed in the urine in this patient must have been due to a hemorrhagic diathesis which became obvious shortly before death. Hemorrhage in the kidneys could not be proved to exist with certainty on macroscopic examination in this case. The author does not mention whether such hemorrhages were present in the renal pelvis, but as they are not spoken of, it may be assumed that they were absent. No bacteria were found elsewhere, not even in the heart. The case was one of endocarditis of the tricuspid with the formation of an aneurysm and rupture of the latter. The clinical course was characterized by chills which occurred almost daily, and by elevations of temperature which were sudden and sometimes very marked. This case is probably unique, and, in my opinion, is still somewhat obscure.

Romberg† points out that malignant endocarditis may be accompanied from the first by the symptoms of a subacute hemorrhagic nephritis, and may continue thus until death. "The disease begins like an acute nephritis, with edemas, uremic symptoms, scanty urine, albuminuria, and at first, usually, a large amount of blood in the urine, which later grows smaller, together with large numbers of desquamated epithelia and numerous casts. The character of the urine and the edemas may improve for a time, but they usually continue with little change. The uremia occurs only temporarily, and is rarely very marked.

"The disease is distinguished from an ordinary nephritis from the very start by the fact that there is fever almost uninterruptedly during the entire course of the case, the temperature usually being moderate throughout; by the very striking softness of the pulse, and by the anemia which is unusually marked even for a hemorrhagic nephritis. In two of my cases a palpable tumor of the spleen led to a correct diagnosis. When an old valvular lesion is also present, it may lead us to think of a malignant endocarditis, because a hemorrhagic nephritis is not usually observed in valvular heart disease and because diseased valves are known to be predisposed to malignant endocarditis."

* "Beiträge zur klinischen Geschichte der Endocarditis ulcerosa maligna," "Deutsches Archiv für klinische Medicin," 1899, Bd. lxiii, S. 217, case B. III.

† *Loc. cit.*, p. 981.

These signs are certainly not very positive. When there is nothing else which points to sepsis, we are almost sure to make a mistake as regards the etiologic diagnosis.

The changes in the circulation which are produced by the heart weakness naturally also affect the kidneys. We have already considered in its proper place the effects of insufficient cardiac activity as such.* It is impossible to say, at present, what disturbances are due to sepsis and what changes are the result of cardiac weakness, when both these factors are present together.

Amyloid degeneration of the kidney has already been spoken of in connection with case XIII, in which there had been a prolonged general loss of nutrition resulting from frequent pregnancies; abundant hemorrhages during the labors, and frequently recurring endocarditis. Von Baumgarten† is inclined to consider the bacteria of sepsis, through the medium of their metabolic products, as the causes of amyloid degeneration. In the few cases—six in all—in which amyloid degeneration appeared with endocarditis, we must admit that no other cause for the degenerative process could be found. I must leave it undecided here, how much reliance is to be placed upon this interpretation. If the view which von Baumgarten represents is correct, that chronic anemia is not capable of inducing amyloid degeneration, then we must again fall back upon sepsis.

The pelvis of the kidney is very frequently the seat of more or less extensive hemorrhages, especially in the severe cases with rapid course. But these hemorrhages have no direct connection with the endocarditis. They are the result of sepsis.

NERVOUS SYSTEM AND MUSCLES.

I have grouped these two classes of structure together, because it is not always possible to make a sharp distinction between them when we seek to prove the origin of certain phenomena in the patient.

The brain symptoms include, in the first place, all those which may be attributed to the general infection. Unquestionably, these include irritations, and paralyses, delirium, loss of consciousness, etc. It is not of any importance, in this connection, whether or not the endocardium is also involved. Very often there is a relation between the cerebral symptoms and the temperature, but this cannot always be demonstrated. In studying these relations we must, as usual, consider both the absolute height of and the rapid variations of the temperature.

The patient in case IX showed a remarkable dulness of the sensorium, a little later delirium, and several days later severe headache, but all these signs appeared without any appreciable variations in temperature. It is true that at that time there had already been a slight albuminuria, but there had been no sign of a true renal affection. The same was true of case XI. Such transient changes are frequently seen in the clinical histories, and may be considered as quite common occurrences. No relation between the cerebral symptoms and the temperature-curve can be noted in these cases.

* See my treatise on "Cardiac Insufficiency" in the present work, p. 129.

† "Ueber Amyloidentartung nach recurrirender Endocarditis," von Hugo Lippert, Tübingen; dissertation (presided over by v. Baumgarten), 1895. Also: "Arbeiten aus dem Pathologisch-anatomischen Institute zu Tübingen," Bd. ii, 3, Brunswick, Harold Bruhn, 1898.

The same is true of the more markedly developed cerebral symptoms. Even when such severe symptoms as occur in a true meningitis appear in these cases of endocarditis, they may again subside, and the patient may regain his full consciousness. If, however, death should occur in the further course of the disease, there is often nothing found, at least with the naked eye, except edema.

A different condition of affairs obtains, naturally, when the pathogenic germs which are responsible for the disease have settled in the brain. We will not consider the diffused surface suppurations—meningo-encephalitis. These can arise undoubtedly as the result of a general infection with pneumococci, which may also produce an endocarditis. Meningo-encephalitis may, however, be the result of infection with ordinary pus-cocci, in the narrower sense of the word, although this is less frequent.

I shall not dwell upon these infections, but shall speak now of the cerebral affections which are immediately related to endocarditis—that is, of those produced by the invasion from the heart of thrombi which contain the bacteria.

Case XII furnishes an example. The patient became unconscious and was found by her bed soon after she had left it. The unconsciousness continued only for about an hour. The muscles of the face which are supplied by the lower branches of the facial on the left side, the left arm, and the left leg were paralyzed. The cutaneous reflexes were increased in the leg. The sensibility was unchanged. There were no disturbances in the speech, nor could any influence upon the temperature be noted. The motor power was restored to the leg, first, as usually is the case, and later on, in a less marked degree, to the arm also. The patient improved somewhat more slowly than usual, but still the paralysis gradually disappeared. On November 7th she still felt quite well subjectively, but in the night from the seventh to the eighth the patient began to "babble nonsense," and tried to leave her bed several times without any reason; she grew more quiet toward 6 o'clock, although she did not respond when she was called, nor did she understand what was said to her. She passed both urine and feces involuntarily. The attending physician found her somewhat somnolent, but she could be aroused from her condition. The same state was noted at 10 o'clock. "The patient reacts but slightly when called, but opens her eyes and stretches her right hand toward the physician. There is a slight ptosis on the left side. When she was asked to look at a finger, she followed it with her eyes, but the right eyeball lagged behind the left. The pupils were small and reacted but feebly to light. The plantar reflexes on both sides were markedly exaggerated. The patellar reflexes were lowered almost to absence; there was no ankle-clonus. There was marked hyperesthesia of the skin, especially in the lower extremities. The patient complained of acute pain when a fold of skin was raised."

At 12 o'clock "the patient is completely unconscious, and reacts neither to the voice nor to pinching. The pupils are very small, the left is somewhat dilated, and reacts a little better. Nystagmus." In the further course there developed complete paralysis on the right side, which began on November 10th and involved both extremities, as well as the muscles of the face supplied by the lower branches of the facial. The coma was so deep that the woman did not even notice the birth of a seven-month's-old fetus. Contractions in the arm and in the leg on the left side, Cheyne-Stokes' breathing, and hiccup from that time on until death. The reflex irritability varied on the left side, but on the right it was almost permanently absent. Trousseau's phenomenon was continuously present. The report of the autopsy has already been given (see p. 224), and the course of the temperature has also been noted.

In this case the first embolus was probably comparatively benign in character, for no inflammatory changes were found, but only softening in the internal capsule and in the lenticular nucleus. The second embolus, however, was softened by suppuration and contained numerous staphylococci. An extension of it, however, did not take place to the meninges. At all events the contrast is noteworthy: As the result of the first em-

bolus, the very short general effect on the brain; the disappearance of unconsciousness after a few hours, and the clearly developed paralysis which appeared at once. As a result of the second embolus, occurring seven weeks later, the cerebral symptoms increased more and more, the paralysis appeared, but became distinct only two days later.

A sudden rise of temperature did not take place immediately after the lodging of the infected embolus. Monakow* says, in speaking on this subject: "When septic emboli occur, we naturally expect a rapid rise of temperature as well as a chill." This is not always so, however, for in a second case of cerebral embolus with severe endocarditis observed in the Policlinic, no rise of temperature was noted at first, but two days after the onset of the attack there was a perceptible rise. In both cases there was a postmortem rise which reached 42.4° C. (108.3° F.) in the second case, and continued for about fifteen minutes. In this patient the state of excitement which preceded the paralysis was still more marked and continued for a longer time.

We know less concerning the condition of the spinal cord in endocarditis. Pribram† has gathered carefully everything recorded in literature concerning "spinal rheumatism," including all that belongs to the subject now under consideration. I cite the following history which bears on the subject:

Case XVIII.—First admission of the patient, who was at that time a man aged thirty years, on November 29, 1894. No other diseases were noted in the previous history. For the last twelve years he had been suffering almost every spring from a stiffness of the joints of the fingers and hands, which for a time appeared swollen and covered with small red spots. At that time he had to stand in water a long time while digging trenches. The attacks came and went without disturbing the ability of the patient to any marked extent.

A few days before admission the "stiffness" for the first time appeared in the knee-joints. The man was unable to work any longer. In addition, he had been constipated for five days, according to his own statement, and had had frequent desire to urinate, but could not always satisfy it. He had a copious stool after a dose of castor oil. Objectively, there was, at first, only a slight resistance to flexion and extension, and pain on motion in both knee-joints. Passive motion was accompanied by unmistakable resistance. The heart and lungs showed nothing abnormal. The temperature was subnormal, and did not reach 37° C. (98.6° F.).

On December 5th there was stiffness and pain in both ankles. The patient walked about quite well in the room, although in a somewhat strained position. The patellar reflex was marked, even on slight irritation. He could stand perfectly well balanced with his eyes closed.

On December 7th the clinical history says: "The patient claims he cannot walk any longer. When he is told to walk his gait shows marked spastic characters: The toes are not lifted from the floor, but glide over it. When his feet are not quite close together, he can stand fairly firmly with his eyes closed." When the patient was put to bed once more after this test he showed for a few minutes contractions of the muscles of both thighs, which were varied in intensity. The patellar and the plantar reflexes were markedly exaggerated. There was hyperesthesia of the skin from the umbilicus downward. The pinching up of a fold of the skin was characterized as painful. There was slight contracture of the muscles of the thigh. When a cold hand was laid upon the thigh, there were contractions in the muscles which increased gradually and which spread also to the opposite limb. The same was seen when the reflexes were tested. The lumbar spinal column was quite sensitive to pressure.

I shall briefly summarize the chief features of the very carefully noted course of the disease. The variation in all the symptoms which occurred from day to day are especially noteworthy. On testing the sensibility for the first time (on December

* "Gehirnpathologie." Nothnagel's "Specielle Pathologie und Therapie," Bd. ix, 1 Theil, S. 832; also table on p. 864.

† See Nothnagel's "Specielle Pathologie und Therapie," Bd. v, 1.

13th) it was found that no disturbances could be demonstrated above a line which was drawn about 5 cm. above the umbilicus. Below this line the following changes were found:

Sense of Touch.—Uncertainty on the part of the patient as to whether he was touched by means of a cotton wad or with the sharp or the dull end of a needle. He is conscious of the wad of cotton only when the contact is more forcible, and his impression comes later than normal.

Sense of Location.—He knows quickly and accurately where the point of a pencil touches his skin, but less accurately when he is touched with the point of a needle. Below the umbilicus he takes a distance of 5 mm. between points of calipers for a single point. At the lower part of the abdomen the distance between the points may be made still wider without changing his impression of a single point. He recognizes as double distances of 110 mm. on the left side, and 150 mm. on the right side, on the skin of the lower extremities. The sense of pressure seems to be diminished on the left lower extremity as compared with the right.

The Temperature Sense.—Heat is usually correctly recognized on testing with tubes filled with water of different temperatures. Cold, however, is more frequently designated as heat.

There was no special abnormality as regards recognition of pain on pricking with needles. On the other hand, the weakest faradic current was able to produce a strong sensation of pain.

The surface temperature at both sides was almost the same. On the right side it was 33.8° C. (92.8° F.); on the left, 34° C. (93.2° F.).

Motor Tests.—The muscular strength of both lower extremities was considerably diminished, and the left leg, measured at different points, was slightly thinner than the right to the extent of from 0.5 to 1 cm.

Active motion: The extended legs could be lifted but slightly from the bed by the patient, but the knee- and hip-joints could be easily flexed. With eyes either shut or open the patient was able to describe a fairly good circle in the air with his left leg. He could not do this with the right. He was able to touch correctly with the heel of the opposite foot any accessible spot which had been previously touched, on either side, with his eyes shut. There were slight and transient contractions of the muscles of the legs whenever these tests were executed.

Passive motion: Flexion was considerably hindered in both ankles; extension was not interfered with. The opposite held good in the knees, flexion being easy and extension difficult.

Abduction was more difficult than adduction. The reflexes: The patellar reflex on both sides was considerably exaggerated. The reflex of the tendo Achillis could not be obtained; the plantar reflex was distinct; the cremasteric reflex absent. There was no abnormal reaction to the electric tests. Defecation and urination were somewhat difficult. There was diminution of sexual desire.

The most prominent sign was always the contraction of the muscles of the thighs, which at times was so marked that it sufficed to raise the bed-clothes in order to bring on contractions which started in the tense muscles of the thighs and involved the muscles of the back, making the patient jump up and down. Even when these spasms were less marked—only fibrillary contractions—it was possible to evolve actual movements of the thighs by contact with a cold object. When the contractions were strongest, there was also increased sensitiveness of the spinal column and hyperesthesia of the skin.

There was a general improvement in the disturbances in the nervous system, beginning with December 22d, and lasting into the first week in January. Then followed three weeks of decline, which ended in the middle of February. Thus the condition fluctuated until the middle of May, when progressive improvement began and lasted until the patient was discharged—in the middle of July.

At first I did not begin to think that possibly an infection, especially a septic one, was present. Everything pointed to a beginning disease of the spinal cord, probably involving also the spinal meninges. The only remarkable feature was the peculiar distribution of the temperature and the relation between the pulse and the respiration. For this reason careful records were kept of these data, the results of which I have given above (see p. 234).

The fact that the heart was not perfectly normal was first noticed in January (on the twentieth), when the pulse became rapid (96) and some-

what irregular with the increase of the nervous symptoms. On February 3d the boundaries of the heart were somewhat enlarged, and the heart-sounds not perfectly clear; but these signs disappeared again on the fourteenth. Only a slight irritability of the pulse remained.

In the middle of May pretty severe general symptoms—sleeplessness and peevishness—came on for the first time, together with quite an extensive multiform erythema, swellings of the joints, and sensitiveness on pressure over the long bones. The heart grew more restless again. These symptoms continued only for a few days, but were sufficient to attract our attention.

When the patient was discharged, these and the other symptoms on the part of the nervous system had not quite disappeared; there were occasionally swellings of the joints, and unimportant eruptions on the skin of the hands—both of short duration.

The second admission took place on February 15, 1899. Without having been seriously ill, the man could no longer pursue his work to the full extent. Walking for any length of time had become difficult, and on severe exertion he often felt pains in the loins and in the legs, as well as a furry sensation in the soles of the feet. He was always constipated, and passed frequent and small amounts of urine. He stated that *potestas coeundi* had long ago disappeared, and that erections no longer occurred. There had been red spots, swelling, and pain in the hands three or four times a year, especially during the cold weather.

The present illness began on February 8th. At first there were red spots the size of a finger-tip on the dorsum of the left hand, together with swelling, redness, and pain in the second and third joints, afterward in the entire left hand, which could be moved only with great pain. On the twelfth the same symptoms appeared over the left ulna, and on the thirteenth over the right thumb-joint.

On admission, there was an extensive eruption, for the most part a multiform erythema, with a slight urticaria. Wherever erythema was present, especially over the joints of the fingers, the patient complained of severe pain on pressure and on motion. There was sensitiveness on pressure over both femurs, and to a less degree over the spinal column. There was a belt sensation at the level of the sixth thoracic vertebra, which, however, was complained of only when the patient stood up. There were slight fibrillary twitchings of the muscles of both thighs, which increased very markedly when the skin was touched with a cold object.

Trousseau's phenomenon was marked in the arms, the thighs, and over the chest and abdomen. The plantar reflex was normal on both sides. The cremasteric reflex was extremely weak. The patellar reflex was increased on both sides—sometimes more, sometimes less markedly. The patient showed a spastic gait, which developed much more distinctly after he had taken a few steps. Romberg's sign was not distinct, yet the patient swayed more on standing with closed eyes than with his eyes open. The circumference of the thigh on the left side was still from 0.5 to 1 cm. less than on the right. The absolute measurements, however, taken on the thirteenth of December, 1894, were from 1.5 to 2 cm. greater. There was constipation and frequent desire to urinate, but comparatively small amounts of urine were passed each time.

I shall further summarize briefly the observations of this case. The eruption continued to the end of February, with frequent new outbreaks, usually in the form of polymorphous erythema, but at times in the shape of urticaria, both usually being quite extensive. Swelling and redness of the skin, extending 3 or 4 cm. from the glabella into the surrounding skin, were the most prominent features of the eruption. Sensitiveness of the bones to pressure was also present—in the ilia, in an unusually marked degree. It had been noticed there a short time after admission. The sensitiveness of the bones had almost disappeared at the beginning of April, while the swelling of the joints was gone a month before. On testing the sensation the sense of touch was found to be undisturbed; the sense of location was considerably impaired, but not so markedly as at the examination in 1894. In contrast with the signs found during that examination, the patient showed now everywhere on the left side a diminished sensibility. Thus, for example, in the middle of the thigh on the right, 82 mm.; on the left, 95 mm. (normal, 67.7 mm.). At the patella, on the right side, 52 mm., on the left, 69 mm. (normal, 36 mm.); distance between the points of the calipers.

The temperature sense was uncertain on the left leg. An increased reaction was produced with the faradic current, even when very weak stimuli were used, but there was no difference between the two sides. I should not care to attribute any great significance to the differences which were shown on testing with a galvanic current, inasmuch as these differences showed considerable variations, at examinations at intervals of from two to ten days. There were no reactions of degeneration. The muscular strength was somewhat diminished, but, I thought, a little less than at the previous examination. I can say nothing definite concerning the motor tests, as these data had not been included in the history. So far as I can remember, however, there was nothing noteworthy.

The twitchings in the muscles of the thigh were also distinct at this examination, especially when the limb was touched with cold objects. In most cases, however, there were only some fibrillary twitchings, and actual movements of the extremities were noted only at first, especially when the cold contact was made in the neighborhood of a nerve-trunk. This symptom disappeared about the middle of March—at least it then no longer was distinct. On percussing with a hammer a bundle of muscle-fibers was raised, which remained prominent for some time.

The reflexes underwent considerable changes. The knee-reflex was maintained until the patient's discharge, May 6th. Ankle-clonus first appeared on February 20th, and did not disappear up to the patient's discharge. These reflexes were always more markedly exaggerated on the left side than on the right, until April 26th. On that day we find a note: "Today the right ankle-clonus is extraordinarily marked, but the patellar reflex on that side is not increased. On the left side both symptoms are unchanged. This continued for the next few days. The plantar reflexes were present, but not especially marked. The cremasteric and abdominal reflexes remained almost absent. There were no marked disturbances of urination or defecation, although both were somewhat difficult. The power of erection and

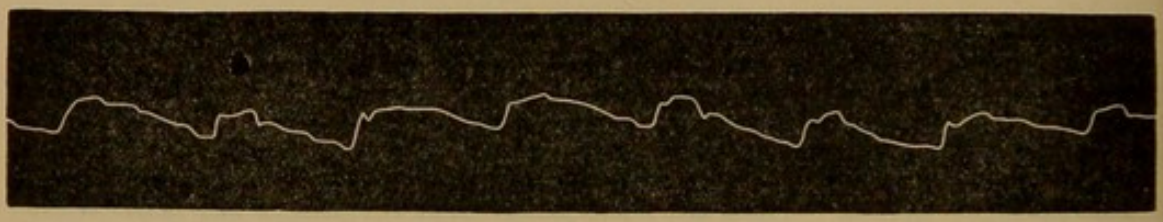


Fig. 52

the sexual desire had completely disappeared. The spastic gait became less marked in the course of time. As early as March 13th it was noticed that the gait had become quite steady, and its spastic character became less marked during the following weeks.

The patient was allowed to get up from bed in the middle of April—at first for a few hours, and then more frequently and for longer periods.

On May 6th, when the patient was discharged, it was noticed that, although there was still a certain amount of trailing of the soles of the feet on the ground, the patient no longer stumbled. The girdle-sensation gradually disappeared, and ceased altogether by April 10th. It reappeared, however, one day for a few hours after the patient had remained out of bed for a long time and had moved about a great deal. Examination with the ophthalmoscope had always given normal appearances.

Disturbances of the heart were very prominent during this attack. On February 3, 1895, a slight extension of the area of dulness, together with a displacement of the apex-beat outward, was noted. The apex-beat projected across the mammary line and was somewhat diffuse. This continued only for about a week, however, and was accompanied by irregularity of the heart's action. On the whole, however, even when the patient was discharged at that time, there was an unmistakable decrease in the force of the heart.

On admission, in 1899, it was noted that the area of dulness was slightly enlarged upward (third rib), and that the apex-beat lay in the mammary line, but did not project across it; and yet there was a marked irregularity in the heart's action. The systolic aortic sound was not clear; it was reduplicated, and the diastolic was exaggerated. A few days later there was an actual intermission of the pulse, and 44, 38, and 46 pulsations were counted successively in as many half minutes. This was sometimes better, sometimes worse.

The tracing of the carotid pulse, taken on February 24th (Fig. 52 was made with

a faster, and Fig. 53 a with a slower, revolution of the drum), shows distinctly the irregularity of the pulse in this case.

On March 1st the patient complained of pain in the region of the heart. Without any special changes in the area of dulness, the systolic tone over the aorta continued to be impure to the end of the observation. The heart's action, on the whole, was weak, and the tones remained faint. Judging by the amount of urine secreted, no cardiac insufficiency could be made out. The patient passed about two liters of urine daily, and the specific gravity was about 1012. I saw the patient once more on December 8th. He complained that he could not work; that when he bent forward, his back would become so stiff, and he would have such a marked girdle sensation, that he was obliged to stand erect as soon as possible. When he walked he grew tired very quickly, his toes dragged even after he had only taken thirty steps—especially the toes of the right foot. This was particularly marked in cold weather, and he was obliged to wear unusually warm clothing—for example, double woolen underclothes. He still suffered frequently from constipation, and was troubled on passing water.

It appeared, on examination, that the spastic gait was, indeed, very marked, and that the toes began to drag after the patient took a few steps. He was able to stand with his eyes closed, but swayed more markedly than before. The fibrillary twitching in the legs was apparent even when he lay perfectly still, but when the muscles were touched with a cold hand, severe contractions appeared which involved chiefly the extensor muscles, but also affected the flexor group, the latter being excited more than formerly. The clonic contractions could not be produced so easily on the posterior surface of the thighs as on the anterior. The adductors were not quite free; still they were comparatively slightly involved. The girdle sensation was present in its old place. The patient was able to describe a circle in the air fairly well with either foot, and was able to touch with accuracy any spot on his body which he could



FIG. 53.

reach with his feet. The plantar reflexes were, in general, weak, especially so on the right side. The cremaster reflexes were absent on both sides. The ankle-clonus was practically the same on both sides, and was not markedly exaggerated. The patellar reflex was strong on the right,—a little weaker on the left, side. The left leg was thinner, to the extent of about 0.5 to 1 cm., but the absolute circumference of both thighs had increased from 2 to 5 cm., as compared with their former size.

The heart dulness extended to the lower border of the third rib, to the median line of the sternum; the apex-beat was in the fifth intercostal space in the mammary line. A slight systolic purr was heard over the carotid, and there was a regular, bounding pulse (pulsus celer). The sounds at the apex were faint and somewhat muffled; the systolic tone over the aorta was not quite sharply defined, and seemed to be preceded by a slight sound. The diastolic sound, however, was loud and ringing, and especially marked over the ascending aorta and the arch. The diastolic aortic sound was louder than the pulmonary sound. The patient looked strong and healthy.

The disease of the spinal cord, which by this time had undoubtedly become permanent, and which must be designated as transverse myelitis involving the lumbar and the lower part of the dorsal cord, was, in my opinion, referable to the same original source which produced the swellings of the joints, the pain in the back, the eruption, and, finally, the heart disease. In addition to the affection of the endocardium, there was very probably in this case a transient involvement of the heart muscle at the time when the carotid sphygmogram was traced. As I have mentioned before, the heart was temporarily affected even at the first admission,

but the disease became gradually localized in the aorta, and did not produce very great changes in the heart.

It may be asked, with good reason, whether this case should be included in this discussion, so far as the affection of the spinal cord is concerned. There may have been here an insidious spinal disease, the development of which was favored by the patient's work in the water.

In my opinion, a very important feature of this case was that the patient positively asserted, without being asked, that the red spots—the septic eruption—which appeared later looked exactly like those which had appeared at first, with the stiffness of the fingers and wrists. The man was intelligent, and observed himself closely. He noticed the appearance of the eruption whenever his spinal disease grew worse in the course of the disease—and this was not an idea which he got from his physician. The fact that the joints of the fingers and wrists had first been affected proves that their stiffness was not a symptom that could be referred to the spinal disease, for the cervical region of the cord and the upper part of the dorsal region were later on found to be free from disease.

The relation of the temperature to the respiration, which has already been discussed (see p. 234), is positive evidence of the fact that the spinal disease was derived from the same source. The same relation between the temperature and the respiration was seen at both the first and the second period of observation. The nature of the cause of the disease was also evident by the involvement of the joints and bones which appeared later; by the prolonged exanthem, and finally by the affection of the heart. We may assume that inflammatory changes occurred in the spinal cord or its membranes similar to those which produced the polymorphous erythema of the skin. That this erythema may occur in mucous membranes in sepsis is well known, and I have myself observed such cases.* The corresponding inflammatory changes are seen often enough in the serous membranes. In the pleura and the peritoneum the changes may be either severe, and manifested by purulent exudates, or mild, with slight deposits of fibrin. I think that possibly an inflammatory process analogous to the erythema, but more narrowly localized, has in this case affected the spinal cord itself, more extensively than the membranes (for the signs of irritation were only short-lived). The observation on December 21, 1894, showed a comparatively rapid development of the principal symptoms. Naturally, we are unable to prove that this theory is correct. We are able only to offer an opinion, which must be tested by further observations.

Endocarditis has, for a long time, been closely connected with certain forms of **chorea,** and both of these diseases are etiologically related to acute articular rheumatism.†

Von Leube,‡ the first to express definite views on this subject, says:

"There is no direct dependence between chorea and endocarditis in the sense that the germ which causes the disease must be transmitted from the affected interior of the heart to the organs of the central nervous system. There is simply an association of local disease which must be traced to the same original cause. For the present we cannot decide

* Compare: Denning, *loc. cit.*, p. 97, cases XVIII and XIX.

† See further details in Pribram, *loc. cit.*, p. 229, and Wollenberg, "Nothnagel's Specielle Pathologie und Therapie," Bd xii, 2.

‡ "Deutsches Archiv für klinische Medicin," Bd. xxv, S. 238 (1880).

whether the germ in question acts by its mere presence or produces its effects through soluble substances which it generates. It is probable that everything that may produce endocarditis may also cause chorea."

Within the past few years I have often had the opportunity of seeing other disturbances of the central nervous system, along with endocarditis and chorea. The first of these which deserve to be mentioned are cataleptic conditions. The following case may serve as a good example:

Case XIX.—First admission, May 26, 1896. The patient was a girl, six years old at that time, whose parents were healthy, and who had had a slight rachitis in infancy. She had not learned to walk until she was a year and one-half old, and had learned to talk quite late. During the last few months before admission the child gradually lost flesh and strength, and complained frequently of headaches and weakness of the legs. These symptoms increased during the last fourteen days before admission. During the night from the twenty-fourth to the twenty-fifth of May the child was very restless and jumped out of bed in her excitement, although she immediately afterward complained of pain in the right foot, which was swollen in the morning. She also complained of severe headache and severe thirst. She was seen for the first time on the evening of the twenty-fifth. The temperature had risen then to 39.8° C. (103.6° F.).

On May 26th, we find the following notes: Severe cyanosis, very irregular breathing; namely, a series of rapid respirations interrupted by one, less often several, deep respiratory movements, at times broken by pauses. The diaphragm was up; and on the left side, above, the vesicular breathing was somewhat faint. The heart's action was very much excited, with gallop rhythm. The dulness was normal, but the systolic sound at the apex was not quite pure. The radial pulse was small, weak, irregular, 140; the jugular veins were distended and pulsated.

The patient was quite apathetic, did not answer questions, but yawned convulsively at frequent intervals. There was a marked chorea of the face and upper extremities. These movements increased considerably when the child was placed on the table. The right ankle was very painful and swollen. The child cried when moderate pressure was applied to the bones of both thighs, and to the cervical as well as the upper dorsal vertebræ. The tendon reflexes on both sides were diminished—more so on the left than on the right side. The plantar reflexes were almost absent on both sides. The abdominal reflex was distinctly present on the right side, and markedly diminished on the left. The urine was free from albumin. Nothing special was noted on examination of the other organs. The child, according to the mother's statements, had pin-worms, which were abundantly discharged after a dose of santonin.

The course of the disease was as follows (the child was observed regularly until the beginning of August, but after that only at such times as were necessary to continue the supervision of the case): All the symptoms changed a great deal from time to time—improvement and decline alternated. The cerebral symptoms consisted of excitement, unrest, and apathy, following each other in rapid succession. These began to disappear on June 8th and slowly vanished. The speech became normal only at the end of July; until then it remained indistinct and the child but rarely answered questions connectedly, although she had spoken very pleasantly to the physicians at first. Sleep became regular at the beginning of July, but until then it had been very much disturbed. The choreic symptoms continued for a long time, but at the end of the year they occurred only transiently.

At first the symptoms were very severe. Thus on June 3d we find the note: "The entire body is always in motion. There are yawning and irregular breathing, interrupted by sighs. The eyes are restless, and there is restlessness of the facial muscles, the arms, and the legs. If the child is allowed to walk a few steps alone, she falls forward with her trunk bent over until she finds a point of support. She stands very unsteadily."

Cataleptic symptoms were first noted on June 1st. "When an arm or leg is lifted, a certain amount of resistance is felt and the limb remains in the position in which it was left for about forty seconds. If the fingers are bent into a fist, they remain in this position with twitchings of the interosseous and lumbrical muscles."

There were variations in the severity of these symptoms. Thus they increased until the third of July, when the limbs remained stiff for two minutes at a time. The choreic movements involved then the upper extremities. The entire upper limbs shook and gradually sank lower and lower. From the sixth to the eighth day

of that month the cataleptic symptoms disappeared. "When a limb was lifted and again dropped, it fell as if dead."

Beginning with the twelfth, however, there was again a noticeable increase in the severity of the symptoms, and on the nineteenth both legs remained for five minutes in the position in which they had been left, while on the first of July they remained thus for eleven minutes. Cataleptic symptoms gradually and permanently disappeared, beginning with August 28th.

The plantar reflexes and those of the quadriceps tendons were absent for several weeks. Later the patellar reflexes were increased, and this continued until September, when the soles of the feet could be tickled even strongly without any response. This followed, however, when the child's attention was called to the fact that she was being tickled. This continued for at least five weeks, but then began to disappear gradually and vanished completely about four weeks later. The exact state of sensibility could not be tested accurately, but at the beginning there had been a rather widespread hyperesthesia. This may have depended upon the sensitiveness of the spine to pressure, which for the first time was noted over its cervical and dorsal portions on June 19th.

The femurs remained sensitive to pressure for a long time, and at times the ribs were also sensitive.

The spleen did not seem to be enlarged on admission, but it reached its largest dimensions on May 30th (8 by 5.5 cm.). After that it diminished rapidly in size. No changes could be found in the lungs, nor any bronchial catarrh. On the other hand, the peculiar change in the type of breathing which had already been noted on admission continued, although it varied in severity. As late as the end of July it was still distinctly present.

On July 28th a number of spots appeared on the skin of the legs, which varied in size to that of a small coin, and were either light red or greenish-brown in color, and well defined. The skin in these places was slightly infiltrated, but not sensitive to pressure. The eruption was diagnosticated as a multiform erythema and disappeared within a few days.

The patient's general nutrition was disturbed considerably for a short time. On June 6th we find this note: "The patient has become markedly emaciated and very pale during the last few days." This, however, did not continue for a long time. On June 12th we find an improvement and an increase in appetite. No further appreciable disturbances in this respect were noted later on. The heart showed great irregularity on admission, with a gallop rhythm and a not quite pure systolic tone at the apex. The boundaries, however, were perfectly normal. In the beginning of June second and third there was an enlargement of the area of dulness to the right and the apex-beat was displaced. The irregularity of the heart's action became still more marked. This continued, with variations, and although the heart-sounds were not quite clear, yet there was no murmur. The pulse was never quite regular in frequency and volume.

On August 20th the apex-beat was one-half finger-breadth outside the mammary line, and the area of dulness had spread 1 cm. to the right. The systolic sound was not clear at the apex and was reduplicated over the aortic area. The pulse was regular. There was a moderate cyanosis of the entire body.

While nothing abnormal could be found in the lungs, the respiration increased very rapidly in frequency, especially during the first three weeks of the disease. On the other hand, the pulse did not by any means increase so rapidly, so that the ratio P ÷ R was considerably diminished. Later, all these symptoms improved. It will be seen how variable were the values on the individual days, and how little they depended upon the temperature.

The patient was observed until February, 1897, but no noteworthy changes occurred. She improved slowly.

Second admission, October 2, 1897: The child had been well until about a month and a half before admission. There had been no general excitement nor twitchings of the face, but at times she complained of headache. The appetite had been poor, on the whole. The gait had been unsteady since the time mentioned, and the limbs gave way very frequently. There were also twitchings in the face and the upper extremities, and general excitement and restlessness. Lately, the headaches had become much more severe and acute pain was felt even when the hair was combed or when the head was lightly touched. The child also spoke "brokenly." Her sleep was restless, and she ground her teeth very often. There were irregular movements of the bowels, and at times pain in the abdomen. During the last few days she had grown very much worse, and become very restless, markedly prostrated, and feverish.

Date, 1896.	Frequency of Respiration.	Frequency of Pulse.	Ratio $\frac{P}{R}$.	Temperature in Degrees.	
				Minimum.	Maximum.
May 29	56	141	2.5	38.1	39.1
30	65	150	2.3	38.0	38.5
31	60	135	2.3	37.7	37.8
June 1	40	144	3.6	37.2	38.3
2	48	135	3.0	37.7	38.0
3	50	138	2.7	37.5	37.8
4	68	147	2.1	37.5	38.0
5	75	144	1.9	37.6	37.9
6	66	135	2.0	37.4	37.8
7	68	140	2.0	37.4	37.8
8	72	129	1.8	37.3	37.7
9	48	132	2.9	37.2	37.6
10	48	124	3.0	37.2	37.6
11	52	118	2.3	37.3	37.6
12	48	129	2.7	37.3	37.6
13	45	135	3.0	37.5	37.6
14	44	130	3.0	37.3	38.0
15	48	124	2.6	37.2	37.6
16	40	128	3.2	37.2	37.6
17	48	124	2.6	37.3	37.6
29	34	112	3.3	37.0	37.5
30	30	120	4.0	36.9	37.3
July 1	28	118	4.2	37.0	37.4
2	32	123	4.0	37.6	38.7
3	33	132	4.0	37.9	38.8
4	36	111	3.0	37.5	37.5
5	36	108	3.0	37.2	37.5
6	36	116	3.2	37.0	37.4
7	33	120	3.6	37.0	37.5
8	32	114	3.5	37.0	37.4
9	39	120	3.1	37.0	37.3
14	33	108	3.3	37.0	37.3
15	30	111	3.7	37.0	37.4
18	40	117	2.9	36.8	37.3
22	36	120	3.3	37.0	37.5

The foregoing table shows the relation between the frequency of the pulse and the respiration together with the temperature.

On October 2d she was found pale and cyanosed. She did not answer questions, and complained of severe headache. There were irregular contractions of the facial muscles, and the eyes moved constantly. When she sat upon the table, it was seen that she never remained perfectly quiet for an instant. The right knee and the left wrist were swollen and somewhat sensitive to pressure. There was no pain on pressure over the long bones. The patellar reflex on the left side was normal, but it was somewhat weakened on the right side. The plantar reflexes were almost absent. There were no cataleptic signs. The lungs were normal; the respiration regular—28. The heart dulness extended to the upper border of the third rib above, to the middle of the sternum on the right, and the apex-beat was in the fifth intercostal space at the mammary line. The systolic sound at the apex was muffled, but there was no real murmur. The pulse was regular—116. The spleen was 5 by 7 cm. The temperature at noon was 39.9° C. (103.8° F.). The following night she was very restless, and vomited in the morning.

The following is to be noted concerning the course of the disease: The headache disappeared entirely on October 5th. The apathy improved, beginning with the eighteenth, and disappeared on the twenty-second. The choreic movements were absent after the seventh. On this day the patellar reflexes were found to be equally marked on both sides, and the plantar reflexes had become distinct once more.

The swellings and pains in the joints had disappeared as early as the fifth of

October. On the eleventh the right wrist was again slightly affected; a multiform erythema appeared at the same time over this joint and was fairly wide-spread over the rest of the body, showing fresh eruptions on the following day, which, however, disappeared rapidly.

The heart remained in the same condition as on admission. It was never irregular, and the pulse was always regular, not very frequent, and not corresponding with the temperature—for example, there were 116 pulsations at 39.9° C. (103.8° F.), and also at 36.8° C. (98.2° F.). The pulse-temperature ratio was rather low, but it was easily changed. The spleen was enlarged on the eighth to 5 by 9 cm. and again diminished in size on the twenty-ninth. The respiration was always regular, did not show anything unusual, and from the few counts that were made, it was probable that it had not by any means increased in frequency. The temperature continued to be 39° C. (102.2° F.) and over until the twelfth. On the eighteenth it was 38° C. (100.4° F.) for the first time, and after that rather subnormal. An irregular distribution was again noticed on various days. The child was discharged, recovered, on October 31st.

Third admission, October 12, 1898: During the past year there had been no signs of illness. On October 9th, however, severe headache appeared, together with lassitude in the limbs and loss of appetite. On examination, on October 12th, the child was found very pale and cyanotic. She complained only of headache. Her speech was somewhat thick, but there were absolutely no choreic or cataleptic phenomena. The lungs were normal; the spleen measured 5 by 7 cm., and the bones and joints were not affected. The heart boundaries extended, above to the lower border of the second rib; on the right side, one finger-breadth beyond the sternal border, and the apex-beat was in the fifth intercostal space, one and one-half finger-breadths beyond the mammary line. There was a loud, blowing, systolic murmur over the apex. The diastolic sound over the pulmonary artery was distinctly accentuated. The pulse was soft, slightly irregular—106; the temperature in the evening was 39.1° C. (102.4° F.).

In the course of this attack the cerebral symptoms remained markedly in the background. The apathy was only slightly marked. The headaches were not very severe, and disappeared completely by October 29th. Traces of chorea were noticed once in a while during the month of November, and somewhat more distinctly during the following months. It was only in February that the symptoms became more severe, extended over the entire muscular system, and especially became noteworthy in the child's gait, which had become very unsteady. These symptoms continued until she was discharged, May, 1899, when they had improved to a great extent.

The patellar and the plantar reflexes did not show any important changes. The joints: From October 13th to October 15th the left knee, the left ankle, and the metatarsophalangeal joints of the great toe became swollen and painful. These signs rapidly disappeared, however, after the use of sodium salicylate—at first, 0.5 gm. three times a day, then four times a day, until October 22d. None of the joints showed any involvement after that. The bones: On November 2d the third and fourth ribs in the neighborhood of the nipple were very sensitive to pressure. On February 12th the left scapula, and on February 14th the right scapula, showed both spontaneous pain and pain on pressure. This all disappeared, however, in a few days.

The heart was irritable; the pulse continuously irregular. The area of dulness was enlarged, and there were constantly recurring variations in the temperature, for which no other cause could be found. All these pointed to a relapse of the cardiac affection. There were, in addition, disturbances of the respiration, and considerable variations in the amount of urine secreted, showing the variable amount of work done by the heart. (Compare my article on Cardiac Insufficiency in this volume, p. 131.)

The table on p. 271 gives a brief summary of all these points, which are of interest here.

Gradually the disturbance in the heart became more and more prominent. In the beginning of January the diastolic sound at the apex became more and more like a murmur, which finally developed to its full extent during the following week, and then was sharply characterized acoustically. The area of heart dulness was not materially altered, but the thoracic wall was somewhat more prominent than normal.

At the beginning of March all these different disturbances of the heart had receded so far that, inasmuch as the slight rises of temperature had ceased some time previously, the present relapse could be considered as at an end. On March 12th I allowed the little patient to get up, and in the middle of May she was discharged from the clinic. There remained a well-developed insufficiency of the mitral valve and a stenosis of the left auriculoventricular orifice, which has remained well compensated until the present time.

The manner in which the endocarditis ran its course in this case was noteworthy, aside from the affection of the nervous system. Slowly, haltingly, and more than two years after the first attack the malady developed into a perfect insufficiency of the mitral valve. During the first relapse the heart seemed to be scarcely involved. During the rather uneventful year which elapsed between this attack and the second, the valvular disease had developed unmistakably, and then progressed.

Chorea, and the peculiar nervous symptoms which accompany it, were also present in the patient, whose history is recorded under case IV. I shall, therefore, consider this case a little more in detail.

It may be noted, in general, that all the symptoms were subject to constant variations

CASE XIX.

Dates, 1898–1899.	Frequency of Pulse.			Frequency of Respiration.			P / R	Temperature in Degrees.								Amount of Urine Passed in C.c.		
								Morning.			Noon.			Night.				
	Maximum.	Minimum.	Average.	Maximum.	Minimum.	Average.		Average.	Maximum.	Minimum.	Average.	Maximum.	Minimum.	Average.	Maximum.	Minimum.	Average.
October 12 to 25*.....	108	78	..	..	36	..	..	37.2	38.4	36.7	37.7	39.1	37.0	37.9	39.1	37.2	..	..
October 26 to November 8.....	102	84	95	32	27	28	2.0†	37.2	37.9	36.8	37.4	38.1	37.3	38.0	39.1	37.5	..	..
November 9–22.....	126	81	107	32	21	27	4.0	37.2	37.5	36.8	37.4	37.7	37.2	38.0	38.8	37.7	..	..
November 23 to December 6	123	78	110	33	18	33	3.3	37.3	37.7	37.0	37.5	37.7	37.3	38.2	38.5	37.8	980	510	767
December 7–20.....	128	96	113	36	18	33	3.4	37.1	37.5	36.8	37.4	37.8	37.1	38.2	38.9	37.8	900	275	670
December 21–January 3	122	105	116	33	28	31	3.5	37.0	37.5	36.8	37.2	37.6	37.0	38.1	38.5	37.3	1000	370	690
January 4–17.....	136	104	114	33	27	30	3.8	37.1	37.4	36.6	37.3	37.7	37.0	37.9	38.6	37.5	890	470	665
January 18–31.....	120	90	108	36	24	29	3.7	37.1	37.3	36.6	37.4	37.8	37.0	38.0	38.4	37.6	860	370	606
February 1–14.....	120	99	113	39	27	32	3.5	37.1	37.5	36.8	37.5	37.7	37.2	38.0	38.4	37.7	800	350	610
February 15–28.....	117	96	112	42	27	32	3.5	36.9	37.6	36.6	37.1	37.4	36.9	37.3	37.8	37.2	870	410	663
March 1–14‡.....	121	96	106	32	27	29	3.7	37.0	37.2	36.6	37.2	37.4	37.0	37.4	37.6	37.2	930	450	663
March 15–28§.....	126	105	115	33	27	28	4.1	36.9	37.2	36.6	37.1	37.4	36.9	37.4	37.6	37.1	730	330	530

* Only six observations of the pulse, one of the respiration.
‡ The patient rose from bed on March 12th for the first time.
† Too few counts of the respiration.
§ Only eight counts of the pulse and respiration during these two weeks.

in intensity. Not that all of these increased or decreased on the same day, but it seemed that each was perfectly independent of the others.

Chorea: On admission, October 27, 1898, we find the following notes: "The patient does not remain quiet for a single moment. Sometimes he has slight twitchings of the forehead, sometimes around the mouth and the cheeks. Besides this, there are strong movements of the eyeballs. The twitchings are noted both in the upper and lower extremities." These motions did not become more marked, but continued for a long time and disappeared entirely at the beginning of May, 1899.

Cataleptic symptoms: On October 30th: "When the legs of the patient were raised, a certain amount of resistance, which was not very marked, was noted. The limbs remained for some time in the position in which they had been left (on the following day as long as six minutes), and did not fall of their own weight. The arms could also be placed in any position desired." This was, indeed, a clear picture of cataleptic muscular rigidity, which disappeared completely at the beginning of April. The arms were free from rigidity for a short time in December, but during the rest of the attack they were involved simultaneously with the legs.

Reflexes: The tendon and skin reflexes of the lower extremities were characterized during almost the entire period of observation by the peculiarity that they were sometimes almost absent; at times they were clearly present, and at other times they were increased in intensity. They varied from day to day. The cremasteric reflexes were stronger than usual from the beginning of the attack to the end. An erection of the penis almost always ensued when cutaneous reflexes were tested, with or without success, on the legs.

Trousseau's phenomenon was present, beginning with the second of November. It appeared here and there over the chest or the abdomen with great intensity, but was variable. At first the ankle-clonus was present; then it disappeared, and finally, beginning with December 20th, the paradox phenomenon of Westphal* was present very distinctly, and continued so until the middle of February.

Westphal says, in speaking of this sign: "In certain diseases of the central nervous system it will be seen, when the foot of the patient in bed is quickly and forcibly flexed toward its dorsum, that after the hand of the examiner has released the foot, the latter will remain in the same position in which it had been left, and will not return to its normal place with the action of gravity.

"If the tendon of the tibialis anticus be more carefully examined while this takes place, it will be seen that it suddenly becomes very prominent and remains so. This usually takes place a little after the act of dorsal flexion. The position of the foot then is one corresponding to the action of the tibialis anticus, namely, in adduction. A further peculiarity noted is that the foot remains in this position for a number of minutes, and then gradually and evenly, or else spasmodically, will sink back to its natural position.

"If we attempt to change the foot from the position which was given it forcibly, and which had become fixed through the contraction of the tibialis anticus, we meet a more or less marked resistance.

"In addition to the contraction noted at the tibialis anticus, we often see a paradox contraction at the extensor pollicis longus, and the extensors of the other toes, when they are vigorously flexed toward the dorsum."

In this case the phenomenon persisted most markedly and for the longest time in the tibialis anticus. It was also noted simultaneously, however, at times, in the extensors of the toes. My colleague, Professor Siemerling, who was good enough to see the patient with me, referred me to the work of Westphal, which had not been familiar to me at the time.

* "Ueber eine Art paradoxer Muskelcontraction," "Archiv für Psychiatrie und Nervenkrankheiten," Bd. x, S. 243 (1880).

He convinced himself of the presence of the paradoxal contraction which he had observed in a number of other cases. [The phenomenon has no diagnostic importance, being found in such diverse conditions as multiple sclerosis, tabes, paralysis agitans, alcoholism and hysteria.—Ed.]

A test of the sensibility, undertaken on November 12th, showed that the sense of location was somewhat diminished for the body in general, especially so in the right thigh. The sense of temperature remained unchanged. There was an absence of any disturbances which pointed directly to the involvement of the brain, and there was only transient headache.

A peculiar behavior of the blood-vessels which appeared very distinctly in case VI was probably referable to a central cause. I found that all the arteries which were accessible to the examining finger remained soft and full, in spite of the greatest amount of chilling. There was, therefore, not the slightest hint of a cramp-like contraction of the vessel. I spoke of these obvious differences on several occasions at that time—1872—during the patient's life, as the basis of a differential diagnosis against intermittent fever. I have had less occasion later on to pay any attention to them, and I do not know how often it may occur.

It is a question whether or not the transient edemas—which affected especially the face, and which occurred without demonstrable inflammation and certainly without nephritis—were connected with disturbances of innervation in the vessels. These edemas were seen quite frequently, as, for example, in cases VII, XII, and XIX. In other cases they were accompanied by a slight redness which seemed inflammatory. And probably in these instances there was only a slight disturbance in the tissues which, in its full development, would constitute multiform erythema.

Neuralgias are comparatively more frequent—especially sciatica. Examples of this are furnished by cases VI and X. According to my own observations, neuralgias, including intercostal and trigeminal neuralgia, are found more often at the beginning than during the further course of the disease. They are probably not connected with the endocarditis, but are concomitants of the fundamental disease. In this group may be included the cases of trigeminal neuralgia reported by Immermann.* The first of these was accompanied with endocarditis, but the other two were not. His interpretation was the same as my own.

It is difficult to decide the question as to whether visceral neuralgias occur, or whether the symptoms which resemble them are referable to actual tissue changes. Infarcts in the intestines undoubtedly produce the most severe attacks of colic, together with signs of irritation, and later of inflammation of the peritoneum. Possibly large infarcts of the spleen and of the kidneys (case XVII) may do the same, but I should like to leave undecided the question as to how much direct influence the cause of the disease has upon the nervous system in these cases. At times one comes across incidents which may give a clue in this direction. Thus, for example, in case VIII:

> The patient became ill after a period of prolonged constipation, and developed signs which pointed to an irritation of the peritoneum, emanating from the cecum, which disappeared rapidly after the use of opium. Three days later he developed signs of sepsis, with a marked involvement of the heart. The movements of the

* "Ueber larvirten Gelenkrheumatismus," "Verhandlungen des Congresses für innere Medicin," Bd. iv, S. 108, 1885. During the discussion similar observations were reported by Edlefsen.

bowels remained regular. Fourteen days after the first intestinal and peritoneal disturbance, from which he had been relieved on May 12th, the patient suddenly was seized again with severe pains in the abdomen, which increased to such extent that the patient literally howled with pain, so that his cries were heard at a great distance along the street, although he lived on the third floor.

Professor Dennig,* who had seen the patient soon afterward, reports as follows: "I found the patient writhing in bed, constantly groaning and moaning. The abdomen was quite uniformly swollen; it was extremely sensitive to pressure, the tenderness more marked on the left side than on the right." He was given forty drops of tincture of opium, which gradually relieved his pain. During the night he vomited thrice. On the following morning he again was seized with severe pains in the abdomen, which were not relieved by small doses of tincture of opium. Only thirty drops taken at one dose had a favorable effect.

An examination became possible on May 14th, and showed: "There was no diffused tenderness on pressure over the abdomen, but several painful areas distinct from one another and separated by free or less sensitive places. Thus, the region of the navel was especially sensitive to pressure, but from this point to the right and below, pain could only be produced by deep pressure, while the pain in the ileocecal region was especially marked. Similar conditions were present on the left side of the abdomen."

No further attacks of this kind occurred, and nothing remained but a lack of appetite, which lasted for eight days. This patient had a great deal of control over himself, and the pains must have been very severe in order to evoke such strenuous demonstrations.

Dennig reports still another case which I have also observed:

A student of medicine, aged twenty-four years. Diagnosis, sepsis with pericarditis and endocarditis, followed by valvular lesions. On the fourth day of the disease the man was very suddenly seized with the most excruciating pains in the abdomen, and at the same time suffered with nausea without vomiting. I found the patient in the knee-elbow position, with a pillow firmly pressed against the abdomen. His skin was covered with cold perspiration. The pulse was rapid, small, and the extremities were cold. He was scarcely able to answer the questions put to him so overcome was he with the pain. The abdomen was quite uniformly sensitive on pressure. Relief was obtained after the application of a large ice-bag and after the administration of large doses of opium—50 or 60 drops of the tincture at each dose. A marked improvement had taken place after four days. Similar events were repeated after eight days, and the same treatment gave the same result.

Not the slightest cause could be found for the origin of the painful attacks, which in this instance constituted the unmistakable signs of a visceral neuralgia. I have seen several similar cases, which were not so well marked, in slight septic infections, with or without endocarditis.

The muscles may be severely involved, may suppurate and form large abscesses, so that their involvement constitutes the central point of the entire clinical picture. We shall not speak here more in detail regarding this part of the subject, but will only mention that some muscles may show very severe, though transient, swelling and sensitiveness, which, however, disappear entirely after a time.

Thus, during the first week of the disease in case VIII, the patient showed a sudden marked swelling of the left biceps, which became extremely painful, and the skin covering it was diffusely reddened. After three days everything disappeared, but twelve days later the left supinator longus was also involved, though less severely, for a space of two days. Again, in case II, we had another picture. It has been already reported above (p. 236) that the respiration was considerably influenced by the painfulness of the muscles. The involvement of the muscles took place in a peculiar manner, concerning which I should like to speak more

* *Loc cit.*, p. 110.

in detail. I may remind the reader that in this case a suppurating angina (bursting on November 8th) began the septic process, which was accompanied by endocarditis. The following condensed notes, taken from the history, will show the way in which the muscular affection appeared:

November 10th: Only in answer to questions the patient complained of pain on the right side during respiration. No signs of pleurisy were present, nor could any be found even at autopsy. The movements of the trunk were slow but painless. The anterior abdominal muscles were very tense, and palpation was very difficult, though not painful, so that deep palpation could only be effected when the patient's thighs and knees were flexed.

November 11th: The patient complained of a vague sensation of pressure and tension in the abdomen, which could not be accurately localized. There was uniform tension, but no sensitiveness on pressure anywhere. November 12th: the symptoms, on the whole, increased in severity, but appeared more distinctly on either side of the middle line of the abdomen. But even there there was no sensitiveness on pressure.

November 14th: The face was painfully contracted. The patient moaned when he tried to rise, which he could do with great exertion. The patient complained at such times of intense pains in the abdomen, followed by pains in the back, which appeared as soon as he lay down. The abdomen was uniformly hard and extremely painful, even when touched very lightly. When the hand was cautiously laid upon it, the patient's face was contracted, and when slight pressure was made he complained of intense pain.

This pain appeared to be most intense, sometimes here, sometimes there, so that its accurate localization could not be determined. The sigmoid region seemed to be the part chiefly involved. The patient also complained of pain on passing urine.

November 14th: The symptoms grew worse. Every contact with the abdominal wall was extremely painful, and the walls were so tense that the bellies of the recti were very prominent. The muscles of the back, especially the lateral border of the quadratus lumborum of the left side, as well as the long muscles which pass upward from the sacrolumbalis on both sides, were also painful on pressure, and the same was true of the adductors of both thighs, as well as of almost the entire musculature of the limbs. All motions were executed with extreme caution and, therefore, very slowly.

November 17th: The symptoms on the part of the muscles remained practically the same, but, in addition, there was wry-neck. The upper part of the sternocleidomastoid was tense and very painful.

On November 18th there was improvement in some of the muscles, but a new symptom, fibrillary twitchings, was noted even on slight pressure with the fingers, and was especially marked in the right thigh. The pain and tension in the muscles gradually disappeared, leaving the abdominal muscles last. At the end of the month they were nearly gone. On the other hand, the excitability of the muscles to mechanic stimuli was still further increased. On November 19th "a slight tap with the percussion hammer upon the muscles of the thigh (extensor surface) produced tonic tension for a number of seconds, not only in the stimulated muscles, but also in those of the other leg and of the anterior abdominal wall."

This phenomenon did not continue for any length of time, but was followed by fibrillary twitchings and by the formation of circumscribed nodes at the point of irritation. These nodes were found in many muscles. The nephritis continued, together with the uremia which it produced and which threatened the life of the patient.

On January 14, 1891, a slightly different form of muscular affection was noted, in addition to the type just described: "A node of about the size of a finger appears on the adductor surface of the right thigh, when strong pressure is made with the finger at some points in the lower third of this part. This node persists for a few seconds, and then the muscle again becomes smooth gradually. The pressure is accompanied with pain."

All these signs had disappeared entirely when the patient was discharged, in a condition fit to pursue his regular work.

It is very probable that these affections of the muscles are inflammatory in origin, and are of the class of acute polymyositis.* An unusual circumstance, however, in these cases was the fact that the pain was at

* Lorenz, "Die Muskelerkrankungen," this series, vol. xi, section iii, pt. I.

first completely absent, and that there was only a feeling of tension. Later the tension became more pronounced, and was accompanied with severe pain. The fibrillary twitchings and the formation of nodes during and after the mechanical irritation may be interpreted as the result of disturbances of nutrition, similar to those which we see in cachexia from any cause. The extreme irritability of the muscles at those times, when a slight tap with the percussion hammer sufficed to produce a fairly extensive tonic contracture (November 19th), is not so easily explained. I think that transmission through reflex paths is very probably a factor in this process. These paths were also very probably involved.

Von Leube* recently contended that "the infectious material of muscular rheumatism represents nothing but a weakened virus of articular rheumatism." He cites two cases which show that an endocarditis may develop in the course of an apparently simple muscular rheumatism. In the first of these cases, however, there was an involvement of the left shoulder and the right ankle during the second week of the disease, in addition to the other features just mentioned.

The cases published by Karl Schnell,† of von Leube's clinic, which, in my opinion, are closely related to septic affections, are very interesting—especially so, the first case of the series. But, unfortunately, no bacteriologic examination was made—at least, none was reported. I have for some time held the same theory as von Leube.‡

In all these affections the question arises, in what relation do they stand to the causes of the disease, and what particular significance has the involvement of the endocardium in their development?

It must be admitted that no anatomic foundation can be discovered in most of the fatal cases of chorea, but the investigations of Nauwerck§ show that such a foundation may be present.

This observation originated in the Medical Clinic of Tübingen:

A girl, seven years old, died after eight days of illness, which set in after suffering for three weeks from vague symptoms, such as a feeling of fatigue, malaise, and indolence, and finally irritability of the heart's action. It was noteworthy that during the observation the temperature had risen only once to 38.1° C. (100.5° F.). On the other hand, the distribution of the temperature was often irregular in the sense already mentioned. "The patient gave the impression of suffering from a severe infectious disease, although fever was absent," says the clinical history. "Chorea was present in 'exquisite' form, and the heart was markedly affected. There was also a striking increase in the frequency of respiration, without any positive signs in the lungs."

At the autopsy (Ziegler), a slight but very acute endocarditis was found on the mitral, at the line of closure of the valves, as well as in the region of its base. Very small, scarcely visible efflorescences appeared at these points and at the points of insertion of some of the tendinous cords, especially on the aortic flap, which was considerably thickened by these changes. The other valves had remained unaffected.

The central nervous system did not show any conspicuous changes on macroscopic examination except edema and anemia, but Nauwerck found the following lesions microscopically:

(1) Inflammatory foci which were most marked on the floor of the fourth ventricle and at the crura cerebelli. They were totally absent in the cerebellum and in the basal ganglia of the cerebrum, especially in the optic thalamus, in the crura cerebri, the corpora quadrigemina, and the cortex of the hemispheres. The

* "Beiträge zur Pathologie des Muskelrheumatismus," "Deutsche medicinische Wochenschrift," 1894, S. 1.

† "Ueber Polymyositis," Würzburg, dissertation, 1892.

‡ "Deutsches Archiv für klinische Medicin," Bd. liv, S. 407.

§ "Ueber Chorea," Ziegler und Nauwerck, "Beiträge zur pathologischen Anatomie und Physiologie," Bd. i, Jena, G. Fischer, 1886, S. 409.

lower half of the medulla oblongata was almost free from inflammation, and the spinal cord was entirely so.

(2) Hemorrhages were most numerous in the pyramids, in the internal capsule on both sides, as well as in the inflamed portions of the medulla and of the pons. There were no capillary emboli, but the effused blood was frequently so rich in white corpuscles that "one could often speak of a true hemorrhagic inflammation."

(3) Degeneration and fatty disintegration of the nerve-fibers, most markedly in the cervical region of the cord, gradually diminishing downward, so that only a few isolated fibers were degenerated in the lumbar region.

The entire cross-section of the spinal cord was involved in this degeneration, but the anterior and lateral columns were especially affected. The degenerative changes were almost entirely absent in the other parts of the central nervous system, and only isolated degenerated axis-cylinders without any fatty degeneration were noted in the internal capsule and in the white matter, especially in the central brain. The peripheral nervous system, examined in the nerves of both arms, appeared unchanged, and the same was true of the roots of the spinal nerves.

No bacteria were found anywhere, although the pneumococcus was particularly looked for, inasmuch as a "slight pneumonic consolidation" was found in the right lung.

These findings show that severe and widely different changes may occur in the central nervous system in chorea. Nauwerck draws the following conclusions, which I think are perfectly justified: "To begin with, admitting the possibility that an inflammation of the central nervous system is capable of producing chorea, it is easy to understand why the clinical picture of this disease is so variable. The inflammation need not be so extensive as in our case (for even the edema which was found at autopsy was probably inflammatory in origin). The inflammation may be more localized—possibly it may be only unilateral, and may either involve the cortex more extensively or else may leave it perfectly free. According to the location and extent of these changes the symptoms will be either predominantly motor and sensory, on the one hand,—for example, hemichorea,—or the psychic symptoms will be more or less pronounced, on the other hand.

"It must be left undecided what rôle the degenerative processes in the nerve-fibers play in chorea, but I am inclined to attribute to these changes a great deal of significance in the disturbance of motion, as they are capable of impairing conduction, or even of totally destroying some portion of the motor paths. At all events it is also possible that the change in the nerve-paths in itself permits contractions to enter into the clinical picture of chorea.

"Small inflammatory foci in the central nervous system, such as I have spoken of, may heal without appreciable permanent changes. We may remember the frequent cases in epidemic cerebrospinal meningitis, in which such foci are almost never absent. In the same way, nothing prevents the degenerated nerve-fibers from returning to normal, at least so long as the degeneration is in its earlier stages. Possibly, the old paths are destroyed and others are formed in their stead.

"On the other hand, the inflammatory foci may persist and may finally lead to the formation of sclerotic patches, or else the degeneration of the nerve-fibers may so extensively involve the conduction paths that they cannot be replaced at all, or only imperfectly."

I think that it is perfectly permissible to apply this fundamental theory to what I have seen and described here, as accompaniments of undoubted infectious endocarditis. It was only in case XVIII that we found possible permanent tissue changes in the spinal cord, which were also peculiar in

the fact that they combined with other signs indicating a renewed infection in producing an aggravation of the local disease. We have general analogies for such a course of events in what is known as *locus minoris resistentiæ*.

In all other cases the resemblance to what is observed in infectious chorea is very close: the transitory nature of the symptoms, their variability, and finally, in most cases, their termination in recovery. Another resemblance is found in the indefinable localization in the central nervous system. Most often the disturbances are only transient. Thus, Siemerling* says that for the present we are justified in assuming that "the paradox contraction should not be regarded as the expression of an organic, but as the manifestation of a functional, disease of the central nervous system."

There is no doubt that true inflammations may also occur in the muscles, which must be directly traced to the germs of the disease that have settled in these structures. How much the other symptoms which have been described above, and which occur quite rarely, depend immediately upon a disease of the muscle-fibers, or upon some or any disturbances in the nerves, is a question which for the present can scarcely be definitely answered.

In the course of time I have learned to pay particular attention to all these symptoms, because they occur in the insidious cases of endocarditis, and possess a by no means inconsiderable diagnostic value. It is true that I could not find an immediate connection between them and the endocarditis, but I believe that they are manifestations of the common cause of the disease, acting, in these instances, upon the nervous system.

THE ORGANS OF THE SENSES.

The Eyes.—Litten, in his work on septic affections, paid a great deal of attention to the disturbances which occurred in the eyes and attributed a great diagnostic value to ocular symptoms in the diagnosis of this infection. We must admit without reserve that he was correct so far as the cases upon which he based this study were concerned.

It is a question, however, how far the conclusions of Litten are applicable to sepsis as a whole. His material included the most severe forms, especially those occurring after childbirth, the latter constituting a group which is peculiar in the intensity of the infection. Possibly, it is on account of this difference in virulence that the affections of the eyes play a less prominent rôle in my own observations. Litten himself† practically admits this to be the case.

In his cases endocarditis was very frequent (63 per cent., more of them ulcerative than verrucous). It is not astonishing, therefore, that affections of the eye occurred quite frequently in these patients. The fact, however, that even the most severe inflammations which destroy the eye have been seen by Litten in patients who had no involvement of the heart teaches us that they may occur without the transmission of the infection from a diseased endocardium.

Thus, we find the following in Litten's principal table‡:

* "Ein Fall von schwerer Neuropsychose, Ausgezeichnet durch congenitale Anomalien des central Nervensystems," "Charité Annalen," Bd. xxii, 1892.

† "Verhandlungen des Congresses für innere Medicin," Bd. vii, S. 325 (1888).

‡ *Ibid.*, p. 380, etc.

"Case VIII.—Eyes: On the left side, hemorrhages with white centers; on the right, at first, hemorrhages; then panophthalmitis. The valves were intact. Bacterial foci were found in the heart muscle.

"Case XI.—Hemorrhages in both retinæ. A panophthalmitis developed later on in both eyes. The heart was intact.

"Case XV.—Panophthalmitis on the left side; on the right, marked chemosis, iritis, and perforation of the cornea. The heart was intact."

On the other hand, the eyes may remain quite free, although the endocarditis has fully developed. This is probably the most frequent event. In Litten's case XXIV, the endocarditis was situated in the valves of the pulmonary artery, but the eyes remained free.

Romberg* expresses himself as follows: "The embolic formations of the retina are usually sure signs of malignant endocarditis, but they are not present in more than a quarter of the cases, and apparently are to be found only in cases running a comparatively rapid course." It is certainly of great importance to pay attention to the eyes, and we must thank Litten for having so definitely called our attention and interest to this, and for bringing out the characteristic features of eye-involvement in sepsis.

Eversbusch† makes the following remarks on this subject: "The so-called septic changes in the retina possess a certain practical significance, and possibly depend either upon intoxication or upon non-infectious capillary embolisms. In some cases they run their course without appreciable impairment of vision and in this respect they resemble the retinal changes observed in severe or pernicious anemia. Ophthalmoscopically, they appear as numerous irregular hemorrhagic foci and yellowish-white spots which are found in the neighborhood of the somewhat indistinct and hazy, but not swollen, papilla; especially at the points of division of the veins. The retina, as a whole, is slightly clouded, while its periphery usually remains free.

These disturbances in the nutrition in the retina which appear soon after the beginning of the general disease, and which reappear in the course of it, with the formation of new hemorrhages, do not possess any prognostic significance, inasmuch as many patients recover completely after the ophthalmoscopic picture has become perfectly normal.

In a case of endocarditis (streptoccocic) a severe involvement of both eyes were noted, not long since, in the Ophthalmological Clinic at Tübingen. My esteemed colleague, Professor Schleich, was good enough to place the clinical history at my disposal:

Case XX.—A girl, aged eight years, admitted November 9th, died November 13, 1899. The child had been well until June of that year, when she was seized, rather suddenly, with fever, pains in the joints, severe headache, and repeated vomiting during the first few days. In the beginning of July there was swelling of the feet, and such severe dyspnea that the patient could scarcely be kept in bed. Her physician diagnosticated heart disease. She improved, but in August had a relapse. Dyspnea, swollen feet and legs, swollen abdomen. A slight remission occurred in September. In October there was a marked increase in the local symptoms, but her general condition had improved.

Six or seven days before admission the girl complained of blurring of vision and of pains in the right eye. No noteworthy external inflammation could be discovered in this eye. Her physician sent her to the ophthalmological clinic.

On examination, November 9th: Left eye: Acuity of vision, apparently normal;

* "Handbuch," etc., pp. 982, 983.

† Penzoldt and Stintzing, "Handbuch der speciellen Therapie," Bd. i, S. 655 (first edition). A detailed account, in Litten, p. 410, etc. See also "Diseases of the Eye in Relation to Other Diseases," by Schmidt-Rimpler, this series (1898), vol. xxi.

at any rate, more than fingers at five meters. The eyes were pale; the media clear, the fundus normal; there was no pain. The right eye: Light-reflex diminished. Cornea clear, anterior chamber shallow; iris thickened and markings quite effaced. The pupil irregular, narrow. A dense, grayish-yellow exudate lay in the pupil, projecting somewhat into the anterior chamber, and completely covering the aperture of the pupil, so that nothing could be seen of the parts of the eye behind it. The pupil did not react to light nor to atropin. There was a circular posterior synechia. There was also slight conjunctival and distinct ciliary congestion. The ocular tension was somewhat diminished, and the eyeball was slightly painful on pressure.

Heart: There was precordial bulging, a pronounced pulsation over the entire cardiac as well as the epigastric region. The dulness extended above to the third rib, on the right side to the right sternal margin. The apex-beat was in the sixth intercostal space, one finger-breadth outside of the mammary line. On auscultation there was a loud systolic and a diastolic murmur over the apex. The systolic aortic sound was impure. The diastolic pulmonary sound was markedly increased in intensity. The lungs did not show any abnormal physical signs. An enlargement of the spleen could not be demonstrated with certainty. There were small amounts of freely movable fluid in the abdominal cavity, and edema on the dorsal surfaces of the feet, as well as of the ankles. The patient complained of pain in the ankles, and the right ankle and the right leg were somewhat sensitive to pressure. The urine was free from albumin, had a specific gravity of 1020, and was dark reddish-yellow in color.

On November 12th the appearances in the right eye were unchanged, but in the left eye the following was noted: There was no external irritation in this eye. The pupil was greatly dilated (atropin on the tenth), and the refractive media were clear. The margins of the papilla were indistinct; there was a marked congestion of the veins, which were considerably twisted toward the periphery. Directly upward from and close to the papilla the inverted image showed two whitish foci, close together and surrounded by markedly dilated and tortuous vessels. A little further toward the periphery a few isolated hemorrhages were noted, and in some places the small vessels seemed to be interrupted. There was marked perivascular hemorrhage in some places. (See Plate II.)

Beginning with November 11th there were increasing disturbances in the brain. The patient became very apathetic; she moaned and cried at times; she ate very unwillingly, and passed urine and feces involuntarily. On the following day the moaning increased and she cried out when she was touched. During the following night the girl was completely unconscious, no longer reacted to the voice, and moaned and cried out whenever she was touched. There were also gritting of the teeth and a slight degree of opisthotonos. The plantar reflexes remained unimpaired—the left somewhat more marked than the right. No Trousseau's phenomenon. This condition remained to the end.

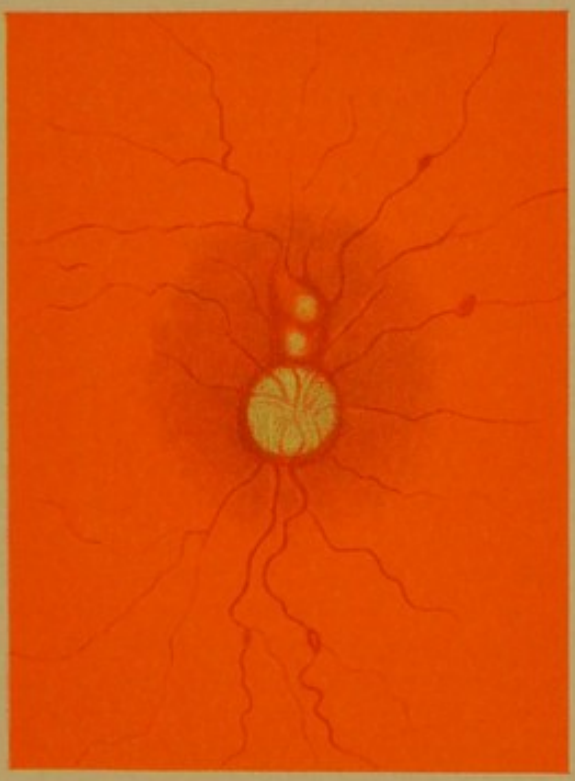
Heart: The signs of dilatation and of heart failure increased. The area of dulness became enlarged, reaching 1 cm. to the right of the sternal border, with the apex extending almost to the anterior axillary line. The heart-sounds and murmurs were very much less sharply circumscribed. There was a thrill over the entire surface of the cardiac area and the neighboring parts of the chest. There was marked pulsation of the veins of the neck and in the epigastrium, increase of cyanosis and of coolness of the extremities and the face.

No new affections of the joints and bones appeared, but, on the contrary, those that had been present rather improved. Swelling of the spleen could not be demonstrated. The temperature, only on one occasion, reached a maximum of 40° C. (104° F.), while the minimum was 37.2° C. (98.9° F.). The distribution of the temperature was distinctly irregular. The pulse was small and irregular, but this varied at different observations. The maximum frequency was 140 to 145; the minimum, 112. The respiration was always frequent and shallow, the maximum being 48, the minimum, 44.

Death occurred on November 13th at 8.30 P. M. The course of the disease was very acute, therefore, from the time when the disturbances in the eye appeared.

The autopsy (Pathologic Institute, Dr. Dietrich) showed the following (abstract): After the sternum had been removed, the lungs contracted completely. The entire space beneath the anterior chest-wall extending over an area equal to that of the palm of the hand was occupied by the pericardium. The apex was at the lower border of the fifth rib in the mammary line; the right border of the heart was approximately in the right mammary line, and the upper border at the level of the second rib.

PLATE II.



Left eye, reversed picture. The white patches and hemorrhages are explained under the case, p. 280.

Heart: Both layers of the pericardium were adherent to each other by a gelatinous mass of tissue, and were covered with fresh, partly fibrous and partly fibrinous, deposits. There was a large hemorrhage in this pericardial deposit over the left auricle. There were, in addition, ecchymoses of pinhead size scattered over the entire heart. The heart was twice as large as the hand of the cadaver. Its anterior wall was formed chiefly by the right ventricle; its apex, by the left ventricle.

Both flaps of the mitral were shortened, thickened, and had thick vegetations at their borders. These excrescences were but loosely adherent to the valve, and were almost purulent in consistence. The tendinous cords, especially those of the aortic flap, were thickened and shortened, and showed some vegetations at the apex of the aortic flap which overlapped other tendinous cords that were inserted there. The endocardium of the left side of the heart showed smaller excrescences at the entrance to the auricular appendage. The other valves and orifices were uninvolved. The left and the right auricle and the right ventricle were dilated, and their musculature thickened.

In the brain, besides diffuse punctate hemorrhages and edema, there was an area of softening with considerable hemorrhage in the posterior part of the third temporal convolution. There were no thrombi, but fluid blood everywhere. There were old and recent hemorrhagic infarcts in the enlarged spleen(12 cm. long, 8 cm. wide, and 3 cm. thick), the capsule of which showed signs of acute inflammation. There were similar infarcts in the kidneys. There was a slight fresh pleurisy, but the lungs did not show any noteworthy lesions. The liver was congested. There was a slight amount of ascites. The stomach and the small intestine showed ecchymoses. The eyeballs were prepared for more careful examination.

Bacteriologic examination: Cultures from the blood, obtained from the fingertip as well as from the small veins at the elbow during life, and from the blood in the heart after death, did not give any positive results. On the other hand, sections from the infarcts in the spleen showed dense masses of streptococci. On examination, the various miliary abscesses gave no satisfactory results.

This is a case which I classed, from the first, not as one of acute articular rheumatism, but as a septic infection in the narrower sense. At all events —and this is the most important point here—we were dealing essentially with an endocarditis localized in the mitral, which continued for a long time, and which brought about death through emboli and finally through heart-failure. The involvement of the eyes which was noted in this instance is quite rare, according to published statistics. A fact which deserves especial mention is that the right eye was involved first, and that the left followed very rapidly, but that the localization of the affected portions in the blood-vessels differed markedly in each eye. Ophthalmologists teach that panophthalmitis may in some cases be the only sign of cryptogenous sepsis, and may often be accompanied by an ulcerating endocarditis.*

A severe puerperal infection is, however, a much more frequent cause of these ocular involvements. Litten found, in his thirty-five cases, five instances with double, and three with single, panophthalmitis. Among these there were five cases of puerperal sepsis; one of abscess of the liver with perforating ulcer of the stomach; one with an uncertain diagnosis (symptomatic anthrax ?), and one with scarlet fever. In four cases there was also ulcerative endocarditis, while in another four this was absent. Romberg saw forty-two cases of acute malignant endocarditis in which one only had suppurating panophthalmitis. I believe that these figures answer the question as to the frequency of eye-complications in the various etiologic types of sepsis.

The Ear.—Aside from functional disturbances, affections of the organ of hearing are not frequently seen in sepsis. Litten collected all the cases which had come to his attention (until 1881), and noted that he had never seen severe purulent inflammations of the middle ear with perforation of

* Schmidt-Rimpler, *loc. cit.*, p. 464; Eversbusch, *loc. cit.*, p. 647.

the drum in the course of septicemia and allied diseases. On the other hand, Bürkner* says: "Septicopyemia not infrequently leads to purulent inflammation of the tympanic cavity, with a distinctly destructive tendency, and especially with a rapid and extensive destruction of the membrana tympani." I can add one case of this kind, and shall report it briefly:

CASE XXI.—A boy, aged twelve years, fell ill very suddenly on May 31, 1886, with a sudden chill. He was admitted on June 1st, when an affection of the heart was already evident, and the further course of the disease proved that the heart trouble was localized chiefly in the aorta. There was a moderate fever, continuing for four weeks, with a maximum of 39.9° C. (103.8° F.). There were also quite wide-spread pains in the bones, but no swelling of the joints. The spleen was slightly enlarged.

On June 6th the child complained of severe pain in the right ear. He could hear the ticking of a watch only at a distance of 2 cm. My colleague, Professor Wagenhauser, found the following appearances in the ear: The drum was intact, but markedly congested, especially at its upper portion. There was an exudate in the tympanic cavity. On June 27th: No more exudate in the tympanic cavity. The drum was pushed outward by masses of granulation tissue, especially by its posterior and lower quadrant. There was no perforation and no congestion. The pains in the ear had disappeared. The hearing had improved, and the ticking of a watch could be heard at 15 cm. July 12th all disturbances had disappeared. Whispered conversation was heard at five meters, with the right ear, and a watch at 20 cm. The patient was discharged cured.

SKIN.

In the majority of cases the symptoms on the part of the skin which occur in endocarditis belong to the primary disease, and not directly to the heart affection. The chief interest in the cutaneous signs lies in the fact that they offer not unimportant aids in the recognition and interpretation of more obscure cases of endocarditis. It is possible, however, that the involved skin ought to be considered as a part of the general disease.

Hemorrhages.—The occurrence of hemorrhages into the skin is probably intimately related to the severity of the infection. Litten found hemorrhages in the skin in twenty-one of his thirty-five fatal cases. We must remember that the "hemorrhagic diathesis" occurs in all severe infections, as well as in some intoxications, in a narrower sense of the word. Hemorrhages also may be found, however, in patients who do not show any signs of a grave general infection.

I have had occasion to observe cutaneous hemorrhages in my own cases, and in some instances they appeared in rather unusual places—as, for example, in the palm of the hand, in case XII. Similar observations were made by Litten. He remarks that "multiple cutaneous hemorrhages may occur from time to time in the course of chronic recurrent endocarditis without any indications of the presence of a general septic infection in the clinical course or in the anatomic findings."

From the seven clinical histories which he had at his disposal it appeared that these cases ran their course without any fever, or with but very slight elevations of temperature.

The hemorrhages in these circumstances may be so limited that they can be noted only on very careful examination with the intent of finding them. This was so in case IV, for example.

From November 1st to 16th there were cutaneous hemorrhages, the largest of which was the size of a nickel. Then there were none until March 9th. From

* In Penzoldt-Stintzing, *loc cit.*, vol. i, p. 581.

this day on until March 14th there was again a hemorrhage of the size of a penny, and after that no hemorrhages were seen until the patient was discharged, June 21st.

A very sharp contrast to this is afforded by the case of Litten (Kaudersky).* The patient was a man with severe septic endocarditis (fatal termination within four days), and within a few hours extensive hemorrhages appeared and changes occurred in the effused blood, from pale red to the darkest brownish-red, literally under the eyes of the observers—the physician, as well as the artist.

Erythema.—All forms of erythema have one feature in common, a congestion of the skin,—which may or may not be accompanied with slight inflammatory exudation. Roseola may also be classed in this group, but we have seen it only in one case (case XVI). A form which is much more frequent is that known as erythema multiforme, together with all the transitions possible toward the other forms of erythema which are known by special names. Of these we may mention, especially, erythema nodosum and urticaria. The anatomic changes in the skin of the involved portions are more important than the classification, and it has been found that inflammatory processes may be present, not only in a mild form, but also in a most severe type, and that different degrees may occur simultaneously in the same case.

I may cite case VIII as an example of different cutaneous lesions existing at the same time.

At the end of the first week of the disease the following appearances were noted overnight: The chest, the arms, the back, the buttocks, less markedly the face, and not at all the abdomen and the lower extremities, were covered with very irregular blood-red spots, the size of which varied from that of the head of a pin to several centimeters in diameter. Some of these were simple hyperemias, which disappeared when the skin was stretched. The majority, however, consisted of ecchymoses with infiltration in their vicinity. These spots projected above the level of the healthy skin; their color varied between a dark-red and a brownish-red, and some of them had a lighter center; a few showed a small pustule in the middle, and still others showed a darker center. The patient complained of burning in the affected portions of the skin. On the following morning there appeared pronounced wheals (urticaria) on the left arm, which at first were pale, with a swollen base. Toward noon they became red, and in the evening they showed distinct effusions of blood. By the following morning several such spots had coalesced, and could not be distinguished from the first eruption. The cutaneous affection continued in variable forms until the end of the fifth week of the disease. It did not leave any traces.

In some cases the erythemas appeared only after the disease had lasted for some time. Thus it was in case XVIII, where they appeared after half a year, and in case XIX, after two months.

In both instances a relapse brought on the same forms of eruption—polymorphous exanthem and urticaria. At some places the erythema continued for weeks at a time, and persisted especially on the glabella (cases III and XVIII). The individual spots, as a rule, disappeared in the course of a few days.

More Severe Inflammations of the Skin and of the Subcutaneous Tissue.—I have not observed these very frequently. For the most part, they were affections which at first resembled erysipelas, which later extended more deeply, and which had to be designated as phlegmons. They occurred as early symptoms, or even as the first symptoms of the affection (case XI), or sometimes they appeared late (case XV). I shall report

* *Loc. cit.*, p. 420, and also plate IV of the second volume of "Zeitschrift für klinische Medicin."

briefly a case in which there was an extensive destruction of the superficial layers of the skin, which occurred rather unexpectedly from within outward.

Case XXII.—A woman, aged forty-three years. Her illness dated from the afternoon of February 4, 1886, when she was seized with a sudden chill, followed by angina. On the sixth a circumscribed edema and redness appeared on the left cheek, the region of the neck remained free, but from the left clavicle downward to the second rib there was doughy edema, sensitive on pressure, which on the seventh of February is said to have extended to the lower border of the thorax on the left side. In the course of that day this edema, as well as the redness and swelling of the cheek, diminished considerably. An eruption appeared during the night from the seventh to the eighth. The color of this eruption was pale red to bluish-red, and its form resembled that of erysipelas, in that the margins advanced in zig-zag outlines. It differed, however, from erysipelas, because there was a great deal of swelling of the subcutaneous tissue, and there were pale islands in the middle of the affected regions. Narrow projections extended into the neighborhood. The region of the body most markedly affected at first was the edematous portion already described, but there were extensions beyond this part, which looked as though the skin had been hit with a whip. In the course of the day vesicles appeared upon the involved portions, which grew to bullæ of the size of a hazel-nut. When these burst they disclosed slightly bleeding granulated surfaces but no gangrenous spots. The skin affection extended further until the patient's death, on February 14th. This extension always took place in such a way that there was at first a slight painful edema, which was followed by redness and the formation of bullæ after a considerable time (up to thirty-six hours). On examining the cadaver (Professor Ziegler) large numbers of staphylococci and streptococci were found.

In this case the destruction of the skin was so extensive, and the healing of the involved portions was so slow, that if the patient had not died as the result of the severe affection, there would have probably been the same signs as those observed in very extensive burns.

A severe pemphigus-like destruction of the skin was also noted in the case of ulcerating endocarditis reported by Litten.* Suppurations of the lymphatic glands draining the portions of the skin involved in the inflammation have also been seen in several cases (case XI).

Bacteria have been found in several instances, either in the involved portions of the skin or in the neighboring capillaries (first by Leube). Litten has also made the same observation. But even in his case (Kaudersky), mentioned above, neither emboli nor bacteria were found within the vessels in portions of skin removed during life, as well as after death. In my own investigations I have always obtained a negative result in those cases in which no pustules appeared. The latter may be truly said to be important in the diagnosis of an insidious endocarditis—as, for example, the following case:

Case XXIII.—A young man, aged seventeen years, was admitted on June 20, 1893. Eight days previously he had been taken ill with indistinct general symptoms, such as malaise, headache, and loss of appetite. At first nothing was found but extensive pain on pressure over the long bones and over the muscles of both thighs. The heart's action was irregular, but the heart-sounds remained clear. The temperature rose to 39.2° C. (102.5° F.).

On the twenty-third numerous circumscribed pustules from the size of a pinhead up to the size of a pea very suddenly appeared upon the skin of the chest, abdomen, and back. The pustules had small vesicles filled with yellow contents in their centers, and were surrounded by red infiltrated areas. The larger ones were painful. They resembled small furuncles. On examination the pustules were found to contain staphylococci (culture). The spleen was enlarged to 9 by 8 cm.

Beginning with June 25th, there was no longer any rise of temperature. Within the next few days the area of heart dulness became larger, especially upward, and the

* *Loc. cit.*, p. 430.

apex-beat was displaced outward, but the tones remained clear as before. It was only beginning with July 5th that the systolic sound over the aorta, and beginning with the eleventh that the same sound over the mitral, became muffled, faint, and impure. There were, in addition, a slight pericardial friction-sound over the base of the heart, and increasing heart failure. There was cyanosis, a venous pulse, and when standing up during the examination, on July 5th, the patient fainted and could not be resuscitated for some time. There were marked variations in the frequency of the pulse when the patient was lying down and when he sat up—as, for example, on July 15th, the pulse showed 84 and 120 beats, respectively, in the two positions.

The area of dulness was increased, and the apex-beat extended one finger-breadth beyond the mammary line, but remained within the fifth intercostal space. When the patient was discharged, the heart weakness had not disappeared entirely, and it looked as though an insufficiency of the aortic semilunar valves was developing. The patient disappeared from observation, but inquiries showed that he was able to continue working at his trade as decorator.

The types of septic erythema which involve the skin surface extensively, like acute exanthems, especially like scarlet fever, are probably due to the products of metabolism of the microbes which circulate in the blood. They resemble a number of skin affections which are caused by the ingestion of certain drugs. Whether such drugs have been given or not is, therefore, an important question to be asked before an erythema can be available for diagnostic purposes.

JOINTS AND BONES.

Affections of the joints and bones are intimately related to the general infection, but in exceptional cases the bone or joint disease may be directly transmitted from the heart. It is difficult to determine when this takes place, and this particular origin of the joint or bone trouble is of very little practical importance.

In the great majority of instances joint involvement is noted among the first, at all events among the early, manifestations of endocarditis. In addition to tenderness on pressure and on motion, these joints usually show redness and swelling. An effusion may be entirely absent in many cases. When an effusion is present, it usually is not very abundant and only in exceptional cases does it become purulent. Acute articular rheumatism offers the well-known picture which it is not necessary to describe more in detail here. It seems to me that in those mild or severe forms of endocarditis which do not coexist with acute articular rheumatism in the narrower sense, it is the epiphyses, the joint capsule as a whole, and the ligaments, which are more markedly affected, and not the serous lining of the joint itself. The transient nature of the articular involvements speaks in favor of the idea that they arise usually only through irritation by the products of metabolism of the pathogenic microbes circulating in the blood, rather than from a localization of the germs themselves. This view is confirmed both by clinical observation and by the fact that the joints usually show very few changes, and that, with a few exceptions, they give entirely negative bacteriologic results. On the other hand, when the germs of sepsis enter into a joint in large numbers and multiply therein, as they do, for example, quite frequently in puerperal infections, it is not at all rare to find suppurative arthritis.

The same remarks apply to the involvement of the bones. In most instances the osseous affections are mild, and often cannot be recognized without a special examination. He who would wait until he sees a case of Chassaignac's "bone typhoid" would probably accompany the grandchildren of the patient suffering with bone sepsis to their grave.

It is ordinarily the diaphyses of the long bones that must be examined; but the bones of the hand, the foot, the ribs, the ilia, and least frequently, probably, those of the skull, must also receive attention. Clinical examples may be found in most of the cases related in this treatise.

SPLEEN, LIVER, STOMACH, AND INTESTINES.

In severe general infections the spleen is markedly enlarged, but even in the milder cases this enlargement is not entirely absent. This must be proved by percussion, and the spleen can be palpated clearly only in those cases in which heart weakness has existed for a long time along with the endocarditis. The friction-sound which is heard during the respiratory movements of the organ, whenever the capsule is involved in an inflammatory process, may be of importance in diagnosis. This friction-sound may be heard, not at all infrequently, even in mild cases of general infection, and even in those instances in which an increase in the area of splenic dulness cannot be demonstrated with certainty. Infarcts of the spleen, as a rule, do not show any special signs, and one must be very much on the lookout for these in order to make a diagnosis of their possible presence. [Suddenly forming infarcts of 2 cm. diameter or more can very often be recognized by pain and tenderness over the spleen. They are often useful in prognosis, with reference to embolisms in other organs. —ED.]

But little is to be said of the liver. As we know, general jaundice may occur in severe sepsis, and is an almost certain sign of death. A slight yellow tinge in the conjunctiva is found at times in the milder infections, but is not of importance. The signs which are observed in the liver in cases of heart weakness correspond to the degree of involvement of the heart muscle in the pancarditis.

The friction-sound over the liver has about the same value as that over the spleen, but I believe that the former is somewhat less frequently observed.

If dyspeptic signs be regarded as evidences of functional disturbances in the stomach, then we must say that this organ is almost regularly involved in endocarditis. Loss of appetite, which may increase to a positive disgust at the sight of food, is a symptom which occurs with especial frequency. I have observed it even in those cases in which there was but a slight general involvement of the body. In cases of recurrent endocarditis, accompanied by severe disturbances in the heart's action, a true gastric catarrh and circumscribed hemorrhages of the gastric mucosa are found in the cadaver—for example, in case XIV.

Severe diarrheas occur in some cases (during the entire second week of the disease, in case XI), but, on the whole, are rather a sign of general severe septic infection, especially of puerperal infection, in which the endocarditis is not a very prominent element. I cannot give general rules as to the intestinal function in chronic endocarditis, but usually constipation is found. In some instances, however, extensive changes occur in the intestines, as, for example, in the following case:

CASE XXIV.—A man, forty-nine years old at the first admission, June 10, 1882, died on January 5, 1885. I shall give only a few short notes on the first period of the disease: The illness began with dyspeptic symptoms of which the patient had complained at frequent intervals. The attack set in after an indiscretion in diet, beginning with chills and severe headache. The fever was very irregular, rising to 39.9° C. (103.8° F.). The spleen was swollen, maximum, 14 by 9 cm. At the be-

ginning of the second week of the disease there appeared inflammatory edemas of the skin, the subcutaneous tissue, and the muscles, accompanied by severe pain. These edemas involved various parts of the body, and usually disappeared within a short time. They persisted for a longer period in the region of the left kidney and in the right leg. It is probable that thrombi had formed in the smaller vessels of the involved muscles. There was also pain in several of the long bones, but only for a short time. The joints were but slightly involved. A variety of erythemas appeared from July 10th to August 15th.

The heart action was at first excited, and later on became weaker and weaker and very irregular. Small amounts of albumin appeared for a short time in the urine. The patient was discharged on August 20th. At that time the signs of heart weakness had not disappeared entirely, but no valvular lesion could be made out. During the following year this man could not do his full measure of work, although he was constitutionally very strong. He continued to be troubled with dyspepsia, vertigo, and vomiting. From time to time his right leg would swell and the eruption would appear on the skin. The patient himself found out that the heart was severely affected, and, especially in the last part of the year, he often suffered from a recurrence of "weakness."

Second admission, from December 3, 1884, to death, January 5, 1885:

The affection of the heart was well marked. The area of dulness had increased but little, and there were no characteristic signs on auscultation that pointed to a valvular lesion. The heart-sounds, however, were very faint, especially the aortic, and the pulse was small and usually slightly irregular. There was, in addition, a marked feeling of weakness, and, not infrequently, fainting spells, while on several occasions there were severe attacks of stenocardia.

There was slight but persistent elevation of temperature (38.6° C. (101.4° F.), maximum). There were scattered petechiæ on the skin. On December 10th there was a severe attack of cardiac asthma, which was accompanied by a severe pain in the left renal region, radiating toward the sacrum and into the left arm. The pain became more severe on pressure over the region of the kidney, and on the left side on pressure beneath the ribs. The pain gradually disappeared until the fifteenth. Beginning with December 17th there was vomiting and pain in the stomach, recurring in varying degrees of severity almost daily, and accompanied by increasing loss of appetite.

Beginning with the evening of January 3, 1885, the patient vomited mucus, bile, and then blood almost every half hour. The abdomen became swollen and increasingly painful. On the afternoon of January 4th, blood also appeared in the stools. The patient died toward midnight on January 5th, with severe signs of collapse and extremely acute pains in the abdomen.

The autopsy, performed twelve hours postmortem (Professor Ziegler), showed the following (abstract): On opening the abdomen that cavity was found to contain a dark, reddish-black fluid and to exhale a gangrenous odor. The loops of small intestines protruded and were diffusely red. Three loops in the left side of the abdomen were dark reddish-black in color; their walls were hemorrhagically infiltrated, and their mesentery, which was rich in fat, was dark reddish-brown and showed a number of hemorrhages. The omentum was rich in fat, was rolled up, and showed enlarged veins, but no infarcts. The veins from the infarcted area itself, as well as from the neighboring portions of the intestine, were filled with soft, dark, reddish-black thrombi, which were slightly adherent to the venous walls, and which showed in their central portions dark, brownish-red, soft, pultaceous masses. No thrombi were found in the infarcted area in the branches of the superior mesenteric artery, at least not so far as one could follow them with the scissors. The large arteries branching from the abdominal aorta were also free. The main trunk of the portal vein contained fluid blood, but adherent thrombi were found in the main branches of the right lobe of the liver. These thrombi consisted externally of a dark, grayish-red mass, and in their centers of a yellowish-red soft substance.

The wall of the intestine in the infarcted area was thickened, and the mucosa and submucosa were hemorrhagically infiltrated. The intestine contained reddish-black blood, and the intestinal contents, even outside of the infarcted area, consisted largely of blood.

The stomach contained a little bloody fluid. Its mucosa was slightly reddened, but showed no infarcts. Profuse hemorrhages were present in the duodenum, and also, mixed with a small amount of feces, in the colon. The spleen was quite firmly adherent to its surrounding tissues and was 16 cm. long by 8 cm. wide. Under the adhesions its periphery showed cicatricial foci with caseous contents. In one of the veins there was a comparatively fresh thrombus with a soft center.

The left suprarenal capsule was hemorrhagic in its medullary substance, and

there was hemorrhagic infiltration of the peritoneal tissue. Nothing of importance appeared in the kidney except a small scar on the left side, due to a healed infarct. The liver was anemic, yellowish-brown in color.

The heart was, on the whole, flabby, only slightly dilated, and did not contain any old clots. On the right side its muscular structure was flabby, without considerable changes; nor did appreciable changes appear in the valves. On the left side the muscle-fibers were remarkably flabby, and on section appeared moist and yellowish-brown in color. The aortic valves showed but unimportant thickenings at their nodules and a slight thickening at the base of the right flap. A small gelatinous vegetation appeared on the posterior flap, below the nodule. Some of the tendinous cords of the mitral were slightly thickened. The flaps were not shortened, and the auricular side of the flap at the line of junction showed a smooth thickening, upon which was found a small vegetation, consisting of five minute nodules. The posterior flap showed a small patch of granulation, and another little growth was noted at the inner surface, where the flap passed into the endocardium. The endocardium of the left auricle was markedly opaque and white. The coronary arteries were dilated, but showed no special changes.

The lungs showed congestion and edema, but no foci. Microscopically (Professor Nauwerck) the heart (left ventricle, sections through the entire thickness of the wall) showed diffuse but slight cloudy swelling. No fatty degeneration was present, but there was a slight increase in the pigment of the heart muscle. There were no small-celled inflammatory infiltrates. The vessels were markedly dilated, and there were small isolated hemorrhages. No germs were found either in the heart or in the mesentery (Gram's stain).

It was clear, even after the first admission, that there was septic infection in this case. This patient was seen at a time when cases of cryptogenetic sepsis were quite frequently seen in our hospitals. Without any clear signs of an involvement of the valves, the heart weakness became marked from the first, and continued as the case progressed. The autopsy showed that the endocardium of the left auricle must have been deeply involved, even at that time. The anatomic examination of the heart-muscle gave a positive result, but was this sufficient to explain the great deficiency in the work of the heart?

The venous thrombi which led to the formation of infarcts in the intestine could not be interpreted otherwise than as a marantic thrombosis. It is not clear, however, why these thrombi should have occurred in the intestine, and why there were no peripheral edemas or other signs of heart weakness. [It seems more rational to assume that the thrombi were septic. The technic used was too rudimentary to warrant the exclusion of sepsis.—Ed.]

While the patient had been suffering from dyspeptic symptoms for years, no anatomic cause could be found for these signs, and we gain nothing by saying that the stomach or the gastro-intestinal tract was a place of least resistance in this patient.

BLOOD. GENERAL NUTRITION.

Great pallor, weakness, and extreme emaciation may occur at the beginning of an endocarditis, even when the signs of a severe infection are absent. This is very frequently seen, but very often we cannot understand why it should be so. It was impossible for me to pursue the necessary investigations, as it is only rarely that I get an opportunity to weigh the patients in the Policlinic, much less to perform experiments in metabolism.

To begin with, we must know that each case must be tested for itself, as we are dealing with a great variety of germs which may have very different effects upon the blood and the general nutrition. Especial interest is attached to the cases in which the infectious general disease falls into the background, and in which but slight temperature elevations

are noted, while the heart's action, although less vigorous than normal, cannot be said to be actually weak. In case IV, for example, in which these conditions obtained, the anemia and the emaciation were pronounced and lasted for a considerable time.

The behavior of the blood in acute articular rheumatism has been described by Pribram.* This author reports, from his own observations, that, as a rule, the number of red corpuscles and the percentage of hemoglobin (method of Fleischl) are not so low as might be supposed from the marked pallor of the skin and of the visible mucous membranes.

This is not so in the cases of heart disease which are accompanied by sepsis. Romberg† reports an interesting case which may be quoted as an example, although, unfortunately, only a short summary of it is given:

"In two cases, I have known the cryptogenetic sepsis which accompanied malignant endocarditis to end fatally in the course of several months, under the clinical appearances of severe anemia. Judging by the examination of the blood, by the palpable splenic tumor, by the enlargement of the liver, and by the fever which was continuous in one case and interrupted by intervals of a week's duration in the other, we were inclined to consider these cases as instances of that group of diseases which is known as pseudoleukemia. The signs on the part of the heart (dilatation, mitral insufficiency) were interpreted as evidences of anemia." Only the autopsy showed the true character of the disease.

[In a cryptogenetic case with no stainable germs in the vegetations, but with streptococci in the late splenic infarcts (blood culture during life negative), I made a correct diagnosis five months before death, the previous diagnoses having been typhoid fever and malaria. But in the further course there was grave doubt as to the correctness of the diagnosis, since there was no change in the heart,—soft systolic murmur without enlargement, no arthritic or skin signs, very low irregular fever,—but anemia and gradual enlargement of liver and spleen, the latter without pain until just before death. Splenic anemia and Banti's disease, tuberculosis and biliary infection, were considered. In the two weeks before death pain in the spleen, later petechiæ, and slight convulsions caused a more distinct picture. The alterations in the heart were almost exactly like those in one of my (streptococcus) cases of pseudomalarial endocarditis. In some cases pernicious anemia has been simulated.—Ed.]

Grawitz‡ pursued detailed investigations on this subject. His chief conclusion was that a marked diminution in the specific gravity of the blood takes place in septic diseases, which is on the average much more pronounced in these cases than in other infectious diseases, and develops within a shorter time.

Both the red blood-cells and the serum of the blood participate in this change. As positive proof of the rapid destruction of red blood-cells of which he speaks, Grawitz cites a case of puerperal sepsis in which the number of red blood-cells fell to 300,000 within the first twenty-four hours, and the total residue of the blood on drying was only 14.5 per cent., instead of 21 per cent. or 22 per cent., as normally found. This observation was made in a patient in whom abortion had been roughly brought on, and who had been infected with staphylococci during the operation. The diminution in the number of red blood-cells and of the dry residue was so marked in all cases that there was no doubt as to the correctness of the author's conclusion. Whenever the dry residue of the

* *Loc. cit.*, note table on p. 213. † "Handbuch," p. 981.

‡ Résumé in his "Klinische Pathologie des Blutes," Berlin, Otto Enslin, 1896, p. 265. The entire literature of the subject is there reviewed.

blood as a whole became less than 15 per cent., every patient observed died.

The serum also became considerably thinner in these cases. Grawitz attributes this to the fact that the metabolic products of the pus-cocci exercise a lymphagog influence, that is, they produce an absorption of the tissue-fluids into the blood, such as has been demonstrated by Heidenhain in the case of the extract of leeches.

The following table is taken from the dissertation of Roscher,* who worked under Grawitz's direction:

Name.	Age, Years.	Day of Disease.	Date of Examination.	Temperature.	Number of Blood-cells in Thousands.		Dry Residue, Percentage.	
					Red.	White.	Blood.	Serum.
Mrs. H.	30	Middle of November, 1893	January 4, 1894	38.0° C.	2575	16.0	14.05	7.24
Girl, L.	28	End of October, 1893	November 15th	38.2° C.	2750	8.8	11.33	7.02
			November 16th	36.8° C.			10.58	6.84
Laborer W.	32	May, 1893	November 8th	39.2° C.	4400	16.8	16.48	9.21
			November 9th	38.6° C.		12.0	15.78	9.33

Three cases of endocarditis are here reported. In each instance the affection was recurrent and was proved at the autopsy to have been present. In the last case the affection was caused by the pneumococcus, which, however, did not seem to be very virulent. Further investigations are necessary to clear up this point.

The number of leukocytes need not be necessarily increased. However, opinions still differ on this point.

[In cases without other causes of leukocytosis the latter gives important aid. But in many cases there is no leukocytosis or a very slight one. "Sometimes pyogenic cocci can be cultivated from the blood, and, if present, may be of the greatest value in a diagnosis always difficult to make. Blood cultures should never be omitted" (Cabot). Besides the pus-cocci and the gonococcus, the Bacillus aërogenes capsulatus (Gwyn, 1900) has been so cultivated.

The opsonin method offers the prospect of great assistance in the diagnosis of endocarditis.—Ed.]

DIAGNOSIS.

Whoever wishes to recognize endocarditis, which always appears as a local feature of a general disease, must, in the first place, determine whether there are signs indicating the presence of such diseases as are known to involve the heart. These affections have already been mentioned above.

* "Blutuntersuchungen bei septischem Fieber," Berlin, 1894.

I should like to lay particular stress upon the discussion of the diagnosis of those cases which begin insidiously and pursue a slow course. In the great majority of instances these are produced by septic infection. In its mild forms the latter offers no characteristic signs, or but very few such evidences that it can be made out at first sight.

I must again emphasize here that I am standing independently in my views on this question. I can vouch for the trustworthiness of my observations, but whether the same observations can be made everywhere with the same frequency, and whether the conclusions which I have drawn from them apply everywhere, are quite different questions.

One thing is certain: All infectious diseases, without any exception, may vary as to their clinical manifestations, both as regards the extent of the general disease and as regards the more or less severe involvement of various organs. These variations are so great that it is impossible to lay down hard and fast rules for this class of diseases as a whole from the limited experience of a single individual. This applies especially to those cases in which the effect of the disease-producing germs is weak. In every epidemic we find cases varying from the mildest to the most severe forms. Scarlet fever is, perhaps, the best example.

On the basis of my observations, as discussed above, I shall briefly summarize the important points in the diagnosis of endocarditis.

1. GENERAL SYMPTOMS.

The Behavior of the Temperature.—Here we must pay attention, not so much to the absolute values, as to the distribution of the temperature over the twenty-four hours, and the irregularity of the temperature-curve, which in healthy persons corresponds so closely to the time of day. We have seen that this may occur both in severe and in mild cases. We also find that in patients who have been kept under observation for a long time, rises in temperature may occur for a shorter or a longer time, which often cannot be traced to any cause. These changes in the temperature of the body are certainly not pathognomonic signs. The same phenomena may be seen—in fact, very frequently—in slowly developing tuberculosis.*

The septic diseases which are complicated with endocarditis are more frequently mistaken for tuberculous diseases with slight local symptoms than for any other maladies. We must always be on the lookout for the possibility of a tuberculous process; especially so, because another set of symptoms is common to both tuberculosis and sepsis: namely, the more marked loss of strength and flesh, the greater pallor of the skin and of the visible mucous membranes, all of which signs are more markedly developed in endocarditis and in tuberculosis than is accountable from the other clinical signs; that is, the fever, etc.

These signs must be utilized cautiously, of course; for the extent of general disturbance which is produced by a disease in each individual varies to a very great degree. The physician who has been treating the patient repeatedly is more apt to be in a position to form an opinion. At all events, it must be admitted that no one can guarantee that nothing else is the matter with the patient except the disturbances which have been recognized.

* Dennig, "Ueber Tuberculose im Kindesalter," Leipzig, F. C. W. Vogel, 1896, S. 187.

2. LOCAL MANIFESTATIONS.

Among these I shall mention, first, cardiac weakness and its results: cold fingers and toes, nose, and ears, which are seen when no other reason exists externally for an increased radiation of heat. When the patient is allowed to stand up or to sit with his arms and legs in a dependent position, the skin of these parts rapidly grows cyanotic. Both cold and cyanosis are observed earlier than the increased irritability of the heart, and are seen before the onset of that irregularity which finds expression in a change in the pulse within a few minutes, or even within fractions of a minute, and much earlier than true irregularity of the pulse-rhythm. Unrest or excitability of the heart usually follows these early phenomena, or may appear at the same time. The two classes of phenomena occur simultaneously, especially when the heart is quite markedly involved. Some attention must also be paid to the complaints of the patient—oppression in the epigastrium, anxiety, and palpitation of the heart.

Later on, changes which may be demonstrated through physical examination appear upon the scene. In most instances these, at first, do not show directly the presence of an affection of the endocardium. Even when such an affection is present, the changes in the myocardium which are developed at the same time are more prominent clinically. This applies also for those valvular lesions which manifest themselves by changes from the normal auscultatory sounds. We know that the heart muscle is, to a great extent, responsible for the closure of the valves, and is not less important in the quality of the tone which arises during the contraction of the valves.

It has been taught, correctly, that the following principle is universally applicable: Murmurs which do not depend upon structural changes in the valves vary constantly in their intensity and quality. There is no doubt that their variations depend upon the variable activity of the heart muscle. This irregularity in the work of the cardiac muscle may also produce another effect: if we can follow the development of an endocarditis from the beginning, we can often note that it is impossible to state definitely the exact place where the process is localized. At times we hear systolic murmurs more clearly and more distinctly over the apex of the heart; at other times, over the aorta. There are days when either the first or the second location is distinctly favored. One thing always remains, and that is, that the murmurs are almost always heard over the entire heart. Only in the course of time after weeks or even months our doubts are dispelled and we can recognize with certainty which particular valve has been affected. We must, therefore, be somewhat cautious in expressing our opinion as to the localization of an endocarditis.

The murmurs are usually heard during the systole of the heart. They must necessarily be systolic, in so far as they depend upon the muscular contraction, and even those murmurs in which the insufficient closure of the bicuspid or the tricuspid valves play a rôle must needs also be heard during this phase of the cardiac cycle.

It may happen, however, that in a case of endocarditis which later on develops into an insufficiency of the semilunar valves of the aorta nothing but a systolic murmur is heard for a long time, is transmitted into the large vessels (the subclavian and the carotid), and is accompanied by a palpable thrill. In rare cases—as, for example, in my case VI—the au-

topsy reveals the cause of this phenomenon. In this case only one of the aortic leaflets was diseased, but it was so markedly thickened that it projected into the lumen of the vessel and blocked the same, thus offering a sufficient cause for the development of a systolic murmur.

In other cases a dilatation of the aortic lumen beyond the valves may develop early. In case of permanent insufficiency of the valves, with its consequent hypertrophy of the left ventricle, this aortic dilatation is by no means rarely seen, and in such cases the eddies which are responsible for the murmur and the thrill occur within the dilated artery. The dilatation, however, cannot be recognized at this time, although it may appear later—as, for example, in my case XIII.

It may also happen that a systolic aortic murmur and a transmitted thrill may be present, although no cause for these signs can be found either in the valves or in the aortic lumen. The autopsy will prove this. Possibly, in such cases, we may be dealing with a phenomenon attributable to the heart muscle itself. It is conceivable that the fibers which compress the beginning of the aorta during the systole may, for a time, exaggerate the contraction of this vessel. It is noteworthy that in these cases the tracing of the pulse, even at the radial artery, shows at times curves which resemble the pure pulsus celer, and at times curves with broad apices, such as are seen in [some—Ed.] cases of stenosis accompanied by insufficiency of the aortic valve. My observations, however, do not permit me to express a definite opinion on this subject.

Diastolic murmurs which do not depend upon tissue changes in the valves or orifices are certainly rare. However, I do not care to agree with von Leube,* who strongly advises that "the diastolic murmurs should be omitted from the category of accidental murmurs, at least for the purposes of diagnosis." The possibility of mistaking a diastolic murmur over the aorta for a blowing sound transmitted from the veins of the neck, to which Leube called attention, is perfectly familiar to me, but I do not believe that I have ever made this error. Therefore, I cannot regard as correct the conclusion which must be drawn, that "diastolic heart-murmurs are trustworthy signs of endocarditis."

We must admit, without reservation, that in exceptional cases—as, for example, when a valve is torn off or is extensively perforated—the sudden appearance of the murmurs which follow the lesion and the uniform continuance of these sounds offer an almost certain proof of the presence of an endocarditis which can be definitely localized. It is also true, however, that in most cases we cannot base an opinion on the evidence of the ear alone; for the ear cannot distinguish which of the signs depend upon an involvement of the muscles and which are due to the involvement of the valves and orifices.

How about the changes in the area of dulness which appears on percussion? These changes are only to be considered in the diagnosis when they are actually produced by hypertrophy. The other signs of hypertrophy must also be present, in addition to the enlargement of the boundaries of the heart. Therefore, we are dealing once more essentially with the heart muscle.

If the heart muscle is seriously involved in cases with a tempestuous course, dilatation of the heart may occur sometimes within a very short time, and may increase in extent until the patient dies. When the disease

* "Specielle Diagnose der inneren Krankheiten," Leipzig, F. C. W. Vogel, first ed., p. 14, 1889.

progresses more slowly, then the dilatation can only be demonstrated in the course of a number of days. The dilatation may also disappear and reappear. Thus the case may run on for some time. Although the work of the heart may apparently be stormy and vigorous in cases of dilatation due to weakness of the muscle, yet when the hand is placed upon the breast of the patient, we know at once that this is not so. Other signs which are also found in such cases are a faint character of the heart-sounds or of the murmur, and a volume of the pulse which is distinctly out of proportion to the apparently vigorous heart's action. These simple and easily recognized signs confirm the diagnosis of the dilatation of the heart, and permit us to exclude the presence of hypertrophy.

Again, we must remember that a true hypertrophy of muscle-fibers requires some time for its development. It cannot appear in one day. Inasmuch as the myocardium is usually involved in addition to the endocardium, the development of a muscular hypertrophy is interfered with from the start; for the circulation in the heart is easily disturbed, and without a sufficient nutrition, no hypertrophy can take place.

We must conclude, therefore, that the hypertrophies of the heart muscle which indicate the presence of a valvular disease or of an orificial stenosis cannot develop so long as the endocardial inflammation which produced the latter is present in marked degree. Muscular hypertrophies are, therefore, useful in the diagnosis of a previous endocarditis or, let us say, more correctly, of an endocarditis which is inactive for the time being. This sufficiently explains the very limited application of hypertrophy of the heart muscle in diagnosis.

When an endocarditis which is localized at the aortic orifice produces an anatomic change which results in the inability of the semilunar valves to close perfectly, then a "pulsus celer" develops. When such a pulse suddenly appears and is really well marked, and when it continues to be present for some time, a diagnosis of aortic endocarditis may be made with certainty. The assumption is, however, that the heart muscle is sufficiently strong to propel a greater amount of blood into the aorta. In such cases the diastolic murmur is but rarely absent. We have here, therefore, two signs, which, when present simultaneously, are evidences of an insufficiency of the aortic valve. When the "pulsus celer" is but slightly developed, then, as Romberg correctly points out, it cannot be used for diagnostic purposes without further evidences.

The uncertain and indefinite nature of the information which we gain from percussion and auscultation over the heart is best illustrated in the cases of recurrent endocarditis, which occur in the presence of old valvular and orificial lesions. In these cases the physical signs on percussion and auscultation are almost useless in diagnosis.

An important fact in such cases is the occurrence of a fresh pericarditis, which does not by any means necessarily occur with a free exudate. It is enough, for diagnostic purposes, if we hear friction-sounds which are, without doubt, due to a pericarditis. I have elsewhere pointed out (p. 225) that this is not always easy to determine, and that pericardial friction-sounds are often distinguished with great difficulty from pericarditic friction-sounds. If a friction-sound which coincides with a movement of the heart is heard in a case in which we have reason to suspect endocarditis, it is certainly more probable that a pericarditis exists than when we are only face to face with indistinct evidences of muscular disturb-

ances in the heart. Still, we must remember we are here dealing only with probabilities.

I have had occasion to observe this once more recently. There was a marked septic general infection; the heart had become dilated considerably within a short time. There was a friction-sound concerning which nothing definite could be said, and which might have been pericarditic or pericardial, but was very loud, sharp, and always purely systolic. It showed about the same intensity from its first appearance to the patient's death. It was heard most distinctly over the apex. I could not consider it as an endocardial sound. At autopsy it was found that the endocardium, especially the valves, appeared entirely normal to the naked eye, but there were fresh fibrinous exudations in the form of bands within the pericardium. The infection was due to the invasion of staphylococci.

To summarize: (1) The diagnosis may be made most accurately through the study of the muscular involvement of the heart. The latter offers signs which may simulate endocarditis, and which may render very difficult a decision as to whether or not an endocarditis is present. From the signs developing in the heart itself, one can detect a fresh endocarditis with certainty only in exceptional cases. One can do this only when the inflammation in the endocardium has involved one valve or one orifice, and has produced there some well-marked and permanent changes.

(2) As regards *affections of other parts of the body* which are produced by emboli, we may distinguish, *first*, the larger emboli which become adherent in the wider arteries of the systemic circulation. These point with great probability to an endocarditis in the left portion of the heart. This applies, however, only in those cases in which the heart is not so markedly weakened that thrombi form within its cavities. Naturally, we must also exclude the possibility of a thrombus-formation within the affected artery itself. Small abscesses do not prove anything in favor of the presence of an endocarditis, as they may just as well be produced by germs circulating in the blood.

(3) Every feature of septic infection, as such, may be indirectly utilized in the diagnosis of a diseased condition of the heart induced by the infection in question. Among the manifestations which often accompany even the mild forms of septic pancarditis with pronounced involvement of the endocardium we may note the following:

(*a*) Disproportion between the pulse and the respiration, the latter being, as a rule, considerably increased in frequency. The fewer local changes may be detected in the respiratory passages, the pleura, or the muscles of respiration, the more important is the significance of this sign in diagnosis.

(*b*) Swelling, redness, and sensitiveness of the joints, as well as pain on pressure over the bones, especially over the long bones.

(*c*) Friction-sounds over the spleen, which is not always necessarily enlarged, and also similar sounds over the liver. The pleuritic friction-sounds which may be present are of less value in diagnosis, because their significance varies.

(*d*) Hemorrhages and inflammatory changes in the skin.

(*e*) Changes in the eye, especially hemorrhages into the retina, may possibly be connected with mild septic infections. In some instances there are isolated petechiæ in the mucous membranes and in the skin. As a rule, these are only found in the severer cases, and in these they are of diagnostic importance.

(*f*) It is well known that we must always pay attention to the heart in patients with chorea. The other signs on the part of the nervous system, *i. e.*, the cataleptic phenomena and the paradox phenomenon of Westphal, as well as the multiform symptoms on the part of the diseased muscles, also deserve full consideration in the diagnosis of endocarditis.

(*g*) The demonstration of bacteria in the blood* is not always possible, even in the severe forms of sepsis, and even after repeated examinations. It is scarcely to be expected that a positive result can be obtained in the mild cases.

I shall not enter into the discussion of the differential diagnosis. In this we must consider, essentially, those infections which may be confounded with the septic processes and, therefore, the differential diagnosis of endocarditis due to sepsis may be better treated in the general consideration of sepsis itself.

Clinical observation cannot determine the variety of bacteria which produce endocarditis from the mere picture of the disease. An examination of the blood, however, can give this information, or else an investigation of the contents of a purulent focus, when such a lesion is present.

We have seen that not infrequently more than one microbe is active. For the present we must say that, practically, the determination of the presence of any special germ is not of any great value. When, however, serum-therapy proves curative, a much greater interest will attach to this question.

The diagnosis of an endocarditis is not easy. We meet everywhere so many gradations in the significance of this or that symptom, that only extensive experience can find the right interpretation. Every physician must pay for such experience, and the older he gets, the more keenly he will feel that, although he has learned a few things, he has still much to learn in order to walk with real assurance upon this slippery path.

PROGNOSIS.

In considering the prognosis of endocarditis we must first determine the character of the infection which has become localized in the heart through the settlement of bacteria which produce it, and which has so severely affected the body as a whole that the local cardiac affection is relegated to the background. The course of the general disease is naturally influenced by the circumstance that the heart's action is directly impaired by the general process. Much depends upon a sufficient circulation of the blood. But the disturbance in one of the organs which are necessary for the preservation of life may be so great from the start that it alone may lead to a fatal termination.

If such a vital lesion does not take place, and if recovery is possible, then the heart becomes the chief factor in the affection. The better this organ is able to continue the circulation under the altered conditions, the better it is for the patient. The less perfectly it is able to do so, the more unfavorable will be the prognosis. When the heart itself is severely affected, then a fatal outcome is probable, which perhaps might have been avoided otherwise, even if the general infection had been equally severe.

It is well to use the expression, "diminution of the working capacity of

* See Grawitz, "Klinische Pathologie des Blutes," S. 261 *et seq.*

the heart." Although this decrease is chiefly caused by deficiency in the muscular tissue of this organ, yet changes in the valves—detachments, perforations, or extensive effusions into the pericardium—may produce a considerable diminution in the functional efficiency of the heart.

The prognosis of an *acute endocarditis* may be made from the following viewpoint:

The more severe the infection itself, the more prominent the part taken in it by the affection of the heart, the worse will be the prognosis. Malignant endocarditis is usually said to have an absolutely fatal prognosis. Such a statement savors of that wisdom of the past which said of a convalescent that he had had "nerve fever," while if he had died, the diagnosis would have been "typhoid."

Experience shows, however, that patients may recover even when the cases seem to be extremely severe, and are accompanied by well-developed signs of septic general infection, as well as marked involvement of the heart. A valvular lesion which develops fully afterward, leaves no doubt as to the correctness of the diagnosis of endocarditis. This occurs but rarely, however, and I have seen it only in a single case, in which eighteen years have now elapsed since the first attack. Frequent relapses occurred, but these pursued a comparatively mild course. O. Fräntzel* reports two such cases observed by himself, one of eleven and the other of twenty-two years' duration.

Possibly the various bacteria have different degrees of importance in the prognosis. The malignant endocarditis produced by gonococci is said to be more favorable than that due to other germs. According to Romberg,† nine cases recovered out of twenty-one, but he doubts the correctness of the diagnosis because no valvular lesion remained, and he is perfectly right in his conclusion. Possibly the pneumococci, the vitality of which within the human body is always rather limited, may give a better chance for the recovery of a heart affected by their presence provided the patient withstands the first charge. It must be added, however, that pneumococci alone, and not in combination with streptococci and staphylococci, must be localized in the heart in such favorable cases. [J. B. Herrick has analyzed a number of reported cases of healed ulcerative endocarditis with three new ones. He thinks the prognosis is not as hopeless as in general if the infectious agent is the pneumococcus or the streptococcus.‡ In the discussion following the reading of the paper several cases of recovery were mentioned by Thayer, Kinnicutt, and Shattuck.—Ed.]

The prognosis of an endocarditis accompanying acute articular rheumatism depends upon the latitude with which the term rheumatism is used (compare p. 182). If we content ourselves with the old-fashioned diagnosis, then the prognosis is favorable in the large majority of cases, so far as the patient's life is concerned. A very extensive experience has taught that there was formerly a sharp contrast between the prognosis of rheumatic benign endocarditis, which anatomically corresponded to the verrucous type, and the prognosis of septic, malignant, ulcerating endocar-

* "Die Entzündungen des Endocardiums und des Pericardiums," "Vorlesungen über die Krankheiten des Herzens," Bd. ii, Berlin, Hirschwald, 1891, S. 36.

† "Handbuch," *loc cit.*, p. 982.

‡ "Medical News," September 6, 1902, and "Transactions of the Association of American Physicians," 1902, vol. xvii, p. 468.

ditis. This statement was true to its full extent in former years, but we must take quite a different view of this question at present.

Litten* has recently published observations concerning a type of endocarditis which occupies a middle ground between the two varieties,—let us retain the expression,—the rheumatic and the septic.

The primary disease itself, the sequel of which was endocarditis, at first always yielded to the action of salicylic acid. Yet the endocarditis which accompanied it always led to a fatal ending, with or without a destruction of the valves. Hemorrhages into the skin and into the retina occurred at different intervals, and effusions of blood appeared in the serous membranes, in the brain, and in the pelves of the kidney. Hemorrhagic nephritis was commonly observed in these patients, but the most important and characteristic feature, according to Litten, was that no suppurations and no thrombophlebitis could be found anywhere, either during life or in the cadavers of these patients. He found an exceedingly small coccus, which, obtained in pure cultures, was pathogenic in mice, guinea-pigs, and rabbits, but lost its extreme virulence in the course of about a week.

I consider these data important because they seem to show, I think, once more that the clinical picture of acute articular rheumatism has changed greatly in our own days. Therefore the prognosis of a rheumatic endocarditis must be made with caution for the present.

What is the prognosis of an endocarditis which has run its course? In the first place, as regards the heart itself: A complete recovery is possible, including a disappearance of the local tissue changes. Endocarditis localized in the walls of the heart, it is true, usually leaves marked opacities and thickenings, but these are at times of but little importance, because they do not have much influence upon the activity of the heart. The fact that parietal endocarditis may also have severe consequences is demonstrated by the observation of Nauwerck, which has already been cited repeatedly in previous pages.

When the endocardial changes are located in the valves and in the orifices, the heart's action is more markedly interfered with. The degree to which this interference takes place depends upon the extent of the lesions of the cardiac mechanism, and, furthermore, on the fact as to whether the heart muscle is able to cope with the newly created conditions and, if so, to what degree. In these cases even severe lesions may be compensated and the work of the heart may be kept up to the standard to a certain extent. (Further details will be found in the article on Valvular Affections.)

My case I offers an example of this kind. Yet this case also teaches the severe danger which always threatens one who has ever passed through an endocarditis—the danger which lies in a possible recurrence of the disease, including both the general infection and the local malady. This danger is so great that the physician, without exaggerating and only giving the known facts their due weight, must always make a doubtful prognosis in a case of endocarditis.

In the relapses, the musculature of the heart is certainly involved just as much as during the first attack, but the endocardium forms the starting-point—the focus from which the conflagration ignites once more and spreads. (Compare the description of the pathogenesis of recurrent

* "Ueber die Maligne (nicht Septische) Form der Endocarditis rheumatica," "Berliner klinische Wochenschrift," 1899, Nos. 28, 29.

endocarditis given above, p. 188). We can never foretell when a relapse may occur.

Romberg says that, in his cases of malignant endocarditis with sepsis, the disease lasted, on the average, fifteen months, and mentions correctly the fact that quite frequently the patients showed intervals free from fever of from four to six weeks' duration. These observations were made in a hospital. In patients who visit a policlinic one has an opportunity to make more prolonged observations, and, therefore, must deal with still longer intervals. The custom of terminology does not, it is true, allow us to speak of the duration of a disease when free intervals lasting for years follow the first attack, and then terminate in a recurrence of the malady. There is nothing to prevent us from considering the recurrent attacks as parts of the same disease, unless we can prove that a new infection was necessary in order to bring about the relapse. This question is still open, and every one is at present free to introduce the term "latent," here, just as has been done in tuberculosis.

Still another point must be discussed. It was formerly the custom to designate an endocarditis as malignant (ulcerating) only in those cases which occurred in one continuous attack and ran their course within a comparatively short time. In speaking of these cases, one usually mentioned that the form was "acute." Yet these malignant forms may extend over a period of months, as, for example, in a case reported by Lender,* where the disease lasted from November, 1858, to February, 1859. O. Fräntzel remarks, in general, that the disease may last for a period of many months.† Recently Ebstein reported the occurrence of malignant endocarditis with a long duration, and similar instances were reported by Romberg.

We need not hesitate, therefore, to look upon the designation of malignant endocarditis chiefly from the prognostic viewpoint, and speak of this form without paying any attention to the duration of the case. Yet it is necessary to add a special designation to this general term, and we must, therefore, distinguish in the customary manner an acute, a subacute, and a chronic malignant endocarditis. A distinction between malignant and "benign" must be applied only in so far as the general infection with its effects upon the several organs of the body occupies the center of the stage in malignant cases.

In the benign cases the chief feature is the interference with the heart's action, with its immediate effect—heart weakness. In these, as in all the processes of nature, we cannot establish a hard and fast scheme of nomenclature; for recurrent endocarditis shows that a case which is originally benign may become the foundation for a malignant affection.

It is scarcely appropriate to go into further details. In the prognosis of an individual case one must consider what particular organs are involved in addition to the general infection and the heart itself, and if so, in what manner the other organs are affected. The discussion of these questions belongs to the bedside.

TREATMENT.

The most important feature of the treatment of endocarditis in all its stages is *rest* for the patient and for his heart. If we possessed a specific remedy which would destroy the germs, or which could materially impair

* "Deutsche Klinik," 1862, p. 119. † *Loc. cit.*, p. 258.

their activity, then everything would be plain sailing. For the present we have no such remedy; for, as yet, we cannot hope to get any results even from serum-therapy. It is scarcely possible that a general "sepsis serum" will be produced. What particular germ is active in a given case? and, if there are several germs, as often happens, in what proportions are they mixed? Neither the first nor the second question can be answered for the present. One thing seems certain, that we cannot combat staphylococci with a serum obtained from streptococci, and even in cases of streptococcus invasion into the skin,—in erysipelas,—absolutely uniform success cannot be obtained with Marmorek's preparations.*

[It is too early to admit that sera can never be applied to the treatment of bacterial endocarditis. The results in the past have been disappointing, but the sera have not always been selected with reference to the germ present, nor has the last word been said upon the preparation of such sera. The practitioner should follow the progress of discovery with care. Soluble silver salts have been recommended by several, and are also worthy of further trial. Wenckebach† has reported two cases improved under collargol; Klotz,‡ one in which recovery followed three intravenous injections of 1 per cent. collargol solution in doses of 9, 8, and 7 centigrams respectively. Manges, who used the treatment in two cases without success, nevertheless recommends further trial, as the method is simple and safe.§ At present the most interesting field is that of opsonin therapy. Barr, Bell, and Douglas‖ report a case in which various sera were used without actual knowledge of the infectious germ, and without benefit. Signs of right-sided endocarditis came on. A streptococcus was found in an abscess in the foot. After some time a streptococcus was cultivated from the blood and a vaccine prepared. The first injection of 10,000,000 cocci was made with the index at 0.4. Improvement soon began, though interrupted by pleurisy and thrombosis of the left iliac and femoral veins. The details of the treatment it is hardly necessary to repeat. In thirty-two days the patient was convalescent. The right heart returned to normal size and the murmur and incompetency disappeared.—Ed.]

What shall we do in cases which are accompanied by very high temperature? If we could do so without injuring the patient, we should certainly attempt to lower the fever in such cases; for every one must admit that the work of the heart is interrupted by shorter pauses for rest when the temperature is high. It is true, however, that, as a rule, the temperature curve shows such marked remissions that the form of fever which is most to be feared, the continuous type, does not develop. Still, although the patient is seriously threatened by a temperature which reaches 40° C. (104° F.) and over, and lasting for some time, we can reduce the temperature either not at all, or, at the best, very little.

This is true even of the very cold baths, the use of which has been systematically pursued in the clinic of my late colleague Säxinger, especially in puerperal sepsis. No very marked success was obtained with these

* Cf. Lenhartz, "Erysipelas": Nothnagel Specielle Pathol. u. Therap., vol. iii, pt. 3, p. 89.

† "Therapie der Gegenwart," 1902, S. 65.

‡ "Deutsche med. Wochenschrift," 1902, S. 524.

§ "Medical News," December 13, 1902.

‖ "A Case of Infective Endocarditis Cured by the Inoculation of a Vaccine Prepared from Organisms Found in the Patient's Blood," "The Lancet," February 23, 1907, p. 499.

baths, and I have, therefore, not employed them in the septic cases which I have had occasion to treat. In many cases the sensitiveness of the bones, the joints, and the muscles precludes the lifting and moving of the patient which are necessary in the use of baths.

Almost the same statement may be made as regards salicylic acid and its preparations. Dennig* reports a temperature curve from the Policlinic which shows that slight influence is produced even by large doses of salicylates administered for a considerable period. In this case 1 gm. of sodium salicylate was administered every hour, and the temperature was taken at the same intervals. The average temperature during the acme of the influence of the drug was 38.9° C. (102° F.), while before and after its administration it was 39.1° C. (102.3° F.). The difference, therefore, was 0.2° C., and the course of the curve did not show much change.

This may be said to be the rule in most cases. We have gathered quite an extensive experience in this respect, at the time when acute articular rheumatism no longer yielded to salicylic acid. Antipyrin and antifebrin were tried only in a few cases, but with exactly the same failures. Nor must we expect much from quinin. Figs. 33 and 34 showed this very distinctly. We are in the habit, at present, of ordering for another purpose (see p. 303, below), 0.5 gm. of phenacetin at a dose, repeated, at most, four times a day, but no visible effect upon the temperature follows, even in the milder cases. It is true that the dose is not very large.

In most cases I do not attempt to lower the temperature, because this does not have much influence upon the outcome of the case, so far as my own observations go.

Some clinicians who are, in general, no great advocates of alcohol in the treatment of febrile diseases, employ this remedy in not very small quantities in sepsis. Romberg (p. 983) remarks, in speaking of the treatment of acute malignant endocarditis, without accurately defining his own position in the matter: "The usual treatment for septic diseases should be employed, especially generous amounts of alcohol (from 50 to 100 gm. of cognac, pure or mixed with eggs, and from one to two bottles of a strong claret or of a dry southern wine, per day)." I have not had occasion to use alcohol in the cases of endocarditis which came under my observation in any other manner than in that pursued by me in other diseases. I must emphasize, however, that large doses are indicated for a considerable time only so long as the patient has high temperature. A patient who is very feverish cannot be easily intoxicated and does not suffer from the bad effects and after-pains of intoxication. Yet a short fall in the temperature diminishes this tolerance, at times very markedly.

In one case of typhoid fever there was a marked morning remission, rather unexpectedly in the third week of the disease. The patient had been taking a considerable amount of wine daily and had not shown any cerebral symptoms. When I saw him in the morning I found him actually slightly intoxicated, although this had not been noticed by the clinical clerk, who had been tempted to make very strange diagnoses. A glance upon the temperature chart showed the cause of the patient's hilarity, and indicated the removal of the wine-bottle. Toward evening the temperature again rose, and the patient was given the same amount of alcohol as before, without any further effects.

This point must not be forgotten, especially because in septic diseases we not infrequently see rather unexpected remissions of temperature,

* "Septische Erkrankungen," S. 212.

and it is all the more important because the unpleasant effects of alcohol upon the heart may be very marked in the presence of low temperatures. In general, I consider it wrong to prohibit the use of alcohol if we suspect a probable endocarditis. Still, everything depends upon the amount which is allowed in each individual case. The fundamental principle is immutable; viz., when alcohol produces unnecessary excitement of the heart, it should be absolutely prohibited. The physician must be the judge as to whether this is necessary, and must act accordingly.

Personally, I do not believe that we can employ an effective treatment in endocarditis beyond what is known as "symptomatic therapeutics." Yet I shall not fail to quote the view of such a good physician as Oscar Fraentzel. He expresses himself definitely enough.*

"Quinin may be given in doses of from 0.5 to 1 gm. twice or three times a day, not exceeding 1 gm. at a single dose. When signs of improvement set in, the same doses of quinin are continued, but at less frequent intervals, according to the favorable effect upon the general condition. In some cases, accompanied by erratic chills, this treatment must be continued for many weeks."

[Fraentzel's advice comes down from the time when quinin was considered a specific for septic diseases. Sometimes marked falls of temperature follow its administration, just as they occur without medication, or under mild saline drafts, but having seen many cases of septic endocarditis in which the diagnosis of malaria had been made and quinin given in large doses and for long periods, I am convinced it has no good effect.—ED.]

Alcohol must be given in not too concentrated form, but in considerable amounts: "As a general rule, I should advise that such patients receive 100 gm. of a good red wine, not too rich in alcohol, every hour." The form of alcoholic stimulant, however, must be regulated in each individual case according to the habits and wishes of the patient.

After expressing the opinion that but little is to be hoped in cases with a tempestuous course, Fraentzel continues:

"I have advocated warmly this quinin-alcohol treatment, for the reason that I have found, on the basis of quite numerous observations, that even the subacute and chronic cases of malignant endocarditis end fatally in the great majority of instances, but that an incomplete recovery, that is, a healing of the acute process with a residue of a chronic valvular defect, is observed only in exceptional cases with other forms of treatment than that just described." Fraentzel certainly does not expect brilliant results, "but we see some successes anyway, and that must satisfy us in such a severe infectious disease and in the present state of therapeutics."

My own treatment is as follows: Whenever possible, I act directly upon the heart when necessary in the manner which has been described in the preceding section on Insufficiency of the Heart. A heart bottle filled with cold water, or, if the heart's action is very excited, an ice-bag, is the only measure which I order regularly whenever the possibility of developing endocarditis appears. A layer of flannel must always be placed under the ice-bag in order to avoid direct contact with the skin. The skin, however, must always be kept cool, and the water or ice must be renewed at the proper intervals for this purpose. The excitable character of the heart's action seems to be favorably influenced by the constant cooling of the skin, and the patients certainly experience a grateful sensation from these applications.

* *Loc. cit.* (see No. 80), p. 39.

If the heart's action is very weak, then we must not use cold, but may possibly employ local heat. I employ stimulants and digitalis in the same manner, and according to the same rules as in cases of heart weakness in general. We should seek, so far as possible, to avoid the loss of nutrition which is so prominent in endocarditis. So long as there are signs of a general infection, the food should remain fluid, and should be given in small quantities at frequent intervals. I prefer the infant foods to milk, without excluding the latter, however, as these foods offer an opportunity to vary the diet to a certain extent. Even when the worst symptoms are over, I avoid large single meals. The problem is to supply as abundant an amount of food as possible, according to the dietetic rules which obtain in each particular case, and to give this food in such a form that it is easily absorbed. Wine may also be given, but never in such amount or in such form that the heart is excited.

The use of stomachics in protracted loss of appetite does not seem to me to be of any value. The movements of the bowels must be watched. I do not think it is right to interfere from the start with the diarrheas which occur as one of the signs of septic general infection, unless these diarrheas be very severe. Frequently repeated and too profuse evacuations may become dangerous. We must not forget, however, that the old observation which considered "intestinal crises" as favorable signs was correct. I avoid, therefore, all interference with the intestine so long as possible, and therefore I do not try to induce a "derivation" through this channel. The experiences of obstetricians have shown what happens when other treatment is employed, when they tried to conquer puerperal fever by means of calomel and drastic cathartics.

We must not tolerate constipation, particularly when it is accompanied by an accumulation of gas in the intestines which interferes with the play of the diaphragm. I prefer, in these conditions, to use mild laxatives, such as the compound licorice powder, rhubarb, or the Friedrichshall bitter water, which is easily taken and is very mild in action. I prefer these remedies to enemas and injections, unless the patient is already accustomed to them, and even then it is advisable to use a certain amount of caution in ordering them.

The most careful nursing is essential in endocarditis. Especial attention should be paid toward keeping everything away that disturbs the patient's rest, while everything must be done to secure his comfort. The patient's position must be carefully watched, so long as he has pains in the joints, the bones, and the muscles. In this, a trained hand can accomplish a great deal of good. When the rest of the body is not disturbed by pain, the brain is able to rest, and then sleep, or at least slumber, is possible.

Here, in securing rest, phenacetin in the doses already mentioned (0.5 gm.) comes into play. I am not averse, however, to employing the ordinary direct hypnotics, which, it is true, must be given with conservatism and caution. They may be required immediately when there is marked chorea, but, as a general rule, I have only seen favorable results from doses of 1 gm. of trional and tetronal given to a sleepless patient every third, or at most every second night. This secures rest for at least a few hours. I avoid hypnotics, of course, when the heart weakness is pronounced. In such cases small amounts of sodium bromid may be ordered with profit, although they do not produce sleep in the proper

sense, but bring about a general relaxation. I am not accustomed to order more than 2 gm. per day.

I regard the effect of rest in bed as a factor more important than any other measure which we can bring to bear upon the course and termination of endocarditis. I am now not speaking, of course, of the severe cases with virulent infection. Owing to their sudden onset, they require rest in bed, anyway, but it is in the milder cases, with a less stormy onset, that rest in bed is to be insisted upon. In such cases the general condition of the patient is not so markedly impaired, and persons who are naturally resistant, and who pay less attention to their well-being than to the continuation of their daily work, will try to keep on their feet as long as possible. The presence of indistinct "drawing" pains, which occur sometimes here, sometimes there, which appear suddenly and disappear just as quickly, is the best proof that "rheumatism" is present. Every one who is looking for it, can remember an occasion when he might have caught cold. The physician is scarcely consulted, and home remedies are used. Thus matters will drag on, until finally the gradual loss of strength or a rapid advance of the disease shows the seriousness of the situation. I have seen, on several occasions, how such a neglected illness has led to death within a few days after the onset.

I make it a rule in my Policlinic that in every case of "rheumatism" the heart be carefully observed, and that rest in bed be ordered at once as soon as a connection with endocarditis is suspected. A few days suffice in order to clear up the matter, and to determine whether the observation should be continued further. Our patients have been content to abide by this rule, and have remained in bed as long as the physicians considered it necessary—long before the mutual benefit societies existed. Deaths which occurred in the manner just mentioned taught them to be obedient.

How long shall the patient remain in bed? In this I have grown more and more cautious as time has passed. A good criterion is the behavior of the temperature, provided one can command prolonged observations with three measurements daily.

I allow the patient to get up only when every deviation from the normal has been absent for at least two weeks. The sensitiveness of the bones and the joints must also have been absent for the same length of time, and eruptions must not have occurred within the same period. I lay less stress upon the nervous symptoms, and the continuation of the choreic movements does not come into consideration as regards the patient's rest in bed. The behavior of the heart itself, however, must be carefully considered. The general rule is that all signs of heart weakness must have disappeared entirely before the patient is allowed to get up. Slight irregularities of the pulse, however, are not of very great importance.

The safest way is to try an experiment. The patient is allowed to get up, or at least to sit for a few minutes with his arms and legs hanging downward. The presence or absence of cyanosis in the dependent limbs will show the state of the heart's vigor.

Even a considerable increase in the pulse-rate, which appears under these conditions or under the influence of other forms of excitement, is not necessarily to be regarded in the question of the patient's getting up, provided these irregularities pass away in the course of a few minutes.

Prolonged palpitation of the heart and cardiac excitement which occur without any cause prohibit the interruption of rest in bed.

The findings on physical examination must be carefully estimated. Older valvular defects are but exceptionally to be considered in deciding this question. Changes in the area of dulness, as determined by percussion, are scarcely met with at this time. They have reached a certain limit before, which remains constant to a certain degree, unless a severe involvement of the heart muscle favors the development of dilatations. These, however, are accompanied by a decrease in the cardiac function, and this is the most important element in deciding whether the patient should stay in bed or not.

It would be foolish to wait for a hypertrophy which might follow the dilatation, and to expect the shrinking of the area of dulness. The auscultatory phenomena need only be considered when they offer something really new, and this is very difficult to decide whenever older lesions are present.

The same may be said of changes in the valves or orifices which occur in the course of endocarditis in a previously healthy heart. It is advisable to give the heart muscle the opportunity to restore compensation by rest of the body under the most favorable conditions possible. One should not forget, however, that a certain amount of cardiac capacity is necessary for this purpose. It is easier to find out when attempts may be made to get up, and when the patient can begin to exercise by watching the heart increase its capacity for work than by percussion and auscultation.

The patient should be watched also in another direction, and if he is allowed to get up, it is best to test whether he is ready for the change or not in the following manner: He should at first be allowed to get up for a few hours, the time being gradually increased. The temperature is taken regularly, if it had been taken before, and if the last traces of the infection have not disappeared, then slight rises are easily observed. This decides the question at once. Everything connected with heart weakness should be considered, and thus the duration of the patient's sitting up, as well as the question as to the length of his walking, driving, etc., are to be regulated.

I believe that in this way we can do a great deal of good, and can lessen the danger to life, as well as limit to the minimum the tissue changes in the heart which are unavoidable. On the other hand, transgressions of these very simple rules of treatment have been heavily avenged.

In the after-treatment, I shall point out only one more thing: we should be careful not to subject a person who has just passed through a fresh attack of heart disease too early to a general treatment, such as, for instance, the Nauheim baths. This warning is especially applicable to the cases of mild recurrent endocarditis.

VALVULAR DISEASE.

BY

THEODOR VON JÜRGENSEN, M.D.

VALVULAR DISEASE.

INTRODUCTION.

CAUSES.

Valvular lesions in the heart are produced by tissue changes which cause an imperfect closure of the flaps involved, or a narrowing of the orifices which they control. The causes of these anatomic changes are either endocarditis or atheromatous changes. For several reasons, no accurate statement can be made as regards the external conditions which favor the development of these changes.

Endocarditis arises as the result of the action upon the heart of a number of causes of disease, perhaps of any that produce general affections. Endocarditis is, in other words, one of the phenomena of the general infection produced by these germs. These general infections may vary, both as to the time and as to the place of their occurrence. The same is true both of the primary disease and also of the secondary affection. Figures which express the frequency of endocarditis in general, and the occurrence of its various causes in particular, are, therefore, necessarily subject to great variations.

One thing is certain, however, and that is that acute articular rheumatism plays a very prominent rôle in the origin of valvular lesions. It is doubtful, however, just how often rheumatism is concerned in the production of heart disease. A few trustworthy observations may be cited here as examples:

In Zurich* (Eichhorst's clinic), 241 cases, of which 65.6 per cent. were caused by acute rheumatism. Jena† (Stintzing's clinic), 210 cases, of which 36.7 per cent. were caused by acute rheumatism. Leipzig‡ (Curschmann's clinic), 670 cases, of which 58 per cent. were due to acute rheumatism. Riga§ (hospital and private practice), 110 cases, of which 27.2 per cent. were due to ("typical") acute articular rheumatism.

Hampeln mentions the difficulties which arise from the fact that the term "acute articular rheumatism" cannot be absolutely defined. He distinguished a "typical" form, and another type which he calls the "atypical and larval," which constitutes not less than 23.6 per cent. of his cases of valvular disease. Just at present, when the question is pending as to the relation of acute articular rheumatism to septic infections, statistical statements are all the more misleading. (Compare Endocarditis, p. 182, etc.)

* "Statistisch klinische Mittheilungen über Herzklappenfehler," dissertation. Zürich, 1889.

† "Statistische Mittheilungen über Herzklappenfehler," dissertation, Jena, 1893.

‡ Mengel in an unpublished dissertation, quoted in Romberg, "Handbuch," p. 801.

§ Hampeln, P., "Ueber Sklerose und entzündliche Schrumpfung der Herzklappen," "Zeitschrift für klinische Medicin," Bd. xi, 1886, S. 487.

Atheromatous degeneration does not seem to be so frequently the cause of valvular lesions. The following figures were taken from the sources already quoted, and express the percentage of cases due to atheroma: Zurich, 12.5 per cent.; Jena, 15.7 per cent.; Leipzig, 12.3 per cent.; and Riga, 39.1 per cent.

The figures of the University clinics do not vary to any extent, but are very different from those given by Hampeln. Possibly the cause of this is that in a municipal hospital a much larger number of middle-aged and old persons find admission.

It may be said, in contradiction to former statements, that valvular defects occur practically with the same frequency in both sexes, and that the female sex is not favored in any way.

The age of the patient is naturally of importance, on account of the fact that atheromatous degeneration occurs but exceptionally before the age of forty, and, therefore, owing to this fact the same is true of the valvular defects due to this cause. It is self-evident that a person over forty years of age may be attacked by endocarditis. Acute articular rheumatism, in the ordinary sense of the word, meaning the typical form, is seen most frequently from puberty up to about the thirtieth year. The largest percentage of valvular lesions falls into this period of life, at least the largest percentage of valvular lesions which come to the physician as such, that is, fully developed. Thus, in Zurich there were patients from eleven to thirty years of age, 59.8 per cent.; in Jena, from ten to thirty years, 50.8 per cent.; and in Leipzig, from nineteen to twenty-nine years, 56 per cent.

The question as to when the valvular lesion arose is quite distinct from that just discussed, and cannot be answered offhand.

Schnitt has tried to do this by considering cases in which an acute polyarthritis had preceded the observation, but was able to utilize only 77 of his 210 cases. He found that in a large percentage, *i. e.*, in 13 per cent., the valvular lesion developed within the first ten years of life. Leuch is less accurate, and without considering the mode of origin, estimates that 11.6 per cent. of his patients in the Zurich hospital developed endocarditis before the age of ten, while three of these had congenital valvular defects. Romberg saw only 0.15 per cent. of fully developed valvular diseases before the tenth year in the cases observed at Leipzig.

This shows that, aside from its fetal form, endocarditis may occur very early in life. However, the fact that endocarditis is capable of producing changes in the valves at its first appearance, but that these changes do not necessarily lead to extensive interference with the valves or to true valvular disease, does not allow us to adopt the standard of Schnitt. We know how slowly and insidiously, and at other times how suddenly and acutely, recurrent endocarditis may set in. Valvular lesions, in their clinical sense, always include a true disturbance in the function of the involved part. So long as this disturbance has not occurred, we can speak of an endocarditis that has not subsided, but not of a valvular defect.

This shows the difficulties which we encounter when we seek to give accurate figures as to the relations of valvular disease of the heart to the age of the patient.

Bamberger, Curschmann, to mention only a few of our best observers, have pointed out that a considerable time must elapse after an endocarditis before the distinct signs of a valvular affection can appear. For this purpose it is not necessary, therefore, that there should be a recurrence of the inflammation; the process which has once developed continues to glow like a spark under a layer of ashes. Therefore, if we desire to be sure of our ground on this question, we must observe suspicious cases for a much longer time and much more regularly than is usually done, especially in the conditions obtaining in hospital practice.

What is the frequency of valvular affections in the individual valves? This question can be decided, as sufficiently large figures are available. The following statistics embrace 2470 cases. From these it appears

SEAT OF VALVULAR LESIONS.

Where Observed. Number of Cases.	Mitral.	Aortic.	Pulmonary.	Tricuspid.	Mitral and Tricuspid.	Mitral and Aortic.	Mitral, Tricuspid and Aortic.	Aortic and Tricuspid.	No Definite Localization.
Prague, Vienna, and Würzburg, 211*	137	45	2	1	12	9	5	..	..
Göttingen, 116†	63	7	..	1	..	30	6	1	8
Rudolfstiftung, Vienna, 93‡	67	13	13	..	..	..	..	..	..
Würzburg, 203§	121	51	7	4	4	8	7	1	..
Breslau, 1396‖	934	283	29	2	10	110	1	..	27
Zurich, 241**	173	21	4	..	9	34	..	..	..
Jena, 210††	121	37	1	2	10	33	5	..	..
Total cases, 2470‡‡	1616	457	56	10	45	224	24	2	35

that the *mitral valve* is by far most frequently affected. On the average, it was involved in 65.4 per cent. of all cases. The extreme values varied between a maximum of 72 per cent. (Zurich and Vienna) and a minimum of 54 per cent. (Göttingen). The valve affected with the next greatest frequency is the aortic, averaging 18 per cent. of the cases, with a maximum of 25 per cent. (Würzburg) and a minimum of 6 per cent. (Göttingen).

It should be noted in connection with the fact that the minimum percentage for both valves was observed in Göttingen, that complex valvular lesions were observed in that city in no less than 32 per cent. of the total cases. On the average, complex cases occurred in 12 per cent. of all cases, and of these, 9.1 per cent. were cases of simultaneous affection of the mitral and aortic.

An *insufficiency of the valves* may develop quite suddenly. It occurs when a perforation takes place in the valve at a spot which has become less resistant, owing to the inflammation or necrosis. The valve, which has previously been strained somewhat, and in consequence has become sacculated (acute valvular aneurysm), yields to pressure of the blood-stream. The tendinous cords of the atrioventricular valves may also burst suddenly, as the result of a previous inflammatory or degenerative tissue-change. In exceptional cases the tearing of a really healthy valve may be produced by an excessive physical exertion, or rather more frequently by a strong traumatism upon the thorax. Usually, it will be found that in such cases the tissue of the valve had not been perfectly normal. The aortic is more frequently thus affected than the mitral valve.

* Clinics, Bamberger, "Virchow's Archiv," Bd. ix, S. 524.

† Ebstein's clinic, Köster, "Beitr. z. Statistik d. Herzklappenfehler," dissertation, 1883.

‡ Hospital, 1882, quoted by Leuch.

§ Gerhardt's clinic, from Hirsch, "Mittheil. a. d. Medicin. Klinik zu Würzburg," Bd. ii, S. 305.

‖ Biermer's Klinik and Poliklinik, Guttmann, "Zur Statistik der Herzklappenfehler," dissertation, 1891.

** Leuch, in the dissertation already quoted.

†† Schnitt, in the dissertation already quoted. Also a case in which all the valves were involved.

In the great majority of cases endocarditis or atheromatous degeneration results in a gradual thickening of the valves produced by an increase of connective tissue which undergoes transformation into fibrous tissue. This is followed by fatty or hyaline degeneration, and finally by calcification.

The further development of a valvular lesion is associated with the *formation of thrombi*, wherever the endothelium has been destroyed and the surface has been roughened. The great importance of this process was first pointed out by Ziegler.* At first there is a deposit of blood-platelets from the blood, flowing in a slow stream over the injured surface of the valve. Next comes a precipitation of fibrin, which is accompanied by red and white blood-cells. Then follows the formation of new connective tissue in that portion of the valve which remains permanently covered by the thrombus. This connective tissue penetrates into the mass of the thrombus, becomes organized, and later may undergo a variety of degenerations. Smaller thrombi usually become replaced by connective tissue, while larger thrombi usually calcify.

A series of changes takes place in the interior of the thickened valve for a long time after the beginning of the process. In virtue of these changes the structure of the newly formed connective tissue is altered, so that there are fewer cells and vessels, and the tissue becomes tougher in consistence. There are also, often, hyaline degeneration, fatty degeneration, and finally calcification (Ziegler).

It is possible, therefore, that a valvular disease may grow worse without the occurrence of a fresh inflammation of the affected parts.

The changes in the blood-supply due to *newly formed vessels*, which arise in the valves in the wake of endocarditis, but not after atheromatous processes, are worthy of attention. They are as follows, according to the researches of Langer.† The semilunar valves normally do not possess any blood-vessels. The auriculoventricular valves have vessels only in the portions which adjoin the endocardium, while the tendinous cords are entirely without blood-vessels. New vessels arise in the course of protracted inflammatory processes in the endocardium, and this is more marked in the valves and the tendinous cords of the left side, than in those of the right. This rule, however, has exceptions. The newly formed capillaries are comparatively large in diameter, and vary greatly as to the thickness of their walls. They are connected with one another in the form of a wide-meshed network of vessels.

These capillaries are apt to remain in the tissue, when no great contraction of the valves follows the inflammatory process. If such contraction takes place, they disappear almost completely. It is uncertain, as yet, what relation the formation of new vessels bears to the nutrition of the valves, and what connection there is between them and the recurrence of the inflammation or the extension of the process.

The diseased valves become less elastic. Even when this occurs but in a slight degree, one change is inevitable. The valve must allow a free passage for the incoming and outgoing blood, and this takes place when the valve is pressed backward against the point of attachment of its valvular ring. This follows in obedience to the pressure of the blood

* "Ueber den Bau und die Entstehung der endocarditischen Efflorescenzen," "Verhandlungen des Congresses für innere Medicin," 1888, Bd. vii, S. 339 ff.

† "Ueber die Blutgefässe in den Herzklappen bei Endocarditis valvularis," von Ludwig v. Langer, "Virchow's Archiv," Bd. cix (1887), S. 465 ff.

which flows in a certain direction. As the time during which the cardiac cycle takes place is very short, the passive movements of the valves must occur with the least possible resistance of their tissue, as is normally the case. In this way only can the passage of blood occur with perfect freedom. Anything that increases the resistance within the tissue of the valves interferes with the sufficiently rapid opening of the orifices. Whenever, therefore, the closure of the valves is no longer possible at the right instant on account of thickened tissues, then the opening of these valves is also no longer possible. Every insufficiency which arises in this manner is necessarily followed or accompanied by a stenosis. The latter increases in proportion as the valves are hindered in their movements, and in proportion as they project into the openings which they are intended to close. The highest degrees of stenosis occur when the valves become adherent at their margins and become stiffened through calcification. In such cases there remain only openings of a few millimeters' diameter for the passage of the blood.

GENERAL PATHOLOGY OF VALVULAR DISEASE.

Every valvular lesion increases the demand upon the muscular mass of the heart which supplies the kinetic energy which is necessary to drive the blood through the circulation.

The simplest conditions obtain in *stenoses of the orifices*. When the same amount of a fluid must be driven through a tube within a given time, the resistance increases in regular ratio with a decrease of the diameter of the tube. The narrower, therefore, the stenosis becomes at one or the other outlet or inlet, the greater will be the tax upon the capacity of that part of the heart which has to drive the blood through this opening. In all stenoses due to valvular lesions there are permanent tissue changes and, therefore, the task of the heart is permanently increased.

If the valves do not close, then a part of the blood which has been driven through them during systole in a certain direction will return into the diastolically relaxed ventricle (aortic and pulmonary valves), or else will be driven into the auricles during systole (mitral and tricuspid valves). The immediate effect will be that a smaller amount of blood will pass through the vessels in the direction with which we are dealing. In order to restore the original state of affairs, that is, in order to make the old amount of blood pass on with the proper speed, the driving force must be increased in a corresponding degree. But, in addition, there must also be an enlargement of that cavity in which the blood temporarily accumulates. (See below, page 319, etc.)

A prolonged insufficiency of valves, therefore, leads to permanent increase in the labor of the heart, just as a stenosis of the orifices does. In order to compensate for these changes the driving force must be increased, and this can be effected only by increasing the power of the mechanism which furnishes this labor.

Wherever a permanent increase in the labor to be furnished is necessary, we find a hypertrophy of the muscle-fibers of the heart. Such a hypertrophy of the muscle-fibers cannot be increased to any degree we like, nor can it continue indefinitely. Every organ of the body, including the muscles, has a limit of activity which cannot be exceeded, and beyond which it cannot increase in function. Therefore, "thus far and no farther" is

the principle, and the most favorable conditions of nutrition can avail nothing in this respect.

Alexis Horvath,* in his remarkable book, teaches, among other things, that "it is ridiculous to say that the muscles become hypertrophic through increased labor and atrophic through disuse." "If the physiologic law, according to which a working muscle grows larger in proportion to the work, were correct, then our heart would have to continue to grow year by year (even after the growth of the body had ceased) and would have to enlarge to such an extent that the heart of a man forty years old or even of a man younger than that would have to fill the entire thorax." It is possible, therefore, that we do not understand the action of one of the fundamental laws of biology, although at first sight this possibility would seem to be remote.

Hypertrophy of the cardiac muscle can only take place when the muscular tissue is capable of growth, or, let us say, of development, and when the material necessary for this increase is available on the spot. In the final analysis, the conditions upon which the ability of muscular growth depends are still obscure. They are manifestations of that which we know as "life."

It is different with nutrition. This may be discussed and even, to some extent, its value estimated. I shall follow here the arguments of Benno Lewy.† When the circulation takes place in normal fashion, a certain amount of blood under a certain pressure enters the aorta in each unit of time. The same amount enters the pulmonary artery, and the intervening capillary region is traversed in both places by the same amount of blood within the same period of time. The driving force of both the left and right side of the heart suffices to overcome the resistances and to communicate the necessary velocity to the blood. Approximately, the work of the normal heart at rest may be expressed as 815 meter-kilograms per hour. When the body is in motion, then the heart must perform from three to thirteen times as much work, according to the demands of the muscles employed for the exertion.

The question is now whether the heart is able to cope fully with the task thus set before it, in spite of the presence of a valvular disease; that is, whether the heart can develop not only the amount of force necessary when the body is at rest, but also that needed for the severest muscular exertion—up to thirteen times the amount of work that it does at rest. We might be allowed to speak of complete compensation, only if this were possible, and if the normal working capacity of the heart could be preserved in spite of the presence of a valvular lesion.

The energy supplied to the body day by day through the food is a constant value, which is estimated at 345,000 meter-kilograms. Of these, 45,000 are allotted to the cardiac activity of a person doing an average amount of muscular work. In every case of a valvular defect the tax upon the labor of the heart is increased; for the amount of vital energy which would be sufficient, under normal conditions, to drive a certain amount of blood, at a certain pressure, through the circulation at a certain speed, within a certain time, is no longer sufficient. The amount of energy which the heart requires, in addition to that normally consumed by it, is to be subtracted from the total (345,000 meter-kilograms), and to be added to the normal amount (45,000 meter-kilograms).

Therefore, it is impossible to get an absolute unconditional compensa-

* "Ueber die Hypertrophie des Herzens," mit einem Vorwort von A. Weichselbaum, Wien und Leipzig, Wilhelm Braumüller, 1897.

† "Die Arbeit des gesunden und des kranken Herzens," "Zeitschrift für klinische Medicin," Bd. xxxi (1897).

tion in the sense that the same working capacity of the heart which existed before the valvular defect developed be perfectly restored. The increased energy required by the heart is lost for the other expenses of the body. This loss is shown chiefly in the muscles whenever they are employed for physical work. The greater the demands which the heart makes for itself upon the store of total energy, the less of this energy will remain available for the muscles. On the other hand, perfect rest of the body, that is, the avoidance of all motion which is not absolutely necessary for the continuance of life, places the entire store of available energy at the disposal of the heart. The latter would then be able to employ 345,000 meter-kilograms; in other words, 7.7 times as much as the normal amount of energy which the heart requires.

Then we have reached the extreme limit. A greater amount of work is not possible, because an increased amount of energy cannot be furnished: "A valvular defect, or any other impediment in the circulation which requires more than 7.7 times the amount of normal energy for the continuance of a normal circulation, even when the body is at perfect rest, is, therefore, incompatible with life. When such a valvular defect is present, a sufficient circulation is impossible" (B. Lewy).

The working capacity of the heart muscle is, as a rule, proportionate to its weight. The weight of the heart is normally said to be 300 gm. How much would the heart have to weigh in order to develop the uttermost amount of energy? Seven and seven-tenths times 300 gm., which equals 2310 gm.

The greatest weight observed in the heart by Stokes (according to Eichhorst) was 1980 gm.; in other words, 6.6 times the normal weight. This is a figure which comes fairly close to the theoretic calculation.

These figures of Benno Lewy's give a general conception of the subject under consideration. It must be noted, however, that he excludes from this calculation all the work which must be done to maintain respiration. If this energy were considered, very complicated conditions would arise; for the activity of the respiratory muscles is indirectly concerned in the circulation of the blood. In my opinion, it would be very desirable to attempt to bring this factor into the calculation. Next to the amount of work furnished by the heart muscle itself, the work involved in respiration is of the greatest importance for the circulation. The absolute values which may be obtained without taking this factor into consideration are satisfactory from the viewpoint of the mathematician, but not from that of the physician. For the physician sees at the bed-side in what pronounced degree the condition of the respiration can become altered in disturbances of the mechanism of the heart-pump, and therefore to what extent the value of the muscular work employed in respiration is to be considered in this connection.

In general, the respiration is increased in frequency. Therefore, the drain upon the total sum of energy which the body has at its disposal is also increased. The diaphragm, and all the accessory muscles which are employed during forced breathing, require a much greater expenditure of energy for the purpose of respiration, and it is a question as to how much of this increased energy is left for the benefit of the circulation. I scarcely think that these questions are amenable to mathematic analysis, but I may be wrong, and I should be glad to be informed if such be the case.

B. Lewy considers a few other points of general interest. He bases his considerations upon the supposition that the normal capacity of the working body remains unaltered, in spite of the presence of a valvular lesion. We must not confound with this working capacity the ability to continue to live. The latter may be maintained even when less blood passes through the tissues under a lessened pressure. B. Lewy asks whether it is not possible that a better employment of the oxygen con-

tained in the arterial blood would be sufficient to maintain organic nutrition in the proper degree, in spite of the fact that only one-half the amount of blood is driven through the tissues within a given period of time? If this question could be answered in the affirmative, the heart could very well diminish the amount of work which it has to do. I have already answered this question in the negative in another connection (p. 143), quoting the deductions of von Noorden, with whom I fully agree.

An organ may temporarily manage to get along with a lesser degree of oxygen tension in the blood which passes through it; but if its tissue shall remain intact, if the capacity of its cells to transform energy into living force shall remain at its height, then it cannot remain contented with a lessened supply for any length of time. This fact must also be taken into consideration in discussing the next question, viz.,

Why does the body require such a high blood pressure and such a rapid circulation?

The answer to this can only be that these are necessary in the interest of the working capacity of the organism. The body must be at all times ready to supply an excess of external work. We know, therefore, that the requirement at such times is an extraordinarily large amount of blood, and we can imagine that without this a rapid transition from the minimum of blood pressure and circulatory velocity which is sufficient when the body is at rest, to the maximum required in a time of activity, would be impossible.

The heart normally works with lavish expenditure of energy, so that the body is ready at any minute to develop its full capacity. While the circulation is normally more active than is necessary, the organs are always immediately ready to furnish at least some measure of external energy. The increased demand for blood, therefore, influences the heart's action less markedly than would otherwise be the case.*

Valvular defects produce an impairment in the heart-pump in still another direction; namely, they decrease the capacity possessed by the normal heart to perform a task set before this organ in more than one way, *i. e.*, so that, no matter which way the work is performed, an excessive exertion is not necessary.

In order to fulfil the requirements of a healthy human body, 4200 c.c. of blood must be driven through the capillaries every minute, with a development of about 815 meter-kilograms of energy per hour—that is, about 1.4 meter-kilograms per minute. Provided this standard is maintained, the number of contractions of the heart, and the amount of blood which is driven through the circulation at each contraction,—the frequency of the pulse, as well as the volume thereof,—may vary to a marked degree.

"It does not matter whether the 4200 c.c. are propelled by 60 separate heart-beats, each having a volume of 70 c.c., or by 70 heart-beats with a volume of 60 c.c. each, or else by 100 beats with a volume of 42 c.c. It is also of no consequence whether the systole be comparatively long or short. If we calculate the heart's action which is developed with a long or a short systole we find that the amount of work done per hour differs only by 30 kg., that is, by an amount which is certainly within the limits of error, and therefore negligible."

Therefore, while the time-relations of the single contractions of the

* Benno Lewy, *loc. cit.*, pp. 355 and 356.

heart are of no consequence provided the valves be healthy, yet the matter assumes a different aspect whenever a valvular lesion sets in.* It depends, however, upon the variety of valvular lesion present. The following examples may be cited, in which it is always assumed that 4200 c.c. of blood are propelled through the circulation per minute.

1. Aortic stenosis, in which the orifice is 0.7 cm., permanently closed by the adhesion of the margins of the two flaps:

60 pulsations per minute—work of the heart, 1000 mkg. per hour.

70 pulsations per minute—work of the heart, 1150 mkg. per hour.

2. Aortic insufficiency, in which about 0.2 cm. of the valves is permanently open:

70 pulsations per minute—work of heart, 1950 mkg. per hour.

120 pulsations per minute—work of heart, 1530 mkg. per hour.

3. Mitral insufficiency, in which there was also about 0.2 cm. of the valve remaining open:

60 pulsations per minute—work of heart, 1900 mkg. per hour.

70 pulsations—work of heart, 2100 mkg. per hour.

All these figures suffer from the drawback common to all theoretic deductions, and this point is brought out by B. Lewy himself. On the other hand, we may conclude from these data how greatly that capacity for adaptation, which is so marked in the healthy heart, is reduced in the presence of valvular disease. It is true, however, that this reduction takes place only in the presence of a certain degree of disease. It appears, from the mathematic considerations, that the altered mechanic conditions begin to gain the upper hand in stenosis only when two-thirds of the orifice is permanently closed, while in insufficiency this takes place as soon as about a fifth of the orifice remains permanently open.

If we continue the consideration of the purely mechanic conditions, we may be allowed to ask a question. The healthy heart is able to adapt itself to the altered condition of such accidents, as, for example, mental excitement, by changing the frequency of its beats without increasing the actual amount of work done. Is it of no consequence, so far as the fatigue of the cardiac machine is concerned, as to whether a greatly increased amount of work is required in a short time, as is the case in valvular disease, or whether this increased labor is accompanied by changes in the heart-beat, *i. e.*, increases in its frequency?

A question of still greater importance is whether a heart with affected valves can obtain the necessary rest and the necessary blood-supply which depends upon this rest. The discussion of this subject has been given in another place (Insufficiency of the Heart, p. 39). Only when the diastole is long enough to allow the amount of blood corresponding to the work of the heart to flow into this organ can the working capacity and nutrition of the heart be maintained. It will be seen that these relations are exceedingly simple.

When, for example, in the presence of an insufficiency of the aortic valves reaching a certain size, a saving in the hourly work of the heart is effected, as has been mathematically shown, through the increase of the frequency of the beats from 70 to 120; when such a saving amounts to 420 mkg., it still remains doubtful as to whether this has done the heart any good; for the simple reason that the periods of rest have been materially shortened in this change. Which is more useful: more work and better nutrition, or less work and worse nutrition?

* B. Lewy, *loc. cit.*, p. 361.

This question cannot be decided in a way applicable to all cases; for, first of all, we must ask whether more work is furnished by such a heart, and if so, to what extent. And this, again, depends, in the first place, upon the size and the structure of its muscular apparatus.

We thus come again to the important rôle of the heart muscle itself. The lessened utilization of the force furnished by the heart muscle, when valvular disease is present, can only be brought closer to the normal working capacity by the generation of vital force by the heart muscle itself. This is possible only by means of an increase in the fibers, that is, by *hypertrophy*. This is the important element. To what extent the enlargement of the volume of the beat, which implies an increase in the size of the cardiac cavity—a dilatation thereof—is of importance for the circulation of the blood is quite another question. Without hypertrophy every dilatation would be injurious; for it would result in a rapid decrease in the working capacity of the heart.

Considered in a general way, the processes which go on after the development of a valvular lesion in the heart appear in the following manner: The increased tax upon working capacity of the heart is at first overcome by drawing upon the reserve force of the organ. If the heart has been healthy until then, a great deal of this force is available, so that, as we have said before, about thirteen times the amount used when the body is at rest is available. The observations of Cohnheim* show clearly to what extent the considerable amount of resistance which is developed may be overcome within a short time. The fact that the heart was subject to a variety of other injuries during his experiments shows still more forcibly how resistant it is.

In a dog which was injected with curare, and in which artificial respiration was maintained, a large window was opened in the left side of the chest by resection of the ribs. After the pericardium had been opened, the isolated trunks of the aorta and the pulmonary artery were each provided with a strong loop of thread which was passed into a Graefe's loop-constrictor. The blood pressure was measured at the femoral artery and at the jugular vein, while the intracardial pressure was recorded by means of a comparatively large glass tube, which was passed into the left or the right ventricle, as occasion required. Thus it will be seen that the heart was abused considerably. In spite of this, a slow and gradual constriction, or one increased at intervals, by means of which the pulmonary artery was markedly narrowed, did not change the blood pressure in the femoral artery or in the jugular vein. It was only when the narrowing progressed still further that the arterial pressure suddenly fell, while the manometer in the vein showed a rapid rise. If the constricting loop had not been soon loosened, and thus the passage of blood through the pulmonary artery again allowed to go on, the work of the heart would have stopped. The heart's action, however, returned to its former state if the narrowing of the constriction was diminished in time.

The observation of the intracardial pressure on the right side showed that when the loop was only slightly tightened about the pulmonary artery, the pressure rose at once, and increased with the increase of the constriction.

The excursions of the manometer increased at each systole. When the aorta was constricted, the same series of events took place, save that a more marked constriction was necessary to increase the intracardial pressure. When the aortic narrowing was continued, the intracardial pressure was both absolutely and relatively much greater than that in the right side of the heart in pulmonary stenosis.

The distances between the systolic elevations and the diastolic depressions became more marked, and the individual contractions required a longer period when they had exceeded a certain limit, so that the total number of contractions per minute was diminished. In correspondence with this, the femoral artery showed infrequent and sluggish, but usually high, pulse.

By perforating or detaching the aortic valves by means of a probe, introduced

* "Vorlesungen über allegemeine Pathologie," vol. i, p. 46 ff of the second edition.

through the carotid,—an operation which is easily performed,—one can obviously produce insufficiency of this valve. Even when this insufficiency reached a considerable degree, the height of the arterial average pressure remained the same as before the operation, and the pressure in the veins was not in the least affected.

Cohnheim concludes correctly that: "The working capacity of the healthy heart increases in proportion to the demands placed upon the organ, and this increase does not take place by means of a complicated mechanism, but any increase of resistance directly excites the heart muscle to more vigorous and more effective contractions."

The length of time during which the heart muscle is able to furnish this increased amount of work without any increase in its fibers depends upon its original inherent working capacity; upon the wear which it has sustained in the course of the patient's life, and upon the question as to whether it has a sufficient blood-supply. These are the same conditions as those which control the development of hypertrophy at the stage when the muscle-fibers are forced to this as the result of permanently increased work.

It is true that such rapid changes as are produced in the experiments quoted occur but exceptionally in the disturbances to which the heart of a patient may be subjected. This makes it more probable that the heart can overcome the increased amount of work which is permanently placed before it by an increase of its working capacity; *i. e.*, by an increase of its muscle-fibers.

The *dilatation of the cardiac cavities* may be of great importance for the regular circulation of the blood. Such a dilatation is often brought about by the defective structure, or the lessened power of resistance, of the muscular wall against pressure, and therefore the dilatation in itself may be a sign of weakness of the heart muscle. Dilatations which arise quickly, even suddenly, as the result of enormous exertion, and are followed immediately by heart weakness, prove this sufficiently (p. 40). They teach that fatigue of the heart which is produced by overexertion of this muscle is the immediate condition of dilatation.

The manner in which dilatation takes place is as follows: After systole the heart muscle becomes flabby and the cavity which it incloses is again filled with blood. The latter flows with increased pressure into the ventricle from the large venous trunks through the auricle. In the last third of the diastole the auricle itself contracts and adds to the volume in the ventricle. The wall of the ventricle undergoes a dilatation, the extent of which depends, on the one hand, upon its elastic resistance, and, on the other hand, upon the amount of pressure exerted upon it. The elastic resistance depends upon the character of the heart muscle. Fatigue renders the muscles much more easily stretched than normally.

The tired heart, therefore, is more widely dilated by the blood which streams into it than the heart which is not fatigued. The increase in the size of the cavity which is thus produced allows the blood to flow into it, provided a sufficient amount of blood is present, until an equilibrium is produced between the force which controls the inflow and the resistance offered by the elasticity of the cardiac wall. An increased amount of blood is certainly present, and the systole which follows is obliged to perform more work in order to propel this increased amount. If the necessary force is not available for this, then the pressure in the aorta can no longer be maintained at its former level. The consequence of this is an insufficient blood-supply of all the organs, which affects the heart most

severely, because it is obliged to perform continuously an increased amount of work, which is indispensable for the maintenance of the circuation.

The principle that the nutrition of the heart depends directly upon its own work is again evident. The less sufficient the heart's action, the less sufficient its nutrition, the greater its tendency to fatigue and to dilatation. It is possible, therefore, that the vicious circle may be completed, that is, that once a dilatation has been produced through fatigue of the heart muscle it can no longer be overcome, and it must lead in the end to a cessation of the heart-beat.

On the other hand—and this is by far the most frequent event—the dilatation is compensated in the following manner: A diminution in the resistance in the greater circulation may be induced largely through the dilatation of the arteries, the smooth muscle-fibers of which are under the control of the vasomotor centers. This, together with the lessening of the physical work of the body to the minimum necessary for the maintenance of life, diminishes the task of the heart. [On mechanic grounds a dilated ventricle will throw out more blood with a given force of contraction than a smaller ventricle.—Ed.] This organ can then perform its work without any more fatigue, and the exhaustion which was present diminishes under the influence of a now sufficient blood-supply in the heart. In this manner it becomes possible again to increase the force available for the emptying of the cardiac cavity. And with this the elastic resistance of the more thoroughly nourished and no longer fatigued heart-wall may increase. Thus gradually a condition is brought about which makes possible a circulation that approaches more or less to the normal.

In valvular defects which develop slowly and gradually the process goes on much less violently. In these cases the deciding factor is, whether the increase in the muscular mass of the heart takes place commensurately with the increased resistance offered by the valvular lesions. If this is the case, then the dilatation produced by fatigue cannot develop, because the fundamental cause—the fatigue—is lacking.

Dilatations of the cardiac cavity which result from certain valvular lesions have a different origin from that just described. The healthy heart muscle is capable of adapting itself in a very complete way to the task set before it.* This applies both to its distention, and to its contraction: Not only does the size of the contraction vary in extent, but also the degree of the elasticity, so that it is possible for the heart to accommodate an excess of blood, and to provide sufficient force for the expulsion of the blood which has thus entered the chamber. If, now, a valvular disease, as, for example, an insufficiency of the aortic valve, permanently produces an increased filling of the left chamber with blood, then the cavity thereof may become dilated and its musculature may increase without necessarily producing any signs of fatigue, with their consequences. Naturally, an upper limit exists, beyond which the heart cannot "compensate," but this limit is different for each individual, and is determined by the capacity of the heart muscle. No further arguments are necessary to show that a heart which is affected with valvular lesions is more subject to dilatation due to fatigue than is a normal heart.

The hypertrophy of the muscular structure of the heart occurs, as in

* For a detailed discussion of the subject and the literature relating to it see Ludolf Krehl, "Pathologische Physiologie," Leipzig, F. C. W. Vogel, 1898, p. 5 *et seq.* (This authority will hereafter be referred to as *A*.)

the other muscles of the body, in response to the law that a muscle which works more than the others correspondingly increases in size. The portion of the heart in which the valvular disease requires the greatest amount of work is, therefore, more apt to be involved in hypertrophy than the less active portions.

In a mechanism so artfully constructed as that of the heart-pump disturbances which affect one portion thereof do not remain limited to that portion. It is the object of pathology, therefore, to gain a more thorough insight into the interrelations of these processes. We shall see, in discussing the individual valvular lesions, to what extent this is possible in each of these conditions.

At this point, however, I should like to discuss the general questions which arise in this connection:

1. Is it possible that in valvular defects which arise in a heart that is capable of hypertrophy the muscular increase does not extend beyond that portion which is forced to perform an increased amount of work as the result of a mechanic disturbance?

This has been denied theoretically.* At any rate, the increased amount of blood which is required for its own increased amount of work by the hypertrophied portion of the heart must, in the long run, stream through the general circulation even when the body is at rest. An increased tax upon both ventricles is, therefore, unavoidable in these conditions. For example, in insufficiency of the aorta, which scarcely places any increased requirements upon the right side of the heart, a slight hypertrophy of the latter is always found. This fact is always cited in support of the view just mentioned.

That a hypertrophy of the entire heart is not always clearly apparent in all valvular lesions is perfectly easy to understand. The required increase in its work is not great, and grows less in proportion as the patient spares his heart. The hypertrophy of the muscle can, therefore, be so slight that it cannot be demonstrated. At all events, this question requires further investigation.

2. Can the groups of cardiac muscle-fibers which are directly employed for the closure of the valves be alone involved to a greater extent in the hypertrophy? It may be assumed, with a fair amount of probability, that a sufficient closure of the valve cannot take place without the assistance of muscles; the muscle-fibers which are specially designed to perform this work have, therefore, a special task. It is possible that in insufficiencies which arise as the result of tissue changes in the valves themselves compensation is favored through an increased activity of the muscular fibers immediately concerned in the closure? If this takes place, then we should expect to find a greater degree of hypertrophy in these constantly taxed muscles. It may not be impossible to demonstrate this anatomically, but the methods of investigation would be very difficult, and it would mean a great deal of work before one could reach a definite conclusion.†

The *assistance of the muscles in the closure of the valves* is of such great importance clinically that it must be considered more in detail. Of course,

* B. Lewy, *loc. cit.*, p. 363 *et seq.*

† *Cf.:* L. Krehl, "Beiträge zur Kenntniss der Füllung und Entleerung des Herzens" (Aus dem physiologischen Institute zu Leipzig), "Abhandlungen der mathematisch-physikalischen Classe der königlich sächsischen Gesellschaft der Wissenschaften," vol. xvii (1891), S. 343 ff. (Referred to hereafter as B.)

the conditions obtaining in the atrioventricular valves are different from those in the semilunar valves.

In the case of the bicuspid and tricuspid valves, as has been known for a long time, the papillary muscles, with their peculiarly arranged tendinous cords, are essentially concerned. Their task consists in tightening the valvular flaps when they contract coincidentally with the systole of the ventricle. If no papillary muscles were present, the flaps would be driven back into the auricle under the pressure of the blood during systole, and thus a closure of the orifice would be impossible.*

But another factor comes into play in the closure of the valve, namely, the specially arranged muscular bundles in the heart which directly diminish the size of the base of the heart during its contraction, and thus lessen the apertures of the ostia. This arrangement is said to have a "considerable part in the closure" (Tigerstedt). Krehl† believes that when the intracardial pressure is great, the mitral is only able to close after the orifice has thus been narrowed.

The following conditions obtain for the valves of the large arteries: "The aortic orifice during systole represents a slit, and the artery is narrowed even below this orifice. The slit-like shape of the opening itself is produced by the formation of the well-known muscular swellings, which form the bottom of the pocket-like valves. The slit becomes narrower as the systole increases. The valves themselves are attached to the edges of these muscular swellings. When the flaps are in place, they lie almost perfectly in the direction of the blood-stream—in other words, *at right angles* to the plane of the orifice, and not, as is usually described, *in* that plane, with a slight bending of their margins. Through the fact that the blood is pressed into the narrow muscular slit and then passes into the dilated space above the valves, eddies must arise which tend to approximate the valvular flaps constantly to one another, although these structures cannot come close to one another because the blood which passes through them, and is under high pressure, forces them apart. When the blood-stream ceases to flow, the valves, as though impelled by a spring, snap together, and the closure of the orifice takes place quickly and without regurgitation. This is confirmed by measuring the pressure in the ventricle, when it will be seen that not a trace of backward flow takes place.

"The closure of these valves is then kept up by the difference in pressure between the ventricle and the aorta, and this difference suffices, once the valves have closed, obviously even when the muscles of the ventricle become flabby and its muscular support is absent. An exact counterpart of this arrangement exists in the pulmonary artery" (Krehl).

Through the results of these newer investigations, we gain in some respects a deeper insight into pathologic conditions. First, we shall speak of "relative insufficiency," and in this connection the tricuspid valve is of special interest. We speak of relative insufficiency "when, as the result of a considerable dilatation of the right side of the heart, the ring of insertion of the still unchanged venous valve is so markedly dilated that this valve no longer is able to close the orifice." This defi-

* Concerning the widely divergent explanations of the details of this process, see the critical presentation by Tigerstedt, in his "Lehrbuch der Physiologie des Kreislaufes," Leipzig, Veit & Co., 1893, S. 39 ff.

† *Loc. cit.*, B, p. 360.

nition is given by Friedreich.* It will be seen that this definition includes two things—the fact itself and its interpretation. Objections have been advanced against the fact itself very vigorously by Bamberger;† Skoda‡ did not explain his dictum: Relative insufficiency is improbable. At present, however, there is no longer any doubt.

In case XIV (p. 240) I have given the results of my own observation on this subject. Tricuspid insufficiency is shown by the tracing of the hepatic venous pulse (Fig. 72), and the fact that the insufficiency was relative here, appeared from the autopsy, which showed no changes in this valve.

In connection with the interpretation of Friedreich's relative insufficiency Kürschner§ remarks as follows: "I have dilated the venous orifice in both ventricles in such a manner as is never seen in the living heart, and the valve was still sufficient to cover a much larger opening." Bamberger refers to this observation and remarks further that the valves always take part in the dilatation of the orifice and of the chamber by becoming thinner and by stretching. This is not absolutely admitted by Freidreich: "The statement that the valves always take part in the dilatation of the right venous orifice by stretching and becoming thinner and thus covering a larger surface is not by any means always applicable, and I have repeatedly seen cases in which the valves had not become correspondingly larger and even had become somewhat shortened in their vertical measurement, and evidently were not in any way able to close the dilated orifice completely. In these cases the dilatation of the orifice was marked and the opening admitted easily five fingers." In his later treatise Friedreich‖ points out that we can at once determine whether or not an insufficiency of the valve exists by employing the trigonometric formula $D = \frac{P}{\pi}$. It is only necessary to measure, in the heart of the cadaver, the length of the opened venous orifice (= P) and the height of the valvular flaps, in order to obtain the figures necessary for a comparison. This procedure, which is frequently employed, is, however, applicable only when rather gross changes have taken place.

Another factor must be taken into consideration in addition to the dilatation of the entire right heart which takes place under these conditions, simply as the result of the insufficient nutrition or of actual changes in its tissue. It is certainly not probable that the muscles which are immediately concerned in the closure of the valves should be excluded from changes which affect the entire heart.

The papillary muscles were accused at first: It was said that they did not set the flaps of the valve in position in time, and thus interfered with their perfect closure. The same charge may now be brought against the muscles that narrow the orifice. We must remember that the time allowed for the closure of the valves is so short that a very efficient appa-

* "Krankheiten des Herzens," "Virchow's Handbuch der speciellen Pathologie und Therapie," vol. v, p. 382, Erlangen, Enke, 1861.

† "Lehrbuch der Krankheiten des Herzens," Wien, Braumüller, 1857, p. 254-255.

‡ "Abhandlung über Percussion und Auscultation," sixth edition, Vienna, Seidel, 1864, p. 324.

§ Wagner's "Handwörterbuch der Physiologie," vol. vi, Braunschweig, Vieweg, 1864, p. 61; see also p. 49. Here occurs the frequently quoted statement that "each individual flap of the valve is nearly as large as the venous orifice" (bull's heart).

‖ "Ueber den Venuspuls," "Deutsches Archiv f. klin. Medicin," vol. i, p. 251 (1866).

ratus is necessary to perform the act without loss of time. Instead of speaking of relative insufficiency, it would be much better to speak of "muscular" insufficiency. We need not have any scruples in applying to the work of the heart the observations which have been made upon the work of the fatigued body muscles. The latter suffer the same loss of functional capacity through insufficient nutrition and insufficient blood-supply as the heart dilated by the stretching of its fibers.

If we watch the changes which take place in the contraction of the gradually exhausted muscle,*—the constantly decreasing height of the curve, the tendency to retardation and prolongation of the stage of contraction, and the slower and slower fall,—we gain a useful conception of the fact that such a muscle can never fulfil the task which is set before it when it is obliged to close a valve within a fraction of a second.

There is a good reason why relative insufficiency was first noticed in the tricuspid valves. The positive wave, synchronous with the systole, which passes through the veins in such cases leaves no doubt that there is a true insufficiency of the valve. On the other hand, in insufficiency of the mitral valve, no such definite signs can be observed. The marked development of the muscular structures of the left ventricle makes it doubtful whether such a marked dilatation and stretching of the insertion ring can take place there, as on the right side. On the other hand, it was necessary to explain the mechanism of systolic murmurs, the origin of which had to be placed in the mitral. Therefore, it became necessary to bring into the foreground the behavior of the muscles which were known to play a rôle in the closure of the valves. These were the papillary muscles, and it was said that they became unable to set the valvular flaps at the proper angle.

This view is stated clearly by Skoda.† "Temporary insufficiency may result in the venous valves as the consequence of a paralysis of individual papillary muscles." We have already mentioned the fact that this author rejected the idea of a relative insufficiency. Bamberger shares this view‡ and devotes the following remarks to this subject: "In general it is impossible to say at present whether functional diseases of the muscles (papillary muscles) which fix the atrioventricular valves,—paralytic and paretic states,—are able to interfere with the closure of the valves, but this is certainly not improbable."

As regards the mitral valve in particular: "In rare cases the valve may have been insufficient during life without showing any changes in the cadaver (such as, for instance, changes in the papillary muscles)."

Von Dusch§ expresses a similar view and thinks it possible "that spastic or paralytic conditions in the papillary muscles interfere with the correct position of the valve at the moment of ventricular systole." C. Gerhardt‖ places the responsibility for the frequent systolic murmurs occurring in the mitral partly upon the slight tissue changes which so often are present in this valve. Yet he expresses the positive opinion that a temporary insufficiency may occur in this valve as a result of a transient disturbance in the function of the heart muscle, and especially in the papillary muscle. This is especially frequent in chlorotic individuals, and may be accompanied with marked signs of heart failure; edemas and catarrhs of various mucous membranes.

Liebermeister** strongly pleads in favor of relative insufficiency of the mitral. "An insufficiency of the mitral valve may also arise as a result of the imperfect action of the musculature of the left ventricle. If the mitral valve, which consists

* See the beautiful curves of Rollett in Tigerstedt, "Lehrbuch der Physiologie," vol. ii, p. 38.

† *Loc. cit.*, p. 210.

‡ *Loc. cit.*, A, p. 183, and B, p. 237.

§ "Lehrbuch der Herzkrankheiten," Leipzig, Engelmann, 1868, p. 203.

‖ "Lehrbuch der Auscultation und Percussion." Fifth Ed., Tübingen, Laupp, 1890, p. 217.

** "Vorlesungen," *loc. cit.*, vol. iv, p. 354.

of a larger anterior and a smaller posterior flap, with two unimportant accessory intermediate flaps, be compared with the orifice which it is intended to close, it may be easily seen that, for the complete closure of this orifice, there are necessary both a vigorous contraction of the ventricle and a proper length of the tendinous cords and of the papillary muscles, inasmuch as the position of the valvular flaps is determined by these structures. When the ventricle or the papillary muscles do not contract in the correct manner, the closure is necessarily imperfect and a so-called muscular insufficiency of the mitral valve may develop."

The functional insufficiency of the mitral valve is fully recognized in the work of Pierre Merklen.* The causes of this condition include the dilatation of the left ventricle, which involves the valvular ring, but this dilatation alone does not suffice, and there must be also changes in the papillary muscles and in the tendinous cords.

Another cause which may possibly come into play is the imperfect action of the papillary muscles and of the muscular rings which contract the orifices during systole. A cramp of the papillary muscles may also take place.

"La tendance actuelle est de rattacher l'insuffisance fonctionelle à un spasme des muscles papillaires, comme l'avait proposé Bamberger, d'ou resultent leur raccourtissement et l'impossibilité pour les deux valves de la mitrale de se relever horizontalement, au moment de la systole.

"Cette insuffisance mitrale spasmodique, ne s'observe par seulement dans les névroses et sous l'influences des émotions. . .

"Cette théorie a été récemment développée par Cuffer et son élève Roger."

The occurrence of relative insufficiency of the mitral valve is, therefore, fully admitted at present, and a good reason for this, which is also frequently mentioned nowadays, is the fact that the phenomena which we are accustomed to regard as positive proofs of an insufficiency may disappear entirely when the entire body, and with it the heart, is brought under better conditions of nutrition. [The subject has been particularly well described from the clinical standpoint by Morton Prince. See especially his article "Physiological Dilatation and the Mitral Sphincter, etc.," "American Journal of the Medical Sciences," February, 1901.—Ed.]

In what manner does the musculature assist in the closure of the semilunar valves? This question naturally arises if it be true that a perfect closure of these valves is impossible without the assistance of muscles. But when we attempt to answer this query, we meet with several difficulties, inasmuch as there are several things which cannot be brought into agreement with that which has been formerly regarded as correct.

The semilunar valves close during the diastole of the ventricle, and remain open during the systole. The older view contends that their closure is effected by the pressure of the blood within the first portions of the great vessels in front of the valvular pockets after the termination of the systole, and which is there under high pressure communicated to it as the result of the ventricular systole. The pressure behind the semilunar valves in the ventricle is now at zero, or is even negative; the blood, therefore, rushes in this direction under pressure, and thus unfolds the valves, the free openings of which lie in the direction of the movement of the blood. This interpretation seemed so plausible that scarcely any doubts were uttered against it. It was only Ludwig who apparently was not satisfied with it, for he remarks: "The contraction of the ventricle does not narrow the arterial orifices, so far as observation shows. We are not quite certain, as yet, how this takes place."† The further development of this question is due to Ludwig. The entire modern

* "Maladies du cœur," vol. vi of "Traitè de Médecine," edited by Brouardel and Gilbert, Paris, Baillière et fils, 1899, pp. 190, 191.

† "Lehrbuch der Physiologie des Menschen," vol. ii, second edition, 1861, p. 82.

Leipzig school stands upon his shoulders, as regards both physiologic and pathologic questions.

The new theory penetrates more deeply and in every direction into clinical medicine and also into diagnosis—the main pillar of medical activity. I shall limit myself here only to the discussion of those points which concern the subject under consideration. In the first place, why is it that we do not hear a systolic murmur over the aorta—limiting the matter thus for the sake of simplicity—under normal conditions? As a matter of fact, the conditions for the development of such a murmur seem to be present. When a slit—a narrow slit—is formed at the cardiac orifice of the aorta during systole, the blood under the influence of this systolic contraction is driven under high pressure into the large space beyond the valves, thus giving an opportunity for the formation of eddies such as are responsible for the production of murmurs. Krehl (see above, pp. 322 and 323) speaks of such eddies, and thinks they favor the closure of the valve.

The further questions which must be asked in this connection are intimately related to that just discussed: Why is it that a systolic murmur develops when the beginning of the first portion of the aorta is dilated? Why does the left ventricle become hypertrophied in aortic stenosis, when normally its function is to press the blood through a much narrower slit which is formed during the contraction of the muscle? Finally, it may be asked does not the propulsion of the blood through such a narrow slit necessitate the expenditure of a disproportionately large amount of the energy of the heart?

I have tried to find an answer to these questions, but I have not succeeded, and so I refrain from offering unripe and undeveloped ideas which cannot satisfy any one, least of all myself. On closer consideration it seems to me that the very complicated conditions which obtain in the heart might be cleared up, at least partially, by experiments. These possibly can give us new ideas, but at present it seems advisable not to venture too far. In any event, we may say this much: If a complete closure of the semilunar valve is only possible with a sufficient amount of systolic muscular work, then we may assume that an insufficient closure of the aorta may be due to an insufficiency of the corresponding muscles.

The phenomena which occur under these conditions at the origin of the aorta are still wholly unknown. One point is of importance: when such a degree of stenosis as has been assumed takes place normally as the result of the contraction of the muscles which close the valve, what will be the result when this stenosis occurs more slowly and is less complete?

COURSE AND DIAGNOSIS OF VALVULAR DISEASE IN GENERAL.

General disturbances are absent so long as the equilibrium remains undisturbed and the heart adapts itself to the altered conditions; in other words, so long as compensation—this is the usual term—is maintained.

It has been remarked above that a perfect compensation is never possible. That there is always a certain loss in the cardiac function as the result of valvular disease. This alone results in a more rapid fatigue of the heart muscle; for the work which is furnished by the organ is

diminished by the amount necessary for the propulsion of the blood, and this discount increases in a certain ratio to the amount of work required of the heart. The smaller this amount, the more the patient with a valvular defect can take care of himself, the longer will cardiac exhaustion be postponed. Yet the heart suffers such alterations as age brings more rapidly than under the same conditions of life in normal individuals. Possibly this is due to the fact that the vessels in the part of the circulation which is taxed more than any other for the purposes of compensation are more easily and rapidly affected by anatomic changes. Hypertrophy of the affected part causes increased pressure upon the walls of the vessels. This is shown most distinctly in insufficiency of the aortic valves, but by far of the greatest importance are the inflammatory changes in the vessels and the heart muscle which are so often found in valvular disease according to the beautiful investigations of Krehl and Romberg,* and which extend gradually in the course of time.

As a matter of fact, the destructive processes, once begun in the tissues, do not always cease immediately. This involves the supposition that, so long as such processes go on, some factor of inflammation must be present. What the nature of this may be cannot as yet be stated, nor can we say in what special relation the degenerative processes stand here toward the inflammatory processes. Such questions must be considered in detail in the discussion of myocarditis, but here it will suffice to point out that the compensation which has been fortunately attained in a valvular disease may be threatened from more than one direction, even though no undoubted fresh infection involving the entire body may supervene.

What phenomena are observed when compensation becomes disturbed—in other words, when "decompensation" develops? The answer to this is simple: cardiac weakness, with all its consequences.

These brief remarks will suffice in order to give a general review of the subject, and further details must be sought in the other portions of this work dealing specially with these subjects.

The *diagnosis* of a valvular lesion varies according to the degree of development and the degree of compensation of the lesion in question. The signs which point to a deficiency of the cardiac function may sometimes be very obscure, while in other cases they may be very prominent. These signs cannot be employed directly for the recognition of a valvular defect.

The physical examination, in my opinion, is best conducted in the following order:

1. **Inspection.**—In this we must see:

(*a*) Whether changes have occurred in the distribution of the blood over the surface of the body—engorged veins; cyanosis—and to what extent.

(*b*) Pulsation in places where it is normal or in abnormal localities. Do the former appear more or less marked than normally?

(*c*) What is the character and intensity of the respiratory movements? A trained eye sees many things which are less easily noted by other methods of examination. In all cases it is advantageous to employ more than one sense for the determination of the conditions present.

* See the recapitulation in Krehl's "Pathologische Physiologie"; also the original work in "Beitrag zur Pathologie der Herzklappenfehler," "Deutsches Archiv für klinische Medicin," vol. xlvi (1890), p. 454.

2. **Palpation.**—Palpation may be used for almost the same purpose as inspection. He who can feel the pulse correctly will usually make a more rapid diagnosis. This art has for a long time been underestimated, as compared with percussion and auscultation, and we are still very far from attaining the skill of the old physicians in feeling the pulse. I endeavor to teach my pupils what I myself have learned in this respect because I realize the great importance of this subject in practice. This is why I speak of it here with such emphasis. Many things that escape the eye, for example, a thrill, are revealed to the palpating finger. Another fact which must be remembered is that this method of examination when practised with expert skill is by far the most easily borne by the patient.

3. **Percussion.**—The findings on percussion are naturally of great importance because they show whether the heart has become dilated, and if so, in what direction. I have described the method which I pursue for this purpose in another place (see Insufficiency of the Heart, p. 59). The level of the diaphragm should be determined on both sides in all cases.

4. **Auscultation.**—A number of points have already been mentioned. I may say, in addition, that the best results are obtained when the examiner places the stethoscope a little outside of the apex, and gradually moves it horizontally toward the left border of the sternum, and thence in a vertical direction, changing its position each time to the extent of the diameter of the bell of the instrument. In this manner one can tell distinctly, by the change in the accentuation of the sounds, when one passes from the region of the mitral into that of the pulmonary artery. I am accustomed, next, to examine the sounds over the aorta, but I never neglect to listen to the ascending aorta (at the sternoclavicular articulation) in addition to auscultating over its place of election (inner border of the second intercostal space and the third rib on the right side).

The next step is the investigation of the sounds over the aortic arch, the subclavian, and the carotid. When necessary, the auscultation of the peripheral arteries follows. In order to determine the intensity of the diastolic tones over the pulmonary and over the aorta one should not listen to more than a few heart-beats in the proper places. I think that in this way it is easier to arrive at a decision than if we listen to a longer series of heart-beats. Possibly the reason of this is that we get tired more easily when we listen intently, at the same time taking into account the quality of the sounds. Therefore we should consider separately the question as to whether the tones are pure, and then should compare their intensity.

The auscultation of the tricuspid valves is the last step. It is not of any great importance in what order one auscultates, but one should become accustomed to employ a certain order and adhere to it. In this way, I believe, one can train one's ear much better.

He who trusts the statements of text-books will not think that the recognition of a given valvular defect is difficult; for so many and such definite signs are given and the recognition of these signs is so simple that even one who is but little skilled can scarcely miss the correct diagnosis. Such is the opinion of the young physician; but he who has grown old at the bedside and who has learned what the autopsy table teaches thinks otherwise. He knows how deceptive is the interpretation of these phenomena which are by no means so easily recognized. Often enough the older man contents himself with a "*non liquet*" when absolute

certainty seems warranted, while the autopsy brings disappointment to the overconfident.

Oscar Fraentzel,* one of the cleverest diagnosticians trained in Traube's school, has expressed this with praiseworthy frankness, and I agree with him perfectly.

Why is this so? The diagnosis of these cases was formerly based upon observations of fully developed, "pure" cases. The observations made in these cases, it was believed, were referable to changes in the mechanic conditions under which the heart had to work with its injured valvular apparatus. Mechanic causes give rise to mechanic effects, and no third factor could be admitted. Therefore the interpretation was invariably the same. Not infrequently the mechanic effects that were thought inevitable were actually referred to mechanic causes which were assumed to exist.

Where does the fallacy of such a calculation lie? This mode of thinking took into consideration the permanent tissue changes only, *i. e.*, the gross anatomic lesions. Sufficient appreciation was not given to the fact that a variable force which is altered within a short time—the action of the heart muscle itself—takes part in the function of the valves. It is not astonishing that such was the view taken, for this mechanism was known far too imperfectly at that time.

Liebermeister† expresses the older view very clearly in the following passage: "In discussing the phenomena and results of valvular lesions, we can employ a mode of reasoning which is, so to speak, the reverse of that usually employed in other pathologic fields; namely, the deductive method, which considers all the individual facts as consequences of certain given hypotheses. With the aid of a few general principles we are actually in a position to deduct purely theoretically, in a manner quite impossible in other sections of pathology, both the principal consequences as well as the subordinate phenomena, simply from a knowledge of a given condition in the valves. Although a number of difficulties are still to be overcome, and although not every author has always been fortunate in his deductions, these things are explained by the extremely complicated character of the subject. It may be said, however, that the conclusions obtained by the deductive method correspond very closely in all essential points to the result obtained by direct observation. . . In addition to the physical condition, we must also consider biologic conditions in the discussion of the results of valvular lesions. These biologic postulates cannot be deduced through mathematic processes, but must be obtained by actual experience. Yet the most important features of these biologic conditions may be reduced to a few principles, and these may be simply included in the premises from which the deductions are made. . . . The most important biologic principles relate to the behavior of the individual sections of the heart in the various valvular lesions, and especially to the hypertrophy and dilatation of the organs."

In my opinion the following explanation is justified by both the old and the new knowledge on this subject: Completely developed and perfectly compensated valvular lesions produce signs which are, for the most part, referable to the existing mechanic conditions, or else are deducible from these conditions. Their biologic sequels are immutably defined, so far as this may be recognized from without, and thus a knowledge of

* "Vorlesungen über die Krankheiten des Herzens," vol. ii, pp. 78, 79.
† "Lectures," vol. iv, pp. 354, 355.

these sequels facilitates and supports our understanding of what has happened.

In such typical cases we can apply typical rules, but these rules do not apply when the following conditions obtain (compare cases I and VI below):

1. A change in the valvular apparatus has taken place, but it is not so considerable that it produces permanent mechanic disturbances in a manner sufficiently marked for their recognition. There may be temporary mechanic disturbances, depending upon the behavior of the heart muscle. A transient fatigue of this muscle as the result of overexertion, no matter how the latter takes place, may suffice. Thus it happens that opinions must needs differ as to whether a valvular lesion exists or not. Not only is this a matter of doubt for the physician who must rely upon single or few examinations, but also for him who observes the case for a long time. Even those who watch the case for some time will often remain in doubt, and will not always be able to reject the possibility of a simple muscular disturbance (compare cases I and VI, below).

2. As soon as compensation fails, there is a change in the conditions which allowed the heart muscle to maintain the circulation in as favorable a manner as possible under the circumstances. If this change takes place rapidly, as, for example, in a newly recurring endocarditis and myocarditis, then the clinical picture will change so vastly in a short time that only those who have seen the patient before can understand it. All the signs which are furnished by our methods of examination change in these cases in a short space of time; and, especially, it is scarcely possible in such cases to be certain whether a new valvular lesion is developing alongside of the old.

If the failure of compensation comes on slowly then the phenomena of the valvular lesion remain perceptible for a certain time, but finally are lost in the general picture of cardiac failure.

3. In contrast to the cases in which the presence of a valvular lesion is not easily recognized, or else is not discernible at all, there are other cases in which a valvular lesion seems to be present, although it does not exist in reality. We have already spoken of this class of cases in another place (Cardiac Insufficiency, p. 61).

If we sum up everything that has been said, we find that it is the heart muscle that creates our difficulties in the diagnosis of valvular lesions. The question, therefore, must be considered somewhat more in detail:

What value shall be attached to the physical signs in the diagnosis of a valvular lesion in general, and in the determination of its seat in particular? Certainly this value must vary considerably, but it cannot be emphasized too greatly that the least value should be attached to the phenomena of auscultation.* And yet these signs are held most highly, as a rule, by physicians with insufficient experience.

Whoever teaches at the bedside knows how difficult it is to make it clear to students that when a "murmur" is heard the diagnosis of "valvular disease" cannot be made from the location where the sound is heard loudest, much less so the determination of the exact valve involved. Why is this so?

1. The sounds which we know as valvular sounds are, acoustically speaking, noises which arise through irregular vibrations. It is true

* Fraentzel says also (*loc. cit.*, p. 84): "The heart murmurs should play the most subordinate rôle in the diagnosis of cardiac murmurs."

that they make the impression of musical tones, because they are repeated at certain intervals, are separated from each other by certain definite periods of time, and are always the same in quality. Yet, each individual observer is bound to form his own conception of what constitutes a pure heart-sound. This conception is, and remains, purely subjective.

Possibly the explanation why more or less agreement exists as to what constitutes a pure or an impure heart-sound, as heard by the trained ear, is as follows: The trained observer has accustomed himself to pay no

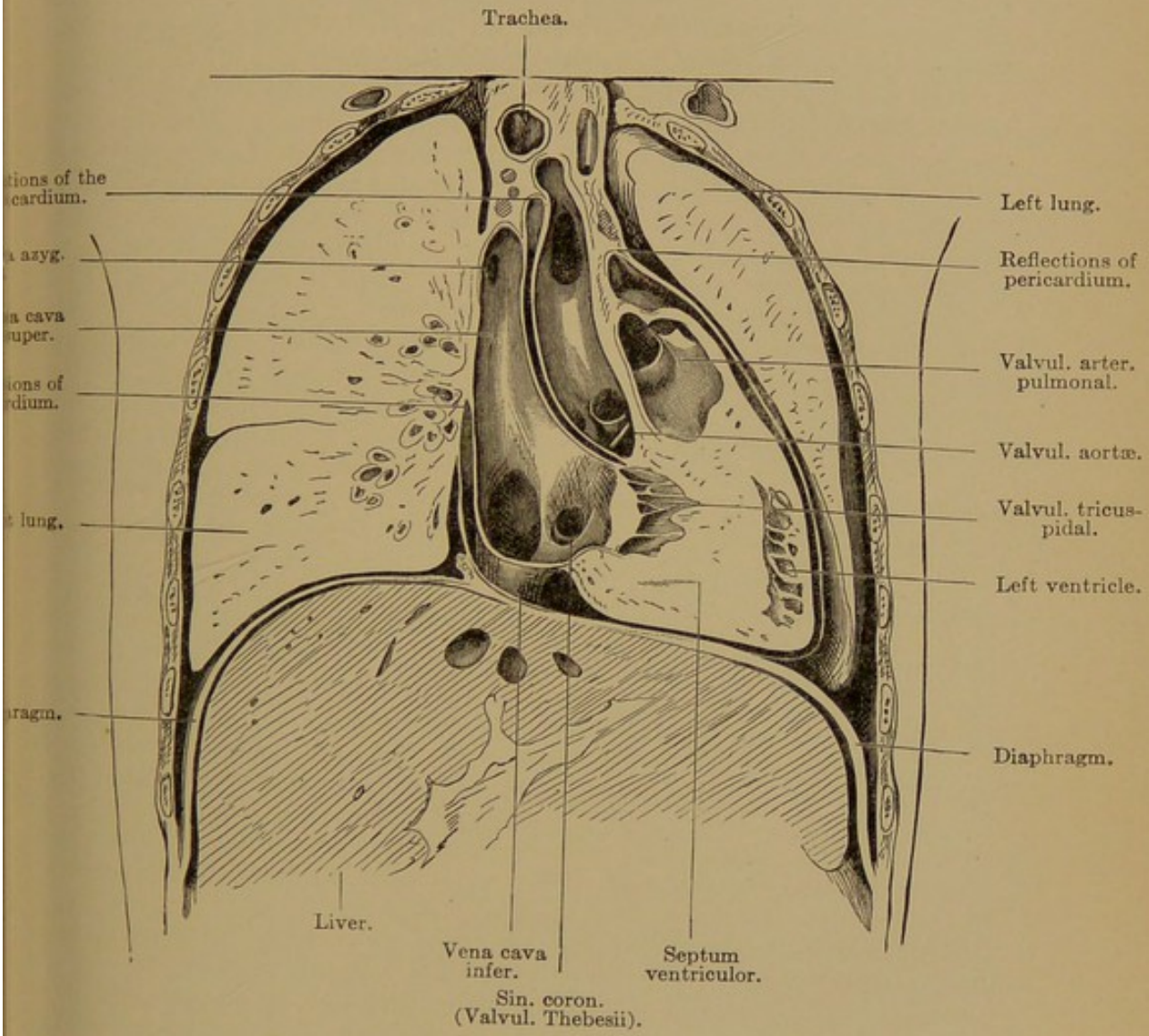


FIG. 54.

attention to minute variations in the sounds, and he habitually compares the auscultatory picture with that which he holds in his memory, which is kept fresh by the repetition of the same sensation. This is probably the reason why a person with an acute ear, but without an intimate knowledge of the heart, can hear "murmurs" more easily. It has happened, not infrequently, in my experience, that I had to admit the correctness of an observation by a beginner who heard "murmurs" which I had at first overlooked, or rather to which I had not paid any attention. The reason of this was that the experienced ear from the first is accustomed to exclude

the unessential features of the sensory impression, with the result that these features of it escape his observation. Analogies may be found in every field of experience.

2. It is not always possible to define the place at which a murmur has its origin. This is rendered difficult through the fact that, owing to the position of the heart, the vibrations which arise in its valves, and which produce auditory sensations, must traverse certain spaces and must overcome certain obstacles before they can reach the ear. At times the path for their transmission is the same or nearly the same. The following very clear description by Merkel* gives a definite idea of the conditions which control the transmission of cardiac sounds:

"In order to understand the relative position of the valves toward each other, and toward the chest-wall, we can begin by remembering that these valves would lie in a horizontal plane if the axis of the heart were placed vertically.

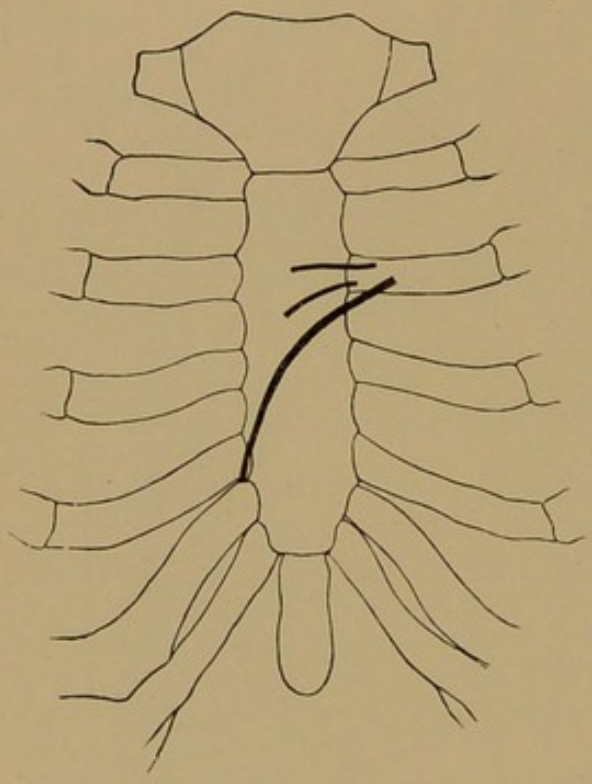


Fig. 55.

"The apex of the heart, however, rises so as to touch the thoracic wall in front. The horizontal level is thereby placed at such an angle that the posterior circumference is lowered and the anterior is raised. This can easily be illustrated with a piece of cardboard. Now, the lower apex of the axis also deviates to the left, whereby the right lateral and posterior boundary of the plane is placed lowest of all. The pulmonary valves lie closest to the front of this imaginary plane, and therefore occupy the highest position of the apparatus. The aortic valves lie somewhat farther back (see Fig. 54). Then come the left and finally the right auriculoventricular valves, the latter being the lowest of all.

"An attempt to define the positions of the valves by means of lines drawn upon the anterior surface of the chest can only be partly successful, for the reason that the attachments of these valves do not represent straight lines. As it is desirable to represent as accurate a picture as possible of their location by the simplest available methods, I recommend the use of the three lines drawn in Fig. 55. The uppermost of these lines lies at the upper border of the sternal end of the third rib on the left side, and one-half of it lies upon the rib itself, while the other half lies upon the sternum. This line indicates the position of the right arterial orifice. The second line begins in the middle of the sternal end of the third rib on the left side, and reaches across the middle of the sternum, running almost parallel to the third line—that is, in a slightly downward direction. This last line corresponds to the position of the left arterial orifice. The lowest

* "Handbuch d. topogr. Anatomie," vol. ii, p. 353, *et seq.*, Braunschweig, F. Vieweg & Son, 1896.

line begins at the lower border of the third rib on the left side, scarcely a finger-breadth away from the sternal border, and arches behind the sternum down to the sternal border of the sixth rib, on the right side. Its lower portion, up to the level of the third intercostal space, corresponds to the position of the attachment of the right venous orifice (tricuspid), while its upper portion corresponds to the left venous orifice (mitral).

"If a heart which is otherwise normal lies somewhat higher or lower than usual, then all we need to do is to determine the position of the apex, in order to be able to judge how much higher or lower the three lines drawn must begin.

"The position of the valves is, to a certain extent, very unfavorable for the purposes of auscultation. In the first place, because the two arterial orifices and the left venous orifice are crowded into a space which scarcely exceeds the width of one rib, and, secondly, because the valves of the left side of the heart are turned away from the thoracic walls. Even the aortic valve is crowded away from the wall of the thorax by the thickness of the pulmonary artery which lies in front of it. But the venous valve of the left heart lies lowest of all, and it can scarcely be expected that it can be reached by auscultation.

"In practice we are obliged, therefore, to refrain from placing the stethoscope accurately over the valves of the left heart itself. We utilize instead the transmission of sounds which takes place in the heart-walls and in the column of blood in order to isolate the tones from each other at convenient points.

"The sounds of the left venous orifice (mitral valve) are best heard over the apex. In listening to the sounds of the left arterial orifice, the stethoscope is placed in the second right intercostal space, directly adjoining the right sternal border. At this point the ascending aorta, and not the valve, is nearest to the walls.

"The valves of the right side of the heart may be reached quite accurately by listening over their thoracic projections. The right arterial orifice is best auscultated in the second left intercostal space, immediately adjoining the sternal border, while the right venous orifice (tricuspid valve) is best heard at the median line of the sternum, at the level of the fifth costal cartilage—a place which lies at about the middle of the insertion of the valve."

In spite of the difficulties due to the peculiar local conditions encountered, we can usually get our bearings, and can localize the sounds heard over the heart with considerable certainty. We succeed in doing this chiefly because in normal hearts the sounds which we auscultate at the "points of election" possess a definite quality, and the mental picture of this quality becomes thus a standard which we can use to detect any deviations that may be present.

These are the facts. Their interpretation, however, is another task. When, for example, the diastolic tone over the pulmonary artery is heard louder than that over the aorta, the reason for this change may lie in the increased tension of the valves of the former, which is induced through an increase in the labor of the right ventricle. But the increased intensity of this sound may also be caused by the diminution of the resistance offered to the transmission of the sound, the diminution being most frequently due to a retraction of the part of the lung which covers the pulmonary artery.

If this applies to the cardiac tones, can the same also be said of the

murmurs? The direct evidences of the senses teaches that the sounds are no longer pure. We assume that these sounds originate in the place which corresponds to the origin of the normal tones; while this is frequently so, it cannot be said that this is an invariable rule. In all probability variations in resistance of transmission also play a rôle under these conditions. But our knowledge as to the behavior of these resistances is extremely scanty. Pure tones are transmitted more readily than the murmurs which develop in the heart—that is about all that we can affirm from experience.

The origin of the murmurs which are most apt to mislead us must be referred to the musculature of the heart. When the heart is hypertrophied or dilated, it changes its position in the chest cavity, as well as in relation to the chest-wall, and very often, also, the adjoining lung expands otherwise, under those conditions, than in normal individuals. Thus, it may occur that the place where the murmurs are formed is transferred to a position which is near enough to the normal position of a valve to allow us to mistake the latter as the point of origin of the murmur.

3. The murmurs which are heard over the heart arise through vortex movements or eddies of blood. These take place in the blood as the result of the collision of two streams moving in opposite directions, or else as the result of sudden and considerable changes in the diameter of the stream. These vortex movements only become strong enough to produce audible murmurs when the velocity of the blood-stream is sufficiently great. The amount of vital force which is supplied by the laboring heart is, therefore, an important item in judging the significance of a murmur in diagnosis. Therefore the decisive factor, once more, is the heart muscle.

Fraentzel* says, correctly, at the beginning of his rules warning the diagnostician: "In the diagnosis of valvular lesions of the heart we should refrain from giving a definite opinion as to the character of the cardiac affection present *sub finem vitæ*—a period which is, comparatively speaking, not difficult to determine."

This principle must be expanded, however, for it applies to cardiac failure, no matter from what cause it may have originated. It applies particularly in severe recurrent endocarditis and myocarditis, no matter whether they terminate fatally or not.

4. It is of great importance for diagnosis to determine whether a murmur is heard during systole or during diastole. This is only possible when the number of contractions of the heart do not exceed a certain limit within a given unit of time. When the beats follow each other in too rapid succession, even the most accurately trained and most acute ear can no longer distinguish the cardiac phases with absolute accuracy. It depends, naturally, entirely upon the individual when this limit is reached. The experienced observer can distinguish diastole from systole longer with more rapid beats than the inexperienced man. There is no question, however, that a considerable impairment in the valuation of auditory sensations for the diagnosis of valvular lesions obtains as the result of this incapacity.

Heretofore it has been deemed sufficient to distinguish the changes perceptible to the ear and to the touch which take place during systole from those that occur during diastole. It is natural, however, that the principal attention should be paid to the left side of the heart, and especi-

* "Vorlesungen," p. 69.

ally to the left ventricle. Some have tried to refine the observations, to extend them to the chief divisions of the cardiac phases, and thus to gain profit for the diagnosis of valvular lesions. Recently Martius, to whom we are indebted for beautiful work in this special field, has summarized those points in this connection which are important clinically.*

He shows that the *systole of the left ventricle* is divided into two periods:

1. *The Period of Closure.*—This begins at the moment when the ventricle begins to contract, and lasts until the blood which is contained in it has been placed under such high pressure that it overcomes the resistance in the aorta and forces open the valves in this vessel which have remained closed until then.

2. *The Period of Expulsion.*—The ventricle remains strongly contracted because it must empty the blood which it contains into the aorta. The period of expulsion is followed by a short period of rest, characterized by the relaxation of the ventricle. The diastole begins only when the contraction of the ventricle has ceased entirely. The apex-beat falls into the period of closure, which, for the human heart, lasts 0.07 second.

As Ludwig† showed many years ago, the ventricle undergoes a change in both form and position during contraction. While this chamber is somewhat elliptic during the diastole, it tends to become spheric during systole. The small diameter of the ellipse must, therefore, be converted into the large diameter of the sphere which is formed in systole, and thus the adjoining parts must be forced outward in this motion. The lungs which are normally easily movable adjoin the base of the heart, and, therefore, we do not recognize any movement there. At the apex, however, which is close to the chest-wall, and is only covered by a very thin layer of lung tissue, conditions are entirely different.

The stiffened apex seeks to place itself at right angles to the base of the heart, but it meets the resistance of the thoracic wall. The muscular left ventricle, which forms a mass of considerable size, with its closely surrounded fluid contents, the blood, gains enough initial velocity in its rapid contraction to disturb the equilibrium of the obstacles before it. The intercostal muscles, therefore, yield and are pushed outward; the ribs are thrown into strong vibrations, and may even be so distended (in hypertrophy of the heart muscle) that they give place to the on-pressing heart. In this manner precordial bulging is formed.

All this can only take place during the period of closure. During this phase of the cycle the auricular valves, as well as the aortic, are closed, and the heart, with its contained blood, forms a unit which loses its greatest mechanic power as soon as its mass becomes smaller. This occurs when the blood can escape from the ventricle into the aorta, and when the heart does not gain a corresponding increase in vital force furnished by muscular action, as may be seen from the pressure-tracing of the ventricle, as compared to that of the aorta.

The heart cannot slip backward; for, as Braune has shown, "the posterior wall of the pericardium is reinforced by numerous fibrous bundles which radiate into it. This wall is strong enough to resist the movements of the heart and to form a base of support against which the posterior wall of the heart can gain a purchase during its contraction" (cited by Martius, p. 10).

* "Methodologie als Einleitung in die Lehre von den Herzkrankheiten," "Die Deutsche Klinik am Eingange des XX. Jahrunderts," by E. v. Leyden and F. Klemperer, vol. iv, 2, p. 1.

† See explanation in his "Lehrbuch," pt. ii, pp. 85, 86, of the second edition.

The apex-beat must necessarily occur during the period of closure and before the ascending (anacrotic) limb of the arterial curve. Martius correctly points out that the clinical value of the apex-beat must include only that which is expressed in the term itself: the concussion or projection of the thoracic wall produced by the contraction of the heart with closed valves.

The manner in which the thoracic wall returns to its normal equilibrium is expressed in the descending portion of the cardiac tracing, but is practically of no importance to the physician.

It should be noted that the part of the cardiac cycle which has been called *"the back stroke"* by James Hope, occurs in the period of expulsion, and, therefore, represents an active process: the shrinking of the ventricle due to its emptying into the aorta (interpretation given by Martius).

The *beginning of diastole* is characterized by the tone which occurs when the aortic valves close. The heart-sounds occur in certain definite phases of the cardiac cycle. The systolic sound occurs in the period of the beginning of closure. R. Geigel, with whom Martius agrees,* concludes from this fact that: "During the period of closure there must be mechanic conditions which produce the acoustic phenomenon known as the systolic sound. During the period of closure there is as yet no propulsion of blood, but the ventricle, which had been flabby until then, suddenly becomes contracted around its contents. The structures surrounding the ventricle (muscular wall and valves) at that moment very rapidly enter into a new state of equilibrium, in which they vibrate, until the inert contents dampen the vibrations. These vibrations form the first sound.

"The first sound, therefore, must be absent when the period of closure fails (or, more correctly, when closure is lacking in that particular phase of the cycle). This is confirmed by thousands of observations. If the auricular valve is open, the contracting heart muscle drives a part of the blood directly into the auricle, and the heart muscle does not enter into a new state of equilibrium about which it can vibrate, but it gradually follows the escaping blood until the pressure of the aorta has been overcome. In place of the sound there arises, then, a murmur due to the fact that the blood which escapes through the slit in the valvular flaps makes these flaps vibrate like the lips of a whistle. This also explains why the murmurs are usually longer in duration than the heart-sounds. In the production of the heart-sounds the elastic walls of the ventricle are thrown out of their equilibrium but once, and vibrate about this new state until external resistances very rapidly produce a dampening of the vibrations.

"On the other hand, when a murmur occurs, the elastic wall is repeatedly brought out of its equilibrium during a shorter or longer space of time, through an external influence, and the dampening of the vibrations takes place only when all the external causes of the murmur—the blood-stream—cease or are no longer sufficiently strong."

So far as I can judge, this explanation is better and certainly more easily understood than those offered by previous authors. Whether it will stand the criticism of physicists I cannot say.

The scheme devised by Martius must also be mentioned here. It gives a clear idea of the relation of the cardiac phases to the tones and murmurs due to disturbances in the valves or in the orifices.

* *Loc. cit.*, p. 9.

MARTIUS' SCHEMA OF THE CARDIAC CYCLE IN RELATION TO SOUNDS AND MURMURS.

A. Mitral Insufficiency.	B. Aortic Stenosis.	C. Aortic Insufficiency.	D. Mitral Stenosis.
Ventricular Systole.		Ventricular Diastole.	
Period of Closure. First sound and apex-beat. (*a*) Closure of the Auricular Valves.	Period of Expulsion. Back-stroke. Beginning of the aortic pulse. (*b*) Opening of the Semilunar Valves.	Second sound. (*c*) Closure of the Semilunar and opening of the Auricular Valves.	Auricular Systole.
a	*b*	*c*	

Martius correctly includes the systole of the left auricle in this scheme; for, undoubtedly, this systole has an influence in the formation of the murmur of mitral stenosis (see p. 364). The presystolic murmur has gained an established place in diagnosis. On the other hand, the same cannot be said of the peridiastolic and prediastolic murmurs which have recently been defended by Senator.* These murmurs occur between the end of the systole and the beginning of the diastole; in other words, while the auriculoventricular valves are still open for the escape of blood. Senator believes that these murmurs are produced as the result of the fact that the papillary muscles do not remain contracted to their full extent, and thus allow the valvular flaps to be so separated from each other that a portion of the blood which is still under higher pressure in the ventricle escapes into the auricle. In the aorta this is said to take place at the beginning of the period of closure, because the blood which still is under high pressure in the aorta flows back into the ventricle.

In considering these possibilities, we must take into consideration chiefly the reinforcement of the valvular closure by the muscular rings which are specially provided for this purpose. A premature relaxation of these rings in the period of expulsion, and a retarded contraction in the period of closure, would be the factors involved.

The murmurs which are conducted by the blood in the direction of the current through the arteries are of great importance in diagnosis. This is due essentially to the fact that the vibrations are, to a certain extent, dependent upon the course of the vessels, and that, therefore, there is less doubt as to their origin. We must not forget, however, that murmurs may occur from dilatation of the aorta itself, as well as from valvular disturbances. When the former condition obtains, other methods of examination must be used to determine their presence. This, however, is not always possible.

The sounds formed in the arteries allow us to draw conclusions as to the force with which the left ventricle is working, and indirectly as to the presence of valvular defects which may have induced hypertrophy (aortic insufficiency).

When vibrations which are perceptible to the ear are also discernible on palpation, we must conclude, in general, either that the force which has produced them has increased, or else that the resistances which were opposed to their transmission have decreased. Both factors are possible, but the former is more frequent. On the whole, in such cases, it becomes

* "On Modified Systolic so-called Peri- and Prediastolic Murmurs in the Heart," von Leyden's "Festschrift," vol. i.

still more difficult to determine the place of origin of the vibrations, inasmuch as the surface over which they are perceptible becomes more extensive. In such cases the auscultation, and even the palpation, of arteries is of assistance. Yet we must be cautious in drawing conclusions, for it may happen that the murmurs are loud enough to be transmitted in continuity, independently of the conduction by the blood-stream. Thus we had in the Policlinic a case of severe insufficiency of the mitral valve, with accompanying stenosis of its orifice, in which, when the heart's action is vigorous, the murmurs may be heard even over the arms,—the left as well as the right,—when these limbs are horizontally extended.

In order to interpret correctly the signs which point to an enlargement of the entire heart, or of one of its parts, the following points should be noted: In the first place, we must consider the behavior of the lungs, *i. e.*, whether they cover the heart in a normal fashion, or whether they extend over a larger or smaller surface than ordinarily. We have already repeatedly spoken of the necessity of determining these facts.

Percussion shows us directly only the extent of the surface over which the heart gives a diminished or dulled sound. Whether this is the result of an increase in its muscular mass or whether it is due to a dilatation of its cavity, and if so, which particular portions are involved, are quite separate questions. In addition to the shape of the area of dulness,—this is a factor of first importance,—we must pay attention to the character of the cardiac action. The points to be noted in this examination need not be considered in detail at this point. I need only point out, once more, that when compensation fails, the rules which apply to the changes in the area of dulness of well-developed valvular lesions lose a great deal of their original value, in proportion as the loss of compensation, that is, the heart weakness, progresses. Conversely, when the cardiac weakness improves, the area of dulness may return into the shape and size dictated by the valvular lesion present.*

The pulse may give indications of great importance, and in some cases of vital importance, in the diagnosis. Yet changes in the pulse are of importance only in diseases of the aorta, for truly characteristic deviations in the type of the pulse are not found in disease of the mitral valve. The pathologic venous pulse may also be mentioned in this connection; for this phenomenon, under proper conditions, may alone be sufficient to indicate the presence of tricuspid insufficiency.

A good old custom is that more than one sign is required for a classic diagnosis of a completely developed lesion. If the diagnosis shall be certain, then the demonstrable signs must be complementary to one another and must furnish an expression for the functional disturbances which necessarily coincide with the anatomic changes. If the disturbances are not confined to one valve, then the clinical manifestations become less characteristic in all directions, and it is very difficult to decipher the exact conditions from the composite picture. Fraentzel,† therefore, is right in warning us that we should not assume the presence of a complicated valvular lesion simply from some deviations from the ordinary picture.

* Compare p. 57, case III.

† "Vorlesungen," p. 83.

GENERAL PROGNOSIS OF VALVULAR LESIONS.

It is often asked whether it is possible, and if so, to what extent, to predict the influence of a valvular lesion upon the duration and the course of life. How long can the patient live and how much of his working-capacity can he retain? Whenever a general answer is demanded, we must decline to commit ourselves. Such an answer cannot be given for an individual valvular lesion, but only for an individual case, and even then it must be given with a great deal of caution.

The facts to be considered foremost are: I. What takes place when a valvular lesion is present which has no tendency to grow worse, and remains unchanged for a long time? Under these conditions our calculation can be fairly accurate. We must consider: (1) The extent of the injury present; (2) the completeness of the compensation of the changes which have taken place in the heart-pump; (3) whether the compensation is likely to be maintained.

First of all, the mechanic conditions which obtain in each case must be considered. A part which is made up of weak muscles behaves differently from one which is strongly built. The auricle, therefore, cannot be placed on the same level as the ventricle. Therefore, even if the heart were so constructed that a certain amount of valvular lesion could be present without injuring this organ, how great would then be the permanent demands upon the capacity of that part of the heart which is chiefly drawn upon for the purpose of compensation? Can such an amount of blood be supplied to this part as is commensurate with its increased activity? In addition, those biologic values which never can be gauged *a priori* must receive full consideration: To what extent is the heart capable of adapting its muscular growth to the task required by the valvular lesions? Much importance depends upon the original constitution of the organ, and also upon the nutrition of the organism as a whole, especially blood formation, in spite of the disturbances in the heart.

In the final analysis, the "constitution" of the patient is of great weight in the prognosis; for the constitution determines his resistance against injuries of all sorts. Another thing which may be cited, merely as an example, is that the prognosis of certain cardiac affections depends almost entirely upon the question as to whether or not the patient is subject to bronchial catarrhs.

Next comes the consideration of the external conditions of the case: What requirements upon the heart are made by the patient's mode of life? Upon this factor depends the decision as to whether the heart is able to secure the periods of rest which are necessary to sufficient nutrition in order to protect it from fatigue and its consequences. All these questions have already been considered in a previous section (see p. 18 *et seq.*).

II. What takes place when the cardiac lesion does not remain stationary for any length of time?

We must not forget that it is always possible that we may have to deal with this question, as the number of recurrences in endocarditis is estimated to be about 75 per cent. of all the cases. We also must remember that atheromatous processes necessarily tend to become wide-spread.

Just when a recurrence may be expected, or just when the process may extend, we cannot tell beforehand, nor can we say how severe the new attack will be. Certainly, it may occur so late, and may be so slight, that it does not produce a great deal of harm, at least, not at first. On the

other hand, it may also happen that an insignificant valvular lesion which has been borne without any trouble for many years leads to death in a short time through recurrent endocarditis (see p. 196).

In general, we must content ourselves with the statement that the more frequent the attacks of endocarditis become, the greater is the probability that the valvular lesion will grow worse; in other words, that the compensation will be disturbed, and thus cardiac weakness induced. On the other hand, the longer the periods of inactivity on the part of the process, the less will be the danger. As regards the atheromatous changes, we must consider, in addition to the severity and extent of the tissue change itself, also the presence and extent of lesions in the coronary arteries, and, therefore, the blood-supply of the heart.

Let us now turn to the other side. Can a valvular lesion disappear? This is said to be possible.*

Of course, the great majority of such cases are insufficiencies of the mitral, and it is extremely difficult to say with certainty in this type of valvular affections whether they are produced by permanent tissue changes or not. However, when such trustworthy physicians as Gerhardt† affirm that they make these statements on the basis of years of observation, one can scarcely imagine that they have been dealing with transient functional disturbances. A diagnostic error, however, is less easily made when the valvular lesion is in the aorta, and in most of the few cases reported the condition was insufficiency. In these instances, also, a long period of observation was assured by experienced observers.‡ But what have been the proofs of recovery?

Von Leyden, and Fraentzel who agrees with him verbatim, believes that "we must understand by recovery not only the disappearance of the murmur, but also the permanent disappearance of all cardiac symptoms. The cardinal point, however, lies in the permanent disappearance of the murmur."

It may be admitted that such a disappearance deserves more attention in insufficiency of the aortic valve, simply because the murmur is diastolic. But is this disappearance a positive evidence of recovery? Von Leyden himself denies this when he says: "It has been shown that the diastolic regurgitation murmur at the aortic valves can disappear without removing the insufficiency and its mechanical results," and confirms this in detail. He also shows that diastolic murmurs which are not the result of anatomic changes may simulate valvular lesions.

From all this we must conclude that the expression "recovery from a valvular lesion" must be understood as follows: A true return of the injured tissue to the normal has not been demonstrated, but there may be such a complete disappearance of the functional disturbances that no trace of them can be found. This is accompanied by the signs which indicate the presence of valvular disease on physical examination, especially the murmurs. It is in reality of but little consequence whether we speak in such a case of recovery or of "complete compensation." The work of

* *Cf.* von Leyden, "Ueber die Prognose der Herzkrankheiten," "Deutsche medicinische Wochenschrift," 1889, pp. 418, 419, where copious bibliographic data occur; also O. Fraentzel, "Vorlesungen," p. 116.

† "Lehrbuch der Auscultation und Percussion," fifth edition, p. 328.

‡ *Cf.* von Leyden, *loc. cit.*; case reported by Senator ("Therapie der Gegenwart," new series, vol. iii, 6) deserves special notice. It is possible that complete recovery took place in this case.

von Leyden, however, shows that the discussion of these questions has been of great value.

"Until very recently a patient who had a perfectly compensated cardiac lesion, and who had been feeling perfectly well, was regarded as a candidate for death, who might die at any minute, whenever a cardiac affection was detected accidentally at an examination. The patient looked upon himself as doomed. The parents of an innocent child saw their dear one condemned to death. These severe pessimistic views have been replaced, as the result of advances in our knowledge, the importance of which should not be underestimated. For we have been more and more distinctly assured, as our medical experience increased, that such a perfect compensation can remain undisturbed for many years, and that the patient can enjoy life unmolested during all that time. . . ."

"Therefore it was a distinct achievement when Sir Andrew Clark,* of London, found 684 cases in which a chronic valvular disease of the heart had existed for at least five years without having given rise to any symptoms which obviously affected the health of the patient."

Such are the views of von Leyden, and every experienced observer must agree with him here, as well as with his further remarks: "We should not tell the patient with heart disease the whole truth, but we should not conceal from him the necessity of regulating his life in a proper manner." In other words, we should not say anything about the danger threatening him from a recurrent endocarditis, because we do not know ourselves whether such a danger is present, nor when it may threaten, any more than we know how one can avoid this danger.

We must tell the patient, however, that a heart whose valves are affected requires careful management, in order to avoid the possibility of its fatigue. The physician must decide the way in which this shall be done. His task, therefore, at the start, concerns itself only with the regulation of the patient's mode of life. When signs of heart failure supervene, then everything else that has been said concerning the treatment of heart weakness comes into play. The various valvular affections present, at times, special problems, which are considered in connection with the description of each of these lesions.

THE INDIVIDUAL VALVULAR LESIONS.

In the days when the valvular lesions—insufficiencies and stenoses—occupied the foremost position among the diseases of the heart, a greater importance was attributed to the anatomic and the clinical details of the individual lesions than is usually done at present. I think it is desirable to review some of the facts, following largely the treatise of Bamberger, which presents the best consideration of this subject. In many places I quote literally, preferring the original to circumlocution. Honor to whom honor is due—and let every one have his own.

Among the newer non-clinical researches, I have considered the experiments upon models as well as the purely theoretic considerations of Benno Lewy. Both these lines of research give important glimpses into the mechanic conditions underlying the different heart lesions. The newer view of this subject places the heart muscle in the center of everything. This means that we are face to face with a biologic problem the

* "Report at the British Medical Assn.," 1887.

solution of which can only be compassed when we gain a thorough understanding of the normal conditions of valvular action. Unfortunately, we are still far from perfect in this respect.

INSUFFICIENCY OF THE MITRAL VALVE.

This is the most frequent valvular lesion (see above, p. 311), occurs chiefly as the result of endocarditis, and is, therefore, frequently seen in young subjects.

Marked tissue changes in the cardiac muscle are often followed by a more or less insufficient closure of the mitral valve. A similar insufficiency takes place when the cardiac muscle is temporarily (infectious diseases) or permanently (cachectic conditions) affected in its nutrition through an impoverished blood. In these conditions, also, there may be a less rapid response to the impulses which depend upon the integrity of the

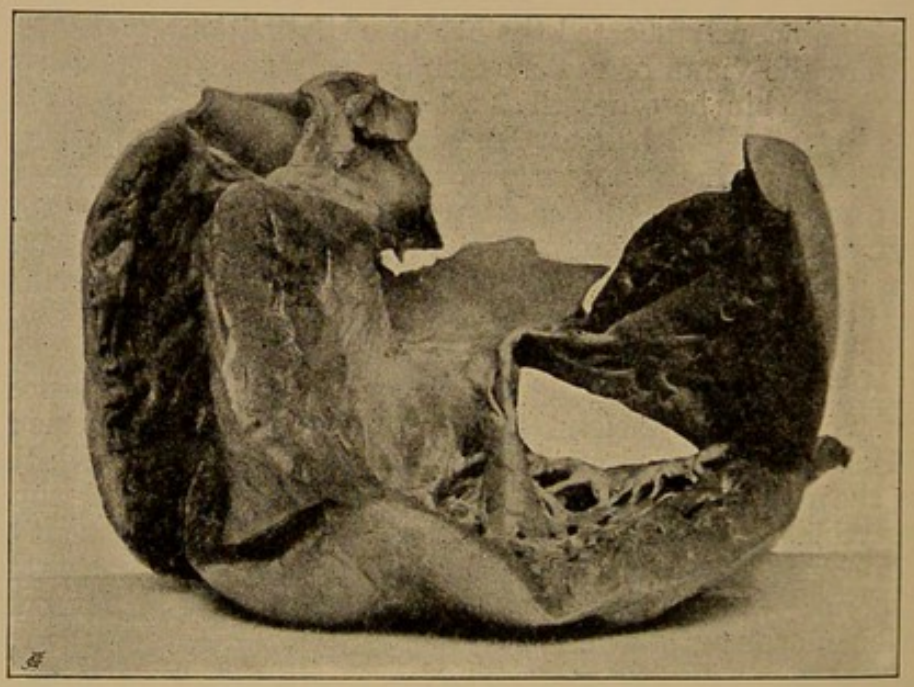


FIG. 56.—Pure mitral insufficiency, seen from the ventricle (from a preparation in the Pathologic Institute, Tübingen).

nervous system. This slower response might be the result of some defect at any point in the path of the nerves.

It is the custom to give a complete description of each valvular lesion, although mitral insufficiency, like the other lesions, according to the general belief, but rarely occurs in its pure form, that is, confined to the mitral valve and without any accompanying stenosis.

It would not be correct, from a clinical viewpoint, to include here the muscular insufficiencies, although their mechanic results are at first the same as those produced by local changes in the valve. The custom is not to include them in the ordinary class of mitral insufficiencies; in other words, they do not belong "officially" to this division of valvular lesions.

The signs which mitral insufficiency produces, so long as it is compensated, are as follows: Hypertrophy of the right ventricle and a less marked hypertrophy accompanied by dilatation of the left ventricle; a systolic murmur which is heard most distinctly over the apex of the

heart; and an increased intensity of the diastolic tone over the pulmonary artery. The general health is not at all impaired, or is but slightly affected, through the fact that greater demands are made upon the respiration, just as soon as such a heart must work more vigorously.

When the heart muscle fails, then all the symptoms of insufficiency are developed in proportion to the degree of muscular failure. These symptoms have been described systematically in another part of this work.

The effects which the insufficiency of the mitral produces upon the circulation as a whole require further discussion. Its mechanic results may be recognized most clearly in a model which is so constructed that it allows us to represent the essential conditions needed for the maintenance of cardiac function, both in the normal state and in the state now under consideration. The apparatus constructed by Fr. Moritz* meets these requirements most satisfactorily. Numeric values, such as those which have been found in experiments, are, of course, only of relative importance. They show the changes which take place when the conditions of valvular actions are altered, and allow us to draw our own conclusions, whether such changes are apt to be slight or marked. These conclusions, however, cannot be opposed.

The following results were obtained in experiments with mitral insufficiency conducted with the aid of the model: The immediate effect is a lowering of the blood-pressure in the aorta, and a rise of this pressure in the pulmonary artery, as well as in the pulmonary vein. This is illustrated in Fig. 57 on page 344. While these changes in blood-pressure take place, the amount of blood expelled from the right ventricle changes to a certain extent (in the experiment of which we are speaking here it changed from 2.7 to 2.2 c.c.). The expulsion-volume of the left ventricle, on the other hand, is markedly increased (from 2.7 to 5.8 c.c.). It must be noted, however, that this figure for the left ventricle is only so high in the model, and must be much smaller in the human heart.

The left ventricle is dilated during diastole (in the experiment the dilatation amounted to not quite 10 per cent. of its contents), while the right ventricle is not at all or, in other experiments, only slightly, enlarged.

The working capacity of the left ventricle increased considerably (from 263 to 322 gram-centimeters). That of the right ventricle in this experiment was slightly lowered (from 181 to 158 gram-centimeters), while in other experiments it remained unchanged. When the expulsion-volume of the left ventricle was increased, then not only was an increase of the aortic pressure to its normal height possible, but also the pressure in the pulmonary returned to normal.

In experiment No. 44, performed immediately after No. 43, the work of the left ventricle was increased from 322 to 410 gram-centimeters. Then the relations of pressure were changed, as Fig. 57 demonstrates.

A complete compensation can, therefore, be produced by increasing the volume (dilatation) and by increasing the working capacity (hypertrophy) of the left ventricle. And the experiments show that the valvular lesion itself gives rise to conditions favorable to the development of these two elements of compensation.

Moritz explains these changes in the following way: "Inasmuch as

* "Ueber ein Kreislaufmodell als Hilfsmittel für Studium und Unterricht," "Deutches Archiv für klinische Medicin," 1899, vol. lxvi, p. 349 *et seq.*

the left ventricle empties a part of its contents backward into the auricle, the amount of blood and the pressure in the latter must increase. The

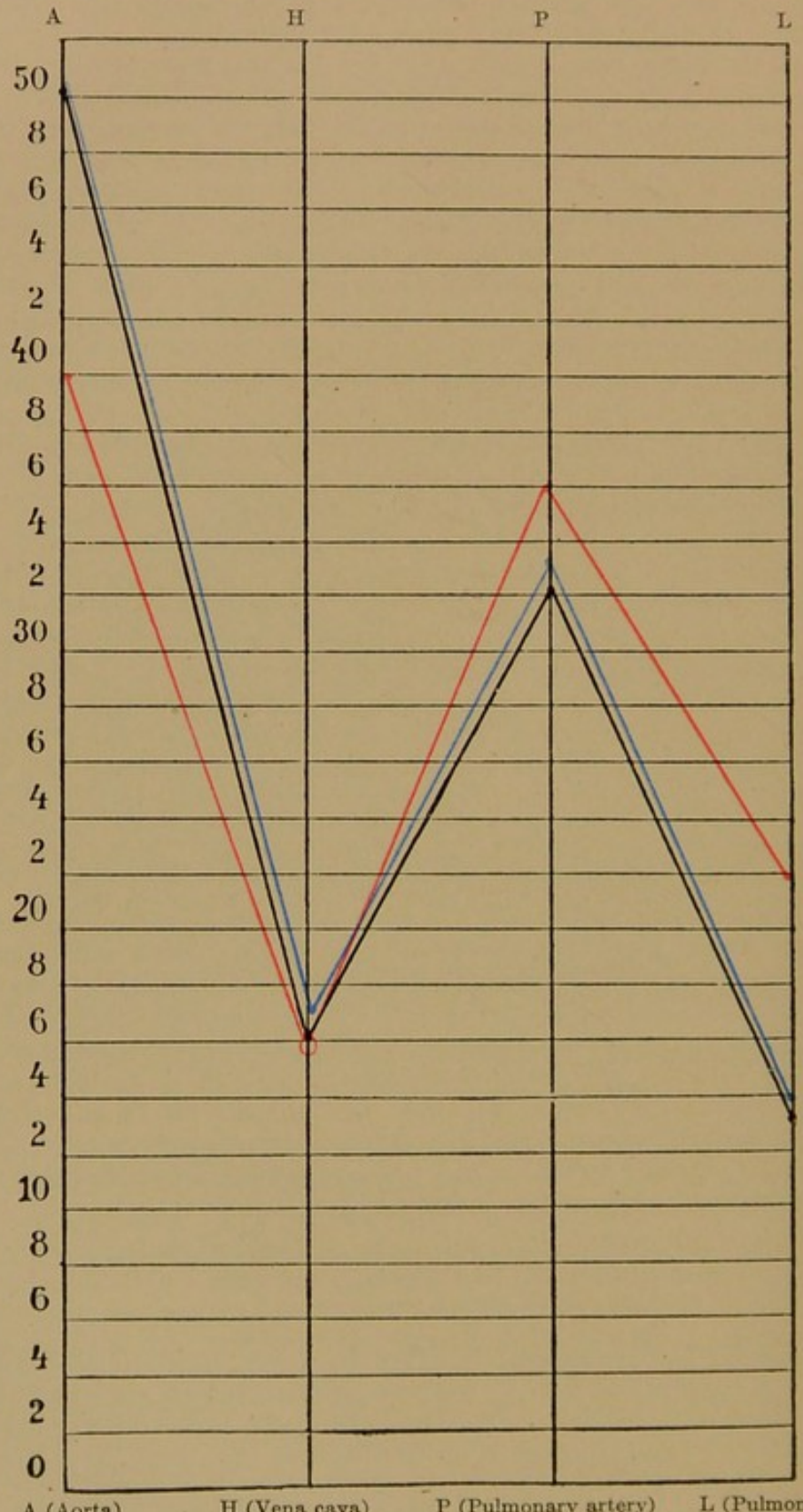


FIG. 57.—Moritz's Experiment 43.

fluid from the pulmonary artery, must, in consequence, accumulate, and, as the result, there is a rise of pressure in the entire pulmonary system. Now, inasmuch as the regurgitated part of the blood sent out from the left ventricle is lost for the aorta, this vessel must suffer the decrease in amount of blood and pressure.

"After the valvular lesion has become established, and after the conditions of pressure have become stationary, the expulsion-volume of the right ventricle, the valvular apparatus of which has remained unaffected, will indicate the amount of the fluid which passes in a cross-section of the circulation between two systoles. For the same amount must be driven from the left ventricle into the circulation, as otherwise no stability of pressure could exist. The difference between the expulsion-volume of the left ventricle and the expulsion-volume of the right ventricle is, therefore, equal to the amount of fluid which regurgitates."

The increase of the expulsion-volume from the left ventricle is produced as the result of the fact that the pressure in the outflow tube of the aorta is diminished, while it increases in the inflow tube of the pulmonary veins, and the volume of the ventricle during diastole increases.

The right ventricle does not change in volume, for the reason that the sinking of the aortic pressure compensates, or even overcompensates, the results of a lessened expulsion-volume of the right ventricle, and thus keeps the pressure in the vena cava at its original level.

The work of the left ventricle is considerably increased, because this part of the heart must propel quantities of blood against the pressure in the aorta and the pressure in the pulmonary veins. On the other hand, the work of the right ventricle is not increased, because it does not have to propel as great amounts of fluid, although it has to work against the increased pressure in the pulmonary artery (in this experiment).

The behavior of the right ventricle is not reproduced in the experiments with the model exactly as we find it in the heart of the patient. This is partly due to the construction of the apparatus, the defects of which in this direction have been pointed out emphatically by Moritz himself. Thus in experiments conducted under other conditions, for example, with a larger amount of fluid in the entire system, there may be a marked increase in the pressure in the vena cava, and a dilatation of the right ventricle (experiments 45 and 46).

The results of these experiments correspond very closely with our experiences at the bedside and the autopsy table. It remains only to discuss somewhat more in detail what occurs in the right ventricle: "In the right ventricle a dilatation need not necessarily take place—at any rate, not from the start; nor must the work of this chamber of the heart necessarily be increased. Inasmuch as we find almost regularly a hypertrophy of the right ventricle in our clinical work, in spite of these facts there remains nothing else to do but to assign the cause of this hypertrophy not to an increased work, but rather to the increased tension under which its contractions take place as the result of the increased pressure of the pulmonary arteries."

This is the view taken by Moritz, and is but another expression for the usual statement that the right ventricle has to work against the resistances which are increased in its vascular dependencies (the increased tension). Such resistances evidently are offered by the increased pressure in the pulmonary artery itself.

There is still another circumstance which affects the work of the right ventricle, and which, I believe, I was the first* to point out. A necessary

consequence of mitral insufficiency is that the normal blood-stream which is led through the pulmonary artery into the left auricle, and the abnormal recurrent stream from the left ventricle, must interfere with each other during the systole of both ventricles—in other words, the two streams literally oppose each other, and therefore uselessly waste a part of their kinetic energy. Now, as the left ventricle is from the first obliged to do a larger amount of work, as a result of the insufficiency of the valve, the right ventricle must, for this reason alone, furnish such an increased amount of work that it becomes hypertrophied in the course of time.

So far as I can see, Sahli† is the only author who indorses this view: "In mitral insufficiency the conditions are simpler, inasmuch as here both ventricles work directly against each other, as the result of the insufficiency, and, therefore, it becomes immediately clear why the right ventricle is allotted more work."

Of course, a great deal will depend here upon the degree of insufficiency of the mitral valve and upon the amount of resistance offered by the regurgitant blood from the left ventricle. If we suppose that the valves had been completely destroyed, then the weakened muscle of the right ventricle would not be able to resist the muscularly strong left ventricle for any length of time. But the possibilities which may be imagined when the different biologic factors come into play are so numerous that a deeper insight into these relations of valvular action and pressure is quite hopeless in my opinion.

As a matter of fact, dilatation is added usually after a variable time to the pure hypertrophy of the right ventricle. Yet we can say that this dilatation already points to a beginning failure of compensation. This was first pointed out by Weil‡ and afterward by Riegel.§

The train of reasoning is as follows: When compensation is complete, exactly the same amount of blood is expelled from the left ventricle as enters into the right auricle. Therefore no accumulation of blood can take place in the systemic veins. If the heart begins to fail, however, either as a whole or in any of its parts, such a venous accumulation does take place. Now, the effect of this is, first, a dilatation of the right auricle, which receives more blood. This increased amount of blood goes into the right ventricle, which is more extensively dilated by the increased amount of blood, which also flows under higher pressure during the diastolic relaxation of that chamber. During the systolic contraction of the right ventricle this increased amount must be emptied into the pulmonary artery. In this manner may be produced fatigue of the muscle, increased stretching of the wall, and permanent dilatation of the right heart.

The right auricle must undergo the same changes. This would be an acceptable explanation of the process. Yet in discussing the processes which go on in the heart, we must never forget that the great determining factor is the question as to whether the changes in the heart which make compensation possible keep pace with the development of the valvular lesion; whether the musculature of the part of the heart affected in each particular case is from the first capable of developing sufficiently, and has

* "Lehrbuch der speciellen Pathologie und Therapie," 1886, first edition, p. 433.

† "Lehrbuch der klinischen Untersuchungsmethoden," third edition, p. 339, Vienna, 1902.

‡ "Zur Lehre von den Mitralklappeninsufficienz," "Berlin. klin. Wochen.," 1881, No. 7, p. 89.

§ "Zur Lehre von den Herzklappenfehlern," "Berlin. klin. Wochen.," 1888, No. 20, p. 395.

been so well nourished under the existing conditions as is necessary to enable it to fulfil all that is required of it.

Both factors are biologic, and they vary widely in different individuals. Therefore, no scheme can be established which would be applicable under all conditions.

The impossibility of formulating such a scheme is still more evident for the following reason: If we suppose that a valvular lesion is present, the pathologic disturbances which caused it are still active, and have not by any means always disappeared. They persist in the musculature of the heart, in the vessels, in the endocardium as well as in the pericardium. In such cases we usually find progressive inflammations in the entire organ, which impair the nutrition of the heart, as well as its capacity to adapt itself to the increased amount of work required of it. To what extent its nutrition is thus interfered with depends upon the behavior of the permanent lesion, and that differs in every case.

Since we have been taught concerning these processes through the beautiful researches of Krehl,* we should be more than ever on our guard against considering only the mechanic conditions in our study of valvular lesions.

The description given by von Basch† is as follows: When the right ventricle retains its capacity of contracting completely; when elasticity of its (hypertrophied) musculature is increased in correspondence with the increased intracardial pressure, the diastolic lumen will at least remain the same, and the systolic contraction will remain unchanged, and as it had been before the valvular insufficiency set in.

When a permanent dilatation of the right ventricle takes place, the question is: Why does this cavity in such cases contain the larger amounts of blood which it permanently holds in its lumen? Does this occur because a larger amount of blood streams into the right ventricle from the veins, or because the ventricle does not empty itself completely, *i. e.*, only a portion of its contents which enters it from the right auricle is propelled into the pulmonary arteries, while a large amount of residue remains in the ventricle itself. Consideration shows that the first supposition seems to be absolutely excluded; for it is impossible that the amount of blood which flows into the right ventricle from the arteries or the veins respectively can be increased, inasmuch as the flow of blood into the systemic vessels is diminished as the result of mitral insufficiency. The question just asked, therefore, can only be answered by supposing that the increased distention of the right ventricle with blood depends upon an incomplete contraction of that cavity. . . .

It must be objected against this proposition, however, that an increased filling of the right ventricle from the systemic veins is only impossible so long as the hypertrophied left ventricle remains efficient. The incomplete emptying of the right ventricle may undoubtedly become important in the origin of the dilatation of that cavity. But then, again, biologic factors—fatigue—play a rôle in the development of this change.

As we have said before, a dilatation of the left ventricle takes place as a direct result of the insufficiency. The heart must adapt itself to the new requirements which are placed upon it—in this instance, in order to propel larger amounts of blood so as to retain the normal circulation (see p. 320).

This form of dilatation, which is styled "active or compensatory" by Krehl, and "primary" by Sahli, must be sharply distinguished from the passive dilatation produced by the stretching of the wall (relaxation dilatation according to Sahli). The active form is not a pathologic process. The passive, secondary form of dilatation occurs in the left ventricle only

* "Beitrag zur Pathologie der Herzklappenfehler," "Deut. Archiv f. klin. Med.," 1890, vol. xlv, p. 454.

† "Allgemeine Physiologie und Pathologie des Kreislaufes," p. 115, Wien, 1892.

when its musculature, for one of the reasons already discussed, becomes less efficient. This most frequently results as a consequence of inflammatory disturbances.

No characteristic changes occur in the pulse so long as an insufficiency of the mitral is sufficiently well compensated. When disturbances in the pulse occur, heart weakness has already set in.

A divergent opinion is held by Carl von Noorden.* He finds, in marked well-compensated cases, that the pulse is of a lower tension than in health; that the dicrotic wave is larger and later, and that the elasticity waves are weak or totally absent.

It is assumed, in explanation, that the arterial system also takes part in the compensation. This would be of interest theoretically, but I think that examination by means of the sphygmograph is of subordinate importance in practice.

To sum up: As a permanent effect of the insufficiency of the left auriculoventricular valve we find an increased amount of blood under increased pressure in the entire pulmonary circulation. It is by no means settled, however, whether this has any effect upon the clinical form of mitral insufficiency, in the sense that this valvular lesion produces respiratory difficulties more easily than other forms. We will be inclined to assume this if we accept the theory concerning the significance of pulmonary swelling and pulmonary rigidity as it has been worked out by von Basch. I have already defined my own position on this particular question (see p. 124). It is possible that the greater accumulation of blood within the pulmonary vessel favors the development of catarrh in some way or another. What we do know, and what we may surmise, have been discussed in another place, where I have explained how much influence heart weakness has upon the tendency to catarrh (see p. 106).

Anatomy of Mitral Insufficiency.—Bamberger describes it as follows:† "The endocardium of the left auricle is markedly thickened, opaque, tough, often tendinous. Not infrequently these lesions are also slightly developed in the serous lining of the other cavities of the heart. The left ventricle, as a rule, is somewhat dilated, while its walls may or may not be hypertrophied. Yet the enlargement of the left ventricle, as a rule, does not reach a very marked degree, unless other complications are present, such as an affection of the aortic valves, atheromatous processes in the arteries, or disease of the myocardium. Not infrequently, even, we find the left ventricle fairly normal in appearance. The left auricle is considerably dilated, often several times its normal size, and its walls are thickened.

"The right ventricle and the right auricle are both dilated, so that the right side of the heart may reach twice or three times its normal volume, or even more. Very commonly we find also that its muscular walls, especially those of the right ventricle, are hypertrophied, although dilatation always exceeds hypertrophy, both in constancy and in degree, and the wall of the right ventricle is rarely thicker than a few lines (a line equals 2.256 mm.).

"The dilatation also affects the pulmonary veins, which empty into the left auricle, the trunk of the superior and inferior vena cava, and the pulmonary artery, which often considerably exceeds the aorta in width. Very often the walls of these vessels are also thickened; their intima is rather opaque and dull in appearance. The pulmonary artery especially,

* "Ueber Beziehungen zwischen Pulsbildern und Herzklappenfehlern," "Charité-Annalen," 1890, p. 188 *et seq.*

† "Lehrbuch der Krankheiten des Herzens," p. 237 *et seq.*

both in its trunk, and even more so in its branches, is often in a state of fatty degeneration.

"The changes, just mentioned, in the size of the various portions of the heart, produce more or less marked deviations in the shape and position of the organ. Through the preponderating development of the right half of the heart this organ loses its rather conic shape, with its apex downward, and assumes a more spheric form, flattened from before backward.

"The entire region of the apex is considerably widened; the apex itself is no longer formed by the left ventricle alone, but the right ventricle, which extends far downward, takes part in its formation; in fact, in some instances the apex is formed by the right ventricle alone. The position of the heart is more horizontal than normally, and the more the right ventricle and auricle become enlarged toward the right and downward, the more will the apex of the heart be crowded to the left. The more marked the enlargement of the heart, the more will the borders of the lungs be forced outward, and even the diaphragm will be somewhat depressed."

These changes in the size and shape of the heart produce manifestations which become evident upon physical examination. We find:

"The precordium is often more prominent than normally, and frequently shows systolic and diastolic movements in the intercostal spaces on the left side. Unless complications arise, the pulsation of the apex is almost always displaced backward, under the left nipple, or more frequently beyond the same, toward the axilla. The apex-beat is diffuse, so that several finger-tips must be used in order to cover it. It is almost always increased in intensity to a variable degree, or is even 'tremulous' in character, but is heaving only in exceptional cases when no complications are present. Not infrequently it is accompanied by a systolic tremor and thrill.

"Another impulse which is felt just as distinctly, or at times even more clearly, than the apex-beat, is the beat which we often feel in the epigastrium, corresponding to the hypertrophied right ventricle. We must not be led by this into assuming that the apex has assumed a vertical position. The movements of the heart may be felt frequently over several intercostal spaces, or even over the entire precordial region, as concussions or as alternating rising and falling. When the affection is present in slight degree, or when complications are present, all the signs thus far mentioned may be absent."

Hardly any essential differences from this description may be found in the works of other authors. It may be noted, however, that some later writers * emphasize the formation of the apex-beat as produced chiefly by hypertrophy of the left ventricle. In general, more attention has been paid of late to the left side of the heart than formerly. The reader is referred to the description given by Fraentzel.† Liebermeister has included hypertrophy and dilatation of the left ventricle among the characteristic signs only in his "Grundrisse," while these signs do not occupy this position in his "Lectures."

"Percussion shows an increase of the area of cardiac dulness, which may vary a great deal in degree. In the milder cases we find that only the

* Romberg, in Ebstein-Schwalbe, "Handbuch der praktischen Medicin," vol. i, Stuttgart, 1899, Enke, p. 812. Krehl, "Die Krankheiten der Kreislauforgane," p. 316, in v. Mering's "Lehrbuch der inneren Medicin," Jena, 1901, G. Fischer.

† "Vorlesungen über die Entzündungen des Endocardiums und des Pericardiums," "Krankheiten des Herzens," Bd. ii, 1891, p. 145 *et seq.*

‡ "Grundriss der inneren Medicin," p. 173, Tübingen, 1901, Franz Pietzcker.

horizontal diameter of the dulness is increased at the level of the fourth, fifth, and sixth rib. In most cases, that is, in the more severe ones, there is also an increase in the vertical diameter, which is quite considerably enlarged, and this is explained not only by the fact that the dilated right ventricle increases in height, but still more by the dilatation of the right auricle, which under normal conditions is quite covered by the lungs.

"Extreme degrees of enlargement, in which the dulness extends for a considerable distance into the right side of the chest and upward to the second left rib, are rarely seen without complications. In the great majority of cases of insufficiency and stenosis of the mitral valves we can assume that the absolute dulness extends vertically from the third to the sixth or seventh rib, and horizontally from the right sternal border to beyond the nipple-line. The dulness in each direction does not extend over less than 6 cm., and does not easily exceed 12 cm. If the outlines be marked by means of silver nitrate, it will be seen that the dulness becomes wider in each intercostal space from above downward, but that the figure, on the whole, is rounded and grows narrower, and is, therefore, not regularly conical, as in pericardial exudates."

The newer methods of investigation, especially radiography, have not produced any essential deviations from these conclusions.

"On auscultation we hear a systolic murmur in the region of the apex (sometimes more distinctly a little above, nearer the nipple). The intensity, pitch, duration, and character of this murmur vary so markedly that no general statement can be made concerning these features. [In general, it is remarkable for its wide transmission to the left, into the axilla, the back, or up to the left clavicle. In their experiments on dogs, MacCallum and Thayer found the murmur sometimes transmitted by the pulmonary veins on both sides.—Ed.*] This murmur may be heard alone, without any systolic tone, or else the latter may be heard along with the murmur, in varying degrees of intensity and pitch [but never normal—Ed.]. The sound often seems to be produced only by the systolic shock to the thorax (metallic click), or else it is a true valvular sound, produced either by the part of the diseased valve which still remains capable of vibration, or by a neighboring normal valve.

"The relations of the sound to the murmur vary a great deal. In most cases the latter follows the former (perisystolic murmur), but the murmur may also precede the sound by a very short space of time (presystolic murmur), or in still other cases the sound itself merges into a murmur at its beginning and at its end. In such cases it is often difficult to determine whether the murmur is not diastolic, unless the heart's action be very slow.

"It occurs not infrequently that the murmur disappears entirely for longer or shorter intervals, and is replaced by a more or less distinct tone or by a muffled sound. Sometimes a reduplicated tone is heard instead of the murmur, but it is rare to find that the murmur is absent through the entire course of the disease, or that neither a sound nor a murmur is heard over the left ventricle. The second sound is heard distinctly over the left ventricle unless a complicating stenosis is present. In some cases it is increased in intensity (and is then usually transmitted from the pulmonary artery). On the other hand, it is sometimes very faint or even inaudible.

"The other valvular sounds usually do not show anything abnormal,

* "Experimental Studies of Cardiac Murmurs," "American Journal of the Medical Sciences," February, 1907.

with the exception of the second pulmonary; but it often happens that the murmur produced in the mitral is transmitted to the other valves, and therefore covers or accompanies the first sound. In such cases it is not always easy to recognize the murmur as a transmitted one and to exclude the presence of complications.

"Yet the transmitted murmur is distinguished through the fact that its quality (timbre) is everywhere the same; that its intensity decreases in direct proportion with the distance from the place of origin, and that we usually are able to hear the normal sounds over the respective valves (aorta, tricuspid) or nearly over them in a fairly pure state, without accompanying murmurs, while we cannot do this, as a rule, when the murmur arises in these valves themselves.

"Such a systolic murmur is most frequently heard over the pulmonary artery, and Skoda formerly explained this by a relaxation of the inner wall of the artery. This murmur very often is transmitted, a fact which may be explained, according to J. Meyer, by the proximity of the pulmonary artery to the outermost portion of the left venous orifice. Yet the murmur is not always transmitted; for frequently it differs in character quite markedly from the murmur which arises in the mitral valve, and it is very probable that it arises in such cases in irregular vibrations of the dilated pulmonary artery itself, the walls of which have lost their tonicity.

"One of the most important signs is the one first described by Skoda, namely, the increase in the intensity of the second pulmonary sound, which is at times so marked that the sound resembles the beat of a hammer, and can even be felt distinctly with the finger applied over the chest. In advanced cases the pulsation of the pulmonary artery is even visible on the thoracic wall. The reason for this lies in the dilatation of, and the increased hydrostatic pressure in, the pulmonary artery, whereby the recoil against its valves is increased. Yet we would be in error if we should believe that this symptom occurs with absolute constancy; for, on the one hand, the dilatation of the artery is not always sufficiently marked, and, on the other hand, the pressure may be lowered considerably even when this is the case, through the lessened contraction of the right ventricle, and partly through the weakening of the walls of the pulmonary artery itself.

"Thus, it frequently happens that this symptom is absent for considerable periods. We find this especially when we examine such patients when their heart's action is excited, and when, evidently, the right ventricle is able to drive only a small portion of its contents into the artery, owing to the short and rapid contractions. If such patients be examined at another time, when their heart's action has become quiet, then the accentuation of the second sound becomes quite distinct. Therefore the absence of this symptom is by no means a valid proof against the presence of a mitral valvular lesion."

The essential facts were noted quite completely by Bamberger, and not many new data can be added. Yet there has been no lack of efforts at other interpretations, and we must discuss some of these.

In the first place, there is the question as to where a systolic murmur can be heard. It is sometimes heard in the second left intercostal space, more distinctly, or at least just as distinctly, as at the apex. This is explained as follows by Naunyn.*

"The origin of the murmur must be sought in the left auricular ap-

* "Ueber den Grund, weshalb hin und wieder das systolische Geräusch bei den Mitralinsufficienz am lautesten in der Gegend der Pulmonalklappe zu vernehmen ist," "Berliner klin. Wochen.," 1868, No. 17, pp. 189, 190.

pendage, which passes around the pulmonary artery at a distance of about two inches (5.4 cm.) beyond the left sternal border, and comes closest to the thoracic wall. The murmur is best transmitted with the systolic recurrent blood-stream which causes it, and is transmitted in the direction of this stream. Therefore it is louder there than at the apex." Naunyn convinced himself, by measurements, that the auricular appendage varies in length in different individuals. When it is shorter, it does not approach the chest-wall so closely, and therefore does not offer conditions which are as favorable for the transmission of a murmur. This explains why we do not regularly hear this systolic murmur loudest over the left auricle. This view has now been generally adopted.

Curschmann* has added the following to this interpretation: According to his observation the systolic murmur is audible at this place chiefly in fresh cases of valvular lesions, and less frequently in cases which have lasted for some time. In the latter it gradually disappears from this localization, and moves once more toward the apex.

He believes that this is due to the fact that the left auricle, and with it the auricular appendage, is often considerably dilated in the beginning, and thus approaches the chest-wall. Later on, when the right side of the heart becomes hypertrophied, the entire left side of the heart recedes from the thoracic wall.

Systolic concussion of the thorax is said to produce systolic sounds, as the result of the vibration of the thoracic wall, *i. e.*, essentially, of the ribs. These systolic sounds are audible with the murmurs. At times a loud, dull sound is heard even at some distance from the patient (1 meter or more). If valvular murmurs are present at the same time, they can only be made out through the stethoscope applied to the chest, while the sound then appears fainter than the murmurs. This sound occurs only when the heart's action is rather vigorous, and is probably muscular in origin.

I have observed this phenomenon by no means as rarely in old fixed mitral insufficiency as in mitral lesions resulting from a fresh endocarditis, and have demonstrated it to my pupils. I have noticed particularly the weakening of the tone when immediate auscultation is practised over the chest. I do not find this mentioned, however, in the exhaustive treatise of Ebstein.†

A metallic tone may arise through resonance in cavities filled with air in the neighborhood of the heart (stomach, intestines, cavities in the lungs, or pneumothorax), whenever the physical conditions are favorable. In general, however, such conditions do not occur very often.‡

What Bamberger states concerning the time-relation of the murmur, and the tone, which possibly may be endocardial also, are facts which are well known to every experienced physician. Many explanations have been attempted for this which it is not necessary to discuss here. On the other hand, I shall mention briefly the relations of palpable thrill to the audible murmurs.

* "Ueber eine eigenthümliche Localisation des systolischen Geräusches, besonders bei frischen Mitralklappenfehlern," "Arbeiten aus der medicinischen Klinik in Leipzig," pp. 231 *et seq.*, Leipzig, 1893, F. C. W. Vogel.

† "Ueber die auf grössere Entfernung vom Kranken hörbaren Töne und Geräusche des Herzens und der Brustaorta," "Deut. Archiv klin. Med.," 1878, vol. xxi, pp. 113 ff.

‡ *Cf.* Leichtenstern, "Ueber einige physikalisch-diagnostische Phänomene," "Deut. Archiv f. klin. Med.," 1878, vol. xxi, pp. 148 ff.

At times we do not feel the thrill, although we hear a rather loud murmur. This may be due to the fact that the force which gives rise to both phenomena is sufficient to affect the more easily excitable acoustic nerve, but not the sensory nerves of the skin. A thrill may occur without an audible murmur, but I believe that such a thrill arises through an irregular contraction of the individual muscle-fibers of the heart. In such cases the conditions of conduction for the propagation of the sounds through the thorax are probably especially favorable. In what way they become favorable we cannot determine.

Diagnosis.—The signs by which an insufficiency of the mitral valve is recognized include an enlargement of the cardiac dulness, rather more markedly in the transverse diameter than in the vertical direction; a slight displacement of the apex to the left; a systolic murmur, loudest at the apex, and an increase of the diastolic tone over the pulmonary artery.

As I have stated above (see p. 346), the enlargement of the cardiac dulness may be regarded as a sign of beginning loss of compensation and of passive dilatation of the cavity of the right side of the heart. Hypertrophy of this side of the heart alone cannot produce an increase in the transverse diameter; for the thickness of the heart-wall is not sufficiently great on that side to do this. In practice, however, we are justified in including this enlargement of the dulness in the symptomatology, because it occurs so frequently, and it does not necessarily coincide with other signs of heart weakness.

Even when all these signs are present, the diagnosis is not absolutely certain. Chlorosis must be named in the first rank of diseases which may be mistaken for mitral insufficiency. A loss of hemoglobin, such as characterizes chlorosis, is also the basis of the symptoms that simulate mitral insufficiency. An enlargement of the cardiac dulness may also be brought about by retraction of the lungs and by the high position of the diaphragm, due to superficial breathing. Corresponding to the position of the heart, the right side of this organ is more markedly affected by this change than the left, and thus a greater enlargement takes place in the transverse direction than in the vertical, although the dulness is also enlarged vertically. The lung also recedes from the pulmonary artery, which may be seen pulsating in the second left intercostal space. The diastolic closure of the valves may also be felt, and its accompanying sound may be clearly heard not at all rarely louder than the sound of closure over the aorta. The apex-beat is also displaced to the left, but rather more markedly upward, into the fourth intercostal space, because the diaphragm is high, than downward, into the sixth.

It is well known that chlorotic patients often exhibit systolic murmurs over the apex and the pulmonary artery [sometimes transmitted to the left axilla and the back—Ed.]. When their heart's action is excited, a systolic thrill can not infrequently be felt at the apex, produced, as I have stated before (see above), in all probability through a not quite regular contraction of the muscles. Thus, we have all the signs of mitral insufficiency on physical examination, and even the subjective symptoms —palpitation, dyspnea, a feeling of anxiety, or at least of oppression, such as are present in a chronic endomyocarditis, also occur in chlorosis.

We can protect ourselves against confounding these two conditions by causing a more marked expansion of the lungs. In most cases it will suffice if we urge the patient to take deep breaths. The patients must be examined out of bed and must remove everything in the way of clothing

that may hinder the expansion of the thorax. They should be told to support themselves against a solid object, so that they may give play to the muscles of the shoulder-girdle which are employed in respiration. They must then be asked to breathe in and out, following the movements of the examiner's finger which is held before them. In this manner we usually succeed in a few minutes in so expanding the lungs, at least transiently, that they again cover the heart and make the area of dulness smaller and the phenomena at the pulmonary artery less distinct. Regular breathing exercises then will lead to a permanent improvement of all the symptoms which depend upon superficial breathing.

In these cases it is also of the greatest importance to determine the boundary-line between the liver and the lung. I have already emphasized elsewhere (see p. 328) how important it is to determine this boundary in any examination of the heart.

This interpretation, which I have followed since 1868, has now been generally adopted. Referring the reader to the lucid and thorough treatment of this subject by Carl von Noorden,* I only add that the measurements of the size of the heart according to Moritz, as they were made in Merkel's clinic,† show that the heart is not enlarged in chlorosis, but is placed in an abnormal position owing to the contraction of the lung and the high position of the diaphragm.

Since attention has been paid to the enlargement of the left side of the heart, especially of the left ventricle, it has become important for the differential diagnosis of the cases which have been caused by pulmonary retraction to determine accurately the position of the apex-beat. We may assume that a true enlargement is present only when the apex has been displaced downward. A displacement outward only is not sufficient.

Other forms of anemia may lead to conditions similar to those in chlorosis, but they rarely lead to a clinical picture which simulates mitral insufficiency in every symptom as does chlorosis.

The physician who lays too great stress upon the systolic murmurs in the diagnosis of mitral insufficiency will often commit errors.

I have already warned (see p. 330) against overestimating the auscultatory signs; but I must again return to this topic because these murmurs are still falsely estimated in the diagnosis of this condition. Not only older physicians find it difficult to appreciate the significance of the musculature of the heart, for the valvular lesion itself still plays the principal rôle, and with it, the question as to whether murmurs are present. Systolic murmurs over the apex are the most important of these murmurs which are regarded as diagnostic, and when they are present, a conclusion is but too easily drawn as to the existence of anatomic changes in the mitral valve.

Let us look a little more closely at this subject, in order to gain a clear conception of the true significance of murmurs in diagnosis. Does the detection of a systolic murmur necessarily inform us as to how this murmur has originated? Various special features have been attributed to the murmur which depends upon permanent tissue changes, which features have been said to give something characteristic to such murmurs. Von

* "Chlorosis," in volume on "Diseases of the Blood," English edition, "Nothnagel's Practice."

† Dr. Wilhelm v. Ebner, "Klinische Mittheilungen über Bleichsucht," "Festschrift," in celebration of the fiftieth anniversary of the Medical Society of Nürnberg, Communication from the General Municipal Hospital at Nürnburg, 1902, p. 39 *et seq.*

Leube* not long since treated this question in considerable detail, but has not solved it. Thus, he says in one place:† "To begin with, as regards the intensity of the murmurs, my experience teaches that it is quite indifferent for diagnosis whether the murmur is loud or faint." Further on he says:‡ "Only the intensity of the murmur is somewhat characteristic as contrasted with the murmur found in functional mitral insufficiency, a louder murmur occurring but rarely in the latter, and when it does occur, it speaks in favor of endocarditis." We see, therefore, that even von Leube has not been able to establish a really fixed rule. The same is true of the other physical signs, which have been mentioned in differentiating functional from organic murmurs: In permanent organic lesions of the mitral the murmurs are said to be constant—that is, to make the same impression upon the ear at all times, while in functional disturbances the impression undergoes frequent changes.

In organic affections of the mitral the murmurs are said to be loudest at the points of election for auscultation, and possibly to be limited to these points, while in functional affections they are more wide-spread over the heart.

The systolic murmur has its full diagnostic importance so long as the insufficiency of the mitral is present in a well-nourished heart which has remained free from other tissue changes. This is changed, however, as soon as the heart muscles begin to take part in the process to any extent. Then, so many and such varying auditory impressions are made by these murmurs that the differential points mentioned above become altogether worthless.

The question as to whether an endocarditis is present, making the mitral insufficient, or whether an endocarditis had been present elsewhere, and now is recurring at the mitral, is very often difficult to answer, or even impossible. In general, I may be allowed to refer to my discussion of the subject in another part of this work (p. 291).

It is certainly of value in determining the character of the insufficiency of the mitral valve to find out by questioning the patient whether he had formerly suffered from acute articular rheumatism or from a septic infection. Yet we should not attribute too great importance to this point, neither when "yes" is answered nor when the history is negative as to these previous diseases.

I append the history of a case which I recently observed, and which shows that I made a gross error in the diagnosis, although I had tried my best in this particular case to avoid all the stumbling-blocks.

Case I.—Mrs. M., aged fifty-three years, laundress, admitted May 16, 1902. During the entire winter she suffered from frequent palpitation, anxiety, and cough. Treatment in the Policlinic for several weeks brought about improvement, though only temporarily. Today she had an attack of suffocation.

Seven years ago this patient had an attack of severe acute articular rheumatism, and one by one many of her joints (the patient says *all* her joints) were affected. She was in bed twenty-one weeks, but does not know whether there was anything the matter with the heart at the time (she was under the care of a physician). Since then she has been subject to palpitation of the heart on slight provocation, and has felt weaker in general, but has never had swollen feet.

The patient presented signs of marked heart weakness. Cyanosis, venous pulse

* "Zur Diagnose der systolischen Herzgeräusche," "Deut. Archiv f. klin. Med.," 1896, vol. lviii, pp. 225 ff.

† P. 227, where "accidental" anemic murmur is spoken of.

‡ P. 230. Here he refers to mitral insufficiency from endocarditis.

at the jugular; very marked edema of both legs; slight swelling of the left hand. The pulse was small, easily compressed, somewhat irregular in both volume and frequency. When counted at the radial, the pulse was 40, while on auscultation of the heart it was 44, both counts being taken in the same twenty seconds. There was no atheroma in the peripheral arteries.

The boundaries of the heart extended on the left side above from the lower border of the second rib; on the right about a finger-breadth beyond the right sternal border, while the apex-beat was somewhat diffuse and the apex itself seemed to lie in the mammillary line behind the sixth rib. On auscultation, a loud systolic blowing murmur, loudest at the apex, was made out, but extending diffusely over the anterior surface of the thorax in the cardiac region. The aortic tones were quite faint; the diastolic tone over the pulmonary slightly accentuated in comparison with that over the aorta. The boundary between the lung and the liver anteriorly was at the lower border of the sixth rib. Pulmonary resonance on percussion behind on both sides at the level of the tenth or eleventh thoracic vertebra.

The respiration was 33, and there was inspiratory dyspnea. The weak respiratory murmur was accompanied by fine moist râles over both posterior thoracic surfaces, while over the right there was a fairly marked dulness extending from the sixth vertebra downward. Above there were, on both sides, occasional sibilant and large moist râles.

There was no ascites; the liver was slightly enlarged, but not painful on pressure. The spleen was of normal size. The urine had a specific gravity of 1019; there was a trace of albumin, no sugar, and no diazo reaction.

The treatment consisted in the proper dietetic regimen, and also in the administration of digitalis. The course of the disease was as follows:

The boundaries of the heart remained unchanged until the death of the patient, which occurred on June 29th. The loud systolic murmur was also essentially unchanged to the end, save that at first it seemed to me that it varied in its quality at times. For a few days when the action of the digitalis was at its height, the sounds over the aorta became louder and the accentuation of the diastolic pulmonary sound became less marked. It was quite distinct, however, during the rest of the time. The signs of cardiac weakness increased on the whole with slight variations, although this increase was very slow. The secretion of urine may be taken as a measure of this. It was as follows:

In the week from May 27th to June 2d, the daily excretion was 860 c.c.
In the week from June 3d to June 9th it was 1010 c.c.
In the week from June 10th to June 16th, 940 c.c.
In the week from June 17th to June 23d, 930 c.c.

The frequency of the pulse also only varied during the entire period of observation between 102 and 120. The respiration varied somewhat more markedly—that is, from 30 to 42 so long as the patient suffered from nothing but the moderate bronchial catarrh. The temperature varied considerably: the maximum on nine days was 38.7° C. (101.6° F.) from May 23d to June 5th. From June 6th to June 24th a maximum of 39.1° C. (102.4° F.) was reached on two days. During the eleven days which preceded death no fever whatever was noticed. Then followed a period accompanied by erysipelas (which led to a fatal end) during which the fever was higher.

For a short time (June 12th to 15th) repeated diarrheas occurred, accompanied on the first day by a sudden rise of temperature to 39.1° C. (102.4° F.). These disappeared later on. An erysipelas developed at various points in the skin on the right thigh and the left leg, where the skin had broken as the result of anasarca. On the day preceding death the signs of an area of consolidation, not quite reaching the pleura, appeared in the upper lobe of the right lung.

I was at first inclined to regard this case as one of muscular insufficiency of the mitral, and only the circumstance that the murmurs could be heard so constantly and uniformly for such a length of time, as well as the fact that there was no doubt as to the previous existence of a severe acute rheumatism, made me doubt more and more. In addition, there was also the question as to whether a recurrence of an old endocarditis had taken place.

The peculiar distribution of the temperature, the apparently uncalled-for variation in the fever, and the fresh focus in the lungs led me to think of this.

The clinical diagnosis, therefore, was as follows: Old valvular lesion—chiefly mitral insufficiency. Recurrent endocarditis(?).

The autopsy (Professor von Baumgarten) showed a moderate dilatation and hypertrophy of the heart, with a displacement outward and downward of the apex, which was formed by the right ventricle. But no changes were discovered at the mitral valve. The papillary muscles and the trabeculæ of the left side of the heart

were slightly hypertrophied. The lungs were moderately emphysematous. The other features of the autopsy are of no importance here.

There was a freshly generalized old primary tuberculosis of the lymphatic glands.

Such mistakes are made often enough. We still venture on the path that leads to the swamp in which we have often enough sunk before. And yet on calm consideration we must say that a diagnosis which is just as frequently correct as incorrect, and in which we find no reason why it is right in one case and wrong in another, should generally be avoided and should be replaced by an "I don't know." We may, however, seize the fact that the mitral valve does not close and may simply set up insufficiency as a clinical diagnosis.

MITRAL STENOSIS.

Stenosis of the mitral orifice is caused, as a rule, by endocarditis. The valves which have become stiff, shrunken, possibly calcified, are no longer able to lie against the wall of the ventricle during diastole. The flaps, therefore, project into the orifice and thus cause a stenosis.

More marked alterations depend upon adhesions of the valvular flaps at their free borders. Then, nothing but a short narrow slit remains open for the passage of blood.

Insufficiency of the mitral accompanies stenosis of this orifice. In proportion as the latter increases, the former becomes less important, until we reach a state in which we can speak of a pure stenosis. The two illustrations (Figs. 58 and 59) reproduced from photographs of specimens in the Pathologic Institute at Tübingen show distinctly the relation.

This subject has been studied in considerable detail on account of its great importance, and a certain degree of agreement has been reached as regards its interpretation. Pure stenosis without accompanying insufficiency can only be assumed to exist "when, at autopsy, the orifice is very narrow and the valves form a slit with smooth and movable borders" (Baumbach-Lenhartz).*

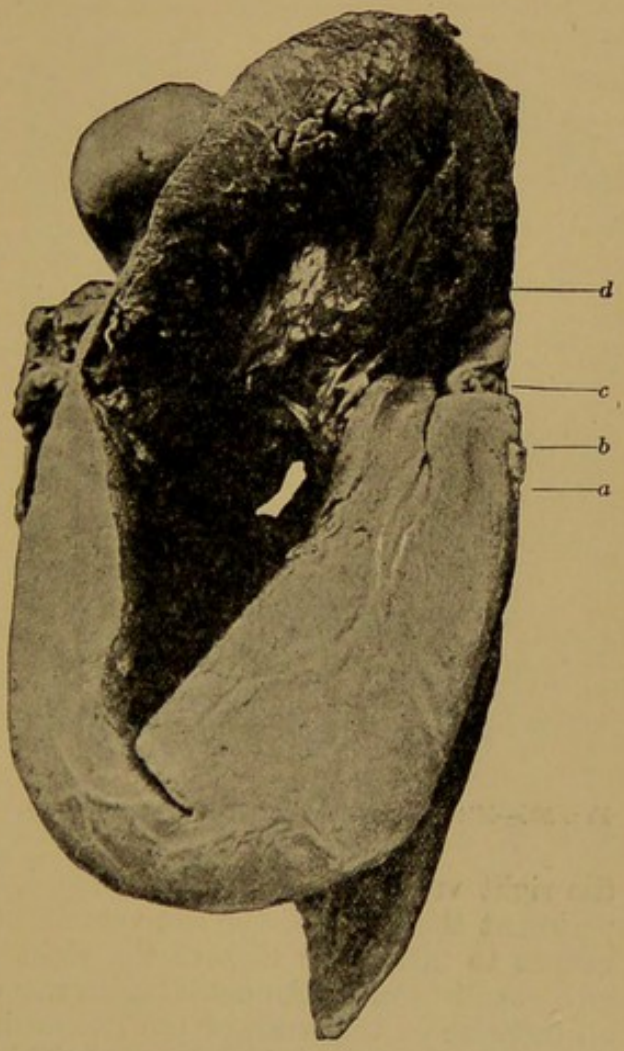


FIG. 58.—Mitral stenosis with mitral insufficiency: *a*, Contracted ventricular orifice; *b*, shrunken flaps of valve with papillary muscle (*c*); *d*, fibrous thickening of endocardium on the anterior wall of the left ventricle.

* "Ueber das Verhalten des linken Ventrikels bei der Mitralstenose," "Deut. Archiv f. klin. Med.," 1891, vol. xlviii, p. 275.

"A pure stenosis can only develop when that part of the valvular border which is not adherent remains comparatively delicate and easily movable and incloses a slit-like opening" (Dunbar-Riegel).*

Yet such cases are rare. Clinically they are distinguished with difficulty; for auscultatory phenomena are the decisive factors in their diagnosis. For we must say: The signs which are produced by a compensated stenosis of the left venous orifice are the same as those of insufficiency of the mitral valve, except that a diastolic or presystolic murmur occurs in place of the systolic.

Experiments on models gave the following results: The flow from the left auricle into the left ventricle is diminished, and with it the flow into the aorta. The pressure in the aorta falls; that in the pulmonary artery rises, as occurs also everywhere in the pulmonary circulation.



Fig. 59.—Mitral stenosis viewed from the ventricle.

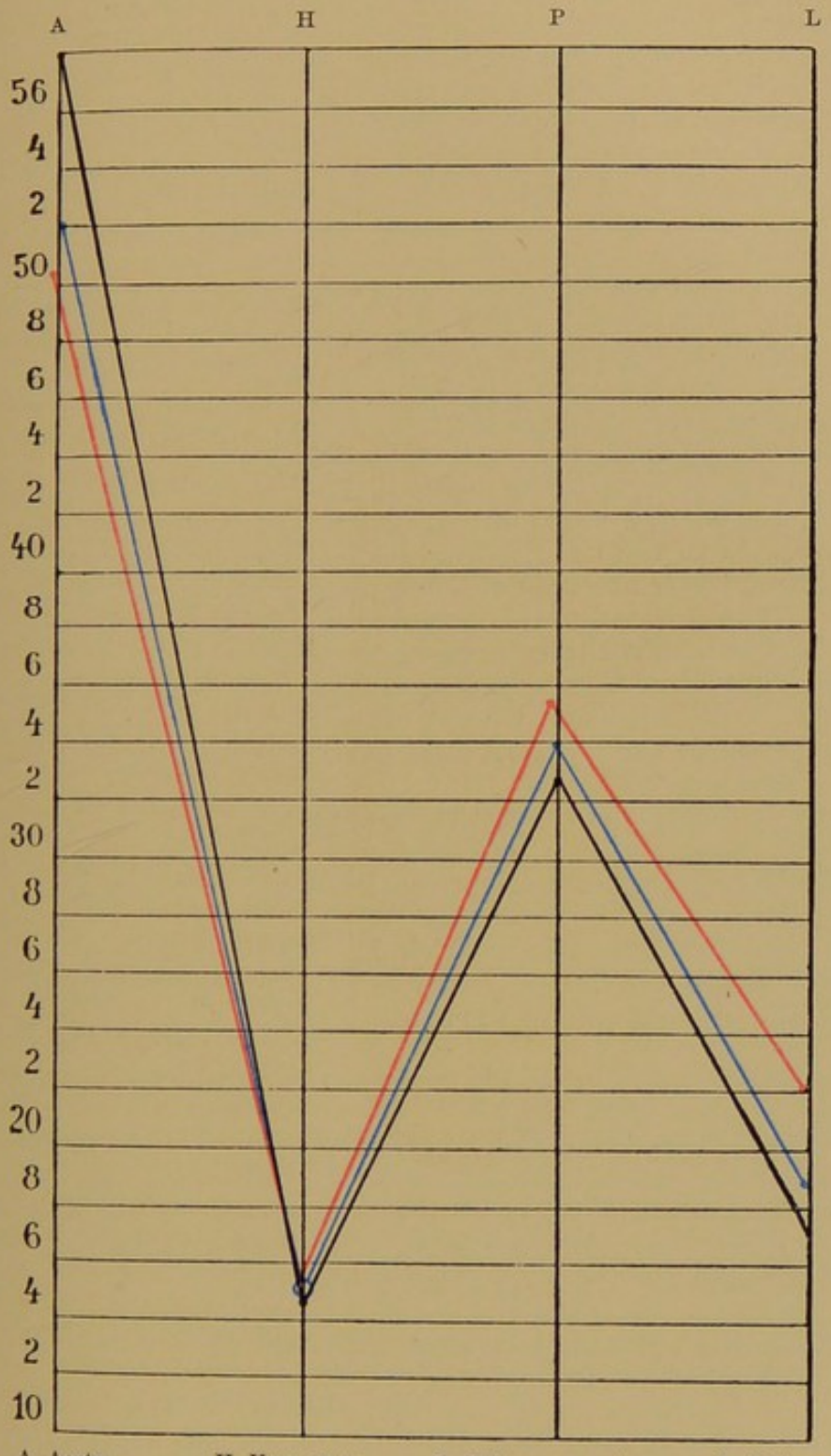
Whether the pressure in the vena cava remains unchanged or is lowered or raised depends upon the following: The right ventricle must regain an equilibrium with the blood flowing from the left if stationary conditions shall be established, and also the amount of blood on the right side must diminish. If less blood is thrown out of the right side of the heart, there will be a rise in the pressure in the vena cava. On the other hand, if less blood is propelled by the left heart, the pressure in the vena cava will sink. According as one or the other condition prevails, the volume of the right ventricle will vary. But great variations should not be expected.

The left ventricle has less work to do in stenosis of the venous orifice. The work of the right ventricle, on the other hand, is scarcely altered. In the experiment the work of the left ventricle diminished from 249 gram-centimeters to 202, while that of the right ventricle sank from 145 to 143 gram-centimeters. An equilibrium was established (see Fig. 60) through an increase in the work of the left auricle, when that became sufficient to secure a more complete filling of the left ventricle.

Moritz notes that the changes in pressure which develop in virtue of the stenosis of the left venous orifice, from the start, give the left auricle a chance to increase its working capacity. The auricle is more markedly distended in virtue of the rise of pressure in the pulmonary veins and contains more blood. The increase in the tension of its walls

* "Ueber das Verhalten des linken Ventrikels bei den Fehlern der Mitralklappe," *ibid.*, 1892, vol. xlix, p. 283.

leads to a more vigorous contraction of these walls, and, in addition,



A. Aorta. H. Vena cava. P. Pulmonary artery. L. Pulmonary vein.
Pressure before experiment. Mitral stenosis. (Experiment 48.) Increased work of the left ventricle.

FIG. 60.—Moritz's Experiment 47.

when the auricle is at rest—that is, during a part of the diastole—the blood flows more easily into the ventricle.

The hypertrophy of the right ventricle which is regularly present is

explained as in insufficiency. The right ventricle must work against the increased tension in the pulmonary artery. The left ventricle does not become dilated in stenosis, but becomes rather smaller, because the work required of it is less. The left ventricle does not need to propel a larger amount of blood; nor does it need to work against an increased tension in the direction of its stream. This, then, is the essential difference between the changes occurring in stenosis and those resulting from insufficiency of the valve. Otherwise, the conditions would be the same, even if a patient would have a pure stenosis, which can be readily enough produced in the model.

What are the conditions in living subjects when both stenosis and insufficiency are present together? It will depend greatly upon the question whether the stenosis develops, in a measure, together with the insufficiency, because upon this depends the question as to which of the changes in the heart will gain the ascendency. Naturally, much depends also upon the extent of each of the two lesions.

Another point to be noted is that insufficiency of the valve lessens the mechanic results of stenosis of that orifice, and, *vice versâ*, both being at first favorable to the circulation. In insufficiency the ventricle systolically throws larger amounts of blood under higher pressure into the left auricle. Thus the wall of this chamber is elastically distended, and this elastic tension becomes transformed into living force during the following diastole. This force then becomes available in overcoming the resistance produced by the stenosis. On the other hand, the stenosis hinders the systolic escape of the blood from the ventricle into the auricle through its more or less injured valve.

But it is difficult to say what takes place later on. The biologic questions come essentially into consideration; that is, the question whether or not the left auricle and the right ventricle are able to increase sufficiently in musculature.

The behavior of the left ventricle has been the subject of much discussion. Theoretically, this ventricle should become smaller in size, but actually this is often not the case.

Baumbach* found rather a hypertrophy of the left ventricle, and by no means an atrophy, in thirteen cases of pure mitral stenosis (classified according to his division already mentioned) in which there was no insufficiency and no other complications.

Dunbar† reported two autopsies in which an atrophic left ventricle occurred along with a marked degree of stenosis without insufficiency. In five other cases, on the other hand, the left ventricle was normal in size or dilated. Of these cases, however, three must be rejected, as they are not pure: phthisis, pericardial adhesion, and slight mitral insufficiency (Nos. 3 to 5, VIII). Baumbach pointed out correctly that the left heart looks smaller on account of the very marked increase in size of the right heart, and this is expressed in the sentence which one often sees in autopsy reports: "The left ventricle appears as an appendix of the right heart." Therefore, accurate measurements should be made.

It was supposed that, when dilatation of the left heart is present, it must be referred to a still present or preëxisting insufficiency of the mitral valve. Lenhartz‡ opposed this, and claims that a dilatation of the left

* *Loc. cit.*, group 1 a, sections from Halle and Leipzig.

† *Loc. cit.*, pp. 284 and 305–307.

‡ "Ueber das Verhalten der linken Herzkammer bei der Mitralstenose," "Verhandlungen des Congresses für innere Medicin," Ninth Congress, Vienna, 1890, p. 478 ff. Completed in the work of his pupil, Baumbach.

ventricle may occur even with pure stenosis. He explains it by laying particular stress upon the diastolic suction-force of the left ventricle. Dunbar's work, however, strongly opposes the reasons upon which Lenhartz bases his argument. Krehl* also opposes this view, and adheres to the old idea. He says, in speaking of this: "How does the left ventricle now behave in stenoses of the mitral? That depends upon the amount of blood which this ventricle receives with the aid of the increased activity of the left auricle and the right ventricle, in spite of the narrowed mitral orifice. In slight degrees of stenosis in which the resistance produced by the narrowing is well overcome by the right ventricle because of its capacity for compensation, the left ventricle may remain unchanged, because then it is filled with the same amount of blood as before the stenosis. But, if a disproportion between the obstacle set by the stenosis and the force of the right ventricle develops, then the filling of the left ventricle suffers, and the amount of work done by it is diminished, so that its musculature becomes atrophied. These assumptions are confirmed by the pathologic-anatomic findings in such cases. When we find, occasionally, a hypertrophy of the left ventricle, this must be explained by an insufficiency of the mitral valve, present simultaneously with the stenosis, or even older."

Carl Hirsch,† who determined the weight of the heart and of its various divisions according to W. Müller's method, draws the same conclusions from his observations, and rejects emphatically the theory of Lenhartz and Baumbach.

When the stenosis of the mitral valve becomes more marked, more severe changes result than when the stenosis is less prominent than the insufficiency. It is evident that, under such conditions, the left auricle and the right ventricle are compelled to perform more work, and are scarcely able to stand the strain for any length of time. The left ventricle not only does not play any rôle in compensation, but its incomplete filling with blood prevents the whole heart from getting such an amount of blood as is necessary for the sufficient nutrition of the parts which are forced to increased labor. The results of this are an insufficient hypertrophy and a rapidly developing dilatation of the fatigued portions.

Disturbances in the pulmonary circulation (see p. 106) then follow. It is an old-established fact that catarrh of the bronchi occurs more frequently in stenosis, is more difficult to cure, is more severe, and that "heart-disease cells" very commonly occur in large numbers. Also that the brown-indurated, congested lung develops to its full extent in stenosis. This is naturally accompanied by a severe disturbance in the respiration.

It is easy to understand from all this that loss of compensation becomes prominent comparatively early, and that the stenosis itself disappears in the general clinical picture.

The sphygmogram does not give us anything characteristic in this direction. Only C. von Noorden (*loc. cit.*) has recorded a few exceptions to this rule. In most instances we find the note that the pulse is small, easily compressed, irregular in volume and in rhythm. If we look at the pulse-curves of patients with mitral

* A, pp. 22, 23.

† "Ueber die Beziehungen zwischen dem Herzmuskel und der Körpermusculatur und über sein Verhalten bei Herzhypertrophie," "Deut. Archiv f. klin. Med.," vol. lxviii, p. 325 *et seq.*, 1900.

stenosis which were published by C. Gerhardt,* and compare them with the curves I have given elsewhere, we become convinced that they indicate nothing but simple heart weakness (compare also Fig. 64).

Anatomic Considerations.—In general, the anatomic conditions in stenosis are the same as those already described in insufficiency of the mitral valve. There are some deviations worth mentioning (Bamberger): "The left auricle shows marked dilatation, thickening of the walls, and



Fig. 61.—February, 1897.

its endocardium is markedly clouded and thickened. The pulmonary veins which enter into it are considerably dilated. The left ventricle exhibits changes which have been described above (see pp. 360 and 361). The right side of the heart shows dilatation and hypertrophy. These are so extensive that the normal size is by no means rarely doubled. The apex of the heart is often formed by the right ventricle only. Both

* "Lehrbuch der Auscultation und Percussion," fifth edition, p. 313.

venæ cavæ and the pulmonary artery are dilated, while their walls are often the seat of fatty degeneration.

"The position of the heart is rather horizontal, and the anterior surface is formed almost entirely by the dilated right heart, while the left portion of the organ is displaced entirely backward, so that nothing, or a very small strip, of the left ventricle is seen from the anterior aspect."

Physical examination reveals the following signs (I am again quoting Bamberger): "The pulsation of the apex is usually felt at a considerable distance to the left of the nipple, and is usually more diffused, but rarely increased in intensity to any extent. On the other hand, the contraction of the heart is almost always felt within a large area, which usually extends over the greater part of the region occupied by the hypertrophied right ventricle, and is, therefore, especially behind the sternum and even to the right of it.

In the region of the apex and toward the nipple, a diastolic thrill is frequently palpable. Sometimes, however, the thrill is only systolic, depending upon the coëxisting insufficiency, and less frequently both a systolic and a diastolic thrill are felt.

Percussion shows a more or less marked increase of the cardiac dulness, particularly in the transverse diameter.

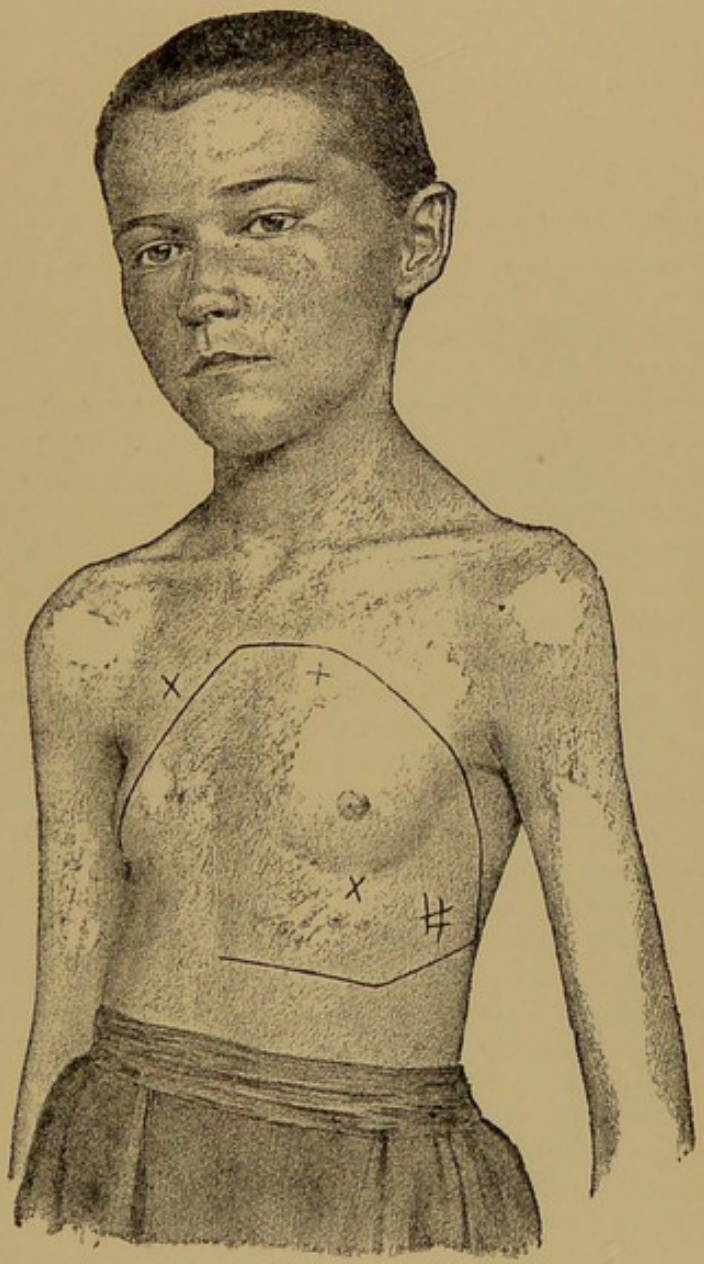


FIG. 62.—July, 1901.

The accompanying Figs. 61 and 62, from photographs, show the area of dulness in progressive stenosis with insufficiency. The patient was a girl, now seventeen years old, who had had a severe attack of endocarditis for the first time ten years ago, and had had several relapses since that time.

On auscultation a diastolic murmur is heard in the region of the apex, but often also over a considerable area. It is more or less prolonged and varies greatly in quality. In addition to the murmur, there may be also

a more or less marked diastolic tone. Very often, however, the murmur is very faint, resembling a short faint breath; or it is entirely absent, either permanently or temporarily, or, instead of it, one hears a short dull sound, a reduplicated tone, or a distinct or accentuated tone, or else all distinct perception of sound is absent in the diastolic period.

"A more or less well-marked murmur can be heard over the left ventricle during the systole, but there may also be a reduplicated tone, or else a clicking [or flapping—Ed.] heart shock [resembling a normal or accentuated second sound—Ed.], a dull indistinct sound, or even no definite sound at all. In the majority of cases, however, a murmur is heard. This great variation in the signs observed requires a few words of explanation.

"The only characteristic acoustic phenomenon on auscultation in mitral stenosis is the diastolic murmur. This murmur is produced by the influx of blood from the auricle into the ventricle, through the rough and narrowed orifice. The more marked this narrowing, the more extensively developed the rugosities, and the greater the blood-pressure, the more intense will be this murmur; while in the opposite conditions there will be only a faint murmur or none at all. The usual cause of the absence of a murmur is a lowered velocity of the blood-stream.

"Even under normal conditions the pressure under which the blood flows into the ventricle from the auricle is much less than the pressure under which the blood is driven from the ventricle into the arteries, and, therefore, a systolic murmur, under any condition, occurs much more frequently than a diastolic murmur as the result of changes in the auriculo-ventricular valves.

"In stenosis of the left venous orifice there are a number of conditions which also tend to diminish the diastolic blood-current and to lessen the velocity of the stream, particularly the stasis and the slowing of the pulmonary circulation. Other conditions diminishing the diastolic current are the passive dilatation of the left auricle, the great rapidity of the heart-beats, which interferes with a complete emptying of the auricle, etc. It is not astonishing, therefore, that in stenosis a diastolic murmur is absent, at least as frequently as it is present.

"In some cases we can convince ourselves that it is only necessary to secure an approximate return to the normal conditions of the blood-stream in order to evoke the murmur. For example, in lesser degrees of stenosis the murmur is often heard only when the blood-stream is somewhat hastened, as the result of physical exertion or of other external influences. In stenoses of higher degree the murmur is often heard only when the heart's action is rendered slower by rest or by the influence of drugs, until a more complete emptying of the auricle into the ventricle can take place.

"If we hear a diastolic tone in addition to the murmur, or else without the murmur, then this tone, in accordance with my view as to the origin of the second cardiac sound in general, can only be the transmitted diastolic arterial tone. This sound does not by any means necessarily belong to the corresponding artery,—in other words, in this instance, to the aorta,—but it may in some cases depend upon the pulmonary artery (especially when the tone is increased in intensity), or else it may be a mixture of both arterial tones.

"When the diastolic murmur is loud, it usually completely masks the second heart-sound. Yet the presence or absence of the second

sound, its reduplication or other modifications, are of absolutely no further significance as regards the condition of the auriculoventricular orifice.

"As a rule, a murmur is heard during systole because the stenosed valve almost always allows of regurgitation, and, besides, is only capable of murmur-like vibrations owing to the complex changes in its texture. Often, however, its character neither allows of the more marked vibrations nor of an appreciable regurgitation, especially when the stenosis is marked, and in such cases we hear either a very indistinct (systolic) sound or no sound at all.

"A pure systolic sound is produced very rarely by perfectly regular vibrations of the sufficient valve, because this is scarcely possible in stenosis. As a rule, the systolic sound, whether it be present alone or in addition to a murmur, is produced, either by a portion of the diseased valve which is still capable of vibration, or else it is transmitted from the tricuspid valve, or is merely the result of a systolic vibration of the thoracic wall.

"Both the systolic and the diastolic murmurs are transmitted to the neighboring valves, especially to the tricuspid valves, and may cover their tones more or less completely. In addition, we find in stenosis of higher degree that the aortic sounds are usually remarkably faint. The first pulmonary sound is accompanied or supplanted by a murmur, as a rule; for reasons which have been given in the section on Insufficiency. The second pulmonary tone, as a rule, is even more exaggerated in intensity than in insufficiency alone."

Bamberger describes the facts just as all who have made a detailed study of these conditions, and the basis for his interpretations must also be accepted: the relatively slight force which the blood-stream passing through the narrowed orifice during diastole has at its disposal. By some authors, among whom Traube stands at the head, the varieties of the diastolic murmur are distinguished even more sharply, and attempts are made to explain* why the murmur behaves in one way in one case and in another, in others. No doubt Traube himself and his immediate disciples, such as Fraentzel, have heard all these fine distinctions, and they may be of diagnostic value; but how often is this the case?

Traube† maintains that the diastolic murmur occurs in four different modifications: (*a*) As a prolonged murmur which grows more intense toward the end and lasts until the next systole. (*b*) In the form of two diastolic sounds which not infrequently resemble tones, while in other and more frequent cases the first is like a tone, the second like a murmur, and lasts until the beginning of the following systole. (*c*) A short presystolic murmur and (*d*) as a non-modified murmur, that is, as a murmur which is separated from the following systole by a pause.

The most practical classification of these varieties of the diastolic murmur in stenosis of the left orifice seems to be the following:

I.—*Not Separated by a Pause from the Following Systole.*—The varieties of this type are as follows: (1) A loud prolonged murmur which grows more intense toward the end of the diastole, and which lasts until the following systole. (2) A short murmur which appears at the end of the diastole—a so-called "presystolic" murmur. (3) A murmur composed of two different sounds, of which the second lasts until the following systole.

II.—*Separated by a Pause from the Following Systole.*—Of this, there are the following varieties:

*Fraentzel, *loc. cit.*, p. 109.

† "Ueber das diastolische Geräusch bei Stenose des Ostium venosum sinistrum," "Gesammelte Beiträge," Bd. iii, S. 251, 252.

(1) A diastolic murmur which is separated from the following systole by a distinct pause, and (2) two diastolic sounds, of which the second is also separated from the next systole by a distinct pause. These diastolic sounds may be either *tones* or short *murmurs*.

The division made by Liebermeister* has the advantage of simplicity; so also his schematic representation (Fig. 63). In this figure — represents the first or systolic cardiac tone, and ⌣ represents the second or diastolic cardiac tone. The varieties distinguished by this author are as follows:

I. *Presystolic Murmur.*—This is produced when the stenosis is somewhat marked and when the diastole does not last very long. Then the blood enters the ventricle at first rather slowly, and only the systole of the auricle which drives the blood with

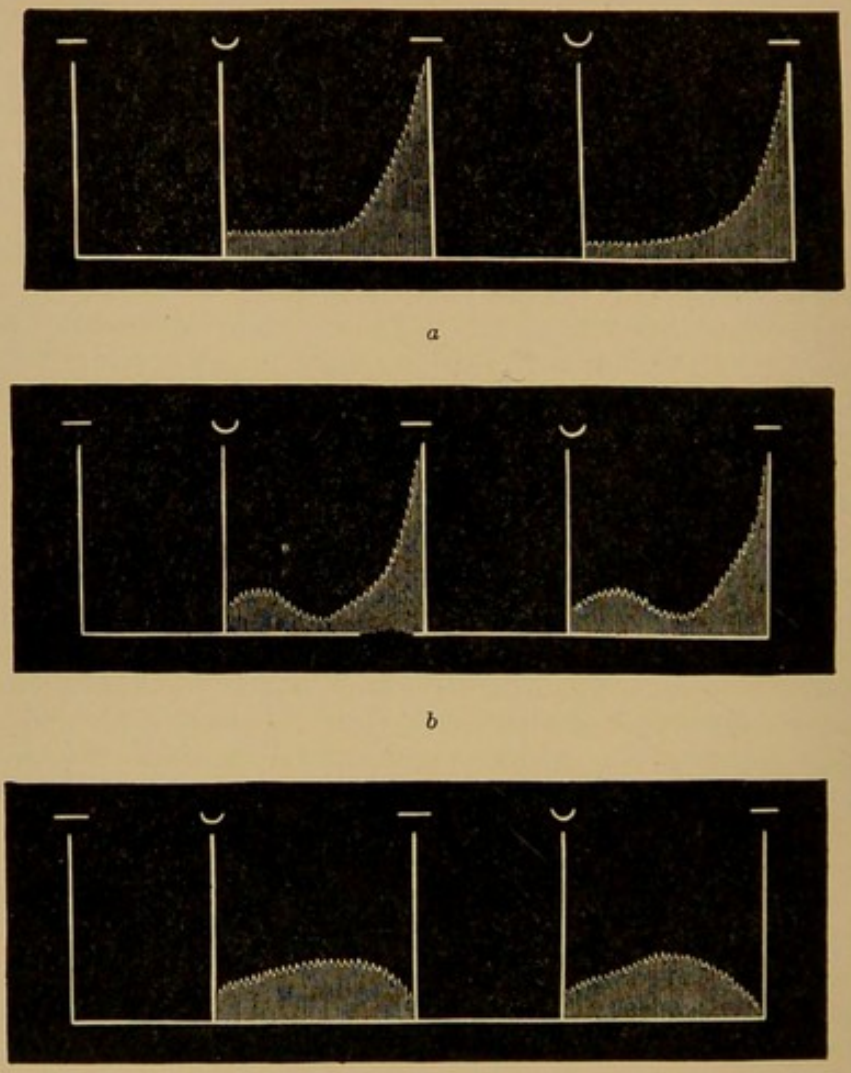


FIG. 63.—*a*, Presystolic murmur; *b*, diastolic-presystolic murmur; *c*, diastolic murmur.

great velocity through the narrowed orifice produces a more intense murmur, which lasts until the beginning of the following systolic sound.

II. *Diastolic-presystolic Murmur.*—This is produced under the same conditions. The stenosis is not too slight, the diastole not too prolonged. The mode of origin of this murmur is also the same. It depends only upon the velocity of the stream in the first part of the long pause. The force which drives the blood through the narrowed orifice is composed of the suction of the relaxing left ventricle and of the elastic tension of the overfilled left auricle. Is it sufficient to produce a stream which at first increases considerably and then diminishes? And this in such a manner that the difference exceeds a certain limit?

* "Vorlesungen," p. 375 and Figs. 4–6.

In this case the muscular contraction of the right auricle, which gives a fresh impulse to the blood streaming through the narrow orifice, may become manifest by an increase in the intensity of the murmur. These conditions are always present, and I do not see quite clearly why Liebermeister still separates this type from the first group (I). Traube, and especially Fraentzel, it is true, lay great stress upon this form of murmur. It constitutes Fraentzel's "interrupted modified diastolic murmur," and, according to his view, only those examiners who can recognize it with facility are sure of their diagnosis of valvular disease.*

III. *Diastolic Murmur.*—The stenosis is less marked; the diastole is prolonged. The blood now flows into the ventricle in such a manner that the cavity is almost completely filled when the auricle begins to contract. The contraction of the auricle, therefore, cannot be especially prominent acoustically. The blood will pass through the narrowed orifice with approximately the same velocity.

On the whole, we can very well content ourselves with the chief divisions made by Traube, whether the diastolic murmur is continuous (I) or is interrupted (II).

The reason why there is such a difference in the auditory sensations is that the character, the form, and the extent of the opening are so very different in the individual cases. Besides,—and this is probably of still greater importance,—the musculature of the auricle is never very strongly developed at first, and yet must furnish the driving force, so that slight fluctuations in its working capacity can very easily occur.

The diastolic murmur is also heard at times in the second left intercostal space, in the region of the pulmonary artery, or a little outward from that point. At times the murmur is heard even a little more distinctly in that locality than at the apex, especially when a systolic murmur is also present at the latter. This is remarkable inasmuch as the blood-stream which produces the murmur is directed from the auricle toward the ventricle (Liebermeister).

As regards the other signs, we must refer the reader to what has been said under mitral insufficiency.

Diagnosis.—As has been stated above, the question, in the majority of cases, is not whether a stenosis is present, but in what relation it occurs to the insufficiency which is present at the same time. We depend essentially, therefore, upon the demonstration of the diastolic murmur for the diagnosis of stenosis. We have already considered, in a general way, the diagnostic value of the sensations perceptible to the ear (see p. 330). With due conservatism, we can say that when a murmur is heard over the apex and is certainly diastolic, and when we can demonstrate, in addition, the corresponding changes in the area of dulness, the diagnosis may be made positively. For it is certainly extremely rare to find diastolic murmurs at the apex due to other causes. The certainty as to the diastolic nature of the murmur becomes greater when the murmur continues in uniform intensity without interruptions. Naturally, the frequency of the heart-beats must not be too great, so that we can distinctly make out the pauses between the individual beats. After the diastolic murmur has been heard without any uncertainty, we must not be led astray later on by the fact that it may have disappeared.

Considerable difficulties arise when a stenosis—even a very marked one—is seen for the first time at a period when the cardiac muscle has become weak. In such cases the murmurs follow each other in a confused sequence, as we have already described elsewhere (Cardiac Insufficiency, p. 61). And the rapid irregular beat of the heart makes impossible the recognition of the phase during which a murmur occurs. The experienced observer then refrains from a definite diagnosis, and is not surprised when

* *Loc. cit.*, p. 112.

a marked narrowing of the venous orifice is found at autopsy. The following case may serve as a good example of what may occur:

Case II.—Marked muscular weakness of the heart which did not allow of the recognition of a mitral insufficiency and stenosis. Slight stenosis of the tricuspid. Absence of dropsy, in spite of severe signs of heart weakness. Death as the result of embolism in the left cerebral carotid. Duration of the clinical observation, about three and one-half years.

Mrs. A.: Died at the age of fifty-eight years. Alleged acute articular rheumatism at unknown dates. First admission, August 16, 1875. Extreme dyspnea, stormy irregular heart's action, but rapid improvement after the use of digitalis. Second admission, February 4, 1876: The same. Third admission, June 29, 1876: A more severe attack.

From June 21st the patient complained of weakness, of slight difficulty in respiration, and of loss of appetite. On the morning of the twenty-eighth severe vomiting of watery material and diarrhea. She complained of black spots before the eyes, vertigo, and severe headache. Admitted on the following day. There was no rise in temperature (37.7° C.—100° F.), but the pulse was slow—a condition which continued until the fifth of July. The minimum pulse was observed on July 3d, with 36 pulsations at the radial and 39 contractions of the heart. The heart's action was so weak that the pulse at the carotid showed a tracing such as that reproduced in Fig. 64. Rapidly developing dilatation of the heart. Improvement occurred after a short time, and the patient was able to walk six kilometers twice daily, beginning on July 9th.

Fourth admission, February 6, 1877: Since the third, she complained of cough and headache. On admission there was a diffuse bronchial catarrh, with a rise of



Fig. 64.—Mrs. A.; carotid pulse (Brondgeest's pansphygmograph).

temperature to 39.9° C (103.8° F.). Marked cardiac weakness. Discrepancy between the radial pulse and the heart-beats most marked on the eighth. Pulse 66 and 98 heart-beats. The fever disappeared after three days, and the patient was discharged, able to work, after six days.

Fifth admission, December 14, 1877: The bronchial catarrh had been troublesome at times and had increased considerably within the last weeks. There was also loss of appetite and general weakness. On admission the patient showed severe dyspnea, widely diffused catarrh, but no rise of temperature. At first the sputum was like that of pulmonary edema. Soon the expectoration diminished. Cardiac weakness the same as at the preceding attack, except that now there was actual intermission. She had improved so much by the twenty-fourth of December that she was discharged, but was again admitted on the twenty-ninth, with the old complaints, and was discharged again on January 6, 1878.

Sixth admission, February 2, 1879: The patient had been unable to work during the whole of the past year, and had remained most of the time in her room, often even in bed. The dyspnea and the loss of strength increased. There was now an increase in all the symptoms. The temperature rose to 39.8° C. (103.6° F.), but this fever only lasted for three days. A slight area of dulness was present in the right lung for a few days. The cardiac weakness was at first very marked, and showed again a tendency to become slower. There were 50 radial pulsations and 62 heart-beats a minute. Improvement followed, although it was very slow, and the patient was able to leave the bed about the middle of March after a severe diarrhea had been abated at the beginning of the month. On March 29th the patient was seized with hemiplegia and with severe apoplectic symptoms, of which she died within thirty hours.

The condition of the heart in the course of the observation is of considerable interest. From February 4 to June 29, 1876, no change occurred in the area of

cardiac dulness. A change occurred then within two days and was quite marked. The following table shows the exact changes:

Date.	Sternal Line.	Mammillary Line.	Apex-Beat.	Right Side of Sternum.	Sounds.
February 4 and June 29, 1876.	Fourth rib, lower border.	Fifth rib, lower border.	Fourth intercostal space, 2 cm. outside of nipple line.	No dulness.	Pure, slightly flapping, somewhat ringing over the aorta.
July 1, 1876.	Third rib, lower border.	Not definite.	Fifth intercostal space; anterior axillary line.	1.5 cm. outside of sternum.	Faint, without murmurs.

The carotid pulse was very weak after the dulness had extended. The frequency of the pulse had diminished considerably at that time (see Fig. 64).

An examination of the boundaries of the heart dulness on February 6, 1877, did not show any changes in the area as observed in July 1, 1876. The heart-sounds were as follows: Apex: systolic tone reduplicated; diastolic, flapping. Pulmonary sound: extremely weak, scarcely perceptible. Aortic sound: more distinct, pure. Right ventricle: systolic sound still more markedly reduplicated.

On December 18, 1877, we found that the cardiac area began at the lower border of the second rib (sternal line), and passed to right and left in a flattened arc. The apex-beat was in the fifth intercostal space, 4 cm. externally from the nipple-line. The heart-sounds were faint and pure, louder on the right side than on the left. On February 2, 1879, the heart dulness extended two finger-breadths beyond the right sternal border. On the left side it was beyond the nipple-line. The apex-beat could not be felt. The cardiac sounds were very weak; there were no murmurs. The diastolic sound over the pulmonary artery was somewhat louder than that over the aorta, and somewhat ringing.

The autopsy gave the following result (von Schüppel; extract): "Both lungs emphysematous, freely movable; catarrh only in the larger bronchi. The heart measures 16 cm. in diameter and is 10.5 cm. long. It is quite flabby in all parts.

"The mitral orifice is narrowed to a slit which allows the tip of the little finger to pass fairly easily. The left auricle is dilated, and its endocardium clouded and thickened. The mitral flaps are markedly thickened and shrunken and are but slightly movable. They are covered near the auricle with thick, chalky masses, some of which appear very distinctly on the surface. The tendinous cords are contracted to at least half their normal length, markedly thickened, but do not coalesce directly with the valvular flap.

"The cavity of the left ventricle is somewhat dilated. Its wall is thickened, its musculature pale, grayish-brown in color, indistinctly striated, and is very brittle and friable.

"The aortic valves are competent; their flaps are slightly thickened, and their points of insertion very slightly rigid. There is extensive but slight atheroma of the aorta, with a marked brittleness of the intima. The right side of the heart contains both fluid blood and white clots, especially in the auricle. The tricuspid orifice is slightly narrowed, but admits both index and middle fingers easily. The elastic valvular flaps have somewhat thickened edges, and the papillary muscles are fibroid at the tips. The tendinous cords are markedly shortened and reach one millimeter in thickness.

"The right ventricle is enormously dilated and almost admits a fist. Its cavity is free from old thrombi. The wall of the pulmonary artery, including its branches in the lung, is thickened, and its intima in many places is the seat of fatty degeneration, is brittle, and is easily torn off in large shreds. The valves are normal. The musculature of the right ventricle is in the same condition as that of the left. Its wall is slightly thickened and is infiltrated with fatty tissue, especially at the apex. There is also some fatty tissue under the endocardium. The muscle of the right ventricle, looked upon from the inside, is indistinctly striated.

The liver showed parenchymatous hepatitis, and was a little smaller in all diameters. The spleen was enlarged to about one and one-half times its normal size,

cyanotically indurated, and contained an old infarct. The kidneys also showed cyanotic induration and old infarcts. There were a few areas of softening in the brain, and a thrombus almost 1 cm. long in the left cerebral carotid, the wall of which was only slightly atheromatous.

The weakness of the heart muscle in this case was very prominent in the clinical picture—so much so, that it masked the considerable changes in the mitral and its orifice during the entire period of almost four years in which this patient was under observation.

The tricuspid stenosis, although slight, is quite noteworthy. Possibly this stenosis was not unfavorable, in the sense that it made the regurgitation of blood from the right ventricle into the right auricle more difficult, and thereby facilitated the outflow of blood from the systemic veins. The auricle, then, would have attended to the filling of the heart itself, although under somewhat unfavorable circumstances. This did take place, inasmuch as the clinical history shows that no venous pulsations were noted. A rather unusual feature in this case was the total absence of dropsy during the entire period of illness.

I refrain from discussing the various less important signs that have from time to time become of value in the diagnosis of mitral stenosis.* I may mention only that the increased intensity of the diastolic tone over the pulmonary artery is of some weight. It has been referred to the lessened tension of the aortic valves as the result of the imperfect filling of that vessel.

Prognosis of Lesions of the Mitral Valve and the Left Venous Orifice.—The prognosis of these lesions may be considered together in a brief way, inasmuch as we have already discussed all the essential points under "cardiac weakness." We must remember that any catarrh of the bronchi is dangerous, in these cases, and that this danger is greater the more extensively the finer bronchi are involved and the more diffusely the process has extended.

Furthermore, it must be noted that when the left ventricle fails to work sufficiently, the blood-supply of the heart itself suffers. Cardiac weakness is thereby directly favored. The stenosis produces an imperfect filling of the left ventricle, and the more marked the stenosis is, the greater this danger becomes. Inasmuch as the right ventricle is affected even more in stenosis than in insufficiency, the predominance of the stenosis may be said in general to be the less favorable feature of this group of heart lesions. If both stenosis and insufficiency maintain a certain balance, then compensation may be maintained for a considerable time.

Treatment.—So long as there are no signs of cardiac weakness, the treatment of lesions at the venous orifice should consist only of a regulation of the mode of life, in order that the dangers that threaten every person with heart disease may be avoided, and particularly those which threaten patients with the lesions under consideration.

Foremost of these are the affections of the bronchi. We should do all in our power to prevent these complications, and to effect a rapid cure of such bronchial catarrh as may develop. Persons who live in favorable conditions should be warned over and over again to regard every bronchial catarrh, even the slightest, as a serious occurrence, and to spare no sacrifice to counteract the tendency to chronicity and to the frequently recurring relapses connected therewith. In patients who are in less favorable circumstances one has, sadly enough, many oppor-

* See also v. Leube, "Specielle Diagnose der inneren Krankheiten."

tunities to see how fatal catarrh often may be, and how little we are able to do for it when the external conditions of life cannot be regulated properly. This is not the place to discuss the methods of treatment of bronchial catarrh, and I have expressed my ideas on this subject elsewhere.*

As regards the use of digitalis, it must be remembered that in mitral disease the work of the heart takes place under the most favorable conditions—first, when the frequency of the contractions is as low as possible within a given period of time, and, second, when the duration of the diastole is as long as possible. These propositions consider merely the mechanic results of the valvular lesions.

The following table shows the differences which are deduced from an increase of the pulse from 60 to 70 according to the calculations of B. Lewy.† In the higher degrees of insufficiency the demand upon the heart's action, even at rest, is very great when 120 heart-beats take place per minute. During muscular activity with four times the circulation that takes place at rest the limit which has been set for the hourly work of the heart (14.315 mkg.) when β : 0.7 has almost been reached. When β : 0.8 this limit has been overstepped.

Work of the heart in mitral insufficiency in meter-kilograms when the body is at rest, and during muscular exertion, requiring four times the amount of blood needed at rest:

β = size of opening in the valve as compared to 1. z = frequency of the pulse.
Duration of diastole when z= 60—0.643 seconds.
Duration of diastole when z= 70—0.5 "
Duration of diastole when z=120—0.2 "

	Perfect Rest.			Exercise.
β	z = 60	z = 70	z = 120	z = 120
0.01	867	873	901	3591
0.05	1080	1125	1254	3859
0.1	1352	1454	1702	4322
0.2	1910	2096	2645	5339
0.3	2495	2782	3656	6441
0.4	3145	3547	4824	7736
0.5	3853	4389	5945	9256
0.6	4662	5357	7502	11133
0.7	5612	6507	9913	13564
0.8	6765	7941	12784	16874
0.9	8230	9762	>15000	21742
1.0	10068	12132	>15000	27760

The actual condition becomes still less favorable through the fact that muscular work increases the burden of both ventricles, to the disadvantage of the weaker right ventricle. According to the calculations, for example, when there was muscular activity and when the pulse was 120 and β was 0.7, the work of the left heart was increased only a little over twice the normal amount, while that of the right had to be increased about five times. The same conditions obtain in stenosis of the left venous orifice. This stenosis apparently offers the best possible conditions for the em-

* Jürgensen, "Handbuch der Therapie innerer Krankheiten," edited by von Pentzold and Stintzing, vol. iii; "Prophylaxe und allgemeine Behandlung der Erkrankungen der Athmungsorgane," p. 3. "Behandlung der Luftröhrenerkrankungen," p. 230 of the third edition, Jena, 1902, G. Fischer.

† *Loc. cit.*, pp. 532 and 553.

ployment of digitalis. It was thought that the more time the blood has to pass the narrowed orifice, the greater would be the amount which would enter the left ventricle. Digitalis increases the duration of the diastole, and at the same time diminishes the number of systoles occurring within a given period of time. The favorable influence of digitalis, which thus becomes reasonable, may be applied also in insufficiency of the valve, yet this does not mean that digitalis is indicated in every case in which there is an increase of the heart's action. A better way to express this would be thus: Lesions at the left venous orifice, in so far as the mechanic conditions which they produce are concerned, present no contraindications against the use of digitalis.

INSUFFICIENCY OF THE AORTIC VALVES.

Arteriosclerosis produces changes in the valves of the aorta much more frequently than does endocarditis. Aortic lesions, therefore, occur

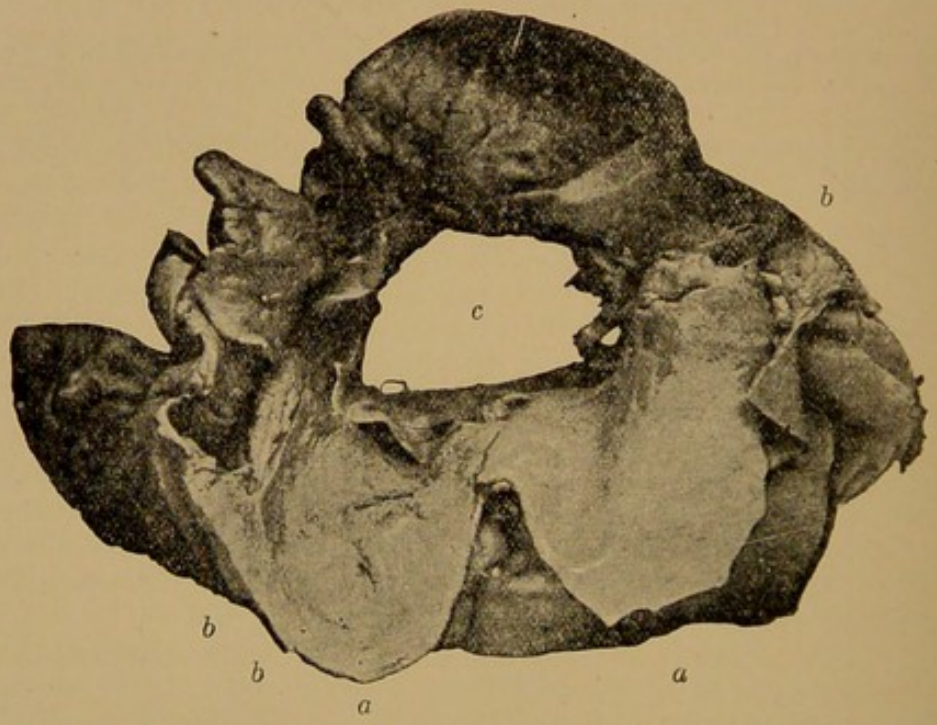


FIG. 65.—*a*, The aorta cut open showing shrunken valves (*b*); *c*, ventricular orifice of aorta (photograph from a preparation in the Anatomic Institute at Tübingen).

usually, it is said, in about four-fifths of the cases in the later periods of life, and undoubtedly are more frequent after the age of forty. They are often complicated with extensive disease of the aorta itself.

Endocarditis may lead to the perforation or the detachment of one or more of the aortic valves. It is also supposed that very violent physical exertion may cause the valve to become detached. Insufficiency of the aortic valves may be pure and not complicated with stenosis of the orifice, but this is not the rule. Fig. 65 shows a case of pure insufficiency.

The manifestations of a well-developed but compensated insufficiency of the aortic valves are as follows: Hypertrophy and dilatation of the left ventricle, *pulsus celer*, and a diastolic murmur over the aorta. The general condition is often but slightly disturbed for a long period, at least when the valvular lesion is not too marked, and when the heart muscle remains

capable of performing its work. When the patient undergoes more violent physical exertion or becomes mentally excited, he usually suffers from palpitation and less frequently from dyspnea.

The behavior of the arteries, especially of the coronary arteries, is a deciding factor in the course of the disease. The degree and extent of arteriosclerosis is the most important factor in determining the fate of the patient. The clinical picture, therefore, at first, is that of atheroma of the arteries, especially of the coronary arteries, and later becomes that of cardiac weakness (see p. 376).

The experiments of Moritz upon his model showed that the diastolic regurgitation of blood from the aorta into the ventricle produces a lowering of the pressure in the less perfectly filled artery, while, on the other hand, the pressure in the pulmonary veins and in the pulmonary artery rises. At the same time the expulsion-volume of the right ventricle is diminished. (It was diminished only from 2.0 to 1.8 c.c. in the experiment.) The expulsion-volume of the left ventricle, on the other hand, is increased (from 2.0 to 4.3 c.c. in the experiment) and the ventricle itself becomes dilated (to the extent of 1.9 c.c. in the experiment). The work of the left ventricle is considerably increased (from 190 to 383 gram-centimeters in the experiment). The conditions in the right ventricle show slight variations in both directions. In the experiment the work of the left ventricle was then increased (from 383 to 630 gram-centimeters or 64 per cent.), and then a relative overcompensation was produced—higher pressure in the aorta and lower pressure in the pulmonary artery. Mechanically considered, therefore, the work of the left ventricle which is dilated during diastole and which is strengthened by an increase in muscular mass, is fully sufficient to compensate for the permanently increased labor required of it. In animal experiments the reserve force of the ventricle is at first sufficient to effect this compensation. But one thing remains, and that is the marked variations in the filling of the arteries, and this constitutes the danger in aortic insufficiency.

We have already explained why the right ventricle, as a rule, is slightly hypertrophied (see p. 321). This explanation is not generally accepted as sufficient. Some doubts may be entertained because we are dealing here only with the very small amount of blood which has become necessary for the nutrition of the hypertrophied left ventricle.

Attempts at other explanations attribute the hypertrophy of the right ventricle to an increased labor which this ventricle itself is obliged to furnish. Liebermeister* expresses his opinion on this subject in the following way. After having explained that complete compensation is only possible when every systole drives such an amount of blood into the aorta that the normal amount remains after subtracting the regurgitated blood, and that the left ventricle must, therefore, become dilated and hypertrophied, he continues: "In what way does dilatation of the left ventricle take place, which is sufficient to receive during diastole not only the normal amount of blood from the auricle, but also the blood regurgitating from the aorta? Such a dilatation of a cavity with strong muscular walls like the left ventricle does not develop easily, and cannot take place without previous and important disturbances in the circulation.

"According to current ideas, this dilatation is suffered to take place as the result of the increased pressure of the blood that flows back from the aorta. But it can easily be seen that this is impossible, for the blood

* "Vorlesungen," vol. iv, p. 368.

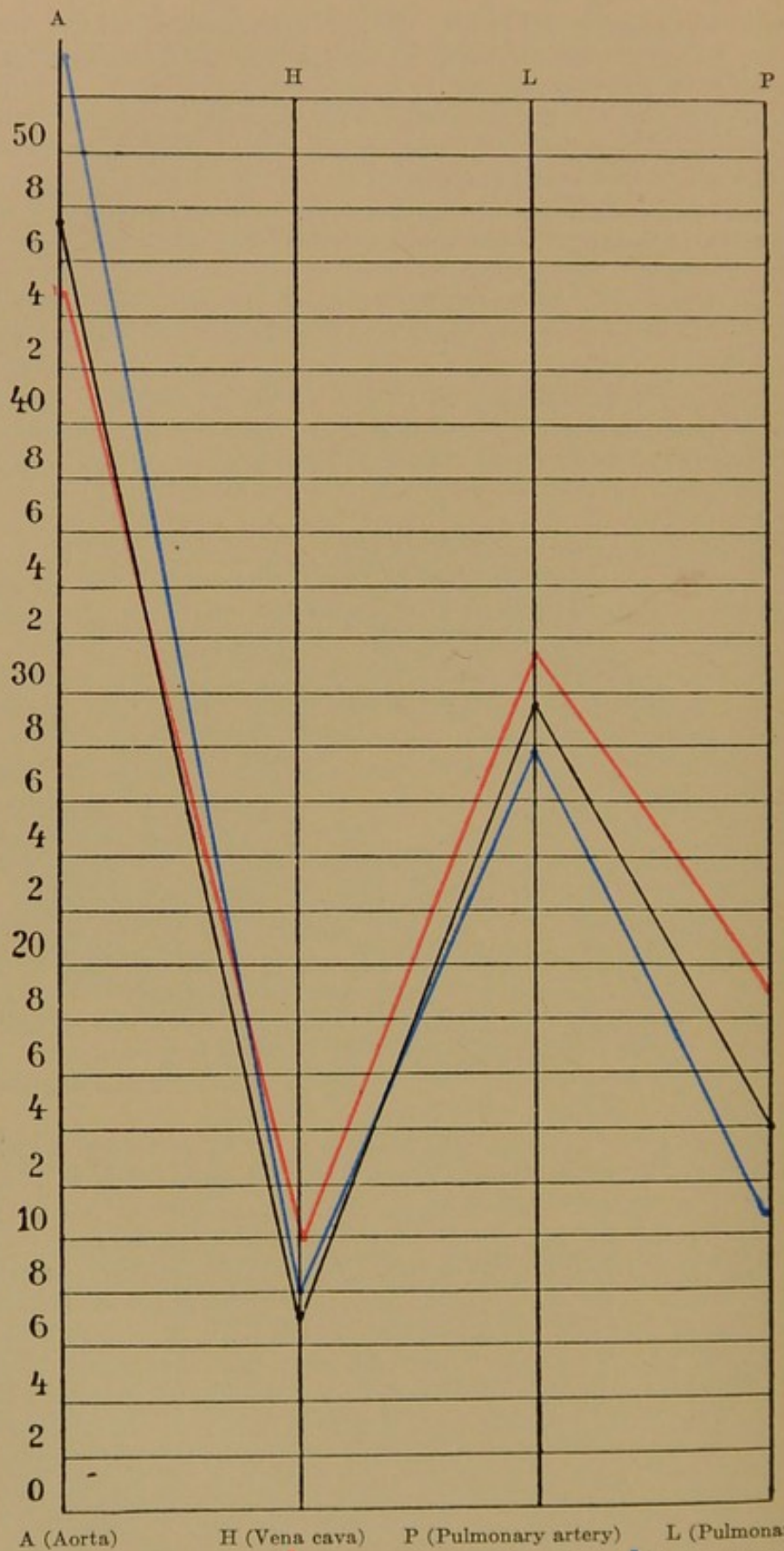


FIG. 66.—Moritz's Experiment 49.

which regurgitates from the aorta will not dilate the ventricle, but will only hinder the inflow of blood from the auricle, which normally takes place under very weak pressure. A dilatation of the ventricle could only arise if the amount of blood flowing back from the aorta and the arteries were larger than the amount propelled into the aorta during systole, and in that case the patient could live only a short time.

"The left ventricle, therefore, cannot be dilated by regurgitating blood from the aorta, except when the blood from the auricle flows in under such strong pressure that the ventricle is thereby distended. The necessary pressure in the auricle is obtained as the result of stasis in the auricle, and in the entire pulmonary circulation, and also indirectly through the increased work of the right ventricle. This latter is the essential cause of the compensating dilatation of the left ventricle. Therefore, every aortic insufficiency is accompanied by considerable disturbances of circulation at first, and only gradually can compensation become established.

"When dilatation and hypertrophy of the left ventricle have once developed to a sufficient degree, the stasis in the pulmonary circulation disappears, the left auricle once more empties into the ventricle without an abnormally high pressure, and the right ventricle no longer is obliged to furnish an increased amount of work. Thus congestion of the pulmonary circulation is not a permanent effect of the compensated valvular lesion, as it is in mitral insufficiency, but ceases whenever a sufficient dilatation and hypertrophy of the left ventricle has taken place."

Although this view contains some truth, it does not include the whole truth. In the experiment of the model a rise of pressure was found in the pulmonary artery and in the pulmonary veins. The right ventricle, therefore, was obliged to overcome an increased resistance. This alone means that it must perform a permanently increased amount of work. On the other hand, its expulsion-volume is diminished, for when the left ventricle is not able to empty all its contents into the aorta, a correspondingly diminished quantity of blood must enter into the right ventricle. Inasmuch as the latter cavity does not expel a larger quantity than it receives, its expulsion-volume is diminished by the amount of blood that flows back into the left ventricle during diastole. Thus the amount of work which it has to do is diminished.

The filling of the left ventricle is produced not so much through the increased pressure in the pulmonary veins and the left auricle, as through an increased pressure in the aorta. The latter pressure is chiefly responsible for the dilatation, as it brings into the ventricle larger masses of blood under high tension. Besides, the increase of pressure in the left auricle certainly also comes into play. The behavior of the living heart must be considered as alone decisive, as it introduces conditions which are not so simple as those in the model.

The relaxed heart is capable of adapting itself to the amount of blood filling it in such a manner that the more or less extensive increase in the elasticity of its muscular walls offers varying resistance to the entrance of the blood. The heart, therefore, possesses a regulating mechanism which acts according to the tasks required of it. And here, when it becomes necessary to fill the left heart with a rather large amount of blood, this mechanism acts by favoring a primary dilatation and by diminishing the elastic tension during the diastole. The same mechanism, however, also possesses the property of so shaping the systole that it commands an

increased amount of driving force (reserve force), and thus is able to drive out a greater amount of blood from the left ventricle.

Whenever this adaptation of the heart does not take place to a proper degree, there may be actually an interference with the pulmonary circulation.

"When the tension of the walls of the left ventricle does not diminish in correspondence with the amount of blood that enters this cavity, considerable difficulties arise for the pulmonary circulation. In order to create such obstacles, it is not necessary that the left ventricle should dilate rapidly and markedly. Then, at the beginning of diastole, while the ventricle is in the stage of suction, the aortic blood that is under such high pressure flows into the ventricle in much larger quantity than the contents of the left auricle. Thus the suction of the ventricle which is so important for the propulsion of this blood only comes into play to a slight extent, and naturally the obstacle will become very marked during the systole of the auricle, because the musculature of the auricles is so extremely weak and can develop only a slight amount of pressure. The result is a stagnation of blood in the left auricle and a rise of pressure in the latter. In fact, this influence of aortic insufficiency upon the pulmonary circulation has been also seen experimentally" (Krehl).*

In their final analysis, therefore, in cases in which circulatory disturbances take place in the sphere of activity in the right heart, the difficulties depend once more upon some form of incapacity of the cardiac muscle. Liebermeister† also emphasized the fact that this incapacity of the heart muscle which leads to cardiac weakness in the end may so impair the pulmonary circulation for a time that hypertrophy of the right ventricle takes place, provided conditions of nutrition are otherwise favorable.

Ingenious though Liebermeister's description may be, it savors a little too much of the mechanic. In the first place, the pulmonary circulation is not always affected, not even in animal experiments. These experiments rather seem to show that a vigorous heart, which has not been too severely affected by the operation, is well able to take care of the new work which is demanded of it.‡ And experience in man certainly teaches the same. Let us rule out those rare cases in which an injury or a great exertion leads to extensive changes in the valves. In these cases one can never say whether and to what extent the muscle will also suffer.

If the destruction of the aortic valves has taken place through endocarditis, then we have to deal with a coccus infection which affects the entire heart and extends also into its musculature. If we are dealing with atheromatous changes, then, I think, we may ask with reason what the condition is in the coronary arteries, and what the state of nutrition of the heart muscle which depends upon these vessels.

We must always reckon with the variations in the working capacity of the heart muscle, which are so great in different individuals, and perhaps these variations are more important than the degree of mechanic change —that is, the amount of valvular destruction. Of course, the importance of the latter must not be underestimated.

* On p. 17, he refers to the experiments of S. Kornfeld, "Ueber den Mechanimus der Aorteninsufficienz," "Zeits. f. klin. Med.," vol. xxix (1896), pp. 344 *et seq.* Important also is the experimental work of Hasenfeld and Romberg (see ‡ below).

† *Loc. cit.*, p. 373.

‡ *Cf.* "Ueber die Reservekraft des hypertrophischen Herzmuskels und die Bedeutung der diastolischen Erweiterungsfähigkeit des Herzens," Hasenfeld und Romberg. "Archiv für experimentelle Pathologie und Pharmakologie," Bd. xxxix (1897), pp. 333 *et seq.*

According to the calculations of Benno Lewy,* in insufficiency of the valve which allows 20 per cent. of the orifice to remain open, the work required of the heart is multiplied by two and one-half, as compared with the normal, while an opening of 83 per cent. would make the continuance of life impossible, inasmuch as the entire sum of the muscular force available in the whole body would be required by the heart. (Compare above, p. 315.) Now, in many cases the space left in the aortic valve is less than one-tenth of its diameter, and up to that point the work of the heart increases, according to the number of pulsations: When seventy pulsations take place per minute, it increases about three-fifths over the normal. When 120 pulsations take place, it increases about two-fifths. When the defects are smaller, as, for example, one-twentieth, the figures are one-third and one-fifth respectively. A heart with a normal working capacity can fulfil these requirements.

In order to secure complete compensation, primary dilatation of the left ventricle, with its following hypertrophy, usually suffices. The left ventricle is the best developed muscular tissue of the heart, and it can stand a great deal. But under the conditions obtaining here, the capacity of the arteries, especially of the aorta, is impaired. Every systole throws a larger amount of blood into the aorta, under higher pressure, and thus produces a marked dilatation of the already dilated arterial trunk. Every diastole, on the other hand, allows it quickly to return to a state of rest. This puts increased requirements upon the elastic wall of the vessel. When these walls fail to perform their function properly, arteriosclerosis, with all its consequences, develops. The coronary arteries which are involved in this arteriosclerosis give rise to severe and extreme disturbances of nutrition, which are accompanied by symptoms of cardiac neuroses, leading up to marked angina pectoris.† The aorta itself becomes dilated, especially in its ascending portion, and later in the arch. Usually this dilatation is tubular, uniform, less often sacculated and circumscribed. [The pathology of arteriosclerosis has been discussed in other portions of this work but not included in the translation.—Ed].

The characteristic sphygmogram of aortic insufficiency will be discussed later. Suffice it here to say that the pulse is hard (durus), tensely filled, and high (altus), that the artery is strongly dilated, while the pulse rapidly rises and falls—in other words, is bounding (celer). When the compensation is good, the average pressure does not exceed the normal limits.

Anatomic Data.—Bamberger describes the anatomy of aortic insufficiency as follows:

"When the atheromatous process is at the bottom of the trouble, the aorta and its branches are usually involved also, and these vessels seem to be the point of origin of the disease. Yet it happens not infrequently that but slight traces of the process are visible in the aorta and its branches, for example, slight gelatinous deposits or an unimportant fatty degeneration, while the valves are very greatly changed. The process not infrequently affects also the venous valves, especially the mitral.

"When endocarditis is the cause of the disease, the aortic valve is rarely affected alone, but usually the mitral, less frequently the tricuspid, valve, is also involved, and if we can trust to appearances during life, the aortic valve, as a rule, becomes diseased only after the mitral valve has been affected."

I should not like to attribute too much importance to the last-mentioned assertion. According to Bamberger's own observation (see table, p. 311), there were

* *Loc. cit.*, pp. 538 *et seq.*

† *Cf.* Krehl, Diseases of the Myocardium.

only nine cases out of forty-five instances of valvular disease localized in the aorta, in which the mitral and the aorta were simultaneously affected. Biermer, it is true, counted 283 cases with localization in the aorta alone, against 110 in both valves simultaneously. A great difference is noted when the results of the observations of different authors are compared. It is impossible to control these data sufficiently to give a definite opinion. The only fact that seems to be established in this connection is the very great frequency with which the combination of lesions in the aorta and the mitral is observed.

The changes in the valve are essentially the same as those already described in the venous valves, modified as the result of the different anatomic shapes of the valvular structures involved. The disease affects all three flaps, or else one or the other of the three. In addition to thickening, rigidity, and impaired mobility, it should be noted that, as a result of the shrinking of the valve and its insertion ring, a markedly shrunken flap not infrequently lies lower than the others, so that the hollow existing in this flap becomes diminished on account of the loss of its concavity, or that the valve may often become perforated or detached from its insertion ring. In the majority of cases the valve is thickened and studded with deposits of new tissue, and thus a certain degree of stenosis is produced, although this stenosis is often so slight that it does not require consideration.

The change which the heart undergoes in this form of valvular disease consists, in the first place, of a dilatation with hypertrophy of the left ventricle, both of which are almost always very marked. The walls of this cavity may reach 2.7 cm. in thickness, while its cavity very commonly becomes large enough to admit a large fist, and the diameter of the ventricle is very often as much as 13 cm. in every direction. The dilatation always predominates, and when no complication exists, especially stenosis or marked atheromatous process in the aorta, the thickness of the ventricular wall rarely exceeds from 1.3 to 1.6 cm.

The character of the dilatation, however, is also quite characteristic for the valvular affection under consideration; for it consists essentially in a dilatation of the aortic portion of the ventricle. If we imagine the left ventricle divided into two portions along its long axis, the inner portion of the two corresponding to the aortic entrance (aortic portion), while the outer corresponds to the mitral valve (auriculoventricular portion), then these will have a fairly equal capacity under normal conditions. In aortic insufficiency, however, the former portion is enlarged so markedly at the expense of the latter that almost the entire cavity of the ventricle seems to belong to the aortic portion.

The papillary muscles also take part in the hypertrophy of the ventricle. They become thick and elongated. The trabeculæ also show similar changes. The ventricular septum in marked cases becomes convex toward the right ventricle, thus lessening the capacity of the latter. The flaps of the bicuspid are frequently stretched and elongated (as the result of a dilatation of the ventricle and possibly also of hypertrophy of the papillary muscles), and this is especially noticeable in the aortic flap. The tissue of this valve is also often involved, but in most cases there is simple thickening or scattered calcareous deposits, especially on the aortic flap. The left auricle, as a rule, is neither dilated nor hypertrophic, and in this differs radically from the ventricle.

A well-marked change in form and in capacity is almost always noted in the right ventricle. Its cavity becomes elongated, scythe-shaped, concave toward the left. The cavity is apparently stretched over the septum

of the ventricle, which is elongated and strongly convex to the right, so that the transverse diameter of the right ventricle is greatly diminished; in fact, in very marked cases the walls of this cavity at its lower portion are almost in contact. A rather imperfect compensation for this loss of space takes place by a dilatation of the upper portion, especially of the cone of the pulmonary artery, and usually by a moderate hypertrophy of this portion.

Any other condition of the ventricle than that described rarely occurs, except as the result of complications, especially affections of the mitral valve, pulmonary diseases, or in cases with a slight aortic insufficiency and its consequences.

The right auricle and the veins which enter into it are usually markedly dilated when the disease has lasted for some time, and this dilatation takes place partly as the result of the lessened space in the right ventricle,* and partly through the effect of the valvular lesion upon the venous circulation, which we shall describe later.

The aorta is dilated often so much that it exceeds the diameter of the pulmonary artery by more than half. The form and position of the heart show essentially the following changes:

"The increase in the size of the heart is very marked—more marked, with a few exceptions, than in all other simple valvular diseases. It involves only the left ventricle, with the exception just noted, while the right ventricle often appears only as a small appendage. Inasmuch as the principal axis of the left ventricle is the longitudinal, the heart appears to be enlarged chiefly in its long diameter. But in all rather well-marked cases the heart also increases more or less, in width, as the result of the simultaneous increase in the size of the transverse diameter of the ventricle.

"The apex becomes more obtuse, and at the same time more elongated, whereby the apex of the right ventricle grows further distant from the apex of the heart than usual, and seems to be pushed up, while, as a matter of fact, the left ventricle has simply grown down further. The heart is turned a little toward the right, on its vertical axis, so that a larger portion of the left ventricle than normally is turned forward, and is in contact with the chest-wall. At the same time the heart lies more horizontally, and its apex is turned more toward the left."

The Physical Examination.—The movements of the heart are visible and palpable over an extended area. On the left side, they may be perceived up to the axillary line or beyond it, not only in one intercostal space, but in several. When there is marked hypertrophy, the thorax moves over a surface of the size of a hand. There may be a permanent distention of the thorax (precordial bulging). The further outward the heart's action is perceptible, the lower downward can the heart-beat be detected—usually in the sixth and, often enough, even in the seventh or eighth intercostal space, although the latter is exceptional.

The apex-beat is broad and heaving. In order to determine its location definitely, it is best to pass the palpating finger from behind forward, from the spine to the sternum, in the intercostal space which we wish to examine. The spot where the finger can be felt to be distinctly lifted by

*This point has been emphasized in the past by various authorities, among them Friedreich. No proof has ever been brought that diminution in the size of the ventricle actually takes place. Later this explanation of the change in the pulmonary circulation disappears from the literature. v. Dusch, in his "Lehrbuch," which appeared as early as 1868, objected to it on theoretic grounds.

the tense ventricular apex indicates the location of the true apex-beat. Besides the apex-beat, we should note changes in position and systolic and diastolic movements which affect the entire heart. During the systolic contractions the corresponding portion of the heart muscle naturally also becomes tense, and therefore can also be determined by palpation from without inward.

The arteries, even the ascending aorta itself, may be seen to pulsate vigorously. The head may be so involved in the motion that we see it move rhythmically from a distance, and even the smaller arteries may be seen to pulsate. Very often, especially in the cases of arteriosclerosis, the aortic arch is found to be situated so high that it communicates its motions to the soft parts above the jugulum. A finger pressed in at that point can feel the aortic arch.

A systolic thrill is said to be palpable in some cases, in addition to the diastolic, in the region of the third left costal cartilage and of the adjoining segment of the sternum. Bamberger noted the diastolic thrill but rarely, and attributed the systolic thrill not to the insufficiency, "but to rough surfaces in the usually stenosed orifice." Accordingly, a stenosis of the aortic orifice might be made out from the presence of this thrill. Gerhardt,* with whom I agree, remarks on this subject:

"In addition to a variably marked pulsation of the intercostal spaces in the region of the apex, we often observe a pulsation at the sternal end of the second or third intercostal spaces, and in these cases we usually feel at this point a diastolic thrill which is distinctly transmitted downward, or else both a systolic and a diastolic thrill." Bamberger probably meant the cases in which there was no dilatation of the ascending aorta, but such cases are less frequent.

A systolic thrill can be felt in the region of the apex-beat, and more especially in the surrounding parts, when the heart's action becomes somewhat excited. This systolic thrill, in my opinion, is derived only from the more rapidly contracting muscular fibers of the hypertrophied heart. The sensory perception itself is characteristic, to a certain extent, for the experienced observer.

Percussion gives an outline of the dulness, which is enlarged especially in the longitudinal direction, although a less extensive increase in the width of the area can also be demonstrated. (See pp. 392 and 393, and Figs. 70 and 71.) When heart weakness develops, the size of the area of dulness naturally increases, and also spreads to the left, where the boundary-line becomes more and more concave inward. Then the horizontal increase may exceed the vertical (Fig. 72). By delicate topographic percussion we can often demonstrate a dilatation of the ascending aorta.

The characteristic sign on auscultation is the diastolic murmur, which is heard loudest at the sternum in the second right intercostal space, but which also is clearly heard beneath the sternum itself. The classic authors, that is, those who, with their contemporaries, laid great and greatest stress upon the diagnosis of valvular lesions, vary somewhat as to the place where this murmur is heard most distinctly, or, in other words, where it is loudest. In my own opinion, these differences are of but slight importance. On the other hand, the conditions of transmission which are dependent upon the varying position of the point of origin of the murmur in relation to the thoracic wall are of great importance. The valves of the aorta must naturally take part in the change of position which this vessel undergoes in virtue of the hypertrophy and dilatation of the left ventricle. Even Gerhardt pays a tribute to the teachings of his youth when he remarks: "In several instances I could not conceal the fact that the murmur was

* "Lehrbuch," p. 315.

louder at the left side of the sternum than at the right, in spite of the fact that aortic insufficiency had been made out by all who had examined the case."

If we pass with the stethoscope from the sternum to the portions of the chest which adjoin it on either side, the murmur becomes distinctly fainter. The transmission is not so good where ribs and intercostal muscles lie side by side, as through the uniformly dense bone. The diastolic murmur is also transmitted into the carotids, and also to the apex of the heart, being led in both instances along the blood-stream. Yet it does not always follow that the murmur is audible at either of these points.

The sensory impression directly produced by the murmur has been the subject of much attention, and, I believe, with good reasons. Friedreich* makes the following remarks on this subject:

"It seems remarkable that the murmurs of aortic insufficiency occurring in different individuals present many features in common as regards pitch and character. While, for example, the murmurs of mitral insufficiency show much greater differences in this respect, and are sometimes blowing, sometimes whistling, and sometimes more rough and uneven, the murmur of aortic insufficiency consists almost always of a long-drawn rustling and hissing sound, which is quite deep in pitch, so that a trained ear usually succeeds in diagnosticating it without reference to the place where it occurs with greatest intensity."

In addition to the whistling sound, a diastolic tone may also be heard. This is possible when a valve has remained capable of opening and of vibrating, and is probably more often heard in valves that have been destroyed by ulcerating endocarditis than in those which have suffered through arteriosclerosis. A still distinct or even an increased diastolic tone has been frequently reported, without further data. Some authors, however, as, for instance, Friedreich, ask whether this tone may not be transmitted from the pulmonary artery.

What can we hear, in addition to the diastolic murmur, when we listen over the aorta during systole? Frequently we do not hear a tone, but merely a murmur, or else both a tone and a murmur together. Here we have a variety of causes for the development of the murmur.

1. If the aorta has become markedly dilated above its bulb,—and this occurs by no means rarely, as we have pointed out,—then the eddying movements which produce the murmur may arise when the blood is driven with great force from the narrow into the wider part of the tube. This is rather common, according to the present view. (Compare the statements made above, p. 326.) I do not quite understand the explanation which Sahli* briefly mentions without naming its author: "The systolic murmur is said to be the result of the impact between the diastolic regurgitating stream and the systolic stream."

2. Experiments have shown that a steadily flowing stream which passes through a glass tube, and which does not give any auditory sensations when its velocity is moderate, will do so as soon as its velocity is increased to a certain extent. Then a murmur becomes audible. Gerhardt's explanation is based upon the following facts: "In explaining the murmur of pure aortic insufficiencies with smooth walls we must remember that the left arterial orifice, even in health, presents a narrow portal which does not give rise to any murmur between the left ventricle and the aorta. When the blood-stream increases in velocity, as happens in aortic insufficiency, the conditions may be favorable for the development of a murmur on the farther side of this natural smooth stenosis."

3. The murmur may be produced when roughnesses, irregularities, in short, anything that increases the friction of the passing blood, and thus produces irregular vibrations, occur upon the valves themselves, or on the inner wall of the neighboring portion of the aorta.

* *Loc. cit.*, p. 371. † *Loc. cit.*, p. 343.

4. A true narrowing of the orifice, in other words, a true stenosis, may also be the cause of this murmur.

Bamberger also remarks, in a general way, that a systolic murmur is always considerably shorter, though not always fainter than a diastolic. This is self-evident from the shorter duration of the systole. The tone that may be audible resembles a dull sound, according to Bamberger—either a murmur or nothing at all.

A pure systolic tone is not very frequently heard over the mitral. This statement is made by nearly all authors, and is no doubt correct, although it can scarcely be satisfactorily explained. It has been thought that tissue changes may occur in the mitral valve, thickenings as the result of the endocarditis which extends to the valve. But the flaps may also be perfectly intact, and then this explanation will no longer suffice. We are reduced, therefore, in the explanation of the "accidental" murmur, to the choice between the possibility that it arises through a disturbed action of the muscles concerned in the closure of the valves, or through a disturbance of the vibrations of the valvular flaps. I think the more common occurrence is the muscular insufficiency of the mitral. When one has occasion to observe the beginning of an aortic lesion produced by endocarditis, which ends in an insufficiency of this valve, one can notice how remarkably variable is the systolic murmur over the mitral. As a rule, it is loud at the beginning of the disease, perhaps so loud that the murmur which is referred to the aorta itself appears of no importance, so that one sometimes is at a loss to know where the seat of the endocarditis is to be looked for. During the further course of the case, however, when the insufficiency of the aortic valves becomes well marked, the systolic mitral murmurs pass more and more into the background. (Cf. also case VI, p. 392.)

I should explain this in the following way: The endocarditis is, as always, complicated by myocarditis, and the latter is the cause of the insufficient closure of the valves, as in all insufficient muscular work in the heart. When the endomyocarditis progresses to recovery,—let us say, more accurately, to considerable improvement,—the muscular insufficiency at the bicuspid also disappears.

When the conditions of the thorax are favorable, we can demonstrate the variations in the cardiac area according to the working capacity of the muscles, day by day, to the extent of several centimeters. The increase in size of this area which characterizes the early stages either disappears entirely or is considerably reduced toward the end.

The fact that the murmur returns when a loss of compensation develops is frequently mentioned and is very true. This and the increase in the size of the area of dulness are also referable, undoubtedly, to the same cause.

What disturbs the regular vibrations of the valvular flaps? The opinions which have been expressed on this subject can, I think, scarcely withstand a critical examination. I should like to except, however, the explanation of Geigel (see p. 336).

Bamberger says: "A pure systolic tone is not very frequently heard at the mitral valve, even when this structure is perfectly free from disease. The reason for this I think is chiefly the rather eccentric position of the venous orifice (see above, p. 379), whereby the direction of the systolic blood-stream is entirely changed and the valve is only hit tangentially by the entire mass of blood which fills the aortic portion." The idea is, evidently, that the valve is set, but is not rendered sufficiently tense

by the systolic impact of the mass of blood. With the aid of a little imagination we can picture to ourselves approximately what Bamberger meant, but this scarcely explains the origin of the murmur. Nor are the "further reasons" more satisfactory: "The hypertrophy of the wall of the ventricle which acts as a bad conductor(?), the increased pulsation at the apex which produces a dull sound at the thoracic wall and the frequently coexisting thickening of the mitral valve." Bamberger is, therefore, obliged to fall back upon the sound produced by the muscle, and forgets that a thickened valve cannot be called "perfectly normal."

Another explanation is given by Traube.* He assumes that the systolic tone heard over the ventricles is not a muscle sound, as Stokes would have it, but a valvular tone in the origin of which the mitral plays a prominent part. He takes the view that "a great part of the acoustic phenomena of the circulatory apparatus, both normal and abnormal, have their origin in the fact that membranes change from a condition of low tension to one of higher tension, and that the intensity of a tone or murmur thus produced depends upon the different tensions which the tone-producing membrane undergoes in rapid succession." When the aortic valves are insufficient, "the differences of tension which the mitral successively assumes fall below the normal; for, on the one hand, the initial tension, that is, the tension of the valve at the end of the ventricular diastole, is increased; and, on the other hand, the final tension, that is, the tension of the valve during the systole of the left ventricle, is diminished. The initial tension is increased because the valve is under the pressure of the aortic blood at the end of the ventricular diastole, and this pressure is evidently far greater than that exercised by the weak muscle of the left auricle. The final tension of the valve is diminished, because the left ventricle is obliged to drive its contents into a system of tubes which has a lower tension than natural. While the sum of the resistances to be overcome by the ventricle is thus lowered considerably below normal, the increase of tension which its wall undergoes during the contraction—and the closed mitral forms an integral part of this wall—must needs be less than under normal conditions.

When the successive differences of tension in the mitral valve become less, then the vibrations of the valve membranes must lose in amplitude. This may become so slight that, under the conditions of conduction which exist in these cases, they no longer can be transmitted to the ear, and thus the tone produced by the valves becomes inaudible.

The following objections may be made against this explanation: Admitting that the systolic tone at the mitral arises through vibration of the tense membrane, and that its intensity depends upon the differences in tension, we may ask: why does the final tension of the mitral diminish? For, how do we know that the sum of the resistances to be overcome by the ventricle is diminished considerably below its normal value? What produces the hypertrophy of the left ventricle if such be the case?

Traube and his immediate followers maintain that "no systolic tone whatever is formed at the mitral in typical cases of aortic insufficiency."* This statement is not confirmed by our best observers. He who wishes to study the question as to the systolic tone or murmur at the apex in aortic insufficiency, in my opinion, should examine cases that are perfectly compensated. If a considerable change from the normal is observed in such cases, then muscular insufficiency of the mitral closure would be less probable. My own material is not sufficient for this purpose, for in the Policlinic we see only patients suffering from heart trouble when they notice a loss of cardiac function. The family physician with a large practice would be in a better position to make observations upon this question.

[The author, like the majority of recent German writers, pays no attention to the presystolic murmur called after Austin Flint, who described it in 1862 as caused by the reflux from the aorta producing a relative mitral stenosis. I believe such a murmur sometimes occurs, but very rarely, so that a diagnosis of organic mitral stenosis is usually safer. For a full discussion and the opposite view see W. S. Thayer, "Observations on the Frequency and Diagnosis of the Flint Murmur in Aortic Insufficiency," "Transactions of the Association of American Physicians," 1901. —Ed.].

* "Ueber zwei eigenthümliche Phänomene bei Insufficienz der Aortenklappen," "Gesammelte Abhandlungen," Bd. ii, p. 793 *et seq.*

† Fraentzel, "Vorlesungen," Bd ii, p. 105.

Peculiar manifestations may be heard over the arteries in aortic insufficiency. Over the large vessels near the heart, for example, the subclavian and the carotid, in which a systolic and a diastolic tone are normally present, we hear a systolic murmur of short duration, usually of a rough character, and often accompanied by a palpable thrill. The diastolic murmur from the aorta is not to be heard here in the majority of cases, nor is the thrill to be felt. The diastolic tone which normally is heard over these arteries disappears, and can be heard over them only when it can be perceived over the aorta.

Over the smaller arteries, as, for example, the radial, which usually do not give any auditory sensations, we hear in systole a short, toneless noise, which has been compared by Bamberger to a fillip. This sound can be heard even with the unaided ear, and if we wish to use the stethoscope then we must take care not to compress the artery, otherwise a murmur would be developed at the point of narrowing, as usual, and would have no significance whatever.

Some unusual sounds may also be heard over the large arteries further removed from the heart. Usually the crural artery is selected for the observation. Normally this artery shows no auditory phenomena, but it can be easily understood that, under these conditions, it may develop a systolic tone just as the smaller vessels. But, in addition, it may also show a sound coincident with the diastole of the heart. When a systolic and a diastolic tone are present, their origin is explained by assuming that the wall of the artery is rendered tense enough during the systole of the hypertrophied ventricle to develop audible vibrations. The diastolic relaxation of the artery (see p. 383, the explanation by Traube), during which in these cases the blood escapes at once in two directions, takes place rapidly and forcibly, and, therefore, may again produce a tone. According to another explanation, a second positive wave is produced in the artery from the remnants of the aortic valve, or, when these are completely destroyed, from the ventricular wall itself. (The chief supporter of this view is Friedreich.)

A greater importance is ascribed to the double murmur which has been named after Duroziez. A moderate pressure with the stethoscope, applied over the crural artery, allows us to discover a murmur synchronous with the cardiac systole and another murmur with the diastole. The systolic murmur arises from the eddies of blood which stream through the narrow portion of the vessel over which pressure is exercised. In the same way the diastolic murmur arises through the rapid return flow which occurs in the column of blood as the result of aortic insufficiency, for the reason that the resistances which are offered to this column by the capillaries are greater than those in the relaxed heart. A great deal has been written about the auscultatory phenomena over the crural, and a great deal of discussion has taken place on the subject. Here we cannot go into details, and I refer those who are interested to the literature* of the subject.

The behavior of the pulse is of great importance, especially in cases caused by endocarditis. In those due to arteriosclerosis the peculiarities of the pulse which are the result of valvular insufficiency are less prominent

* Traube, *loc. cit.*, and "Gesammelte Beiträge," vol. iii, p. 80. The most important articles are in "Deutsches Archiv für klinische Medicin," Talma, vol. xv; Gerhardt, vol. xvi; Bamberger, vol. xix; Friedreich, Winternitz, vol. xxi; Matterstock, vol. xxii; Schreiber, vol. xxviii; Friedreich, vol. xxix. The last contains the entire literature.

and there may be almost no difference in the pulse observed as compared to that occurring in pure arteriosclerosis, which, it is true, may be accompanied by eccentric hypertrophy of the left ventricle which may not affect the aortic valve.

The pulse in aortic insufficiency is, in the first place, a *pulsus celer*, that is, bounding in character. It rises rapidly and sinks just as rapidly again. ["Water-hammer," or "Corrigan's pulse."—Ed.] The marked filling of the aorta which is produced by the dilated left ventricle, which throws out larger amounts of blood during its contraction, produces the rapid rise. The ventricular systole also leads to a more vigorous and more marked dilatation of the arteries, because their walls are less markedly tense at the beginning of systole.

At the moment when the contraction of the heart muscle begins to lag, and when the pressure in the aorta exceeds that in the ventricle, the blood regurgitates into the ventricle, the tension of the arterial wall is relaxed and returns to the position of equilibrium it had before the systole. When the insufficiency of the valve is great, this happens within a short time, and the pulse sinks quickly to a corresponding degree.

A further peculiarity of the pulse is that the slight renewed rise (recoil-wave) which occurs during the relaxation of the artery is very weak. This may be explained in the following way: The recoil-elevation is caused by a wave thrown back from the periphery after being stopped by the closed valves of the aorta. Another explanation is as follows: "The blood which flows backward in the first portion of the aorta (at the end of the ventricular systole) unfolds the semilunar valves, and thus closes them. The closure of these valves impedes the return flow of the blood, and produces a partial rehabilitation of the suddenly diminished positive pressure in the first portion of the aorta. This new increased pressure is then propagated as a positive wave through the arterial system."

Both of these explanations concern intact aortic valves. If these do not close, then the recoil-elevation must be less, in proportion to the degree of insufficiency.*

[As Janowski† has shown, a dicrotic wave can occur in aortic insufficiency from diminished tension of the arterial walls. This may be due to weakening from infectious diseases, loss of compensation, vasomotor disturbance as in exophthalmic goiter, and some intoxications, as amylene nitrite.—Ed.]

A rare occurrence which deserves mention is that the liver, the enlarged spleen, and the kidneys (Gerhardt) may pulsate. Pulsating movements of the palate, which Fr. Müller first described, have also come under my observation in one case.

The movement of the blood may be visible in the capillaries, and is then parallel to that in the arteries. During the systole of the ventricle the surface becomes red and it grows pale again during diastole (Quincke). This may be observed without any trouble in the retina during an ophthalmoscopic examination. The best way of demonstrating the *capillary*

* Tigerstedt, "Physiologie des Kreislaufs" (p. 394), in these words summarizes Grashey's explanation of the normal dicrotism of the pulse. In the same place he also gives a review of the present state of the doctrine of the pulse, from which it appears that, for the present, there is small prospect of its proving of any practical value. For a more detailed discussion of these questions the reader is referred to M. v. Frey's monograph, "Die Untersuchung des Pulses, etc.," Berlin, Springer, 1892.

† "Zeitschrift für klin. Med.," Bd. lxi.

pulsation in the skin is to render a small area of skin on the forehead hyperemic by rubbing, and then to observe the behavior of this area in contrast to the less congested surrounding skin. Formerly, it was the custom to employ the finger-nails for this purpose by pressing upon their upper (anterior) surface, thus allowing the nail-bed to show through the nail, and observing the pulsation in this temporarily anemic area.

Diagnosis.—When endocarditis has preceded the valvular lesion, and when the cardinal signs are present, the diagnosis may be made with considerable certainty in young people. These cardinal signs are: hypertrophy and dilatation of the left ventricle; a bounding pulse, and a diastolic murmur over the aorta.

On the other hand, when marked arteriosclerosis exists, the diagnosis becomes more difficult, and this in proportion to the degree of the arteriosclerosis. In such cases it is by no means easy to decide with certainty whether or not insufficiency of the aortic valves, which results from the sclerosis, is actually present. In most cases, in elderly working people, the disease of the arteries develops quite insidiously, and the symptoms which it produces are referred to old age. Thus the physician often sees a fully developed picture of disease which may be interpreted in several ways.

But older and newer authors lay a decided stress upon the diastolic murmur. Von Leube* gives the most positive expression to this view: "The loud diastolic murmur over the sternum is the most important of the diagnostic signs, without which a diagnosis cannot be made, and, conversely, in the presence of which the diagnosis must be made, even when other symptoms of the valvular lesion are absent."

This may be admitted without reserve provided errors of interpretation of the sensory impression, which are possible, can be unquestionably excluded, and provided we admit that a relative insufficiency of the aortic valves may occur. The following case may serve as a proof of the correctness of this view:

Case III.—A man, aged sixty-nine years, was admitted on May 21, 1902. Since last summer dyspnea, palpitation, and troublesome cough. Since the end of March dropsy of the legs and confinement to bed on this account. No important previous diseases. He has worked hard all his life.

On admission, the temperature was 37° C. (98.6° F.), the pulse 78, rather regular, bounding and high, the respiration 27. Distinct venous and arterial pulsation at the neck and marked signs of arteriosclerosis in all peripheral arteries. Slight cyanosis; anasarca of the legs and of the abdomen up to the level of the umbilicus. Slight enlargement of both liver and spleen.

The apex-beat was in the sixth intercostal space, two finger-breadths outside the nipple-line, and slightly heaving. The dulness extended above to the upper border of the third rib, and on the right side to the right sternal border. The first sound at the apex was murmurish. During diastole a fairly distinct blowing sound was heard at the apex. The diastolic murmur was loudest at the place of election over the aorta, and was transmitted with approximately the same intensity to the left border of the sternum over its upper half. The systolic tone was impure. Nothing abnormal was heard in the pulmonary area. The lungs were distended and reached on both sides down to the eleventh dorsal vertebra, giving a loud, box-like tone. There was a catarrh of the larger bronchi. Vesicular breathing pure everywhere. The urine always showed a small amount of albumin. Its amount and its specific gravity varied considerably, but the details, which were interesting enough, do not belong here.

The signs over the heart remained unchanged until the patient's death, which occurred on June 28th. The diastolic murmur, especially, was always distinctly audible over the aorta. The cardiac weakness increased, although with marked

* "Specielle Diagnose der inneren Krankheiten," i, p. 31.

variations. Days with over two liters of urine excreted alternated with days in which less than one liter was passed. Similar variations were noted as regards the feeling of anxiety, which almost suggested stenocardia. The clinical diagnosis was diffuse arteriosclerosis and insufficiency of the aortic valve due to the same cause.

The autopsy (Privatdocent Dr. Dietrich, Pathologic Institute) showed the following findings in the heart:

"A small amount of fluid in the pericardium. Both pericardial layers smooth and shiny. The heart was considerably enlarged and measured 15 cm. in length as well as in breadth. The left ventricle was almost exclusively concerned in the formation of the apex. The mitral almost admitted three fingers; the tricuspid more than three fingers. The aortic valves were competent when water was poured into them, and were delicate and thin, like the other valves. Both ventricles were dilated. The aorta was markedly distended, even directly above the valve, so that the entire ascending portion of the arch was enlarged. The entire inner surface was covered by arteriosclerotic growths, irregular ulcers, and calcareous plates. It was so rigid that it tore on the slightest bending. The wall was somewhat thinner at the site of the dilatation. The circumference of the aorta was almost normal at the origin of the vessels of the neck. The arteriosclerotic changes (ulcers, plaques), however, extended into the thoracic aorta as far as the beginning of the renal vessels, when they ceased abruptly. Most of the abdominal aorta was free, but the iliac arteries showed a large number of arteriosclerotic foci."

The dimensions of the aorta were as follows (circumference): At the line of closure, 9 cm.; at the bulb, 14 cm.; at the arch, 7.4 cm. I append here a tracing of the radial pulse taken on the twelfth day of June, seven days before death. The steep ascent and the weak recoil-wave appear distinctly, as well as the anacrotic wave. The latter is known to occur quite frequently under these conditions (Jacquet's sphygmograph).

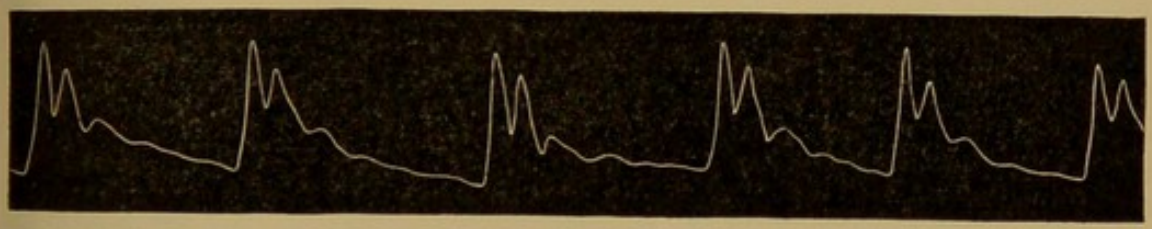


FIG. 67.

There is no doubt that we had to deal in this case with a relative insufficiency of the aortic valve. I believe also that it is impossible to separate the insufficiency from the atheromatous changes in the valves themselves.

Von Leube (cited above) quotes Groedel as authority for the statement that relative insufficiency, excessive dilatation of the aortic mouth, is characterized by a diastolic murmur of unusual intensity and of musical quality. He himself confirms this observation, and considers it of great importance, but warns us against too sweeping deductions, inasmuch as the same phenomena may be observed at least temporarily, in health.

It is known to many that a murmur which appears to be diastolic may occur over the aortic valves at the place where we are accustomed to auscultate, and may lead to mistakes. It is to the credit of von Leube that he has explicitly called attention to this occurrence.

The murmur is the so-called "Nonnensausen" (venous hum or bruit de diable). The veins in which it arises are: On the right side, the jugulars, the subclavian, possibly also the innominate and the descending vena cava. These veins lie so close to the point at which auscultation is customarily practised that their hum may be heard distinctly. A venous hum may also be heard on the left side between the articulations of the

third and fifth ribs with the sternum. This hum is referred to the ascending vena cava, and it is still doubtful whether or not it can give rise to errors in diagnosis.

The venous hums heard on the right side at the point of election strike the experienced ear by their peculiar quality. If we then pay close attention, we notice that the venous hum does not exactly correspond in point of time to the second tone, which always can be heard along with this abnormal sound, and that the venous hum becomes reinforced and more distinct when the second sound begins. The relation of the venous hum with the movements of the lung may also be easily recognized, when we make the patient breathe deeply and at prescribed intervals. The venous sounds then become louder during inspiration, for the latter causes the blood to flow more rapidly toward the heart. The same event occurs during the diastole of the heart, so long as the ventricle exercises its suction-force. For this reason the venous hum is only distinguishable in connection with the closure of the semilunar valves, if the hum is not marked, and if the patient breathes faintly. The examiner who knows this source of error cannot easily mistake the venous hum for anything else.

The *hypertrophy and dilatation of the left ventricle* are, of course, also of very great importance for the diagnosis; yet they may be absent. This may be understood without any further explanation so far as the hypertrophy is concerned, in the cases due to endocarditis which are rapidly fatal; for in such cases time is too short and the nutrition of the heart is too markedly impaired. An example of this kind is furnished by case VI, p. 211 (endocarditis). Yet the same statement applies even to well-developed cases in which complete compensation exists for the time being.

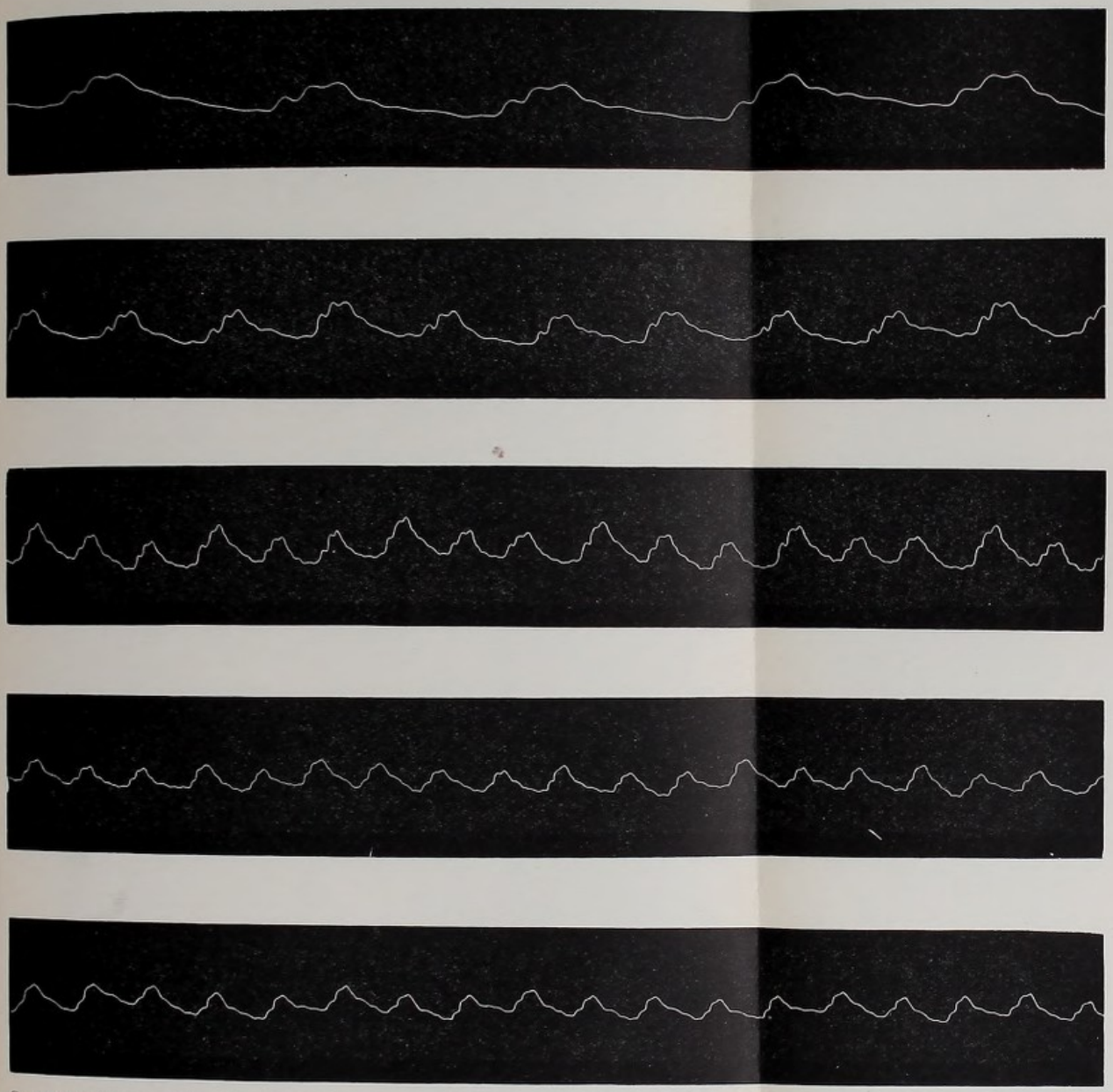
Krehl* remarks correctly: "Another widely disseminated error must be also mentioned here; namely, the idea that every aortic insufficiency must needs be accompanied by a marked, or at least an easily demonstrable, enlargement of the left side of the heart. The extent of the hypertrophy in a heart with good power of contraction is directly dependent upon the amount of work demanded of the organ, while the latter depends upon the amount of blood which passes the wrong way. If the insufficient aortic valves, for example, allow a third or a quarter of the pulsation-volume to flow back, then a marked hypertrophy of the left ventricle must take place. On the other hand, the insufficiency of the valve may be demonstrated quite clearly clinically, even when only a few cubic centimeters of blood return from the aorta. For the characteristic diastolic murmur is heard while dilatation and hypertrophy of the ventricle are very slight, and cannot be made out in any way by the physician."

Benno Lewy† gives figures on this subject. I content myself with quoting one of his remarks: "The theoretic amount of work of the heart in a case with an opening of 0.5 sq. cm. (which no longer can be considered a very small one) is 1069 meter-kilograms per hour when the contractions are 70 per minute. The difference between this figure and the normal number of 815 meter-kilograms lies within the range of errors of observation, while the acoustic phenomena of insufficiency can easily be developed under these conditions."

That hypertrophy and dilatation of the left ventricle are not necessary

* "Pathologische Physiologie," pp. 19, 20.

† "Die Arbeit des gesunden und kranken Herzens," *loc. cit.*, p. 540.



Carotid sphygmogram taken with Hürthle's tambour with decreased velocity of the kymograph. Illustrating Case IV, p. 389. Insufficiency of the aortic valves (taken May 22, 1899).

signs in insufficiency of the aortic valve is also proved by the experiences of the autopsy table, which find a satisfactory explanation in these theoretic remarks. It is true, however, that the close relation of hypertrophy and dilatation to arteriosclerosis is very important in the diagnosis of insufficiency of the aortic valves which develops in arteriosclerosis. This applies especially to the appreciation of the pulse. Certainly a *bounding pulse* belongs to the clinical picture of aortic insufficiency. But what are the conditions in arteriosclerosis?

It can scarcely be doubted that even the experienced observer cannot distinguish by the touch between arteriosclerosis combined with aortic insufficiency, and the same process existing without this insufficiency. The sphygmograms corroborate this. The following cases may be cited as proofs of this statement.

Case IV.—Joseph A., aged forty-three years. Was treated in the Policlinic for three years before his death in 1901. The details of the patient's history have no special interest here. We may mention only that he had been working hard in a brick factory, and had passed through a severe pneumonia and a nephritis before admission to the Policlinic.

On admission, September 20th, his pulse was regular—93; his respiration, 27; his temperature at noon, 36.8° C. (98.2° F.), and in the evening, 37.8° C. (100° F.). The pulse-wave struck the palpating finger vigorously, lifted the finger, and fell quite rapidly, even with strong pressure over the artery. The vessel itself (radial) felt hard and rigid; was tortuous, as was the brachial, which pulsated visibly; likewise the temporal, and other vessels.

The area of cardiac dulness was enlarged. It reached, on the right side, to the middle of the sternum; on the left side, at the sternal line, to the lower border of the second rib. The apex-beat was in the fifth intercostal space, one and one-half finger-breadths outside of the nipple-line, and heaving.

On auscultation over the apex a systolic murmur and a diastolic tone were heard. The aortic tones were both muffled. The diastolic sound over the pulmonary was accentuated.

In the further course of the disease no rises of temperature were noted (three observations a day).

The changes in the heart, however, developed in the following way:

October 1st: The second sound over the aorta was murmurish; the systolic, faint and muffled. The systolic murmur at the apex was still heard distinctly.

October 20th: The diastolic murmur over the aorta became more distinct; the systolic tone, muffled. The systolic murmur over the apex remained.

November 10th: A soft, flowing diastolic murmur was heard over the aorta and the systolic sound also became murmurish.

An insufficiency of the aortic valve, therefore, developed completely with all its signs in the course of about six weeks. The patient remained in the ward until about the beginning of March, after which he continued to come to the dispensary from time to time and felt comparatively well for about a year.

Early in April, 1901, he was admitted again. He suffered then from marked attacks of cardiac asthma, which increased to veritable angina pectoris. There was a rise of temperature, reaching 39.5° C. (103.1° F.) without any locally demonstrable cause. The bronchitis which had been present before was somewhat more severe. The findings in the heart were essentially the same, except that the area of dulness had extended both to the right and to the left. A thrill, chiefly diastolic in character, was felt over the entire area of dulness adjacent to the thorax, most markedly at the point of auscultation of the large vessels. The aortic murmurs were louder, the diastolic being always more marked than the systolic. Death occurred under the manifestations of cardiac failure.

The autopsy (von Baumgarten) showed the following findings in the heart and vessels: "The apex reached several centimeters beyond the mammillary line, and down to the lower border of the sixth rib. The right border of the heart reached the parasternal line. The heart was more than twice as large as the fist of the cadaver, and was rather more hemispheric than conic. The apex was formed by both ventricles, and the longitudinal sulcus was displaced to the left. Two fingers could be passed through the mitral; three fingers easily through the tricuspid. The cavity of the right ventricle was dilated; its wall was thickened (6 mm.).

"Water, poured upon the aortic orifice, trickled into the ventricle. The aortic valves were retracted especially in their vertical direction, and especially the middle flap, while the others showed only slight thickenings and retractions at the lines of closure. In addition, the flaps were adherent to one another at their points of contact near their insertion into the aortic wall. The cavity of the left ventricle was considerably dilated; the trabeculæ were hypertrophied; the wall measured 1.5 cm. in thickness. The beginning of the aorta showed only a very slight fatty deposit in the intima, and was not dilated. The wall of the aorta was of normal thickness. The arch of the vessel was not dilated, but showed marked fatty degeneration of the intima and small spots of beginning arteriosclerosis. The thoracic and the abdominal aorta were normal."

Here, then, was the not unusual occurrence that the peripheral arteries showed a marked arteriosclerosis which had not been transmitted to the aorta. Naturally, the sclerosis of the radial may have had an influence upon the appearance of the sphygmogram. The slight stenosis of the aortic orifice scarcely had any importance.

Case V.—Anton E., fifty-three years old, had been treated in the Policlinic from November, 1901, until his death, which occurred on November 28, 1902. He had been used to hard work from early age, and during his military service he had suffered from a felon, which was followed by the general signs of sepsis which confined him to bed for some time. In 1887 and 1888 he had attacks of pneumonia; in 1900 a pleurisy which kept him in bed for twelve weeks. Since May of the present year (1901) he complained of dyspnea which he attributed to overexertion in loading coal. The dyspnea took the form of paroxysms when the exertion was very severe, and a paroxysm often occurred shortly after he fell asleep at night.

On admission (November 4th) the pulse was 72, small, regular, easily compressed, and not markedly bounding. The respiration was 33; the temperature 36° to 37° C. (96.8° to 98.6° F.) in the course of the day. The radial artery was rigid. The markedly tortuous brachial pulsated visibly, and the carotids showed a marked *pulsus celer*. A loud systolic tone was heard over the femoral artery, while on pressure a double murmur appeared over this vessel (Duroziez).

The area of heart-dulness was enlarged. On the right side it reached to the right sternal border; on the left side, to the third rib at the sternal line, and the apex-beat was diffuse, indistinct, and palpable only in the fifth intercostal space, 2 cm. externally from the nipple-line. On auscultation the sounds over the entire heart were very faint, murmurish, but it was not possible to distinguish a murmur that could be accurately localized. To sum up, the signs over the heart were those of an organ enlarged in both directions and weakened in its musculature. The symptoms on the part of the heart took the following course as the case progressed:

November 7th, rest in bed and an ice-bag over the heart lessened the symptoms of heart weakness. A loud blowing murmur, chiefly systolic, was heard over the aorta. On November 15th the same signs were observed: a systolic murmur over the aorta, especially distinct in the direction of the blood-stream. The second sound was also impure.

The patient recovered gradually, and no longer suffered from a feeling of cardiac anxiety. He remained under treatment in the dispensary service, and only on October 15, 1902, again entered the ward.

On admission, his pulse was 86, regular, easily compressed; his respiration 36; the temperature at noon 38.2° C. (100.7° F.). The heart dulness on the right side extended 2 cm. beyond the left sternal border; above, to the sternal line at the third rib. The apex was indistinct and was best felt in the fifth intercostal space, about 1 cm. beyond the nipple-line.

On auscultation the heart-sounds as a whole were faint; the systolic over the apex, impure. A distinct systolic murmur was heard over the middle of the sternum and over the aorta, and the same sound was heard, in addition to a faint diastolic tone, over the carotid. There was a faint, not easily circumscribed dulness also over the upper portion of the sternum, in the region of the ascending aorta and its arch.

These signs did not undergo marked variations up to the patient's death. A distinct diastolic murmur over the aorta could not be heard, while a loud systolic murmur remained quite marked over the middle of the sternum. Even on the day before death nothing could be heard over the femoral artery when the stethoscope was placed lightly over the vessel, while on stronger pressure a systolic and diastolic murmur were audible at that point (Duroziez).

During the latter part of the disease there were signs of cerebral disturbances, such as occur in cardiac failure: Unconsciousness, Cheyne-Stokes breathing, with pauses reaching fifty seconds in length. Then followed fever, reaching 39.8° C. (103.6° F.), for which no satisfactory cause could be found

The autopsy (von Baumgarten) cleared up this point also: A fresh disseminated miliary tuberculosis which had extended from an old cheesy focus in the retroperitoneal glands. The clinical diagnosis was: atheroma of the arteries, especially of the beginning of the aorta, including the valvular apparatus, and dilatation of the aorta. The autopsy showed that the beginning of the aorta was calcified. The lungs were, therefore, removed first. When the heart was removed later, and when the aorta and its principal branches, which were removed at the same time, were examined, the following was found: The heart was moderately enlarged, especially the left ventricle. The aortic valves showed a slight thickening at their free borders and a slight shortening in their vertical diameters. The sinus of Valsalva was much dilated. The ascending aorta, the arch of the aorta, and the thoracic aorta were totally calcified, and were slightly dilated in a diffuse cylindric fashion. The petrification was so marked that the artery tube could only be opened with difficulty. The inner surface of the calcified aorta showed numerous irregular

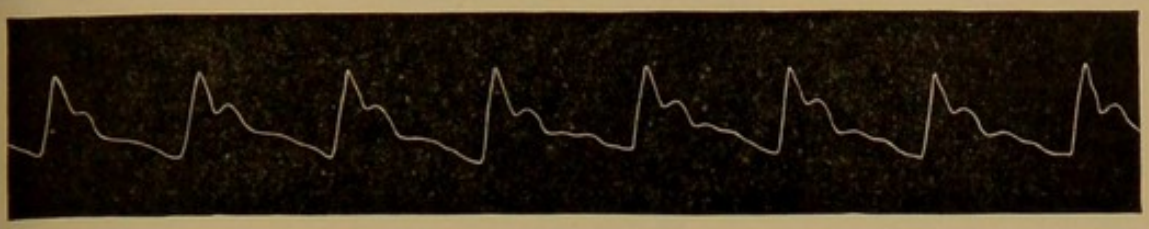


FIG. 68.—IV. Joseph A.

prominences, which also were, for the most part, calcified. The disease of the vessel decreased gradually in intensity from above downward, so that the abdominal aorta was more normal down to its division into the common iliacs, although its inner surface showed a certain number of endarteritic prominences.

Analogous changes to those found in the ascending aorta and the arch of the aorta were found in the trunk of the innominate, while the carotid and the left subclavian were soft. The right carotid, from its origin to the base of the skull, was filled with an adherent thrombus.

The "slight insufficiency" of the aortic valves, so designated in the anatomic diagnosis, and which was demonstrable at the autopsy, had not given any signs during life. The diastolic murmur over the aorta and the *pulsus celer* could not be detected. The moderate enlargement and



FIG. 69.—V. Anton E.

dilatation of the left heart were also sufficiently accounted for by the arteriosclerosis.

The sphygmograms of case IV and case V present marked differences. Both were taken at a low tension of the spring with Jacquet's sphygmograph, at the radial artery. They show the abrupt rise, the anacrotic wave which forms part of the rise, and the weak dicrotic recoil-wave. If the tension of the spring be altered, the tracing of the pulse is changed considerably, though not characteristically. What I intended to show here, especially, was that the sphygmogram of the radial under certain conditions, perhaps at all times, cannot be employed in a differential diagnosis of aortic insufficiency.

I may insert here another report of a case of aortic insufficiency, produced by endocarditis in a young man, the features of which are of diagnostic interest.

Case VI.—Duration of the treatment at the Policlinic, fourteen years. Julius W., born 1880. History: 1881, extensive capillary bronchitis; 1884, slight attack of

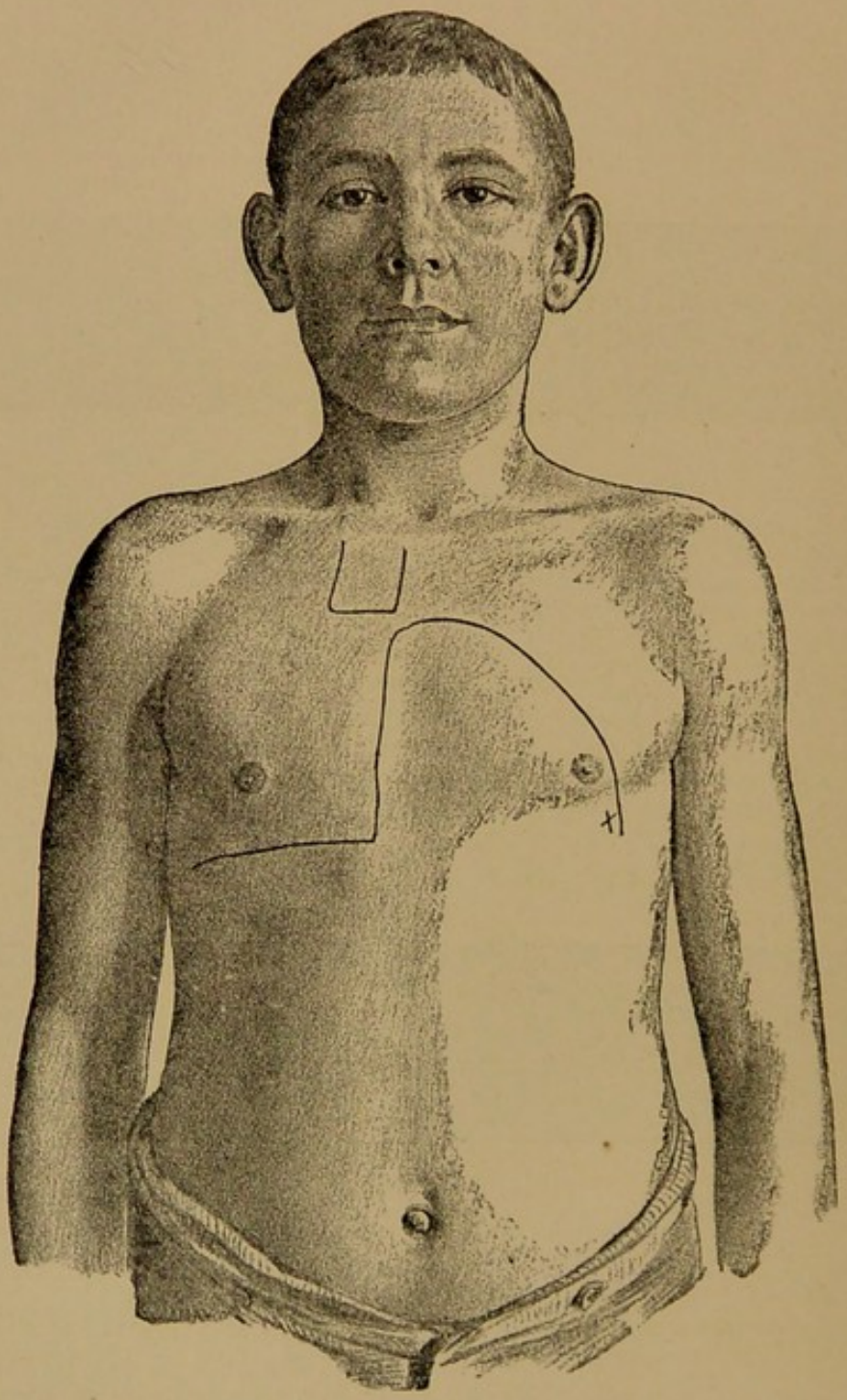


Fig. 70.—Julius W., March, 1897.

scarlet fever; 1885, severe pleurisy; 1888, sepsis. At first pains in the bones and muscles, then a slowly developing endocarditis which did not produce any other general symptoms, but was followed by a valvular lesion. The patient felt well, but every slight exertion, especially walking upstairs, always produced dyspnea and palpitation.

In 1890, after a period of vague illness, he complained of pains in various parts of the body. Fever was present three days before admission; the spleen was markedly enlarged. The pains varied quickly and sometimes were very severe. This relapse was short and mild. The maximum temperature was 38.5° C. (101.3° F.). Similar complaints were noted during the following years, together with signs of cardiac weakness: palpitation and edema of the ankles.

In 1894 all the symptoms became more severe, but only for a short time—a few weeks. In 1895 he had another mild relapse. Then he felt well until 1897, when he was treated from February 9th to March 30th. The symptoms were not threatening in any way; the maximum temperature was 40° C. (104° F.). From that time on until the end of 1902 there were no signs of illness. The man is now

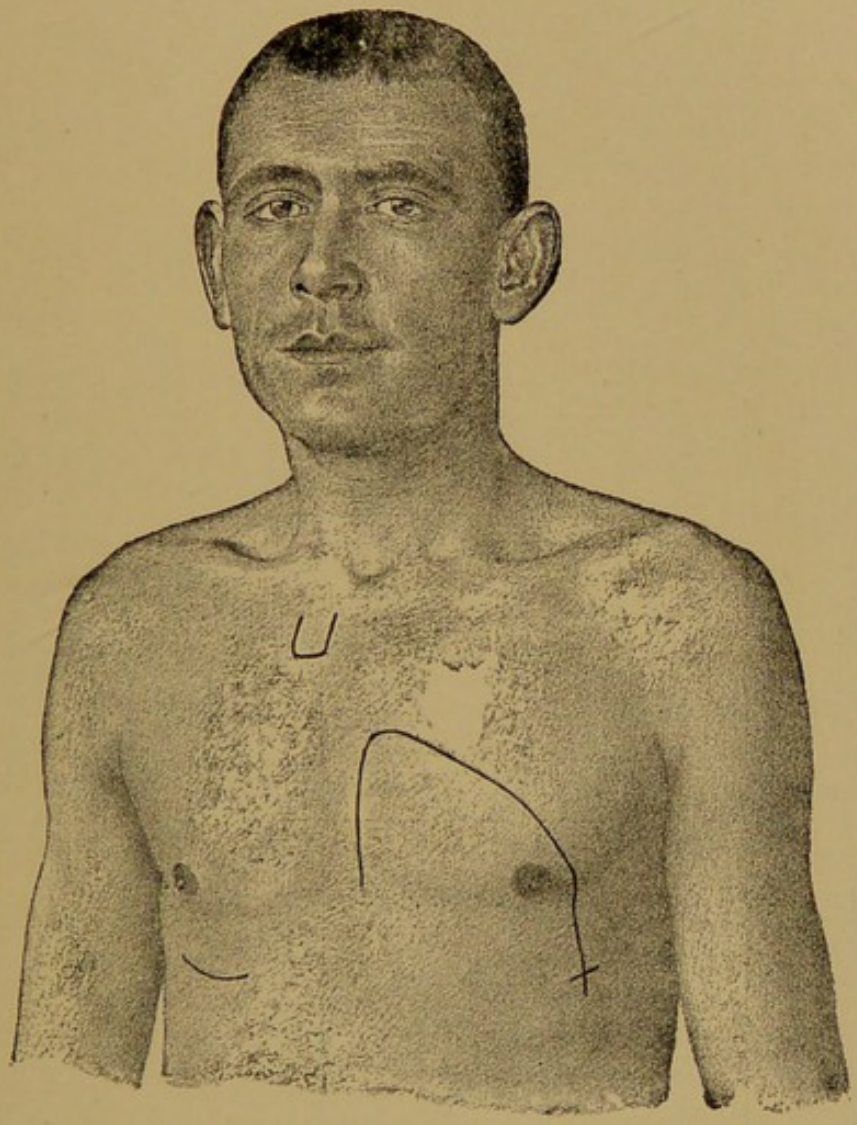


Fig. 71.—December, 1902.

strong, and, according to his own statement, can pursue his trade as a mason with full working capacity.

The table on pp. 394 and 395 and the accompanying illustrations (Figs. 70, 71), as well as the diagram (Fig. 72, p. 396), show how the valvular lesion gradually developed in this patient.

We find: (1) At first, disturbances on the part of the heart muscle dominated the picture quite markedly. The right heart was also involved. The signs of aortic insufficiency were already well marked. (2) During the following four years the heart weakness improved, in spite of

Dulness.		Apex-beat.	Mitral and Tricuspid Areas.	Aortic and Pulmonary Areas.	Remarks.
Sternal Line.	Nipple-line.				
Above, second intercostal space on the right, as well as on the left, side. The right border approximately in the parasternal line. July 15, 1890.	Extends to the left of the nipple - line, from the level of the second intercostal space down.	Diffuse; most distinct a little outside of the nipple-line, in the intercostal space.	Loud systolic and diastolic murmur over the apex.	Aortic: The diastolic murmur is louder than the systolic, and has a different sound from that of the mitral diastolic murmur. Pulmonary: The diastolic sound is slightly "clapping."	Slight precordial bulging. Distinct pulsation visible over carotid and subclavian. Double murmur over femoral artery (Duroziez's sign). Capillary pulse not present. Over the carotid a marked systolic thrill is felt, and occasionally a weaker diastolic. The pulse is quick (pulsus celer, water-hammer pulse) and quite irregular. Feet cold and cyanotic. Temperature: Maximum daily, 38.5° C. (101.3° F.).
Above, on both sides, lower border of the second rib. The right border extends about 1 cm. beyond the right edge of the sternum. June 21, 1894.	Extends to left of the nipple-line at the upper border of the fourth rib, and from that point in a curved line to the apex-beat.	Diffuse; most distinct a little outside of the nipple-line in the fifth intercostal space.	Over the apex the first sound is stronger than the second; a little farther out (to the left) a loud systolic murmur which obscures the (first) sound. At times the diastolic sound "also is somewhat impure."	Aortic: "A very loud diastolic, and a slightly softer systolic murmur," which is distinctly different from that heard at the apex. Pulmonary: Systolic murmur identical with that heard at the apex, the diastolic sound is "feeble."	Slight precordial bulging. Pulsation in carotid fairly well marked. Capillary pulse faintly seen in the nails, and distinctly in the soft palate, especially the uvula. Double murmur over femoral artery. Indistinct sounds heard over the radial. Distinct systolic thrill over carotid. Pulsus celer, not intermittent. The skin generally somewhat cyanotic. Extremities cool to the touch

Above, upper border of third rib on both sides. Right border a little to the right of the right edge of the sternum. February 9, 1897.	On the left, at the level of the fourth rib, extends to, but not materially beyond, the nipple-line.	Diffuse, heaving, 1 cm. outside of the nipple-line in the fifth intercostal space. The sixth rib is also slightly elevated.	Over the apex, indefinite systolic sound; the diastolic sound is very loud and clapping. The auscultatory signs at the apex vary: February 12th, distinct systolic murmur; March 6th, no murmur, only an indistinct muffled sound.	Aortic: Loud systolic and softer diastolic murmur, with a clapping second sound. Pulmonary: The diastolic sound strikingly clapping.	Slight precordial bulging. An area of dulness, about two finger-breadths wide to the right of the sternum, extending from the lower border of the second rib upward to the clavicle. Forcible pulsation over carotids and ascending aorta at the site of the dulness. No capillary pulse; no pulsation in the soft palate. Thrill at apex, in second intercostal space to the right of the sternum, and over the carotids—systolic in time everywhere. Pulsus celer, regular. Cyanosis of the skin on the trunk and extremities.
Above, lower border of the third rib on the left side. On the right, it does not extend beyond the left edge of the sternum. December 8, 1902.	From the fourth rib down, extends slightly to the left of the nipple-line.	Distinctly heaving; extends a distance of 4 cm. in the sixth intercostal space, outside of the nipple-line.	Over the apex, the heart-sounds are distinct, although feeble. Both sounds are distinctly heard.	Diastolic murmur, heard in the sixth intercostal space at the sternum, and attaining its maximum intensity in the second right intercostal space. The diastolic sound is everywhere distinctly audible through the murmur. The systolic sound is somewhat impure and muffled. Pulmonary: Nothing of moment.	Slight precordial bulging. Slight dulness, 2 cm. wide and 3 cm. high, at the upper border of the second rib and costosternal junction on the right side. Forcible pulsation over carotids, arch of aorta, and subclavians. No pulsation over dull area. Capillary pulse visible in the nails. The systolic sound of the heart over the large arterial trunks is somewhat impure; over the carotid a diastolic murmur is also heard, and the (second) sound is distinct. A systolic sound is heard over the femoral with the stethoscope lightly applied; when some pressure is employed, a double murmur is heard (Duroziez's sign). No cyanosis.

repeated mild relapses; especially the transverse diameter of the heart was diminished. Some of this must be attributed to an increase in the hypertrophy of the left ventricle. (3) With the onset of puberty the transverse diameter of the heart dulness, which must be dependent upon the dilatation of the right heart, diminished more and more. The left heart increased in muscular mass and its total volume seems to have remained the same. At present the compensating changes of the heart have restored full working capacity to the body. (4) A severe disturbance of the semilunar valve is certainly absent, for the diastolic tone always remains audible.

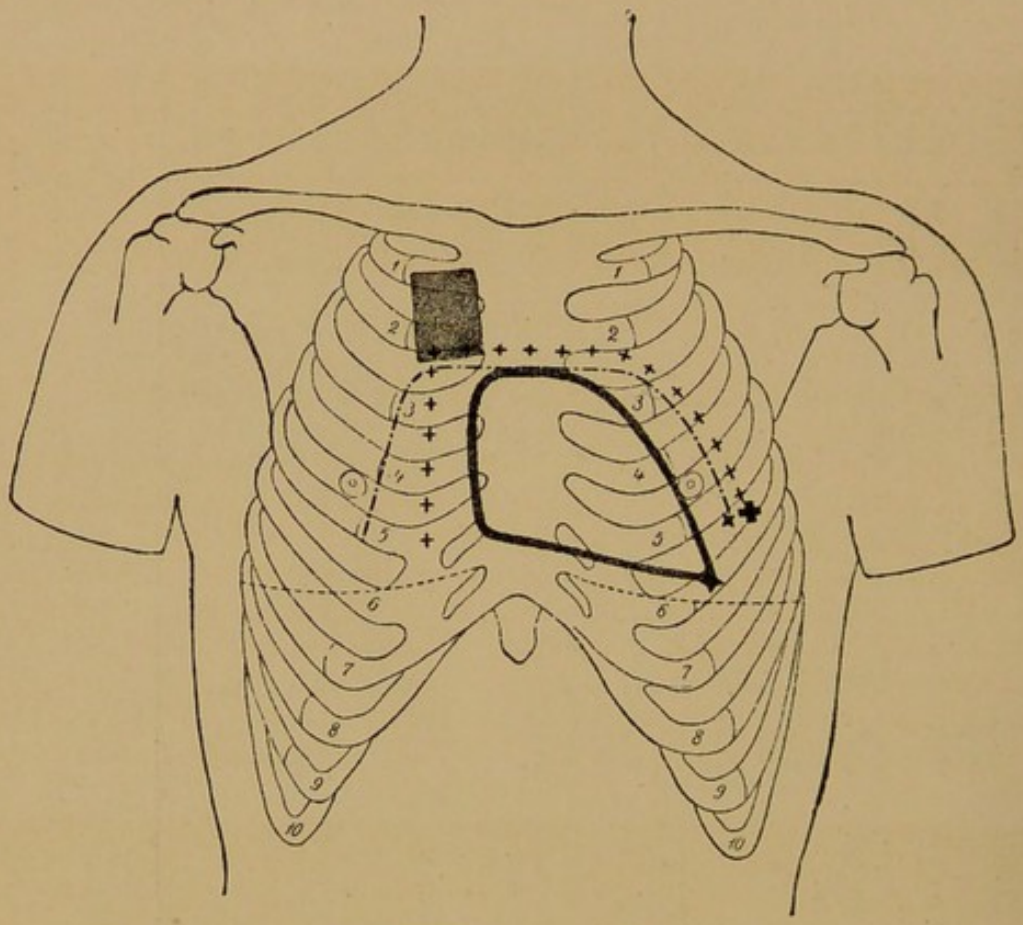


FIG. 72.

We can scarcely say what part a stenosis of the orifice took in the development of the symptoms. In judging this we must largely rely on the systolic murmur over the aorta, but there is a dilatation of the ascending aorta, and it is not clear what influence it might have.

A disease of the mitral or of its orifice seems to be excluded in this case. The shape of the cardiac dulness as it is noted makes this probable. The case evidently belongs to that class in which the insufficiency of the valve is by far the most prominent feature. The two pictures, reproduced from photographs (Figs. 70, 71), show the extent of the left heart five years ago, as compared to the present, after a long time in which

the patient was free from relapses. The dilatation of the ascending aorta has decreased to a certain extent. This, I think, can be easily understood, as we are dealing with a vessel that is still quite elastic in a young man. This also may be the reason why a systolic murmur could not be heard either over the apex or over the aorta at the point of election during the last examination.

I reproduce two sphygmograms of the radial (Jacquet's sphygmograph), taken in 1897 and 1902 respectively (Figs. 73 and 74).

Of the other signs which have been considered important, with more or less emphasis in the diagnosis of insufficiency of the aortic valves, there is none which can be recognized as pathognomonic. The double murmur

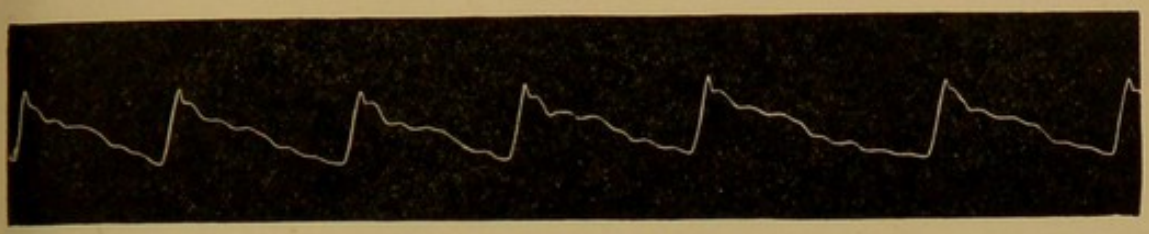


FIG. 73.—February 19, 1897.

in the great arterial trunks, especially in the femoral (Duroziez), has aroused the greatest amount of discussion.

Friedreich* speaks thus of its value: "It has been agreed long since, that the double murmur by no means can be considered as a pathognomonic sign of insufficiency of the aortic valves.

"And yet the double murmur is an almost constant phenomenon which occurs sometimes with and sometimes without similar murmurs, over the larger arterial trunks, so that it may be considered, at any rate, a very valuable sign in the diagnosis of the cardiac lesion under discussion. Matterstock found it absent only once in 12 cases. Von Bamberger only 4 times in 31 cases. I myself (Friedreich) have missed



FIG. 74.—December 8, 1902.

the double murmur only twice permanently out of over 100 cases of aortic insufficiency since I have known Duroziez's sign (1867), and since I have examined the behavior of the femoral arteries.

"At times the phenomenon disappears temporarily after complete rest in bed. It can be produced again at once, however, when the heart's action is accelerated, simply by walking about the room."

Friedreich enumerates the following conditions in which he has also found the double murmur: Atheroma of the aorta and of the larger arterial trunks; aneurysm of the aortic arch; hypertrophy of the left ventricle as the result of interstitial nephritis, and also in fevers (typhoid, miliary

* "Deut. Archiv f. klin. Med.," 1881, vol. xxix, pp. 343, 344.

tuberculosis), as well as in some cases of Basedow's disease and of "celiac neuralgia." Matterstock also heard it in chronic lead-poisoning.

The capillary pulse is an accompaniment of the *pulsus celer*, especially when the heart works vigorously. It has nothing to do with aortic insufficiency as such.

While the diagnosis of this valvular lesion may be said to be simpler than that of the other cardiac diseases, yet it is not so simple as has often been claimed.

STENOSIS OF THE AORTIC ORIFICE.

Stenosis of the left arterial orifice is also more often the result of arteriosclerosis than of endocarditis. It is not very often present alone, nor is it often more marked than the insufficiency when both conditions are present.

The classic symptoms are: a systolic murmur at the aorta; a simple hypertrophy of the left ventricle, and a retarded [slow—*pulsus tardus*—ED.] pulse. The experiments of Moritz upon his model showed that the expulsion-volume of the left ventricle diminishes; that the pressure in the aorta and in the venæ cavæ is thereby lowered, and that the pressure in the pulmonary artery and in the pulmonary veins rises. The expulsion-volume of the right ventricle is diminished in correspondence with the decrease of that of the left ventricle. The extrinsic labor of the left ventricle, which is somewhat dilated, is quite markedly diminished. (In the experiment it was diminished from 227 to 180 gram centimeters.)

When the work of the left ventricle was increased in the experiment, complete compensation was effected. In man, hypertrophy of the left ventricle takes place for the reason that the work demanded of this part of the heart is increased, because:

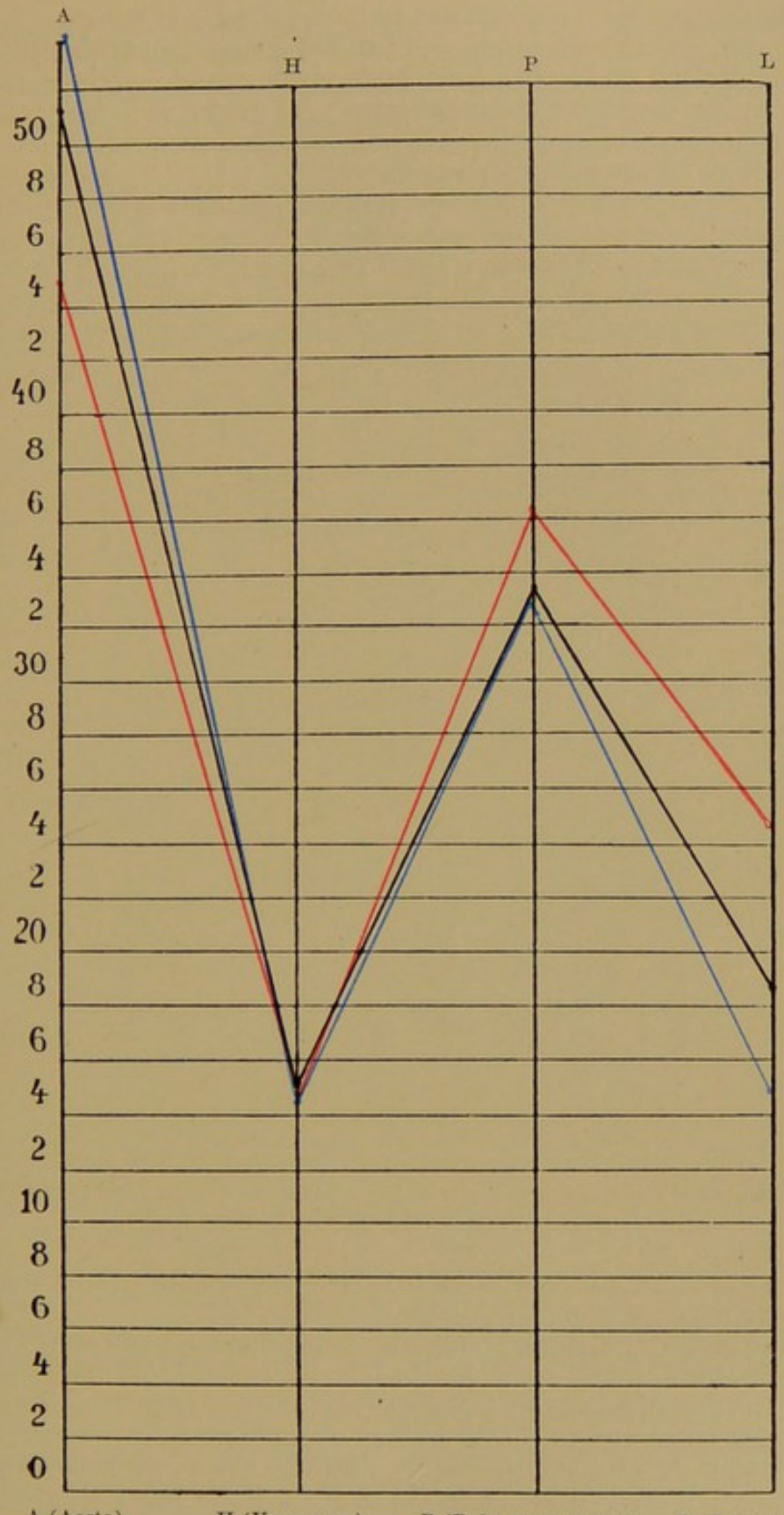
1. The pressure in the pulmonary veins and in the left auricle becomes greater, and consequently the tension of the left ventricular wall becomes more marked during diastole. This occurs the more readily when, from greater resistance at the narrowed orifice, a portion of the blood contained in the ventricle must remain in that cavity.

2. The narrowing of the outlet directly produces an increase in the labor of the left ventricle. A dilatation of the left ventricle may occur when the reserve force is no longer able to furnish the increased labor, after the hypertrophy fails to supply the needed increase in force. But this dilatation is a passive one, is secondary, and is a sign of weakness on the part of the cardiac muscles, quite the opposite from the primary dilatation, which favors compensation in aortic insufficiency.

In view of the universally acknowledged comparative rarity of "pure" aortic stenosis it is of importance to consider this lesion in connection with aortic insufficiency.

Anatomic Conditions.—I shall follow Bamberger's description:

"Stenoses of moderate degree are usually produced as the result of the fact that the degenerated aortic valves make the orifice narrower, in proportion as the valve itself becomes thickened and projects from the wall. In stenoses of high degree the valves, however, coalesce into a ring-like irregularly formed and almost immovable mass, which usually projects upward in the shape of a dome. Within this we find a central opening, which is often scarcely large enough to admit a quill. In the most pronounced degrees the edges of the degenerated valves are so closely adherent that they seem to occlude the orifice entirely.



A (Aorta) H (Vena cava) P (Pulmonary artery) L (Pulmonary vein)

Pressure before the experiment. Stenosis of aortic orifice. Increased work of left ventricle (Experiment 52).

FIG. 75.—Moritz's Experiment 51.

"Slighter degrees of stenosis are, as a rule, combined with insufficiency, because the stiff and thickened and usually shrunken valvular flaps allow the regurgitation of blood. Yet there are not infrequently exceptions, and it may happen that the flaps approximate pretty well under the pressure of the column of blood in the aorta; and so the regurgitation may be completely or almost completely prevented.

"In marked degrees of stenosis, however, the rule is that there is no insufficiency, or that there is such a slight degree of insufficiency that it may be neglected. These facts are of great importance with reference to the sequels of aortic lesions. If a large number of specimens be examined, one may become easily convinced that the thickness and volume of the left

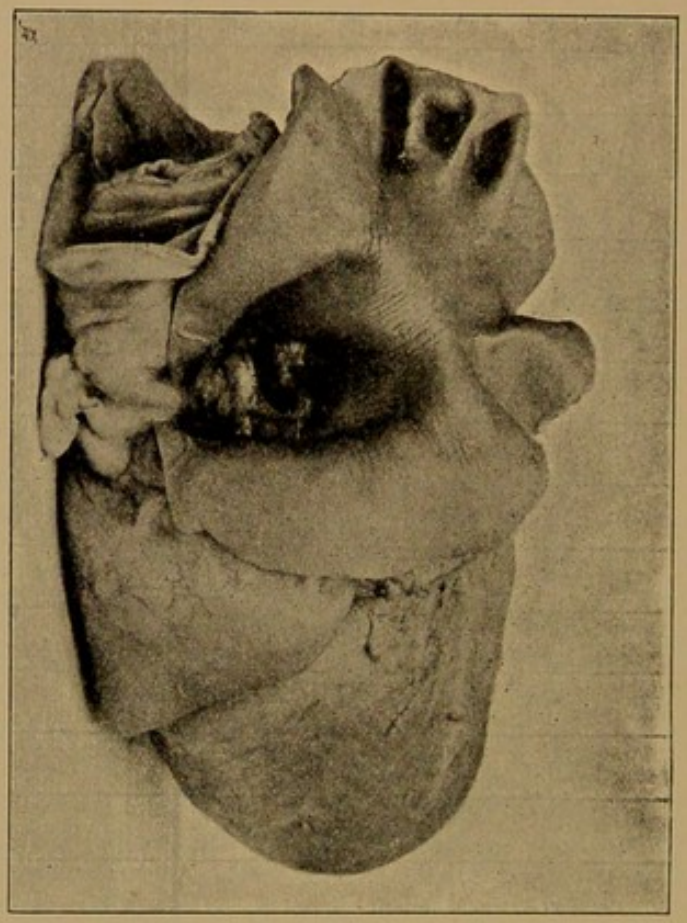


FIG. 76.—Stenosis of aortic orifice, viewed from the aorta (from a preparation in the Pathologic Institute, Tübingen).

ventricle varies greatly, according to the preponderance of insufficiency, on the one hand, and of stenosis, on the other."

Basing his conclusions upon about fifty specimens, Bamberger classifies the cases as follows:

"1. Insufficiency of the aortic valves with very slight stenosis. These cases behave in the same way as those of pure insufficiency. The left ventricle is always markedly dilated. (The ventricle measures from 8 to 11, or even 13 cm. in height and in width, while the hypertrophy of its wall is moderate—about 0.9 to 1.6 cm. at the thickest part.) More marked hypertrophy occurs only in the presence of a complication; such as, for example, an advanced atheromatous condition of the aorta.

"2. Insufficiency of the aortic valve with moderate stenosis (ad-

mitting one finger, approximately). Such cases show the most marked enlargements of the left ventricle. The latter is not only dilated in the same degree as in the preceding case, but its walls become very markedly thickened and not infrequently measure 1.8 to 2.2 and even 2.7 cm. in diameter.

"3. Very marked stenosis with little or no insufficiency. In these cases the hypertrophy predominated, and the thickness of the walls varies from 1.4 to 2.7 cm., although it almost always comes closer to the former figure than to the latter. Either no dilatation at all or a very slight degree is observed in these cases. As a rule, on more careful examination, we find again that the septum is pushed with its convexity toward the right ventricle, and thus the left ventricle gains the space which would otherwise be lost through the considerable increase in the thickness of the walls. Thus it is very rare to find even slight degrees of the condition usually known as concentric hypertrophy (hypertrophy with narrowing of the cavity). The shape of the left ventricle, and of the heart as a whole, is almost globular. The aorta is narrowed in marked degrees of stenosis, while in slighter degrees with preponderating insufficiency it is very often dilated."

Physical Examination.—According to Bamberger, two sets of conditions must be considered:

"1. Moderate stenosis, with preponderating insufficiency. The apex-beat is displaced considerably toward the left, often even to the axillary line, and is also displaced downward. The apex-beat is markedly exaggerated in intensity; is heaving, and often also shakes the chest-wall. The cardiac contractions are often felt distinctly over several intercostal spaces above the apex, and may be visible. Often the thorax moves over a considerable area, and there may be a concussion of the entire trunk and of the head during systole. Very marked increase in the cardiac dulness, especially in the vertical direction, is also noted. A slight systolic thrill is felt very often in the region of the second left or right costal cartilage at the sternal border. Less frequently there is a double systolic and diastolic thrill at the same place.

"On auscultation two murmurs are heard in the region of the aortic valves, of which the systolic is usually louder, but shorter, and the diastolic fainter, but longer. In the left ventricle (over the apex) the same murmurs are audible, but somewhat fainter, and in rare cases also a rudimentary systolic or diastolic tone is also audible. The other sounds of the heart are normal, unless they are masked by the transmitted murmurs. The same phenomena as in insufficiency are usually found in the carotid and the subclavian, namely, a harsh systolic murmur without diastolic sound. The pulse in the smaller arteries is also the same as in pure insufficiency, but usually it is not quite so large, nor so bounding, nor does it give such distinct sounds, and the arterial tones may even be entirely absent.

"2. Stenosis of high degree with slight or no insufficiency. The apex-beat is less markedly displaced to the left and downward, and while it is usually heaving in character, it is less vigorous than in the first class of cases. The heart's action, especially, is not so widely diffused. The cardiac dulness is enlarged only moderately, and about equally in the vertical and transverse diameters. At times there is no increase in cardiac dulness. A systolic thrill which is very distinct and well marked may be

felt at the aortic entrance, over the entire sternum and over the end of the upper intercostal spaces.

"On auscultation we hear at this point a very loud murmur, which sounds like a whistle or groan, or even like the sound of a high-pitched musical instrument. This murmur is transmitted over a considerable area of the thorax and covers all the first sounds in the cardiac region completely. In diastole we hear either a very faint murmur, or nothing definite, the systolic murmur ending quite abruptly, and beginning again after a short pause. A rudimentary second sound is but rarely heard.

"In the carotid a short, dull, indistinct sound or a murmur is heard in the first phase of the cycle, while in the second usually nothing is heard, or rarely there may be a rudimentary second sound. The arterial pulse is small and retarded (*tardus*). It is usually of considerable resistance, not easily compressed, and never produces sounds. If the stenosis is extreme, or if the heart is no longer able to contract vigorously, as in the last stages of the disease, the pulse is small and easily compressible, but slightly resistant (soft), and sometimes it can scarcely be felt. In some advanced cases there is considerable difference between the time of the apex-beat and of the radial pulse."

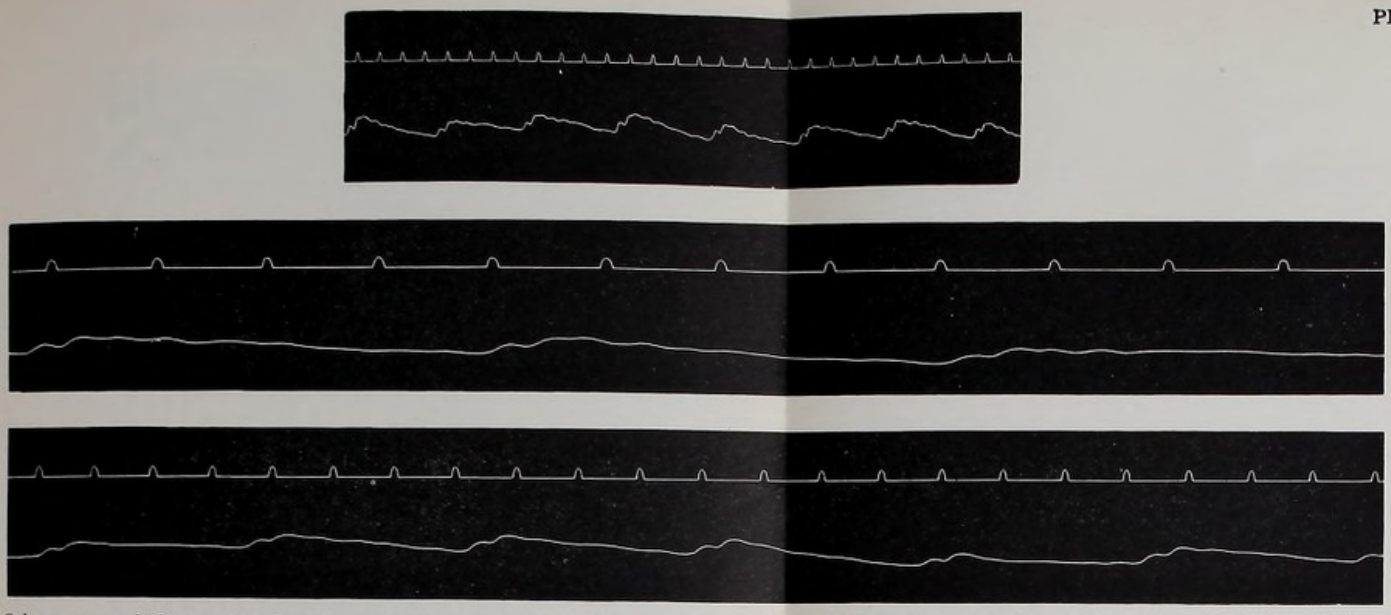
No important additions have been made since this description was written. As an example, I shall describe a case observed in the Policlinic.

CASE VII.—Abraham K., born in 1879. The man states that he had influenza in 1892. He was treated in the Policlinic at that time and was told that his heart was affected. His mother tells us she had noticed that her son looked ill for a year before that attack of influenza. He also often complained of a stabbing pain in the region of the heart, and became tired very easily. When he worked in the field he could not carry anything, as he became exhausted immediately, so that he was unable to breathe easily. The patient remained in the ward for four weeks, on account of the influenza, after which he presented himself, now and then, at the dispensary. After his recovery he found that he became dyspneic and had palpitation of the heart on exercising. In spite of this he worked hard in a brick factory from his fourteenth to his nineteenth year. Usually he was able to do his work, and only on two occasions he was obliged to remain at home for a fortnight at a time, on account of cardiac pains and dyspnea.

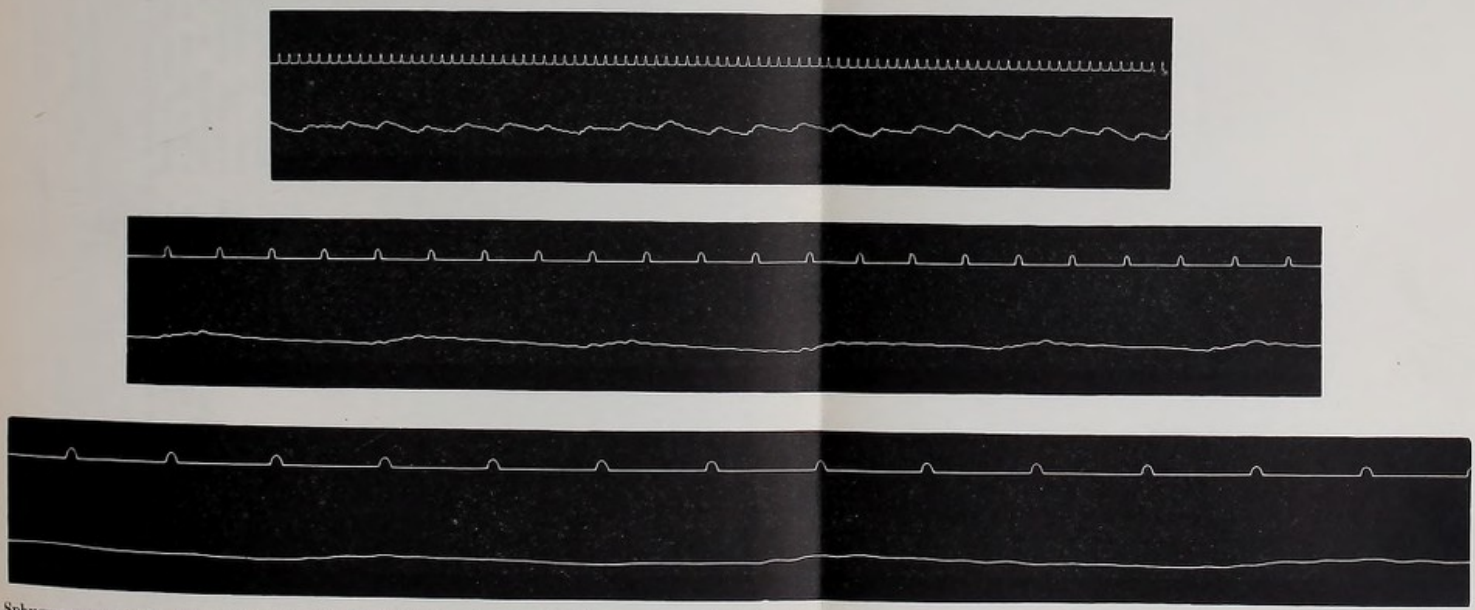
At that time I showed him repeatedly to my students in the clinic. The valvular lesion had become fully developed and was, so far as I can remember, in about the same condition as at present, except that the boy looked extremely ill and certainly did not seem able to do any hard work. I succeeded then in persuading the parents that their son must be allowed to do easier work, and he has now been employed for the last four years at a press in a large printing establishment where he does not overexert himself. His general condition has now improved, and I was astonished to find what had become of this former weakling.

The following table shows the heart signs, and Fig. 77 represents the outline of the heart dulness, drawn after a photograph. Fig. 78 shows a sphygmogram of the radial pulse taken with Jacquet's instrument, and of the carotid pulse drawn with Hürthle's tambour, with a rapid and then a slow movement of the drum. There is no doubt that there was in this case a well-developed but not pronounced pure stenosis of the aortic orifice, as the result of a very slowly progressing endocarditis. There were no symptoms of insufficiency of the valves.

Diagnosis.—The physical signs which belong to a well-marked pure stenosis of the aortic orifice are rather characteristic: A simple hypertrophy of the left ventricle, a loud systolic murmur, loudest over the aorta, and a *pulsus tardus*. But this simple picture is rarely seen on account of the great frequency of a combined insufficiency. In such cases it behooves us to estimate the significance of the signs according to their



Sphygmogram of the carotid pulse taken with Hürthle's tambour with decreasing speed of the kymograph. Time recorded at one-fifth second. Gärtner's tonometer shows 116 mm. Hg. Illustrating Case VI, p. 392. Insufficiency of the aortic valves.



Sphygmogram of the carotid, taken with Hürthle's tambour, with decreased speed of the kymograph. Time recorded at one-fifth second. Gärtner's tonometer shows 89 mm. Hg. Illustrating Case VII, p. 402. Stenosis of the aortic orifice.

Sternal Line.	Nipple-line.	Apex-beat.	Venous Orifices; Mitral and Tricuspid.	Arterial Orifices; Aortic and Pulmonary.	Other Data.
Dulness.					
Above, upper border of fourth rib, left side. On the right side a little beyond the left sternal border. December 21, 1902.	Begins at the level of the fifth rib on the left side.	Diffuse. Palpable over an area of 5 cm. Outermost point at the sixth intercostal space, a little beyond the nipple-line.	A loud systolic tone over the apex, and a faint murmur accompanying it. A loud, clear diastolic tone. The systolic murmur increases in clearness from the apex inward, while the tone grows less distinct and is but faintly heard in the fourth intercostal space. The murmur grows louder up to that point.	A loud systolic murmur in the second right intercostal space, close to the sternum. A flapping, clear diastolic tone. The maximum intensity of the murmurs at the part of the sternum corresponding to the insertion of the third right rib. Nothing heard over the pulmonary save the transmitted murmur.	Slight precordial bulging. The thorax heaves slightly in systole from the upper border of the fourth rib to the anterior axillary line (left). No increased pulsation of the carotid or subclavian. The murmur is systolic over the ascending aorta, the subclavian, and the carotid on the right side, and everywhere there is a pure diastolic tone. The systolic murmur is very loud at the supra-sternal notch, while the diastolic tone there is weak, but pure. The pulse is remarkably poorly filled, in comparison to the apex-beat. Scarcely can be felt as *pulsus tardus*. Peripheral arteries not hard or tortuous. No tone or murmur over the crural. Systolic thrill over the entire extent of the cardiac dulness and in the right and left second intercostal spaces. Very faintly over the carotid.

distinctness, for it is impossible to measure them directly. At the same time it is not always possible to make an absolute diagnosis; for we cannot know whether, in addition to the two elements,—the changes in the valves and those in the orifice,—there may not be also other factors, such as a dilatation of the first portion of the aorta.

When insufficiency and stenosis exist simultaneously, the following features must be considered in detail:

1. *Hypertrophy of the Left Ventricle.*—Does this change coincide with a dilatation, and in what way does it become manifest? The first question must be answered at once in the affirmative; for dilatation is a direct and necessary result of insufficiency. As regards the second part of the question, however, I do not think it is probable that any one who has

the picture of this condition before him can judge definitely as to whether primary (active) dilatation alone is present. If this were possible, then we could have a good measure for the degree of the insufficiency. The greater the insufficiency, the more pronounced the dilatation of the left ventricle. But how do we know whether passive dilatation may have occurred during the development of the disease, and could not thereafter be completely compensated? He who observes the development of a val-

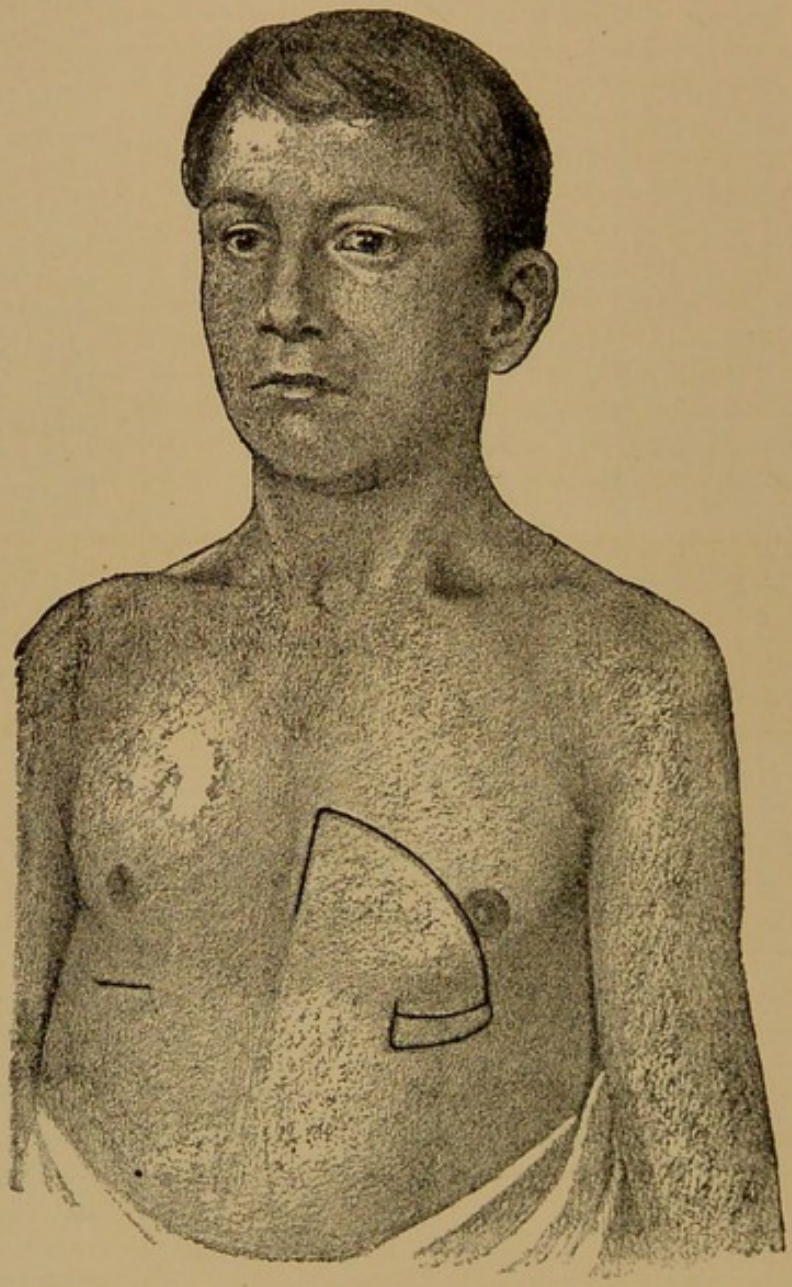


Fig. 77.—Abraham K., age twenty-two, December 21, 1902.

vular lesion in the aorta as it appears in my Case VI will admit that the doubt which I just expressed is perfectly justified. Both in the arteriosclerotic and in the endocarditic form the musculature of the heart may at any time be affected. If one has followed the development of the lesion, one may possibly be entitled to a positive opinion, but even then his opinion will not be conclusive. This is all that it is necessary to say on this subject.

2. *A loud systolic murmur*, loudest over the aorta. We have already discussed the occurrence of a systolic murmur in the aorta itself, and in the large arterial trunks which spring from it, in pure insufficiency of the aortic valves (see p. 381). Therefore such a murmur alone cannot be regarded as pathognomonic, but it cannot be denied that this murmur may be regarded as characteristic, when it is really very loud and has a markedly peculiar quality.

I am inclined to place the marked *systolic thrill* even higher in diagnostic value. This thrill is felt most distinctly beside the sternum in the second right intercostal space, but is usually palpable over a much wider area, and is transmitted with the blood-stream, so that it is quite audible as a murmur over the carotid.

Murmurs as well as thrills are the expression of the eddies which arise as the result of the passage of the blood from the narrower into the wider arteries. The same physical conditions obtain, however, when the aorta is dilated above its orifice, which has remained normal in diameter. In such a case the same results must follow, for it makes no difference whether there is a normally wide aorta above a narrowed orifice, or a dilated aorta above a normally wide orifice. Experience teaches us that it is particularly the ascending aorta which is often markedly dilated, as the result of the pressure exercised upon it by the blood forced into this vessel by an eccentrically hypertrophied left ventricle. A dilated aorta may cause the



FIG. 78.

sounds to become less distinct, and its systolic dilatation may be felt or certainly may be heard on auscultation. At times, however, its irregular systolic vibration may also become perceptible to the touch. We must always remember the possibility of this dilatation, and must, therefore, be careful as to what importance we attribute to the systolic murmur, for it is not always possible to determine by percussion or palpation whether a dilatation of the aorta has taken place. This may, in fact, be prevented by the overdistended lung between the aorta and the thoracic wall.

3. *Pulsus Tardus.*—Theoretically, it seems unreasonable to doubt whether such a pulse can be absent when the stenosis amounts to anything. For this lesion offers all the conditions necessary: the narrow orifice through which the blood can pass but slowly, and therefore a gradual dilatation of the artery—in other words, a slow rise, and a broad apex in the curve of the pulse. It certainly cannot be denied that the finger of the trained observer can recognize the form of pulse known as "slow," but this alone is not enough to make a definite diagnosis of aortic stenosis. (Compare the remarks of Krehl, p. 474.) In the pronounced cases the sphygmogram, which can be taken with a convenient sphygmograph, becomes rather characteristic, and especially do we note the contrast between *pulsus tardus* and *pulsus celer* (see Figs. 74 and 78).

The tracings of the carotid made with accurate apparatus (Hürthle) and shown in tables I and II in his work show in the anacrotic portion

regularly marked waves. Inasmuch as these occur in insufficiency as well as in stenosis, they cannot be regarded as of diagnostic value for the latter condition any more than can the harsh systolic murmur at this place (pp. 384 and 402).

Von Frey,* to whom we are indebted for a thorough treatise upon the pulse, says on this subject: "Sphygmography and the palpation of the pulse are not rival methods, but simply complements of each other." . . . . "The pulse-tracing cannot supplant the simple palpation of the pulse, because the latter detects qualities which remain hidden to the sphygmograph. On the other hand, the sphymograph is far superior to the finger in recognizing the form of the pulse." It is especially advisable to employ both methods of examination in determining whether a pulse should be designated as "tardus." I, for example, was not able to recognize with certainty the retarded pulse in case VII by palpation, and, conversely, as Krehl, for example, has also emphasized, a pulse may appear to be retarded in arteriosclerosis although the sphygmogram does not show this quality. Von Leube† says correctly "that the characteristic quality of the pulse may be completely absent," and that even a marked *pulsus altus* and *celer* may be exhibited in the sphygmographic tracing when the insufficiency markedly predominates over the stenosis. How could this be otherwise, so long as the heart works vigorously?

The conclusion, therefore, must be that we have no absolutely characteristic signs as to whether an insufficiency of the valves and a stenosis of the aorta are simultaneously present, and in what degree each of these elements exists in the same case. We are, therefore, obliged to content ourselves with an estimate which may be approximately right, but which may also be quite incorrect. It is especially advisable to employ caution when a distinct arteriosclerosis can be demonstrated.

Course and Prognosis of Aortic Lesions.—So long as compensation continues, aortic lesions produce but few symptoms. If the patient who has such a lesion lives a sensible life, his condition is not really disturbed. Some complain of headache, of bleeding of the nose, of palpitation of the heart, especially after physical exertion, sometimes accompanied by annoying pulsation of the carotid. If, as is usually the case, the insufficiency is the dominant factor, then the capacity of the heart for muscular work is conserved in a great degree. This fact is demonstrated in the following table, according to the experiments of Benno Lewy.‡

β-Degree of Insufficiency in Fractions of a Unit.	Daily Working Capacity in Hours.
0.01	9.96
0.05	9.86
0.1	9.74
0.2	9.47
0.3	9.14
0.4	8.77
0.5	8.39
0.6	7.87
0.7	7.31
0.8	6.57
0.9	5.59
1.0	3.81

* "Die Untersuchung des Pulses und ihre Ergebnisse in gesunden und kranken Zuständen," Berlin, 1892, Jul. Springer, p. 41.

† "Specielle Diagnose," etc., p. 34.

‡ *Loc. cit.*, p. 550.

The calculation here was made for 120 pulsations, both at rest and during muscular work. For every hour of work the labor developed is assumed to be 30,000 meter-kilograms. All this is dependent upon the great working capacity of the left ventricle, the muscular mass of which is so well able to become hypertrophied. But this is only possible when there is sufficient nutrition, that is, a sufficient blood-supply, and for this the condition of the coronary arteries is of cardinal importance. Now, it is certain that the alternating tension and relaxation of the entire aortic region which takes place when the valves are insufficient favor the development of arteriosclerosis in a high degree. If these variations become less marked, then the danger of arteriosclerosis diminishes. It is for this reason that a stenosis which exists simultaneously with an insufficiency has, in general, a favorable effect upon the course of the disease.

Many authors agree with Bamberger* when he says: "Of all possible diseases of the valves, the one that is most favorable from the viewpoint of prognosis and which promises the longest duration of life, is insufficiency of the aortic valves, accompanied with a moderate stenosis." Yet at first glance the opposite should be expected—an earlier breakdown of the left ventricle, because the stenosis of the aorta causes an increase in its burden. The theoretic conclusions of Benno Lewy also support this view. The complete destruction of the aortic valves (see table, $\beta = 1.0$) still allows of a working capacity of 3.81 hours daily, but when stenosis also develops, this working capacity diminishes, so that when the stenosis becomes greater than 0.75 of the diameter, all possibility of muscular work is excluded. Only 14,375 meter-kilograms per hour are available as a maximum for the work of the heart. This maximal value can only be reached when the entire reserve force over which the human body has command could be expended upon the heart.† According to the calculations, when 0.01 represents the insufficiency and 0.7 the stenosis, 12,174 meter-kilograms are required, while with a stenosis of 0.8 the heart needs 16,963 meter-kilograms.

With the increase of arteriosclerosis the patient is exposed to the development of heart weakness, with all its concomitants. Heart weakness is also far more frequently the cause of death in aortic lesions than any other factor. That cerebral hemorrhages are especially to be feared appears rather from the prominent elements of these cases—hypertrophy of the left ventricle and arteriosclerosis—than from actual clinical experience.

Bamberger‡ says on this subject: "The frequency of cerebral hemorrhages, on the whole, is much less than would appear *a priori*. Among fifty cases of this affection which I treated (chiefly insufficiency), which were not complicated with other valvular lesions, and of which twenty came to autopsy, only one cerebral hemorrhage was noted, and one small apoplectic cyst, the latter not having given any symptoms during life.

In a third case the patient stated that he had had an apoplectic attack some time before, but no evidences of this could be discovered. I do not know whether more extensive statistics exist on this subject. I have not found any, and I have not thought it necessary to search further into the subject.

The heart weakness is very often accompanied by the signs of cardiac neuroses. The arteriosclerosis then comes into play (see p. 674). The fact that Bright's disease frequently accompanies these lesions results in

* "Lehrbuch," p. 284.

† B. Lewy, *loc. cit.*, p. 358.

‡ "Lehrbuch," p. 274.

an important tendency to dropsy which may reach a marked degree even when the kidneys show predominatingly interstitial lesions. Bamberger, in his text-book, speaks of the slowly developing renal degeneration, and the altered blood state resulting therefrom, as "the causes of the dangerous symptoms." But it is not clear whether he is speaking of anything else besides the dropsical symptoms.

When cardiac weakness has once attained a considerable degree, the patient dies in a comparatively short time, for on one point all observers agree; namely, that the condition may become stationary, but it can never really improve. The permanent changes which occur in the sclerotic coronary arteries account for this.

Does a marked narrowing of the aortic orifice produce phenomena which change the clinical picture in a characteristic way? I shall again quote from Bamberger ("Lehrbuch," pp. 284, 285): "Stenoses of marked degree present less favorable conditions; yet even these, as I have often observed, may be very well borne for a long time, and, in general, the lesions are somewhat more favorable than stenosis of the mitral of equal extent. Compensation is comparatively easily obtained by the increased pressure of the left ventricle, and even the normal filling of the arteries requires no other aid than a little longer duration of the systole. Naturally, under all conditions, the velocity of the blood-stream suffers most of all.

"On the other hand, stenoses of high degree become dangerous and fatal after a shorter or longer period, either as the result of a considerable impairment of nutrition and dropsy, or through venous stasis and secondary pulmonary affections. A marked anemia of the important organs may also become dangerous. I have seen sudden death in one case following epileptic convulsion, in which the autopsy showed no other cause than extreme anemia of the brain.

"In general, stenoses of the aortic orifice show a rather long stage of latency, during which the patients complain only of attacks of palpitation of the heart, of a moderate sense of oppression, with a rather dull pain over the chest; of slight interference with respiration, headache, dizziness, nose-bleed; at times, attacks of pulmonary hemorrhage—all of which symptoms may appear for a time or may be entirely absent. In some patients we find exquisite attacks of stenocardia, at first rarely, but later recurring with increased frequency.

"The more severe symptoms of the second stage almost always begin with respiratory difficulty, and this continues to play a prominent rôle during the entire further course of the disease. The patients are particularly tortured by severe attacks of dyspnea and orthopnea; a severe cough develops, and attacks of pulmonary hemorrhage occur from time to time. In addition, pneumonias and symptoms of pulmonary edema may occur.

"Bright's disease occurs rather less frequently in these cases than in the purer forms of insufficiency. The frequency of pulmonary hemorrhages is also very striking here. In fifteen cases of marked stenosis (usually most of them with insufficiency), seven showed either attacks of pulmonary hemorrhage during life or hemorrhagic infarcts in the lung at autopsy."

According to this description, the greater involvement of the right side of the heart is a very important feature of these cases, for it produces the respiratory symptoms which are so strongly emphasized here. No

reason for this can be found, so far as I know, unless we fall back upon the limited possibility of a perfect compensation in cases of marked stenosis and the consequent early occurrence of insufficiency of the cardiac muscle. This may be the case in some individuals, but a general deduction cannot be made concerning the majority of the patients.

Treatment.—No amendments or additions need be made to the principles which have already been discussed in the section on the Treatment of Cardiac Insufficiency (see p. 145). Yet we must emphasize the fact that the chief danger for the patient with aortic lesions lies in the extension of arteriosclerosis. Anything that may favor this must be avoided, so far as possible. In the first place, therefore, the patient should avoid every marked exertion of the heart by an excess of physical labor or mental excitement. In this, each individual must have his own limits, according to his position in life; but the patient can keep within these boundaries by adhering to a definite order of his daily work, avoiding every excess in eating and drinking. Aside from abstaining from the use of stimulants,—alcohol, coffee, tea, tobacco,—the work of the heart can be lessened essentially by a proper distribution. The patient should not take one principal meal, but preferably three or four meals, each about the same size, during the day. Nor should he flood his arteries with fluid. This would, indeed, be a deluge for these patients.

The use of digitalis requires special consideration. The frequency of the contractions of the heart have quite a different significance in insufficiency of the aortic valves than in stenosis of these structures. The conclusions of Benno Lewy, based upon the results of the mechanic features susceptible of measurement, have a bearing only upon the property of digitalis to diminish the frequency of the heart-beats. He found in insufficiency* that the value of the amount of work to be furnished by the heart for the propulsion of the blood depends upon the duration of the diastole. The latter again depends upon the frequency of the beats.

Muscular activity corresponds to increased pulse-frequency. Its influence is so great that in high degrees of insufficiency, that is, about two-thirds and more, we have the apparent paradox that the force necessary during muscular work is less than that needed in muscular rest—in other words, that the heart must do less work to make 120 contractions per minute and to propel 16.8 liters of blood through the capillaries than when it has to make seventy contractions to propel 4.2 liters of blood. Of course, the cardiac labor may also be lightened through increased frequency of contraction during muscular rest. The increase in the pulse-frequency is, therefore, a means of saving the heart muscle, which, then, proves very effective when the insufficiency grows more marked.

WORK OF THE HEART PER HOUR IN METER-KILOGRAMS IN CASES OF AORTIC INSUFFICIENCY.

The following table shows the figures obtained by experiments as to the work of the heart in aortic insufficiency. The sign β indicates the permanent aperture of the valves. The duration of the diastole for 70 pulsations per minute is 0.5 second; that of the systole is 0.3 second. The duration of the diastole for 120 pulsations is assumed to be 0.2 second, while that of the systole remains the same, 0.3 second.

* *Loc. cit.*, p. 538.

β.	Seventy Pulsations per Minute.	One Hundred and Twenty Pulsations per Minute.	Fewer
0.01	864	847	17
0.05	1,069	983	86
0.1	1,342	1160	182
0.2	1,952	1532	420
0.3	2,607	1924	683
0.4	3,334	2404	930
0.5	4,420	2877	1543
0.6	5,868	3501	2367
0.7	8,072	4285	3787
0.8	12,134	5259	6875
0.9	>15,000	6530	..
1.0	>15,000	8563	..

The body itself employs the increase of pulse-frequency for sparing the heart muscle, and, therefore, according to most authors, the pulse is usually more frequent than normal in aortic insufficiency. Among the writers who speak of this, Corrigan* deserves prominent mention. He stated that the pulse-rate is usually between 90 and 110 beats per minute. He added some remarks which, according to Benno Lewy, quite closely coincide with the latter's own view. Basing his opinion not only upon this, but also upon his experience at the bedside, Corrigan warned, in general, against the use of digitalis in this valvular affection.

So far as can be judged from his writings, he opposed the use of digitalis only when it is given solely for the purpose of reducing the increased frequency of the pulse, in a patient who otherwise does not show any symptoms on the part of his diseased heart. "The pulse which varies from 90 to 100 in this disease, or even higher, should not be interfered with for the sole reason that it is more frequent than in health."

This is exactly the view taken by Traube, whom B. Lewy criticizes, and also by every other scientific physician. Traube says,† in a general way:

"It seems wrong to give digitalis in cases in which valvular disease is detected in an apparently healthy man. To order digitalis in such a case means to lead the patient toward a disturbance of compensation; for in such cases the slowing of the pulse produced by digitalis easily causes a loss of compensation."

It is also true that when once cardiac weakness has developed in consequence of aortic insufficiency, it will not always be favorably influenced by digitalis.‡ But this is due to the fact that in this lesion the strong hypertrophic left ventricle must be extensively involved to become so damaged in its working capacity. On the other hand, if the irregularity of the heart's action is present, which I classify as a separate type (see Cardiac Weakness, p. 68, etc.), then I have not seen any more or any less success with the use of digitalis than can be got with this drug in these conditions under any circumstances—a success which, by the way, is by no means to be underestimated. In aortic stenosis, quite conversely,

* Cited by Benno Lewy, p. 541 *et seq.*, who gives a detailed communication.

† "Gesammelte Beiträge," vol. iii, p. 74.

‡ Of the moderns who approve the old methods *cf.* Carl v. Noorden, "Eulenburg's Realencyklopädie," Art., "Herzklappenfehler," vol. x, p. 440; Romberg, "Ebstein's Handbuch," vol. i, p. 870.

it is desirable to obtain a cardiac action which makes the sequence of the individual contraction less rapid, for thereby we facilitate the overcoming of the obstacle, provided, however, that the duration of each individual systole be also lengthened. The following table shows of what importance this is:

WORK OF THE HEART PER HOUR IN METER-KILOGRAMS IN AORTIC STENOSIS.

α	Complete Rest.							Moderate Work.
	Z = 50 T = 0.5	Z = 60 T = 0.5	Z = 60 T = 0.4	Z = 60 T = 0.3	Z = 70 T = 0.3	Z = 100 T = 0.3	Z = 120 T = 0.3	Z = 120 T = 0.3
0.00	813	813	813	815	814	813	813	3,401
0.01	813	813	813	815	814	813	813	3,402
0.05	813	813	813	815	814	813	813	3,406
0.1	813	813	814	816	814	813	813	3,411
0.2	815	814	815	818	817	814	813	3,452
0.3	817	816	818	824	820	816	815	3,539
0.4	823	820	824	835	828	820	818	3,720
0.5	835	827	837	857	845	827	823	4,068
0.6	858	844	862	902	878	844	834	4,788
0.7	991	936	1,006	1,158	1,066	936	898	8,884
0.8	1,088	1,003	1,112	1,346	1,204	1,003	945	11,897
0.9	2,115	1,716	2,226	3,526	2,659	1,716	1,440	43,578
0.95	6,836	4,681	6,861	11,566	8,713	4,681	3,500	175,426
0.96	9,612	6,920	10,361	17,788	13,248	6,920	5,056	274,986
0.97	16,806	11,912	18,164	>20,000	>20,000	11,912	8,524	. .
0.98	Always over 20,000 Meter-Kilograms.						17,558	. .

It will be seen from this table that the stenosis is markedly influenced by the lengthening of the systole only after the narrowing has become rather marked. The symbol α indicates the degree of stenosis, Z the number of pulsations per minute, and T the duration of the single systoles in seconds. It appears from these figures how little the increase of the labor of the heart is due to the degrees of stenosis and how little influence the frequency of the pulse has in such cases. Even on the duration of the systole it has scarcely any effect before we reach a stenosis of 0.5. After that, however, these factors assume an increasingly important character, and the increased pulse frequency makes it possible that even in high degrees of stenosis the cardiac labor can be furnished, at least theoretically.

All this, however, applies only when the body is at perfect rest. The moment muscular activity is demanded, the task of the heart increases to such an extent that the limit of capacity is reached early. Even then a change in the pulse-rate and a lengthening of the systole may be useful under certain conditions, but this is possible only to a very limited extent.

In well-compensated stenoses we usually find a low pulse frequency. C. von Noorden* even states that the frequency varies from 60 down to 40 beats per minute.

In any event, there may be circumstances in which the use of digitalis may seem contraindicated; at least, if we should be tempted to deal with the purely mechanic conditions. Personally, I think it is not necessary to deviate from the general rules in the use of digitalis in aortic stenosis,

* *Loc. cit.*, p. 421.

if for no other reason than that the pure cases are so rare that few physicians have the opportunity to test at the bedside any conclusions drawn from theoretic considerations.

We are still but too imperfectly acquainted with the mode of action of digitalis to be able always to make accurate therapeutic calculations based upon the changes in the cardiac activity—that is, the slowing of the pulse and the lengthening of the diastole—which it produces. For the present, we must adhere to the rules obtained from the experience of our trustworthy observers, and these may be summed up as follows: In lesions of the aorta we should use digitalis with more caution than in any other forms of heart disease.

INSUFFICIENCY OF THE TRICUSPID VALVE.

With very few exceptions insufficiency of the tricuspid valve exists in combination with general cardiac insufficiency. Endocarditis may be the cause of this lesion, but in extra-uterine life such is the case only when endocarditis had previously existed and had involved other valves of the heart. In either case this "valvular" lesion scarcely ever is seen "pure." It is better to speak of it as a disturbance in the closure of the valve which appears as a part of the results of the insufficient labor of an incapacitated heart. (Compare pp. 323 and 324.)

The general symptoms are those of a marked insufficiency of the heart. In cases in which an insufficiency of the heart is not already present before the tricuspid lesion develops, the signs of such an insufficiency will probably soon develop. The mechanic effects of tricuspid insufficiency have been constructed upon theoretic considerations. It is said that in insufficiency of the tricuspid the systole throws back a part of the blood contained in the right ventricle into the right auricle. Therefore, so much less blood enters into the pulmonary artery, in which the pressure and the velocity of the stream correspondingly diminish.

Inasmuch as the left heart also receives a smaller amount of blood, the pressure and the velocity of the flow in the aorta also diminish, and blood consequently accumulates in the systemic veins; all the more because its entrance into the right auricle is rendered difficult by the fact that the right auricle is filled by the right ventricle at each contraction of the latter.

The result of all this, therefore, is an overfilling of the systemic veins, a lowering of the blood-pressure in the pulmonary artery, as well as in the aorta, and a slowing of the general circulation.

The effect of these changes upon the heart itself consists, in the first place, of a dilatation of the right auricle, which receives blood from two different directions, followed by the hypertrophy of this part of the heart. The same changes follow in the right ventricle. Compensation must needs always be imperfect and of short duration, because the right auricle is too weak muscularly to maintain it. The dilated and hypertrophic right ventricle is able, it is true, to throw an increased amount of blood at increased pressure into the pulmonary artery, but, at the same time, the amount and the velocity of the blood which escapes back into the auricle increases steadily, thus increasing the dilatation of the ventricle during the following diastole.

It is evident that in tricuspid insufficiency the right and the left ventricles of the heart work in opposition to each other. This is shown unequivocally by the return wave, presystolic-systolic in character, which

passes from the veins toward the capillaries; in other words, by the presence of a true pathologic venous pulse (see p. 85).

The signs obtainable on physical examination are said to be as follows: marked increase in the area of the cardiac dulness toward the right. A wide-spread but weak pulsation of the lower part of the sternum, and a systolic murmur heard at the right border of the sternum or over this bone itself, as well as a slight thrill, which may be felt over the site of the murmur. The cardiac tones are everywhere faint. The pulse is small and not well filled.

These phenomena should be used with caution in **diagnosis.** The extension of the cardiac dulness indicates that the right side of the heart in both its divisions is more abundantly filled with blood, and therefore distended. This extension of the cardiac dulness allows us only to conclude an increased massing of blood in the veins of the systemic circulation. Inasmuch as this may occur with any marked cardiac weakness, and inasmuch as it may, indeed, lead to a muscular insufficiency of the tricuspid, but does not necessarily do this, this sign cannot be given much prominence. We can only say that an insufficiency of the tricuspid cannot exist without an extension of the cardiac dulness, and that, therefore, the latter is a sign which must always be present.

The concussion observed over the lower right side of the thorax, beginning at the middle and passing outward, can only occur when the heart is comparatively vigorous as to its musculature. This is naturally more frequently to be expected when a more slowly developing condition, either in the lungs or in the heart, gives time to the right side of the heart to become equal to the tasks set before it by a hypertrophy of its muscular structure. But in such cases insufficiency of the tricuspid naturally does not occupy the foreground, and even would scarcely be considered. The sign, therefore, is of no value in diagnosis.

And now as to the systolic murmur. In order to make use of such a murmur diagnostically, one would have to show with certainty that it coincides with the closure of the valve. The rules which have been set up come to this: The murmur must be heard on the right side, out to the nipple-line, and must have a peculiar quality. But, unfortunately, such a variety of systolic murmurs may be heard in so many places in the degrees of cardiac weakness which are considered here that the greatest caution must be exercised, even in the diagnosis of a mitral insufficiency.

Personally, I scarcely trust myself to determine tricuspid insufficiency in its ordinary form by auscultation alone. I would not be able to distinguish anything definite from the confusion of dull and obscurely timed murmurs. I remember one case in which I succeeded in distinguishing the murmur of tricuspid insufficiency; but in that case there was a structural change in the valves, as the autopsy afterward proved.

It goes without saying that we must be on the lookout for a confusion of this murmur with a true murmur of stenosis or insufficiency developing in the left side of the heart. This is a little less difficult when the muscular tones do not become too insistently loud.

The pulsations in the systemic veins, especially in those of the liver, offer trustworthy diagnostic signs. They must be presystolic-systolic in character. This subject has already been discussed in detail elsewhere (see p. 62).

As regards the differential diagnosis, the only condition which needs to be considered is insufficiency of the aortic valves with its systolic pulse

in the liver. The latter sign may be distinguished simply by the fact that it can only occur with a very vigorous action of the heart.

A sign to which a great deal of weight was attributed formerly was the less vigorous diastolic tone over the pulmonary artery. This is scarcely of any value in diagnosis. It was said that when the blood was free to pass through the auricle into the veins, then a smaller amount entered into the pulmonary artery and the systolic pressure in this vessel was lowered. Thus a lessened intensity of the diastolic sound over the pulmonary valve resulted. If we remember upon how many circumstances it may depend, as to whether the right side of the heart works relatively more vigorously than the left, we will agree with Bamberger,* who characterizes this sign as one established *a priori* and devoid of any significance.

The **prognosis** is decidedly unfavorable. In cases with organic change in the tricuspid, we should always bear in mind the great improbability of sufficient and permanent compensation. In functional cases the insufficiency of this valve alone constitutes a sign of a high degree of cardiac weakness. Of course, this condition may improve, at least for a time, and thus its consequences may disappear; possibly for a considerable time, when, for example, a marked bronchial catarrh or a curable general illness produced the weakness of the heart.

[TRICUSPID STENOSIS.

This lesion occurs rarely alone, and when it does, can usually not be distinguished from mitral stenosis, even when suspected. When combined with other lesions, as it more frequently is, its detection is still largely a matter of chance. The signs have been succinctly described by Herrick† as follows: "Tricuspid stenosis can be suspected when the patient is a female; has a history of dyspnea, palpitation, edema, often with remissions and exacerbations; is cyanotic, has rapid, arhythmic, feeble pulse; has mitral stenosis with enlarged right heart, particularly if auricular enlargement can be made out. Especial value attaches to persistent cyanosis. If, in addition to this, there is a presystolic or diastolic murmur, heard best over the ensiform or over the right fifth and sixth cartilages, and particularly if this murmur differs in intensity, quality, or pitch from the mitral diastolic, or if there is an area between the mitral and tricuspid areas where no murmur is audible, the diagnosis becomes reasonably certain."—Ed.]

The changes in the valve at the right side of the heart and the pulmonary artery are so very frequently congenital, and then are accompanied by other disturbances, that it seems more correct to consider them separately. The treatise of H. Vierordt, contained in Nothnagel's "Specielle Pathologie und Therapie," vol. xv, pt. ii, is the best and most complete.

[Attention should be called to the "ease with which murmurs may be produced in the pulmonary artery" in experiments on animals, as pointed out by Thayer and MacCallum.‡ "Very slight pressure over the conus arteriosus or over the artery results in the development of a systolic thrill and murmur immediately beyond the point of pressure. The ease with which these murmurs are produced by pressure suggests immediately that a similar process may account for some of the frequent basic pulmonary murmurs which are heard in man. If systolic murmurs can be

* "Lehrbuch," p. 258.
† "Boston Medical and Surgical Journal," March 18, 1897.
‡ "American Journal of the Medical Sciences," February, 1907.

produced in man by a degree of pressure over the conus arteriosus as slight, relatively, as that necessary to bring about the same phenomenon in the dog, it is but natural to assume that the resistance offered by the chest-wall alone may, in many instances, result in systolic murmurs in the pulmonary area.

"In two instances a pulmonary systolic murmur developed after considerable hemorrhage.

"In one instance with a low general pressure following nitroglycerin, a pulmonary systolic murmur developed. The administration of adrenalin and nitroglycerin had, however, but little effect in modifying the pressure in the pulmonary artery.

"The administration of adrenalin resulted in an increase in systolic pulmonary murmurs when present, during the first strong beats which followed, but during the period of very high pressure occurring in the general circulation the pulmonary murmurs as well as murmurs at the aortic area tended to diminish, returning, however, again, if present before, with a fall of pressure at a period when aortic murmurs still remained absent.

"Infusion of salt solution resulted commonly in the development of systolic pulmonary murmurs.

"In a general way, conditions which resulted in especially abrupt action of the right ventricle, with a large excursion of the pulse-wave in the pulmonary artery, seemed to favor the development of systolic murmurs at the pulmonary orifice, especially if there had been a preceding hemorrhage, followed by infusion of salt solution.

"Murmurs were produced more readily in the pulmonary artery than in the aorta, notwithstanding the very slight variations in pressure which occur in the lesser circulation. This was probably due, at least in part, to the fact that with a more or less indistensible ring, the pulmonary artery beyond is much less resistant than the aorta, and capable of greater dilatation with ventricular contractions of moderate force, thus producing, just beyond the pulmonary ring, those conditions favorable to the development of fluid whirls, and consequently a thrill and murmur.

"In general, our observations seem to show that a change in the character of the circulating blood, such as that which results in the replacing of a considerable quantity of blood by salt solution (diminished viscosity?), is extremely favorable to the development of systolic murmurs at the base."—Ed.]

COMBINED VALVULAR LESIONS.

The frequent coexistence of insufficiency of the valves and stenosis of their orifices on the left side of the heart has already been considered in detail in connection with the description of these conditions. The same is true of insufficiency of the tricuspid as the result of a marked degree of cardiac weakness.

But cases occur also in which the venous valve, as well as the arterial valve, become diseased at the same time. The proper field of the compound (combined or complicated) valvular lesions is thus more accurately circumscribed. Those who wish may also include here the valvular lesions occurring on the right side of the heart, together with those on the left side, but in such cases the lion's share will be taken by the "muscular" insufficiency of the tricuspid.

The frequency with which the different valvular lesions are complicated

is shown in the table on p. 311. Yet I cannot refrain from expressing some doubts as to the accuracy of the various data given there. The diagnosis of combined valvular diseases in the living is so difficult that only statistics based upon the results of autopsies can be recognized as of positive value. Do such statistics exist? This must be answered in the negative, so far as some of the selections of figures are concerned; as, for example, those of Breslau, in which both hospital and dispensary material was used.

It cannot be regarded as extraordinary that lesions often simultaneously appear in the venous and the arterial valves of the left side of the heart. These lesions, it must be remembered, represent by far the largest number of valvular affections observed, and the proximity of the valves favors an overlapping of the tissue changes from one to the other.

The effect of complex valvular lesions upon the circulation of the blood has been reasoned out by some writers; but very little has been gained by this for a true understanding of the conditions, and nothing has resulted beyond relapsing into a schematic method of thinking which has no value in treatment and but a very slight value in diagnosis.

We must not forget that, in addition to the mechanic conditions, the work of the heart-pump depends upon its musculature, and that the latter is actually the most important element. Since we know that the musculature largely controls the play of the valve, and that it is capable of directly adapting itself to the increased or decreased task set before it, it is no longer possible to hold the gross changes in the valves and orifices solely responsible for everything that occurs.

The earlier authors understood this quite well, especially from the clinical standpoint. "In view of the great variety of combinations of valvular diseases that may occur, it is natural that there should be a great difference in the disturbances on the part of the heart and the circulation that may be observed in such complicated cases. Each of these cases, properly speaking, has its own characteristics, and a general description can be given only in very broad terms" (Bamberger*).

He then attempted to classify these cases in such a way as to divide the combined valvular affections into: (1) Those that supplement each other in their effects. Example: a stenosis of the aortic orifice combined with insufficiency of the mitral. (2) Those that mutually compensate each other. Example: Aortic insufficiency with a considerable degree of stenosis of the left venous orifice. (3) Lesions which complement each other in one direction while they compensate each other in another. Example: stenosis of the left venous orifice with insufficiency of the tricuspid. The final conclusion of Bamberger, however, is as follows: "It would be erroneous to suppose that this classification includes every possible combination, but the scheme outlined above is very useful in diagnosis."

One fact to which Bamberger calls attention should be remembered as a warning to all those who are inclined to judge *a priori:* "The conditions which occur when very extensive lesions are present are worthy of note, especially those of stenosis of all valves except the pulmonary artery. In such a case we would expect *a priori* to see the most marked local disturbances and the greatest increase in the volume of the heart. Judging from a quite extensive series of specimens which I have examined in various museums, I may be allowed to state positively that such is the case only very exceptionally, and that the heart in such instances is but slightly enlarged, or may not even be hypertrophied at all."

The importance of the musculature of the heart shows itself very distinctly in the picture of combined valvular disease when we have the opportunity to follow the development of such valvular defects, together with frequently recurrent endocarditis and myocarditis. I have reported two such cases in a brief manner in this treatise (Endocarditis, p. 222), and have included reports of the autopsies:

Case XII.—Stenosis and insufficiency of all the valves on the left side of the heart; dilatation of the ascending aorta.

* "Lehrbuch," p. 292.

CASE XIII.—Stenosis and insufficiency of all the valves of the left heart; slight stenosis of the right venous orifice; dilatation of the ascending aorta.

The diagnosis of these conditions is, in a great measure, obscured through the variable working-capacity of the heart.

In the first place, the increase of cardiac dulness is by no means always the same, the area enlarging and decreasing from time to time. Thus, in case XIII, we find that on October 29, 1886, the dulness extended one finger-breadth beyond the right sternal line. On November 3, 1886, it extended three finger-breadths beyond the right sternal line. Then it steadily decreased in area until June 14, 1887, when it was found one finger-breadth beyond the right sternal line, and finally it increased again and on December 13, 1887, was found four finger-breadths beyond that line. There is no doubt that these variations were produced by the more or less marked resistance which the heart muscle offered against the dilatation, and that this resistance again depended upon the degree of working capacity of the heart. We must remember this fact when we attempt to utilize the increase of the transverse diameter of the dulness in diagnosticating the involvement of the right heart when a valvular disease is compensated.

The left ventricle is much less exposed to such variations in its volume. At least these variations are not demonstrable; the irregular activity of the left ventricle is manifest through the changes in the auscultatory phenomena and, unfortunately, we are obliged to depend chiefly upon these signs in the complicated cardiac lesions (see above, p. 330). Experienced physicians are rather conservative on this question. The most emphatic expression of opinion is that of Oscar Fraentzel,* who justly says: "The inexperienced observer cannot be too strongly warned against assuming the presence of complicated valvular lesions." And, further on: "The physician who is constantly accustomed to control his diagnoses by autopsies will be reluctant to assume the presence of complicated valvular lesions, and will prefer very often to express a '*non liquet*' rather than to attempt to make a positive diagnosis."

Fraentzel himself believes that, aside from stenosis and insufficiency of the mitral valve, characteristic signs sufficient for an easy diagnosis can only be expected in mitral stenosis combined with aortic insufficiency. The determining factor is as follows: "A diastolic murmur at the apex existing in one of its numerous modifications (see above, p. 365), combined very frequently with an exquisite diastolic thrill, while on the left side of the sternum at the fifth costal cartilage, or at the right border of the sternum in the second intercostal space, we hear a loud single diastolic murmur. The pulse is more or less small, and yet its qualities of pulsus *altus* and *celer* can scarcely be mistaken."

Other signs may either be absent or present; as, for example, cardiac dulness and apex-beat, both of which may vary to a great extent. Therefore, the distinguishing characters of the diastolic murmur, together with the quality of the pulse, are the decisive factors in these cases, and when they are both clearly marked, a definite opinion may be expressed.

I do not care to enter into details, and I repeat what Gerhardt says:† "In all combined valvular lesions, thorough study of the case makes all the rules worthless." Another point to be mentioned is that the symptoms of a combined valvular lesion may disappear wholly or partly

* "Vorlesungen," pp. 80 *et seq.*, and pp. 168 *et seq.*

† "Lehrbuch der Auscultation und Percussion," p. 327.

for a time. There is nothing remarkable in this, if there is also cardiac weakness, but it happens at times that it is only with the development of cardiac weakness that the lesion becomes clearly marked. This is what happened in the case reported by von Leube.*

The patient, a man aged fifty-five years, showed the picture of a pure mitral insufficiency on admission into the Julius Hospital. Three weeks later the first signs of an insufficiency of the aortic valves appeared, and soon increased in intensity, while a stenosis of the left venous orifice could also be made out. In addition, there was insufficiency of the heart with edema of the ankles, swelling of the liver, and, finally, insufficiency of the tricuspid. The autopsy showed, in addition to the changes upon the left venous orifice, the following lesions of the aorta.

"The first valve was deformed, thickened, and calcified. The same changes appeared in the second, which was adherent to the third."

These changes in the aorta, therefore, did not develop during the patient's stay at the hospital, and did not give rise to any symptoms so long as the heart was comparatively strong. They developed, however, when cardiac weakness ensued.

The **prognosis** is certainly not improved by the presence of several cardiac lesions. Possibly stenoses that are combined with insufficiencies may in one way be considered as favorable, at least when they remain within moderate limits (see above, pp. 360 and 406). Yet it is doubtful whether the work of the heart is facilitated and whether an early collapse of the organ is thereby prevented.

The fact that a number of foci of infection are present in valvular lesions due to bacterial endocarditis is probably not unimportant. Do these infections not tend to recur more readily?

* "Spec. Diagnose," first ed., pp. 41, 42.

DISEASES OF THE MYOCARDIUM

AND

NERVOUS DISEASES OF THE HEART.

BY

DR. L. KREHL.

DISEASES OF THE MYOCARDIUM

AND

NERVOUS DISEASES OF THE HEART.

INTRODUCTION.

The consideration of the subject of diseases of the heart at this time presents a great many serious difficulties. Authorities are not even agreed on the conditions that are to be included among diseases of the heart. As a matter of fact, the term should include those conditions in which a functional abnormality of the heart constitutes the starting-point of the morbid symptoms observed in the organism. It is not advisable, however, to restrict the term to these processes in a discussion on diseases of the heart. Disturbances of the cardiac function are very frequently secondary to diseases of other organs which represent the cause of the cardiac trouble. According to the above definition, all such conditions would have to be omitted from the discussion. But in not a few of these cases the cardiac symptoms predominate to such a degree that none but an expert is able to recognize their secondary nature. In forming his judgment of a disease the physician must begin with the symptoms. On the symptoms he bases his diagnosis, as well as his classification, of the disease. The question to what extent diseases of organs other than those which give rise to the symptoms form the starting-point of the pathologic process is merely secondary. For these reasons it seems advisable to include in a clinical presentation all morbid conditions characterized by pronounced cardiac symptoms, and, indeed, such a course is absolutely necessary in a discussion of the differential diagnosis.

A comprehensive discussion of circulatory symptoms occurring in all forms of disease would absolutely guard against omission; but to do that would carry one too far afield and would be, in fact, absolutely impossible, because in many cases phenomena emanating from the heart can only be understood when taken in connection with the complete clinical picture of the disease.

It is evident, therefore, that we must choose a middle path, and for the present we can find no better criterion than custom. To a certain extent a writer will, no doubt, be swayed by his personal views; for on many subjects a diversity of opinion is allowable and will probably always continue to exist. My purpose to discuss, under the head of diseases of the heart muscle and nervous diseases of the heart, all those conditions which, by common consent, based on our present conceptions, are included under these heads, is confirmed in this case by the consideration that, in the preparation of the present text-book, the field of circulatory disturbances, which should form a composite whole, has for practical reasons been broken up and assigned for treatment to several different physicians. This consideration alone makes it imperative for each individual contributor to follow custom in the classification of his material. There is no occasion in this work to give a comprehensive view of the entire field.

One great difficulty encountered in the treatment of this subject is that the heart and the blood-vessels have not been assigned to the same individual. We have come to recognize more and more the functional unity of these two structures, and our efforts should be directed toward developing and elaborating a pathology of the circulation. In health the most wonderful contrivances exist for controlling the circulation in a certain definite manner to meet the needs of the organism by mutual adaptation between heart and blood-vessels, and in disease also the etiologic factors in many cases do not act upon the heart or vessels separately; on the contrary, disease involving both heart and vessels plays a very important rôle, greater, indeed, than is generally supposed. The same is true also of therapeutic agents.

The assignment of the material to a number of workers must explain and excuse the presence of many repetitions. As in the contemplation of biologic processes in general, artificial divisions cannot be carried out beyond a certain point; nor is diversity of opinion in the general conception of individual matters avoidable. Many questions are still under discussion and, therefore, can, and indeed ought, in the interests of science, to be approached from various points of view. At all events this diversity of opinion will do no harm; for the work is intended for physicians, and in the course of their practice they have long ago found out the harm that may accrue not only to the progress of science, but also to the actual effectiveness of a physician's work—in view of the ever-changing and complicated relations of daily life—from any attempt at schematic dogmatism or deception in regard to what we really know.

As this is not the proper place, nor the present the proper time, to introduce new boundaries for the field as a whole, I have avoided any innovation in the classification of the various individual conditions which I shall discuss and, agreeing entirely with E. Romberg, have adopted a classification in which the various groups of diseases are jumbled together according to clinical, pathologic, or etiologic considerations. In doing so things which in many respects belong together have had to be separated, and others, which ought to be considered apart from one another, have been frequently discussed together; digressions and repetitions are unavoidable.

From a theoretic standpoint all this is very unfortunate, but I know of no other practicable method. Any attempt to erect a pure, etiologically circumscribed clinical picture had to be given up, because to do so I should have been obliged either to omit certain things altogether or to do violence to the facts and bring them in at altogether unsuitable places.

The physician will find what he wants in spite of the classification, and perhaps this sad state of affairs will impel the reader to contribute something toward clearing up the clinical phenomena. True progress can only be achieved by coöperation, and if the entire work is left to the clinician, as has been done in the past, a certain one-sidedness will continue to be unavoidable, for it is a necessary consequence of the distribution of the clinical material. On certain very important and interesting matters, especially the course and duration of many diseases, the only one who can give useful information is the practising physician who has at his disposal a well-established private practice.

We are living in a period of practice and "therapeusis." One is almost afraid to indulge in theoretic considerations, and the world is ever ready to brand the man who does so as "unpractical" and a "poor doctor." For all that, I consider it my duty not only to devote some attention to theory and controversy, but also never to attempt to hide my lack of knowledge. The only hope for progress in science and for

securing the necessary coöperation of the practising physician is to keep the latter thoroughly instructed as to the status of the questions in controversy. But in my opinion the physician cannot dispense with theory even in his every-day practice, for his theoretic knowledge alone enables him, in view of the endless varieties of pathologic phenomena which, strictly speaking, are different in every new case, to be sure of his judgment and ready to act.

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EXAMINATION.

In order to describe diseases of the heart it is necessary, first, to set forth how and according to what principles the diagnosis is made. For, although we are agreed on the general principles of diagnosis, there are, nevertheless, a number of individual differences of nomenclature, usage, and habits of thought, and an observer's descriptions cannot be clear unless one is acquainted with his own special diagnostic methods.

THE ANAMNESIS.

It is extremely important, in examining heart patients, to take an exhaustive history of the case. The force of this becomes more and more apparent the older one gets and the more precise one tries to be in diagnosis. The history of the present disease, the history of former diseases, the patient's social and family conditions, both present and past—all such matters must be carefully inquired into in this as in other diseases.

There are two questions that demand special attention. The first is to what extent the patient, up to the time of the present disease or possibly after its inception, was able to meet the various demands of daily life, both physical and psychic. To determine this point the patient must be closely questioned in regard to every possible detail. For, since we must have information about the condition of the circulation in our patient and the methods for testing it are still very imperfect, the patient's ability to meet the demands made on his heart must be determined as fully as possible from the history.

In the second place, we ought to make ourselves acquainted with the patient's personality, and the best way to accomplish it is by taking a careful history. A thorough acquaintance with the individual is the most important point, both in the diagnosis and in the treatment of heart patients. If the time-honored saying that we treat patients and not diseases is ever to be strictly applied, it is in the case of heart patients. To do this it is obvious that we need as full a knowledge as can be obtained of the patient's whole personality. How far the physician will be able to obtain this knowledge will depend, in the first place, on the patient's relations to his physician in general, on the latter's penetration, and, to a large extent also, on a number of minor matters. Thus, the physician

must carefully note the patient's manner in relating his trouble, the judgment he has himself formed of his condition, what his ideas are about its cause, and his relations to men and things in general in the outside world. This is not the place to discuss these things in detail, for they are essentially the same in heart patients as in any other class. I merely wish to point out the great importance of these matters in the consideration of the subjects with which we are dealing.

EXAMINATION OF THE PATIENT.

Heart patients may be examined either in the semirecumbent position in bed or in the standing or sitting posture. In the latter case the examiner must also stand or sit in front of the patient. A good general view of the body is obtained by having the patient sit on the edge of the table, the physician standing in front of him, as Curschmann is frequently in the habit of doing. Very often, however, we have no choice, the patient's dyspnea forcing us to make the examination in a certain definite position. It is, therefore, important to master the technic so thoroughly that one is able to examine in any position. Whenever a choice is possible, it is often very useful to make the examination in several different positions, because some phenomena, as, for example, auscultatory signs, change with the position of the body.

In heart disease, as in every other morbid condition, a careful inspection should be made of the patient's exterior, noting the expression of the face, from which alone much information may be obtained, whether there is anxiety and dyspnea, grief and worry, or whether the inner personality has remained untouched by the disease. We shall learn that in a variety of conditions, such as angina pectoris, for example, the expression of the face is unmistakably altered.

Other things to be noted are the color of the skin and mucous membranes, the patient's position and attitude, the type of breathing during rest as well as during movement, as, for example, when the patient is talking. After that the individual systems are examined; for a conscientious physician must include them all in his consideration of the case before forming his opinion. But it is not possible to give general rules as to how minute this examination of every individual part of the body must be; that must be determined by special conditions and by the requirements of the individual case.

In this discussion we shall, of course, include only the examination of the circulatory apparatus.

INSPECTION AND PALPATION OF THE HEART.

Examination of the Heart by Inspection and Palpation.—We desire to learn the position of the heart, its size, the condition of the individual portions, as well as the action of the valves at the various orifices. In addition we must satisfy ourselves in regard to the condition and ability to perform its work, both of the general circulation and of the circulation in each individual organ.

We have at our disposal a great variety of methods of examination, and these methods are all necessary; for, on the one hand, a certain object is attained by means of various procedures, and, on the other hand, the same method may solve several different questions. In presenting the subject we may either take up the data that are to be determined in their

order, or discuss the individual methods and emphasize what they are able to teach us. Like most authors, we elect the second alternative, because, by doing so, we shall at the same time describe the process of clinical examination in its proper order.

First of all, the entire precordia, as well as the anterior chest-wall, must be subjected to careful inspection. For this purpose it is necessary to remove the shirt altogether, as otherwise it is impossible to get an exhaustive view. The shape and arching of the thorax first demand our attention. In the great majority of healthy persons the chest is symmetric in both respects, there being no difference between the right and the left side. In some cases there are differences due to anomalies in the bones, as, for example, after rachitis, and much more frequently in kyphoscoliosis of the thoracic vertebræ, the etiology of which is so mysterious. For the kyphoscoliosis may be of almost any degree, and there are individual cases in which the condition produces a deformity in the precordial region—either a bulging or a depression. These are the only deformities that interest us here. The same thing is sometimes seen in cases of unilateral distortion of the ribs at their insertion to the sternum, which may also cause bulging of the precordia. Other causes are mediastinal tumors and aneurysm of the aorta, which, with due care, can always be differentiated from cardiac precordial bulging.

In all these cases the anomaly in the shape of the thorax is primary and must be carefully distinguished from cases in which a functional anomaly of the heart causes the change in the thorax, or, in other words, in which the functional anomaly is primary and the change in the thorax secondary.

The heart itself is capable of increasing the circumference of the chest by forcibly pressing against yielding costal cartilages.

Thus the position and the action of the heart, as well as the degree of flexibility of the costal cartilages, are of importance; the relation between the two factors appears to be fundamental. It is the cartilaginous portions of the ribs that must be considered chiefly from this point of view, as their flexibility varies at different periods of life; but the most important change that they undergo is calcification, the degree of which varies exceedingly in different individuals. The deposit of lime salts in any quantity obviously offers a considerable impediment to the bending of the ribs.

In many cases the absolute strength of the heart's contraction is greater than normal. But this is not a necessary condition for the production of a prominence in the precordial region; the essential thing being that the heart must be brought forcibly against the chest, which, as will be shown presently, may happen even when the strength of the cardiac contractions is absolutely diminished, providing the situation of the viscus in relation to the chest-wall is changed by dilatation.

The thorax in these cases either bulges forward or is drawn out laterally, so that the left nipple is farther away from the sternum than the right; not infrequently both changes are found associated.

Unquestionably, the most marked cases of bulging of the precordia are found in children or young persons as a result of intense dilatation and hypertrophy of the heart affecting either the left or the right ventricle. As a rule, increased action on the part of the left ventricle is of more importance because its contractile strength is much greater than that of the right. But if the costal cartilages are sufficiently yielding, the pressure of the right ventricle may also be sufficient to produce bulging.

The influence of pericarditic processes on the production of a bulging precordia need not be discussed in this connection. In the literature the effect of a recent exudate has often been overestimated, as Schrötter quite correctly points out in this work. When the condition is solely due to the presence of an exudate, it is probable that the changes more frequently consist of a certain degree of edema of the precordia and a certain degree of bulging of the intercostal spaces rather than true protrusion of the ribs. On the other hand, the adhesions that follow an exudative pericarditis occasionally produce enlargement of both ventricles, and, as a result, bulging of the precordia; in these very conditions, however, retraction of the chest-wall is also not infrequently observed.

As the ribs become firmer and the cartilages undergo calcification, the effect of even marked enlargement of the heart on the shape of the precordia becomes less and less.

Whenever an enlargement of the heart has encroached upon and diminished the size of the left lung, the respiratory excursions of the left half of the thorax are lessened, assuming that the thorax in the affected individual shows distinct respiratory movements.

The methods of inspection and palpation cannot be dissociated in the examination of the **apex-beat.**

The term "apex-beat" or "heart-beat"—we use these terms synonymously—is used to describe the bulging of a point on the chest-wall at regular intervals coinciding with ventricular systole. In a healthy adult of middle age this point is found in the left intercostal space, more or less close to and within the nipple-line,* and exactly corresponds to the apex of the heart or to a point a little to the median side of the apex.

In small children, up to the tenth year of life, the apex-beat is frequently found outside of the nipple-line, and quite often in the fourth intercostal space. As the time when the apex-beat begins to coincide with the mammillary line varies greatly in different individuals, it is often difficult to say in the case of children near the upper limit of childhood whether the position of the apex-beat is normal or abnormal.

Old age in itself does not lead to displacement of the apex-beat, but it is often associated with such conditions as arteriosclerosis and emphysema, which cause downward and outward displacement. In arteriosclerosis the left ventricle is dilated; in emphysema the position of the diaphragm and that of the entire heart is low.

Sometimes the apex-beat is observed in, or even just beyond, the nipple-line in perfectly healthy middle-aged men, and very rarely it may be found in the same class of individuals in the fourth intercostal space. The thorax is then usually abnormally short.

This displacement is much more frequent in women,† it being observed in 12 per cent. of the cases. The position of the apex-beat as regards the horizontal and the vertical bear a certain relation. When the diaphragm is high, the long axis of the heart approaches the horizontal; when the diaphragm is low, the long axis is more nearly vertical. For this reason an apex-beat felt in the fourth intercostal space is frequently found in or beyond the nipple-line. Even when the apex-beat is in the fifth intercostal space in women it is sometimes found beyond the spot which in the male corresponds to the nipple-line.

Very rarely, and only when the thorax is abnormally long, the apex-beat is seen in the sixth intercostal space in perfectly healthy persons.

Obviously, therefore, it would be altogether wrong to follow a rigid scheme when interpreting the position of the apex-beat, and I cannot

* *Cf.* Eulau, "Archiv für klinische Medicin," vol. xxxiv, p. 258. Kirchner, Merkel-Bonnet's "Anatomische Hefte," i, x, p. 127.

† Schultess, "Archiv für klinische Medicin," vol. lx, p. 320.

caution my readers too emphatically against drawing absolute conclusions from slight abnormalities in the position of the apex-beat, especially when there is any anomaly of the thorax. For abnormalities of the thorax give rise to the most bizarre displacements of the apex-beat even when no disease of any kind is discoverable in the heart.

It has been shown that in a healthy individual the apex of the heart, or a point very near it, during the apex-beat actually forces itself in the intercostal space. As the apex of the heart is behind the tongue-shaped process of the left lung, the apex-beat must be effected through that portion of pulmonary tissue which has an important bearing on the explanation of some changes that are observed in the apex-beat. It explains, for example, the frequent diminution of the apex-beat during inspiration.

So far as I can see, there ought now to be no more doubt about the *way in which the apex-beat* is produced.* The heart occupies the left anterior portion of the dome of the diaphragm, the long axis running from the right above and behind, to the left forward and downward, so that the apex-beat, as a rule, reaches as far as the fifth intercostal space. During diastole the shape and position of the heart are directly dependent on the arrangement and tension of neighboring organs; for then the viscus is exceedingly soft and has no characteristic form, yielding itself absolutely to the mechanic conditions of its surroundings and to the action of gravity. During diastole the long axis of the heart is inclined toward the left anterior chest-wall in such a way that the chest-wall forces the apex backward.

During systole, on the other hand, the heart has a definite shape, namely, that of a cone with the apex approximately above the center of the base. The long axis of the ventricles undergoes very little or no diminution† in comparison with the length of the organ during diastole. As the heart becomes extraordinarily hard during systole, it is able to persist in its effort to assume its characteristic shape even in the face of considerable resistance. During diastole the apex, as has been mentioned, is forced slightly backward by the chest-wall from its position above the center of the base; during systole it has the power of maintaining its position at that point. A study of the relations of the thorax in any good illustration of a cross-section (as, for example, illustration No. 13 in the large edition of Braune's "Atlas") makes it at once apparent that there is a systolic bulging of the apex in the fifth intercostal space. In addition it has been shown‡ that during ventricular systole the apex of the heart moves to the right, upward and forward.

In all essential points this corresponds with the old theory,§ thought out by C. Ludwig, and, so far as I can see, satisfies all reasonable requirements. It tallies perfectly with the more recent views in regard to the position and the contraction of the heart. As Martius points out, with good reason, it is now time to discard the numerous other hypotheses that still exist in regard to the production of the apex-beat, especially

* For the literature relating to the mode of production of the apex-beat up to 1880 see Rollett, in Hermann's "Handbuch der Physiologie," fourth edition, i, pp. 182 *et seq.* Martius, "Zeitschrift für klinische Medicin," xiii, p. 478. *Ibid.*, "Volkmann's Vorträge," N. F., No. 113.

† Hesse, "Archiv für Anatomie," 1880, p. 328.

‡ Filehne and Penzoldt, "Centralblatt für die medicinischen Wissenschaften," 1879, pp. 465 and 481.

§ Ludwig, "Zeitschrift für rationelle Medicin," vol. vii, p. 189; "Lehrbuch der Physiologie," second edition, vol. ii, p. 83.

the theories which deal with the movement of the blood as it leaves the heart and enters the large vessels.

These theories are untenable for two reasons: in the first place, the apex-beat is produced even when the heart is empty, and, in the second place, it begins long before the blood leaves the heart, that is, during the so-called closure or tension period of the systole, as was pointed out by Landois* and Martius.† The time occupied by the bulging of the apex coincides in part, but only in part, with this period of closure or tension, and the determining factors in the production of the apex-beat are, in the first place, the contractile induration of the heart and, in the second place, the position of the apex above the center of the base without any change in the longitudinal diameter.

The fact that the apex-beat is dependent, on the one hand, upon the position and action of the heart, and, on the other hand, upon the position and action of the lungs, explains a good many variations in its position and behavior. In the first place, it may be altogether absent in healthy individuals, either because the layer of lung overlying the apex is unusually thick or because the apex-beat comes in relation with a rib instead of an intercostal space; or, finally, because the longitudinal axis of the heart, even in diastole, occupies such a position that the apex is not forced downward.

In is often of advantage in determining the position of the apex-beat to make the examination with the trunk bent over forward. In this position the heart falls forward and downward, the edges of the lung recede, and the apex-beat either becomes more distinct or may be detected when it was apparently absent before.

As the position of the heart changes with that of the body, the apex-beat is often found to be displaced to the left when the individual lies on the left side. The degree of displacement to the left is usually slight‡—a few centimeters—in most healthy individuals; in some cases, however, the displacement is so great that the term "wandering heart" is justifiable.§ It is probable, however, that in such extreme cases the conditions are not normal and that the extreme mobility of the heart depends on great loss of intrathoracic fat (starvation cures, tuberculosis). In these cases the cardiac displacement causes more or less distress when the patient lies on his side, possibly on account of traction on or kinking of the large vessels.

These questions have recently received much attention.‖ In nervous persons abnormal mobility of the heart appears to be not uncommon and causes peculiar sensations. A comparison with movable kidney naturally suggests itself.

When the position of the diaphragm is high and the heart is abnormally movable, so that a larger portion of its area comes in contact with the chest-wall, a dilatation may be simulated. Hoffmann believes that

* Landois, "Lehrbuch der Physiologie," seventh edition, p. 92.

† Martius, "Zeitschrift für klinische Medicin," vol. xiii, p. 478.

‡ See Livierato, "Centralblatt für klinische Medicin," 1891, p. 201. Pick, "Wiener klinische Wochenschrift," 1890, No. 39, 40. Tausyk and Vas, "Wiener medicinische Presse," 1891, No. 29, 30. Ref.: "Schmidt's Jahrbücher," p. 193.

§ Rumpf, "Congress für innere Medicin," 1888, p. 221.

‖ A. Hoffmann, "Acute Herzdilatation und Cor mobile," "Deutsche medicinische Wochenschrift," 1900, No. 19. *Ibid.*, "Wiener medicinische Wochenschrift," 1899, No. 12, 13. Determann, "Ueber die Beweglichkeit des Herzens," "Deutsche medicinische Wochenschrift," 1900, No. 15.

many cases of so-called acute dilatations are in reality only examples of this condition, that is, an unusually large portion of the heart coming in contact with the chest-wall. Error can be avoided by testing the mobility of the organ.

In the right lateral position the apex-beat in healthy individuals either remains unaffected or is displaced only a very little to the right.

The change from the horizontal to the vertical position and vice versâ has no appreciable effect on the apex-beat.

In examining for adhesions it is sometimes useful to remember that the apex-beat is displaced upward during forced expiration and downward during forced inspiration.

Whenever the apex of the heart is displaced, providing the conditions for the production of an apex-beat are present, the latter is found in an abnormal position. Hence, displacement of the apex-beat is utilized in the first place for recognizing a dislocation of the heart. With this we have nothing to do here, because dislocation of the heart is caused by diseases of the lungs, of the pleura, or of the thoracic cage.

If the apex-beat is displaced and the position of the heart itself is normal, it indicates a change in the size of the heart, especially of the left ventricle.

It is true that there is very rarely a distinctly noticeable displacement of the apex-beat to the right when the heart is diminished in size; but that is because, under normal conditions, only a small portion of the left ventricle enters into the formation of the anterior surface of the heart. Hence it may atrophy to a certain extent without necessarily producing any material change in the outline of the anterior surface of the heart, more particularly as in many of these cases the right ventricle is enlarged. Generally speaking, this point has but little practical importance.

Much more commonly we see the apex-beat displaced to the left beyond the nipple-line in dilatation of the left ventricle; sometimes it is found beyond the middle axillary line, and not infrequently it is displaced downward to intercostal spaces lower than the fifth. In the great majority of cases the cause really is a dilatation of the left ventricle; but occasionally a normal left ventricle is displaced by an enlarged right ventricle, or the enlarged right ventricle may alone form the entire apex of the heart and extend toward the left, so that the small left ventricle practically becomes merely an unimportant appendage of the right. This possibility must be borne in mind in every case, as it is only by carefully weighing all the conditions entering into the problem that error can be avoided.

Dislocation of the apex-beat downward as well as outward depends partly on the position of the heart and partly on the kind of enlargement that is present.

Not infrequently the pulsation of the heart, instead of being seen only at a circumscribed spot, is diffuse; that is to say, the apex-beat is broader than normal, and pulsating movements appear in other intercostal spaces besides the fifth. This condition may be present in the absence of any abnormality of the heart or its activity, as it may be due to shrinking and retraction of the edges of the lung. It is seen not infrequently in disease of the left upper lobe, which can be readily detected by percussion and auscultation, thus making it possible to guard against the error of assuming that the heart is abnormal.

Diffuse pulsation in the precordial region is also observed, however, in many cases of enlargement of the right or of the left ventricle, from pure

dilatation or hypertrophy, or a combination of both. The phenomenon depends partly upon retraction of the edges of the lung, partly on a large area of contact between the heart and chest-wall. The former condition is present in any case of enlargement of the heart; the second depends on certain factors our knowledge of which is as yet imperfect. It is certain, at least, that abnormally wide-spread pulsation is not seen in all cases of dilatation or hypertrophy of the heart. Up to a certain point this is not difficult to understand. Ludwig* long ago showed that the base and cross-section of the heart in diastole have an oval outline, which is gradually transformed approximately into a circle during systole. In certain positions of the heart, therefore, bulging must take place in the intercostal spaces; and the more readily, of course, the greater the displacement of the lung. The occurrence and extent of the pulsation, therefore, depend on a variety of conditions: position of the edges of the lung, position of the heart, and change in the shape of the heart. A great variety of different combinations may conceivably be present in individual cases.

Whenever several pulsations due to the action of the heart are seen on the left side of the thorax, the one which occupies the lowest and outermost position is to be regarded as the apex-beat; hence in every case careful palpation of the precordial region is necessary.

The apex-beat also affords accurate information in regard to the character of the heart action.

I shall not here refer to frequency and rhythm, although these two elements can also be very readily determined by inspection and palpation of the apex-beat; but the information obtained by auscultation is at least equally reliable; and, besides, these two aspects of the cardiac activity will be discussed in a separate portion of this work. In many cases, however, the strength of the cardiac contractions, especially those of the left ventricle, can be directly determined by the strength of the apex-beat. An idea of the intensity of the ventricular contractions is obtained by general clinical deductions, such as the consequences of increased or diminished cardiac action that manifest themselves in various organs. The strength of the heart is also determined by auscultation of the second sounds at the base, a point that will be discussed more in detail later. So far as the left ventricle is concerned, the most important factors undoubtedly are the arterial pulse and the apex-beat, which must, therefore, be carefully studied.

The arterial pulse in turn depends on two factors: the condition of the vessels and the action of the left ventricle. As the character of the apex-beat depends directly upon this one factor of the action of the left ventricle, it is manifestly of the greatest importance in judging the strength of the heart as a whole. There are certain factors, however, that must be rigorously borne in mind.

The *resistance of the apex-beat*, as measured by the force required to obliterate it, is a direct and *reliable sign of the strength of the heart* in the same individual at various times, provided the position and size of the left ventricle have not changed.

It is incorrect to take the force of the apex-beat as the measure of the strength of the cardiac contractions under all circumstances, especially without regard to the two above-mentioned conditions. Such a method of reasoning is not permissible because it follows, from the above explanation of the mode of production of the apex-beat, that the position of the

* *Loc. cit.*

heart has an important influence on the strength of the apex-beat, while the position of the heart again to a very large extent depends on its size. This point has been repeatedly and justly emphasized by Martius. When the heart is dilated, the apex may, on account of its altered position, be displaced during diastole farther than usual from its position above the center of the base, and, consequently, although the contractions of the muscle may be relatively weaker, the apex may enter the intercostal space with greater force than in the case of a smaller heart which may contract more vigorously. One may say, therefore, that a portion of the force of the cardiac contraction, which varies with changes in the position and size of the heart, manifests itself in the apex-beat. Unless these two elements remain unchanged, it is not justifiable to assume that aliquot parts of the force of the heart appear in the apex-beat. When that is the case, the resistance of the apex-beat affords a direct means of measuring the force of the heart.

Hence, provided the above-mentioned conditions are fulfilled, we have in the resistance of the apex-beat a most valuable means of determining the strength of the left ventricle in the same individual at different times.

In a non-dilated heart, even if its normal conditions are not known, a greatly resistant apex-beat is practically a certain sign that the strength of the heart is increased.

When the heart is dilated, however, great care is necessary, because under such circumstances an abnormally resistant apex-beat, for reasons mentioned above, occurs even when the heart action is weak. Martius lays great stress on this point. But even in cases of this kind the apex-beat, when continuously observed for the purpose of noting its changes, affords a most valuable means of judging the strength of the heart.

When the apex-beat is examined for the purpose of determining the strength of the heart, an additional point of some importance is the estimation of the blood-pressure—the hardness of the pulse. The association of a resistant apex-beat with a hard (high tension) pulse is a positive sign that the heart action is increased, and very important in the present connection, for we may disregard cases of valvular lesions causing increased activity of the left ventricle without increase of the blood-pressure, and it is rare to see a left ventricle vigorously propelling its contents into abnormally relaxed vessels.

The question as to when an increase in the resistance of the apex-beat indicates merely increased heart action and when it may be taken as a sign of hypertrophy of the muscle will be discussed in connection with cardiac hypertrophy.

I must remind the reader once more that the above remarks apply solely to the quality of the resistance in the sense defined above. The best way to test it is by applying one or two fingers to the site of the apex-beat and attempting to obliterate it, at the same time measuring the force necessary to do so. The method is valuable only in the hands of experienced observers and, on account of its importance, necessitates much careful practice.

Abnormal weakness of the heart's action is very difficult to determine by observing the apex-beat.

Such a deduction is perfectly justifiable, however, if an apex-beat the resistance of which is known to the observer becomes softer without any material change in the position or size of the heart.

When, after dilatation of the left ventricle, the apex-beat is found to be high but abnormally soft and the general clinical picture justifies the assumption of hypertrophy of the left ventricle, and when, as appears from the height of the apex-beat, the conditions for the production of a vigorous apex-beat are present—under such conditions a diminished resistance may confidently be regarded as a sign of weakness of the left ventricle, particularly if, at the same time, the pulse is soft and indicates a low arterial pressure.

On the other hand, it is not justifiable to assume that the heart's action is abnormally weak merely because no visible signs of cardiac activity are present in the chest-wall. While the cause may reside in diminished intensity of the heart action, it may also be that the intercostal spaces are abnormally narrow, that the apex during diastole is considerably above the center of the base, that the apex is hidden behind a rib or an abnormally thickened layer of lung, or is forced away from the chest-wall by pericarditic fluid and is only on that account unable to enter the intercostal space. It is needless to say that edema or emphysema of the chest-wall may have a similar effect.

The *height of the apex-beat*, by which is meant the length of the excursion of the apex as it bulges forward, should also be observed. Here again I may remind the reader of the characteristics of the arterial pulse and what we mean by the terms "height" or "size," which we carefully distinguish from "resistance."

A high apex-beat is produced whenever the conditions for a marked systolic displacement of the apex are present; that is to say, when the intercostal spaces are broad and the long axis of the heart is applied to the left wall of the thorax in such a way that during diastole the apex is displaced relatively far backward from its systolic position. Thus even healthy persons may, owing to individual conditions, exhibit an abnormally high apex-beat. The phenomenon is most frequently produced, however, when there is dilatation of the ventricles, especially of the left, without any change in the usual direction of the long axis of the heart, because under such conditions the backward movement of the apex during diastole is specially marked and the systolic excursion is accordingly also quite considerable. Thus, a high apex-beat is observed in cases of dilatation of the left ventricle, and, as Martius* has quite correctly pointed out, the phenomenon is not by any means a sign that the left ventricle is acting vigorously. A high apex-beat may be associated with weakness of the heart and may then even be extremely resistant in the sense defined above. But not infrequently in cases of cardiac weakness the apex-beat, although high, is soft; at all events it is less resistant in the same individual than it was before the heart began to weaken. Systolic pulsation of the entire precordia has the same clinical significance as a high apex-beat, with which it is often associated along with diffuse pulsations in other intercostal spaces. Concussion of the precordia may, however, be present when the apex-beat is not visible at all, that is, even when the lung covers a considerable portion of the heart. It may be produced by increased action and hypertrophy of the ventricles, especially of the left. The phenomenon is also observed when the heart is very large, even if the cardiac contractions are feeble. For an explanation of the phenomenon we refer to what has just been said about the "high" apex-beat.

* Martius, "Volkmann's Vorträge," N. F., No. 113.

Finally, the *duration and shape of the apex-beat* must be noted. The information obtained by the purely clinical methods of inspection and palpation is comparatively meager; for, while it may be possible to determine by ordinary palpation whether change in the shape and position of the heart has taken place very rapidly or very slowly, as, for example, in stenosis of the aorta, finer details can only be ascertained by means of the graphic method. The latter consists in a mechanic contrivance which follows the movements of the apex with the greatest accuracy and without being exaggerated by its own vibrations, recording the movements on a revolving drum (see Cardiography).

When the action of the right ventricle is increased, particularly by hypertrophy, diffuse and more or less vigorous pulsation is frequently visible in the third, fourth, and fifth intercostal spaces to the left of the sternum. These pulsations may be due to the systolic change in the outline of the cross-section of the heart—that is, its transition from an oval to a circle. The more vigorously this change is effected, the greater the bulging of the intercostal spaces, especially when the anterior border of the left lung is at the same time displaced.

In the highest portion of the epigastrium pulsation is frequently observed, which must be attributed to concussion by the right ventricle. It may be present in perfectly normal individuals, and its production is favored by a low position of the diaphragm and enlargement of the right heart. This "*epigastric pulsation*" is not to be confounded with pulsating movements also occurring in the epigastrium, but produced by the abdominal aorta and not directly due to the action of the heart. These pulsations, as a rule, are not found above, at the apex of the epigastric angle, but occupy a position a few centimeters lower and usually a little to the left of the median line. The fact that in some individuals the pulsation of the aorta is visible at this point is probably due to lordosis (forward bending) of the vertebral column; it is somewhat remarkable that the phenomenon is observed chiefly in nervous individuals, who themselves feel the throbbing and are distressed by the sensation. It is almost needless to say that this form of pulsation is greatly increased by aneurysmal dilatation of the abdominal aorta—an extremely rare occurrence—and by tumors overlying the vessel. If any difficulty is experienced in distinguishing these aortic pulsations from true epigastric pulsation, it must be borne in mind that the former are separated by a perceptible interval of time from the apex-beat.

Pulsations not dependent on the action of the heart itself are occasionally observed in the region of the large arteries, particularly in the second intercostal space, to the right or the left of the sternum. Such pulsations are produced when the trunk of the aorta or of the pulmonary artery, whether the vessel be normal or in a state of dilatation, has pushed the corresponding lung aside and throws parts of the chest-wall into vibration, either directly or through the medium of some solid body, such as a tumor or infiltrated pulmonary tissue. Pulsation to the right of the sternum is almost always due to a dilated aorta or to aneurysmal change; pulsation to the left of the sternum is frequently produced by the pulmonary artery. In the latter case the immediate cause usually is exposure of the vessel by some process in the left upper lobe. These pulsations are, of course, a little later in time than the first heart-sound or the beginning of the apex-beat, and the difference in time can sometimes be determined; frequently, however, the interval is so short that it is not appreciable by

the unaided senses. If the fingers are applied to the upper intercostal spaces during the entire duration of the cardiac cycle, a shock is not infrequently felt at the beginning of ventricular diastole and especially over the region of a pulsating pulmonary artery. This vibration is due to movements associated with the closure of the semilunar valves—"one feels the closure of the valves." This is particularly the case in the presence of conditions increasing the pressure in the pulmonary artery and thus intensifying the closure of the semilunar valves.

In not a few cases of heart disease there is a palpable thrill in the precordial region, and important conclusions may be drawn from its character and the time of its appearance. But as the significance of these things is almost exclusively confined to valvular lesions and pericarditis, the reader is referred to the respective sections in this work.

Cardiography.*—In a great number of normal individuals the movement of the heart as shown by the apex-beat is susceptible of direct observation. The information which the physician can gain by examining the apex-beat without the aid of special instruments has been discussed. Early in the history of the development of the graphic method an attempt was made to study, by means of a recording instrument, the character of the movement of the heart as manifested at the bulging point in the intercostal space. It was hoped that in this way a more minute knowledge of the action of the normal and of the diseased heart could be obtained.

The cardiographic curve is the product of a number of factors: changes of pressure within the cavities of the heart, changes in the form and position of the surface of the organ, and movements of the lungs and of the chest-wall. It is seen, therefore, that a number of independent factors take part in this production.

As we have seen, the apex-beat is produced during the change that takes place in the shape of the ventricles during systole. For reasons explained on page 428 the apex of the heart, or a part of the right ventricle near the apex, is forced into the intercostal space. But there is no guarantee that identical portions of the surface of the heart enter the intercostal space in exactly the same way in different individuals; and the uniformity certainly ceases when the size or position of the heart undergoes a change. Now it has been found that the movement varies in different portions of the surface even of the same heart; that is to say, the different portions yield different cardiograms† even when the curve is taken directly, without the intervention of the chest-wall. In addition, the force with which the corresponding portion of the surface of the heart is forced into the intercostal space varies greatly in different individuals; for it has been shown that that portion of the force of the heart which appears in the apex-beat is not the same in different individuals and under different circumstances. There are also great variations in the rigidity of the soft parts connecting the ribs, changes in their elasticity due to respiration, and, finally, variations in the movement of the lung over the heart. In short, a number of factors tend to diminish somewhat the value of a cardiographic investigation of the more minute peculiarities of the movements of the heart.

Another point is important: the forces that are called into action during the bulging of the intercostal space are often quite considerable, and the apparatus used for recording the movements must, therefore,

* For the literature see the end of the article.

† v. Frey, "Die Untersuchung des Pulses."

be designed to record the most rapid and extensive changes in form with absolute faithfulness. This point has not received sufficient attention in most recording contrivances, which accounts in part for the numerous differences in the apex-beat curves found in the literature. The recording instruments fail to give a faithful reproduction of the changes in the shape of the chest-wall because the tracing is affected by oscillations of the instrument itself due to inertia. This is clearly shown in Hürthle's* exposition of the subject.

However, this accounts for only part of the differences,† and they can be avoided by the prudent use of suitable apparatus. The differences in the cardiograms obtained from different individuals are in part due to the action of the many factors that influence the movements of the chest-wall and which were enumerated above. In other words, even with the best recording apparatus the curve obtained in healthy individuals is not uniform; it does not present a normal type. This, so far as I can see, may be stated positively, as it not only tallies with numerous statements found in the literature,‡ but also coincides with numerous experiments of my own. In 1887 I recorded numerous cardiograms of individuals in health and in disease, and on the advice of my friend, M. v. Frey, used a double-armed lever,§ which had been carefully tested with reference to its power of recording the movements of a given point. I could not discover any definite type of curve for the apex-beat. As in the use of curves the form is the most important feature, this absence of a distinct type is a great disadvantage of cardiograms and renders their interpretation difficult. In fact, v. Frey and myself discarded the curves at the time because we could not draw any conclusions from them. The difficulty is equally as great in pathologic as in normal conditions; a definite anomaly in the cardiac activity does not produce a definite type of curve. These few words show the state of the question.

Nor, in my opinion, is anything gained by, at the same time, recording certain definite factors in the heart's action which can be positively determined; it has no effect on the shape of the curves, and certainly does not obviate the difficulty that the shape of the curve depends on several different and indeterminable factors.

Thus, the carotid pulse may be recorded at the same time for this purpose. If this is done, the moment when the aortic valves open can be found, since the velocity of the arterial pulse is determinable. Or the time of the first and of the second heart-sound may be recorded, which gives the beginning of ventricular contraction and the time of closure of the semilunar valves. These data are obtained by auscultation and registration of the heart-sounds—a method employed by Donders, Marey, and Landois, and chiefly elaborated by Martius.‖ The latter also discusses the theoretic questions involved. The operator makes his records in time with the sounds as he hears them and *not after* he has received the auditory impression; this is an important fact and represents both the strength and the weakness of the method, because the irregularities in the action of the heart so frequently present even in healthy individuals render delicate measurements less reliable than would be the case if the

* Hürthle, "Pflüger's Archiv," vol. liii.

† The mechanic requisites for such an instrument are accurately known (see the works of v. Frey and Hürthle).

‡ See, for example, v. Frey, Hilbert, Hochhaus, Martius, A. Schmidt.

§ *Cf.* v. Frey, "Die Untersuchung des Pulses."

‖ "Zeitschrift für klinische Medicin," vol. xv.

recording represented merely a reaction to the auditory impression. Hürthle,* Einthoven and Geluk† succeeded in recording the heart-sounds by mechanic means.

The technic of the procedure employed by the three last-mentioned authors is complicated, but by its aid the time relations of the heart-sounds can be determined with absolute accuracy. It is my opinion that even Martius' procedure affords perfectly adequate data for the decision of a number of questions. But I hardly think that the recording of certain phases of the cardiac cycle is destined in the near future to increase our ability to interpret the individual features of the cardiogram; for I see little prospect of this until we learn to recognize and control the conditions which determine the shape of the curve. On the other hand, it is not unreasonable to hope that the careful and circumspect employment of methods for analyzing the purely temporal relations of the cardiac cycle may yield data of some importance, and I believe the method of Martius is quite adequate in a great many respects because the sources of error are not so great as to render the determination of intervals of time valueless.

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* "Pflüger," vol. lx. † "Pflüger," vol. lvii.

PERCUSSION OF THE HEART.

The indirect method is now exclusively used for the percussion of the heart. Whether digital percussion alone, or a pleximeter, either with the finger or with a plexor for a hammer, is used, the result will probably be the same in most cases. Custom and practice unquestionably decide the choice of method in the individual case. Personally, I prefer to use a

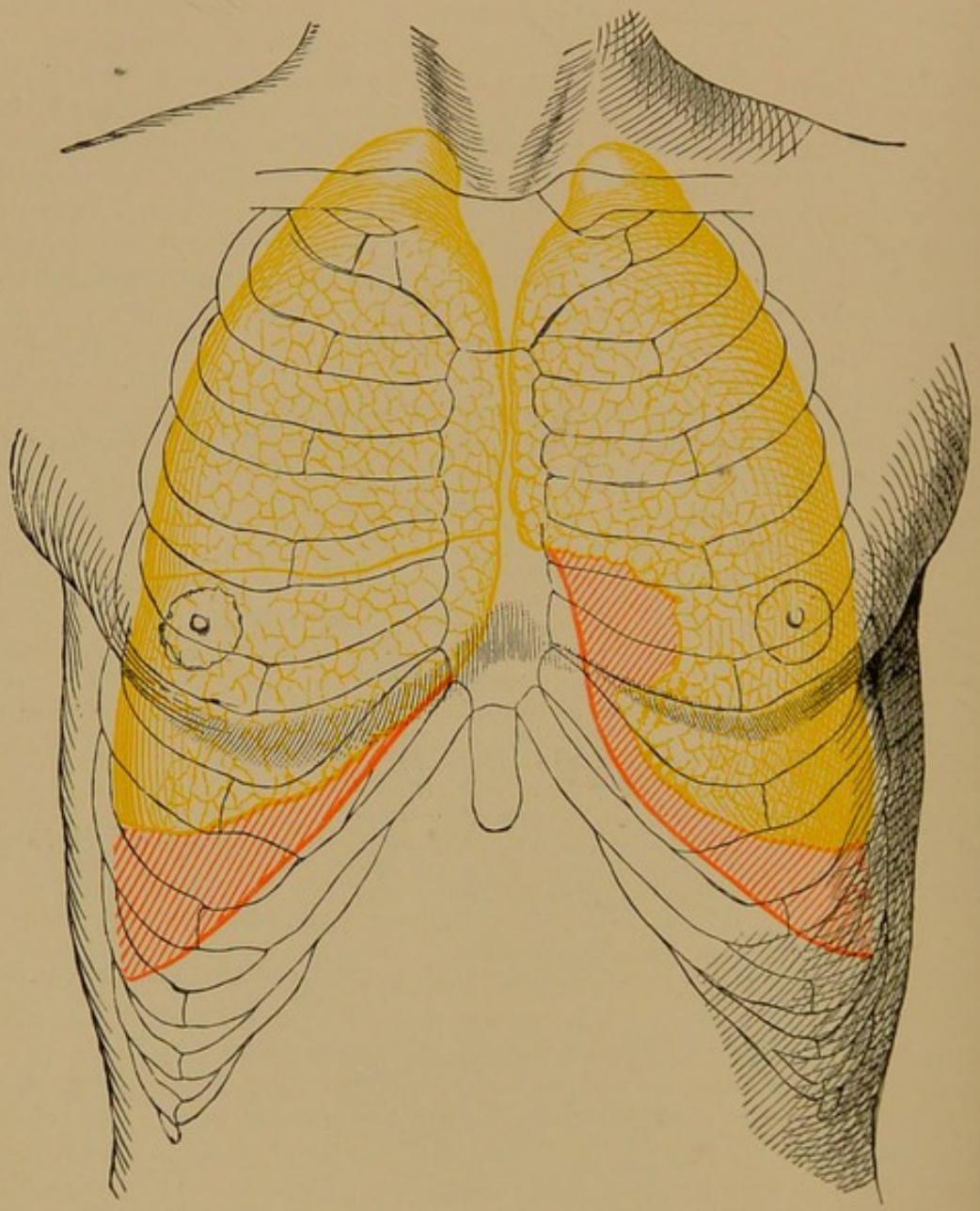

FIG. 79.—The outlines of the lungs and pleura on the anterior surface of the chest-wall, after Merkel, "Handbuch der topographischen Anatomie," ii, Fig. 122.

light hammer and thin pleximeter for the accurate determination of the pulmonary and cardiac outlines, rather than digital percussion. Every physician, however, must, above all, be able to examine without any instruments whatever in order not to become the slave of his instruments. As the terminology of percussion nowadays is very much dominated by the personal equation, it is incumbent upon an author, if he

wishes to be understood, to indicate what significance he wishes to have attached to the expressions he uses. Personally, I follow the

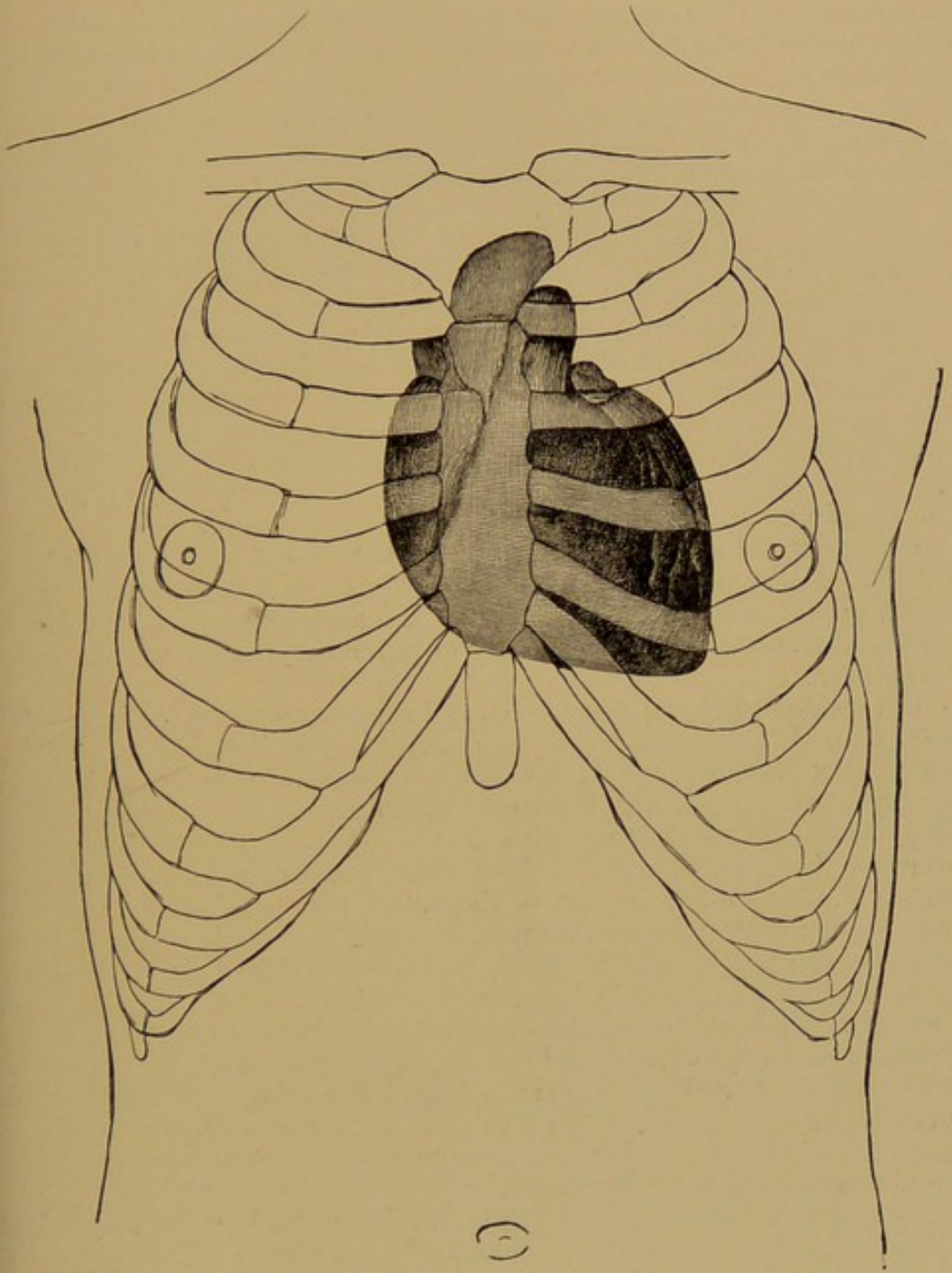

FIG. 80.—Relations of the heart and its divisions to the anterior surface of the thorax, after Merkel, "Handbuch der topographischen Anatomie," ii, Fig. 95 (diagrammatic).

time-honored expressions used by Skoda,* and somewhat modified by Weil in his excellent text-book.† The pulmonary note is described

* Weil, "Handbuch und Atlas der topographischen Percussion," Leipzig, 1880. See the excellent treatise on the subject by Matterstock in the "Würzburg Festschrift," Leipzig, 1882, vol. ii, p. 247. Biernacki, "Zur Revision einiger Capitel der physikalischen Diagnostik," "Volkmann's Vorträge," N. F., No. 154, 155.

† Skoda, "Abhandlung über Percussion und Auscultation," fifth edition, Vienna, 1854.

as resonant and the sound obtained over airless (solid) organs dull or flat; a decrease in the resonance of the percussion-note is described as "relatively dull."

Percussion is used to determine the position and size of the heart and of its separate parts. Both hearing and touch are utilized in arriving at a conclusion.

The outlines of the heart can be determined to the right, above, and to the left by means of percussion because at these points the resonance of the air-containing lung contrasts with the dulness of the heart. Below, where the heart is bounded by the solid liver, the outline can, of course, not be determined by percussion.

The size of the heart can be determined by means of percussion only when the lung contains air; the cardiac dulness cannot be differentiated by means of percussion from areas of infiltration in the lungs or from a pleural effusion.

If the above-mentioned conditions are fulfilled, the size of the heart may be determined by means of percussion in one of two ways: either by determining the entire (largest) outline of the heart projected on the anterior chest-wall, or by determining the size of a portion of the heart which possesses certain definite characteristics and deducing from the changes in this portion of the heart the changes that are present in the entire heart or in certain other portions.

Whatever method be adopted, only the size of that portion of the heart which is turned toward the anterior chest-wall can be determined; for the entire posterior surface of the organ is inaccessible to examination on account of the overlying thick layers of pulmonary tissue and the vertebral column.

The right auricle, right ventricle, and a narrow strip of the left ventricle take part in the formation of the anterior surface of the heart. The accompanying illustration (Fig. 80), which we have borrowed from Merkel's excellent "Handbuch der topographischen Anatomie" (Braunschweig, 1896), shows that the right ventricle forms by far the greatest portion of the anterior surface of the heart and alone comes in immediate contact with the chest-wall, every other portion of the anterior surface being covered by lung. There is no need to describe in detail the course of the anterior borders of the lung above the heart, because they are so distinctly shown in the illustration. The left lower border of the lung, at the point where it changes its vertical course and is deflected outward behind the sternum, is subject to marked individual variations even in middle-aged persons; for the deflection may evidently take place behind the lower border of the fourth rib or directly above the fifth rib. As the illustration also shows, the course of the pleural borders permits a considerable degree of variation in the boundaries of the lungs.

By means of percussion we first try to determine how large an extent of the heart's surface is in direct contact with the chest-wall, and, secondly, the condition of the thickness of the lung edges that cover a large portion of the organ. In this way two outlines are obtained: the so-called absolute (small) and relative (large) cardiac dulness, and from a change of the outline as a whole or only in any one direction we deduce the existence of changes in the size of the entire heart or of individual portions of the organ.

In determining the **absolute cardiac dulness** our object is to outline that portion of the heart which is in direct contact with the chest-wall. Almost the whole of this area can be determined—all, in fact, but the small portion which is covered by the sternum; for the bone acts approxi-

mately like a pleximeter, at least when the subjacent organs have their normal configuration—that is to say, the bone vibrates as a whole and, therefore, yields a note like that obtained over the organs which occupy the greater portion of its posterior surface, in other words, the lungs. Hence, in a normal individual of middle age one would expect to find the right border of the "absolute cardiac dulness" at the left edge of the sternum, the upper border at the fourth or fifth rib, and the left approximately in the left parasternal line or a little outside of it. The outer boundary will always be more or less variable, because the anterior edge of the lingula is often so extraordinarily thin that it can only be found with very light percussion and then not always with absolute certainty.

In advanced age, when the edges of the lung have expanded, the outline of this cardiac dulness diminishes above and on the left side, and in children up to ten years of age—and not infrequently even longer—the dulness is larger for reasons that are well known; it often begins at the third rib and extends to the left as far as the nipple-line.*

In this discussion the customary, well-known vertical lines, and, in the horizontal direction, the ribs, are utilized for purposes of orientation on the anterior surface of the thorax. Individual peculiarities of these structures must, however, be carefully borne in mind, and if this is done, I believe the results are perfectly satisfactory for all practical purposes.

Others prefer to give absolute measurements. Thus Riess,† for example, determines the distance in centimeters of the boundary of the relative cardiac dulness from the median line in each intercostal space. Although the figures obtained in 200 normal individuals show a very close agreement, I cannot see that any great advantage is gained in this way over the former method.

Bearing in mind the above-mentioned details, if the chest is lightly percussed and the points where the typical, resonant pulmonary note disappears completely are determined, the boundaries of the lungs as determined by this method of examination are found to coincide with the anatomic boundaries except at the above-mentioned point (where the sternum covers the heart).

Both directions must be strictly followed if the boundaries of the absolute dulness are to be found correctly; and two observers, if they will pursue the same method, will obtain identically the same boundaries. It is not a matter of "opinion" or of "taste" or of "practice" where the boundaries of the absolute dulness are to be placed; for the principles which underlie their determination are perfectly clear and rational. If these principles are disregarded, mistakes will be made. Percussion must be linear and very light, because the edges of the lungs at the boundary of the heart are extremely thin. (For a detailed exposition of the reasons for this see any good text-book on methods of examination, as, for example, Vierordt, Sahli, Gerhardt, Weil.)

It is also important to note attentively where the pulmonary note ceases altogether; for these points are, in the case under discussion, identical with the boundaries of that portion of the heart which is not covered by the edges of the lung. It is wrong to wait until the sound is absolutely dull, because, owing to the proximity of the heart to the stomach and intestines, the cardiac note very frequently is not absolutely dull, but merely dull with a tympanic element. It is never resonant, however; hence, if the principles just laid down are strictly observed, the result will always be uniform and positive. The conditions, therefore, are perfectly clear; but we admit that the term "absolute cardiac dulness," which has been generally adopted, is by no means a happy one, for reasons which we have just explained; and many of the numerous other designations, as, for example, "small" (lesser) cardiac dulness would be very much better because they would not be so apt to give rise to misunderstanding.

* See Sahli, "Die Percussion im Kindesalter," v. Starck, "Archiv für Kinderheilkunde," ix, p. 241.

† Riess, "Zeitschrift für klinische Medicin," xiv, p. 1.

It is evident that retraction of the edges of the lung, the result of processes leading to contraction of the organ, and a more extensive apposition of the heart to the chest-wall when the diaphragm is high, must cause an enlargement of this cardiac dulness. But in either case the true cause cannot escape recognition if all the factors in the case are duly taken into account.

As the edges of the lungs change their position with respiration and certain changes in the position of the body, the same factors must influence the absolute cardiac dulness; it is smaller during inspiration and larger during expiration, is increased in the left lateral position and when the trunk is moved forward, and diminishes when the individual lies on the right side; in the latter position a small absolute cardiac dulness may make its appearance to the right of the sternum. Suitable combinations of these factors may lead to considerable variations in the size of the dulness. We purposely give no absolute figures for the extent of these changes in the dulness because individual fluctuations are very great.

In some cases these phenomena due to passive movement can be utilized in making the diagnosis. Thus, as Gumprecht* has pointed out, an enlargement of the left heart in emphysema of the lungs can often be determined from the increase in the width of the absolute cardiac dulness when the body is bent over forward.

In what way may we now infer changes in the size of the heart from variations in the outline of the absolute dulness? Generally speaking, the answer is: when one of the ventricles enlarges it pushes the edges of the lungs, which are normally movable, aside, a larger portion of the heart comes in contact with the chest-wall, and the absolute dulness is, therefore, enlarged. The necessary condition, therefore, is that the edges of the lungs be perfectly normal; retraction of the lungs may simulate an enlargement of the heart-wall; distention (emphysema), on the other hand, may conceal the fact that the heart is enlarged. Another condition is that the position of the heart be normal. For, if the organ as a whole is pushed upward, as so frequently happens when the diaphragm is high, a larger portion comes in contact with the chest-wall, even though the heart itself be not enlarged, and the absolute dulness becomes broader.

In the case of the left ventricle the conditions are perfectly clear. If the ventricle is greatly enlarged, there will be, in the great majority of cases, in addition to the outward and downward displacement of the apex-beat which was mentioned earlier in this discussion, an increase of the absolute dulness which will extend almost or quite to the apex-beat.

On the other hand, the converse of this, namely, that an increase in the width of the absolute dulness to the left is always caused by an enlargement of the left ventricle, does not hold. Although it is true, without exception, in cases of great enlargement, it is far from being strictly true when the enlargement is slight; for a slight increase in width may also be due to an enlargement of the right ventricle, as under such conditions the left ventricle is slightly displaced to the left by the right ventricle.

This is not likely to cause any difficulty in diagnosis because, when an increase in the width of the absolute cardiac dulness to the left is due to dilatation of the right ventricle, the dulness is, at the same time, increased to the right also. It is true that the increase in the dulness does not necessarily show itself in the enlargement of the absolute cardiac dulness, and this is one of the most cogent reasons that induces me always to determine both forms of cardiac dulness. For, if the anterior edge of the right lung

* Gumprecht, "Archiv für klinische Medicin," lvi, p. 490.

is displaced to the right, the displacement first occurs underneath the sternum. In that case the resonant note over the lower portion of the bone is weakened so that relative dulness is obtained at that point. Absolute dulness of cardiac origin, extending to the right of the right edge of the sternum, is only produced when the enlargement of the right ventricle is very great or the right auricle is also enlarged.

An increase of the absolute dulness to the left is frequently associated with a similar increase upward toward the clavicle. This probably depends chiefly on the position of the organ. The absolute cardiac dulness may also be increased by an effusion in the pericardium; in such a case the boundaries are usually displaced in both directions in proportion to the increase in the size of the exudate, producing the familiar triangular outline of dulness. The distinction from enlargement of the dulness due to dilatation can be made only by taking all the symptoms into account, and even then the question presents great difficulties. It is not our province to enter more fully into this question.

So far we have spoken only of enlargement of the heart. Is the increase in the width of the dulness caused by dilatation or by hypertrophy of the chambers? Dilatation is unquestionably the determining factor; for hypertrophy in itself affects the size of the chambers so little, especially as regards their surface projection, that the difference can hardly be recognized by such a comparatively crude method as percussion. Hypertrophy must be determined by other signs, such as the apex-beat, the strength of the second heart-sounds, and the pulse.

Let us add a few practical observations. It is best to begin by determining the upper border of the absolute dulness; the chest is percussed gently from above downward, beginning just outside of the left edge of the sternum, and the point where pulmonary resonance disappears altogether is noted. Percussion is then begun at the point where the absolute dulness was found along the left border of the sternum and carried first to the left and then to the right; or in the opposite direction, from without inward. Which of these two methods is adopted is chiefly a matter of habit. Either way correct results are obtained provided the examiner firmly adheres to the principles given above. He must always be on his guard, however, not to look for the absolute cardiac dulness higher than he may expect to find it. This error may be avoided by percussing from right to left along the lower border of the right lung.

Diminution in the size of the absolute cardiac dulness does not indicate a diminution in the size of the heart as a whole or of its parts; for, if the heart is diminished in size, it merely does not extend so far underneath the edges of the lungs as under normal conditions. On the contrary, the principal cause of diminution in the size of the dulness is an extension of the edges of the lungs, that is to say, emphysema or **volumen auctum.**

In pneumopericardium the absolute dulness disappears; we shall not discuss the subject at greater length because the condition is treated elsewhere in this work.

When the chest is percussed forcibly in the direction from the lungs toward the heart, a change is observed in the sound long before the boundary of the lung has been reached; the note becomes less resonant or relatively dull (**relative cardiac dulness**). The point where this change becomes distinct depends in part on the thickness of the edge of the lung and in part on the force used in percussion. The first factor is the determining one in a given case; the second, it is true, is variable, and that is the reason why two equally competent and equally experienced examiners do not necessarily find the boundaries of this dulness at the same

point. For this reason the difficulties are great, both for the learner and for the teacher.

Nevertheless, these limitations should never prevent us from determining the relative cardiac dulness. For, if the examination is properly made, the possible fluctuations in the results amount to only a few millimeters—a negligible quantity in the ultimate value of the result. On the other hand, a great deal is gained by finding the relative dulness, not so much for the purpose of detecting an enlargement of the left ventricle,—we have other aids for that purpose, such as the position of the apex-beat and the absolute dulness,—but because an enlargement of the right heart practically cannot be determined without finding the relative cardiac dulness.

Just as the enlarged heart pushes the soft yielding edges of the lung aside, so it diminishes their thickness by compressing them against the chest-wall. The combined effect of these two factors is to push the boundaries of the relative heart dulness away from the heart toward the lungs. And this is a valuable diagnostic point in connection with the right heart because, as has already been pointed out, enlargements of the right ventricle can rarely be recognized by other methods, particularly by determining the absolute dulness.

In a healthy adult the upper border of the relative dulness is found somewhere between the third and fourth ribs, the left at the level of the apex-beat, the right either at the right border of the sternum or—and I almost believe usually in perfectly healthy individuals—about 1 cm. to the right of the right edge of the sternum. The right border is given differently by different authors and, indeed, it varies in individual cases. In some individuals the note elicited over the lower portion of the sternum is distinctly less resonant than that over the upper portion; in others the difference is slight, and in some the sound is everywhere the same. Hence a relative dulness over the lower half of the sternum is probably pathologic in some cases, but for obvious reasons the sign cannot be utilized. In old persons with emphysema of the pulmonary edges the relative dulness is smaller, and in children up to the tenth year or even older it is larger than normal. In children the dulness regularly extends a finger-breadth to the right of the right border of the sternum, attains the position of the apex-beat, and often begins at the second rib.

When the right heart is enlarged, however, the anterior border of the right lung is displaced to the right and then a more extensive and more intense relative dulness is very frequently found to the right of the sternum. Thus an increase in the size of the relative dulness to the right most frequently indicates enlargement of the right heart. But here again the conditions are not entirely in accord with theoretic rules; for cases are not infrequently encountered in which a much enlarged left ventricle displaces the right heart to the right and thereby causes an enlargement of the relative cardiac dulness in that direction.

To determine the limits of relative dulness deep percussion is required, beginning over the lungs and approaching the heart in various directions, and marking the points where the first change from the pulmonary note is heard. As in finding the absolute dulness, it is best to begin along the left border of the sternum and then proceed from right to left along the lower border of the lung. The term "deep" percussion in this connection must be taken relatively in comparison to the degree of force used in finding the absolute dulness, which requires the lightest possible percussion. One should never use sufficient force to make the procedure painful to the patient.

Utilizing the Sense of Resistance.—In determining the absolute and relative cardiac dulness we base our judgment chiefly on the auditory impressions received; but every expert examiner will, at the same time, take account of pressure and tactile sensations, as they constitute an integral factor in the make-up of his opinion. That is why the impressions gained by one's own percussion are much more distinct than those obtained merely by listening to another.

Ebstein has elaborated the determination of the sense of resistance into a special method of examination.

Before him various attempts had been made to find the true outline of the heart by means of the sense of resistance; Piorry, and from time to time Traube, laid great stress on the sense of resistance. But Ebstein was the first to evolve an actual method from these first efforts.* He percusses either with the finger or a hammer and glass pleximeter. When a hammer is used, the index-finger of the right hand may be kept on the head of the hammer. Light percussion should be employed and the examiner should concentrate his attention on the sense of resistance as he percusses from the nipple-lines toward the median line on both sides, and from above down toward the precordial region. The point where increased resistance is felt indicates the boundary. With this method the outline obtained in a healthy adult male, either in the dorsal or in the erect position, extends about two to three centimeters to the right of the right edge of the sternum. To the left the boundary in women extends, on the average, 7.3 centimeters beyond the left edge of the sternum, while in men it is safest to assume that it never extends beyond the left nipple-line and very often the left boundary lies just within the nipple-line. Instead of giving the distance of the borders of the resistant area from the edges of the sternum, the measurements may be given from the median line; if this be done, one-half the width of the sternum must be subtracted to make the figures tally with those given for the other method.

Ebstein maintains that he has proved, by numerous experiments on the cadaver, that he has learned by practice to determine the true outlines of the heart by means of his method to within one-half a centimeter. I have satisfied myself that the method is easy to learn and to teach, and unquestionably deserves to be recognized and employed more than it has been so far.

[In "Die Tastpercussion," Stuttgart, 1901, Ebstein has given an account of the method and its application to the diagnosis of various organs. He practises direct as well as mediate palpatory percussion, in the latter employing a rubber-tipped thimble of his own invention instead of a hammer. Those who have not thoroughly tried palpatory percussion can gain much by the perusal of Ebstein's articles.—Ed.]

Critical Review of the Methods Described.—Ebstein claims that, by means of his procedure, the size of the heart can be directly determined. Large deposits of fat and edema unquestionably render its application difficult; emphysema of the lungs interferes but little, and I agree with Ebstein that one who has thoroughly mastered the technic can gain very

* Ebstein, "Berliner klinische Wochenschrift," 1876, No. 35, and 1894, No. 26 and 27. Lüning, "Dissertation," Göttingen, 1876. Schläfke, "Dissertation," Göttingen, 1877. Hornkohl, "Dissertation," Göttingen, 1887. Busse, "Dissertation," Göttingen, 1888. Ebstein, "Archiv für klinische Medicin," pp. 275, 392. Burgtorf, "Dissertation," Göttingen, 1897. Hafke, "Dissertation," Göttingen, 1892.

accurate information by its means, at least in determining the size of the heart.

However, the same claim may be put forward in favor of the method of examination by which the absolute and relative cardiac dulness are obtained. This method also meets with difficulty in various anomalies of the chest-wall (accumulation of fat, edema, deformities) and of the lungs (emphysema, contraction of the left upper lobe). Aside from these conditions, however,—and even when they are present, a careful weighing of all the factors in the problem, as a rule, leads to a correct conclusion,—it is, nevertheless, possible, by determining the absolute and the relative cardiac dulness, to gain a very correct idea of the size of the heart and its several parts. The value of this method is not diminished by the fact that one only determines the size of portions of the heart; for I am firmly convinced that it is possible to draw accurate conclusions in regard to variations in the size of the heart as a whole and of each individual ventricle from the information thus gained. The values are not absolute, but an approximate estimate is obtained which, in actual practice, is perfectly adequate in the majority of cases.

In speaking of the relative dulness reference was made to the subjective character of the boundaries obtained; but even this limitation does not, in my opinion, detract from the value of the method in determining the size of the heart. This is clearly proved by the great differences in the method employed by excellent investigators and diagnosticians in determining the boundaries of the heart. Many of the keenest observers have their own, so to speak, personal way of percussing the heart, and these individual differences in technic result in the production of mixed forms of cardiac dulness gained partly according to the rules for determining the absolute, and partly according to those for finding the relative, dulness. Again, the observers base their judgment partly on tactile and partly on auditory impressions and I am sure the examiner himself is not quite clear to what extent one or the other of these factors enters into the result. No other examiner would find a heart dulness of identically the same shape, nor could the first examiner teach his method of determining it to any one else because he does not proceed according to clearly defined principles, and yet his judgment in regard to the size of the heart may be eminently correct. The modern disciple calls this "artistic intuition." Our own more sober opinion is that, by adhering to a method which is definite so far as he himself is concerned, and is, therefore, uniform, a keen observer capable of correlating all the different phenomena is enabled to deduce from his subjective conception of the size of a certain portion of the heart, which, however, is based on a uniform technic, the variations in the size of the entire organ and of each individual ventricle.

I do not wish to be misunderstood: I am the last one to advocate arbitrary methods in percussing the heart. On the contrary, I am strongly in favor of a strict observance of the rules discussed above. By adhering to these rules it is possible to obtain uniform judgments which can be clearly demonstrated and can be imparted to others; and I wish German internists would unite in the interest of medical instruction on the method and terminology to be employed in determining the size of the heart. [This pious wish might well be extended to include all nationalities.—Ed.]

It appears, therefore, that it is not possible, by determining the absolute and the relative cardiac dulness, to find an absolute measurement of

the size of the heart: nevertheless, the methods afford information which in most cases, suffices for the needs of the physician as to whether the heart or its individual portions are enlarged or not, and whether the enlargement is to be regarded as a slight, a moderate, or a great enlargement. This, as a rule, suffices for the physician; and the final verdict in regard to the utility of the method must depend on whether it accomplishes the purpose which it is intended to accomplish. So far as I can see, this purpose is accomplished by determining the absolute and relative cardiac dulness, and the reason why, in my opinion, Ebstein's excellent method has not been more extensively adopted is that the older procedure approximately satisfies the physician's needs. All the same, an easy and absolutely certain method of determining the actual size of the heart, and unanimity on the part of teachers in regard to the methods to be employed, would constitute a real advance.

Unless I am mistaken, the use of the Röntgen rays to examine the size of the heart in the living subject is really destined to enlarge our knowledge. Already experiments of considerable value are available.*

In my opinion these experiments have led to the most interesting results. I need only refer to the illustrations in the beautiful and careful work of Moritz. The small size of the projection outline of the heart in the living subject fills one with astonishment. The true shape of the heart in these illustrations really corresponds quite closely with the outline obtained by Ebstein's method, and with the outlines of the relative cardiac dulness as I frequently find them in healthy individuals. After seeing Moritz's beautiful work on this subject, which I received after finishing this section, I was confirmed in the hope that, by means of palpatory percussion of the relative dulness as indicated by Ebstein, we shall find out, at least approximately, if not accurately, the true size of the heart, and, as a result, a unity of method in regard to this subject, so important and difficult for all clinical teachers, will be achieved.

[Since the above was written a great deal has been added to our knowledge on the subject, the details of which must be sought in works on diagnosis and *x*-rays. It may be said here that both fluoroscopy and röentgenography are valuable aids to the study of the heart, but do not replace or lessen the importance of the physical methods of exploration, and in many cases they do not add to what may be learned by inspection, palpation, percussion, and auscultation. In some obscure cases *x*-rays reveal enlargement or forms of enlargement not made out by other methods, but, owing to the possibility of distorting the heart shadow, such changes can only be accurately demonstrated by operators with special experience, and usually only with the aid of a diaphragm and skiagraph. Orthodiagraphy, however, has two important sources of error: one, technical difficulties not yet cleared up by operators; the other, more serious, leading to the belief that the absolute size of the heart is to be sought. We know that this is less important than an approximate knowledge, such as can be gained by older methods, and the ability to discover changes from day to day. *x*-Rays can rarely be used for this essential purpose. The fluoroscope is valuable in enabling one to recognize abnormal pulsations, such as dissociation of the auricles and ventricles, or of the two sides. —Ed.]

* Compare v. Criegern, "Congress für innere Medicin," 1899, p. 298. Moritz, "Münchener medicinische Wochenschrift," 1900, No. 29.

AUSCULTATION.

Auscultation of the Heart.—Indirect auscultation by means of the stethoscope is generally employed. It has the advantage over immediate auscultation with the ear that it enables the examiner to listen to a smaller, more circumscribed area on the chest-wall. It is, however, desirable that every physician should be able to form at least a general opinion in regard to the quality of the heart-sounds from what he learns by direct auscultation, so that on important occasions he may not be quite helpless without his instruments.

The kind of stethoscope used appears to me quite immaterial. As a rule, every man hears best with his own stethoscope, however it may be constructed, because he is used to it. Binaural stethoscopes and phonendoscopes, according to my experience, are of no direct advantage to a physician with normal hearing, although those who are hard of hearing will find them very useful.

In every case the stethoscope must at first be lightly applied, because heart patients are particularly sensitive to pressure on the chest.

The auscultation may be performed while the patient is seated or lying down. It is often advisable to make the examination in both positions because the results are frequently different in each, and it may be useful to combine the two results in arriving at a diagnosis. Not infrequently it is well to have the patients make a few muscular movements before proceeding with auscultation if they are in a condition to do so. In cases of heart weakness a very guarded opinion should be given. In all complicated cases is it advisable not to give a final diagnosis until the patient has been examined repeatedly.

In order to interpret the auscultatory phenomena in the heart and assign their true diagnostic value, it is necessary to determine their time relation to the contraction and relaxation of the ventricles. When the heart rhythm is normal, this presents no difficulties, because the time occupied by diastole is longer than that of systole. If merely listening to the rhythm is not enough, the apex-beat is felt for; or if that is impossible, the carotid pulse. The apex-beat and the carotid pulse almost exactly coincide with ventricular contraction.

By means of auscultation we ascertain the conditions of individual portions of the heart, especially of the valves, but, as will appear, of the action of many of the muscular parts also. In addition the method gives us an excellent idea of the rhythm, for the human ear is very well adapted to the perception of differences in time.

Two sounds are heard over the heart—one at the beginning of the contraction of the ventricle and the other at the beginning of its relaxation. If we go back to the source of the sounds, we must speak of four; for during systole a sound is produced in the right and another in the left ventricle. The important element in their production is, in the first place, the systolic tension and pull of the muscular fibers.* The sound-waves thus produced unite with those which emanate from the systolic tension of the auriculoventricular valves.† It is this valvular element that gives to the true muscle sound the tone which we actually hear.

It is stated in many books that a sound is also produced in the initial portion of the large arteries caused by the tension of the walls. This phenomenon is assuredly rare‡ and, in general, any sound heard on the chest-wall is to be referred to the heart.

The second sound is due to the tension of the semilunar valves in the large vessels at the beginning of diastole.

Probably no one doubts these facts at the present time, and I shall

* Williams, "Diseases of the Chest," Germ. edition, 1835, S. 171. Contains almost all the facts. Ludwig and Dogiel, "Berichte der Sächsischen Gesellschaft der Wissenschaften, Mathematisch-naturwissenschaftliche Classe," 20 (1868), S. 89. Krehl, "Du Bois' Archiv," 1889, p. 253.

† Bayer, "Archiv der Heilkunde," x, pp. 1 and 270. *Ibid.*, xi, p. 157.

‡ Compare Frey, "Die Untersuchung des Pulses," Berlin, 1892, S. 6. See, also, Geigel, "Virchow's Archiv," cxl, p. 385. *Ibid.*, "Münchener medicinische Wochenschrift," 1896, No. 15.

not go into a detailed discussion of their causes, as it would lead me too far afield.

The auricles also give forth a sound during the contraction of their muscles,* but this sound at once merges with the systolic sound of the ventricles and cannot, therefore, be distinguished from the ventricular systole when the rhythm of the heart is regular.

In the case of systole, as well as diastole, the two sounds are produced simultaneously, or at least within such brief intervals of one another that the ear receives but a single auditory impression; hence we only hear two sounds in the heart. Of these, the first or systolic is always lower in pitch, more dull, and materially longer in duration than the second or diastolic; the latter is often heard loudest at the base, the former at the apex and the lower portion of the sternum.

When auscultation is disturbed by the presence of sounds emanating from the respiratory apparatus, the patient is asked to stop breathing after deep expiration. It must, of course, be borne in mind that heart patients are apt to be dyspneic and, therefore, frequently find it difficult to stop breathing.

It follows from the way in which the heart-sounds are produced that any variations of time in the contractions of the heart or its individual portions must also affect the rhythm of the heart-sounds; this point will be taken up again in a special section on Abnormalities of Rhythm.

Quality (timbre) and intensity of the heart-sounds exhibit extraordinary variations in different individuals in health; the first ventricular sound especially is very variable in character, even under entirely normal conditions. It is only by long practice that the normal can be distinguished from the pathologic. The strength of the sounds depends, in the first place, on the thickness of the integumentary tissues; it is obvious that a heavy layer of fat or edema of the chest-wall, as well as pulmonary emphysema, may considerably diminish the audibility of the sounds, and that, on the other hand, the intensity is increased whenever the chest-wall is very thin or the large vessels lie very near the surface. Thus, when the pulmonary border is retracted on one side, or when the large arteries are dilated and in close contact with the chest-wall, the two heart-sounds are specially loud and distinct over the position of the corresponding vessel.

The strength of the first sound is increased by rapid contraction of the ventricles; the second, chiefly by high blood-pressure in the vessels, which increases the difference between the pressure in the aorta and in the ventricular cavity. Both sounds are weakened and may be very greatly weakened when the ventricular contraction is inadequate and the arterial tension therefore falls—that is, chiefly in cardiac weakness. The statement is very frequently encountered that a loud first sound at the apex, as a rule, accompanies vigorous contraction of the heart and is, therefore, found chiefly in cases of hypertrophy. This statement is probably based on the association of a vigorous systole with a loud tone. Careful observation, however, in many cases shows rather the opposite, as Williams has pointed out. In hypertrophied hearts, when the ventricular contraction is vigorous, the first sound is often distinctly weak or impure, a phenomenon that is well known to be present in cases of aortic insufficiency. From all that we know, hypertrophied hearts contract slowly, and it is probable that loudness of the ventricular sound depends on the rapidity with which systole is effected and the tension of the muscular

* Krehl, *loc. cit.*

fibers, which probably explains the loud first sound in mitral stenosis and in many nervous individuals.

In disease the *loudness and quality of the sounds* are also subject to wide variations, which is a point that must be carefully taken into account. I shall purposely refrain from explaining our reasons for referring auscultatory phenomena heard over definite regions of the surface of the chest to certain definite portions of the heart, because the subject has been discussed at length in connection with valvular disease.

Modifications of the second sound. In most healthy individuals the semilunar valve sound heard at the sternal extremity of the second right and left intercostal spaces is uniform and equal in quality; at least the two sounds, as a rule, appear to be equally loud to the unaided ear.

H. Vierordt compared the two sounds by diminishing their intensity and found * that the sound on the left side was louder than that on the right, and it is also stated that the second aortic sound has a different quality from that of the pulmonary.†

As the blood-pressure and the difference between the pressure in the artery and in the ventricle are much greater in the aortic than in the pulmonary artery, one would expect, in accordance with the principles of physics, to find the second aortic sound louder than the pulmonary. Actually, this is not the case, however, and the reason why the two sounds are alike is not at all clear. In my opinion, however, it is hardly possible to attempt anything like a thorough discussion of this problem. For, in order to investigate the physics of the question, the sounds would have to be auscultated and compared under absolutely identical conditions. But I know of no such method of examination, and, owing to the close proximity of the two orifices, one would have to overcome the difficulty of separating the auscultatory phenomena produced in one vessel from those produced in the other. [Thayer by the auscultation of the exposed dog's heart, found a striking difference between the sounds over the aortic and pulmonic rings. "The second sound at the aortic orifice is of very much greater intensity than that at the pulmonic ring, of a more ringing and musical quality, while the second sound over the pulmonic valves has a deader, more wooden character."‡

According to investigations conducted by Cabot, "the relative intensity of the two sounds in the aortic and pulmonic arteries depends primarily upon the age of the individual, the pulmonic sound predominating in youth and the aortic in old age while in the period of middle life there is relatively little discrepancy between the two. . . . . Pathologic accentuation of the pulmonic sound must mean a greater loudness of the sound than should be expected at the age of the patient, and not simply a greater intensity than that of the aortic second sound," and conversely.§—Ed.]

We physicians describe as the second pulmonary sound that which we hear during cardiac diastole to the left of the sternum, and as the aortic sound the one we hear to the right of the sternum in the second intercostal space. When we speak of changes in the loudness of these sounds, such a statement is perfectly justifiable and is of value for the clinical diagnosis. This, however, does not prove that the sound produced at the aortic or pulmonary orifice respectively actually does undergo such a modification. We are not, as yet, in a position to discuss the theory of these things, and what we say here refers solely to practical matters for medical use.

In disease the sound of the second semilunar valve is often considerably louder on one side than on the other; to adopt the usual terminology, the second aortic or pulmonary sound is accentuated.

Accentuation of the second pulmonary sound is the classic sign of increased tension in the pulmonary artery, and this statement is fully justified.

For the present, however, we are quite unable to offer any explanation of this remarkable fact. For, no matter how much the pressure in the pulmonary artery may be increased, it is certainly considerably less than in the aorta, and the difference

* H. Vierordt: "Die Messung der Intensität der Herztöne," Tübingen, 1885.

† Heitler, "Die Localisation des zweiten Aorten- and Pulmonaltones," "Wiener klinische Wochenschrift," 1894, No. 50.

‡ *Loc. cit.*

§ "Physical Diagnosis," 1905, p. 176.

in pressure between artery and ventricle is also much less on the pulmonic than on the aortic side of the heart. Hence the second pulmonary sound ought, under all circumstances, to remain weaker than the aortic sound. One simple and obvious solution of the riddle is that when the pressure in the pulmonary artery is increased, the trunk of the vessel displaces the lungs and lies close to the chest-wall, and that the sound to the left of the sternum is, therefore, louder on account of the greater proximity of the vessel. The objections to this explanation, in my opinion, are that the first sound in the second intercostal space on the left side is by no means accentuated at the same time, and the fact that the second varies directly with the intensity of the blood-pressure.

So far as I can see, therefore, we cannot as yet hope to find an explanation for this curious fact.

It is probable that we would have made more progress in our knowledge of the changes in the heart-sounds, their production and their significance, if we had at our disposal more accurate reports of examinations so as to furnish a solid foundation for a strict analysis of the data collected. Mere auscultation and relative comparison of heart-sounds without mensuration and based solely on memory and so-called experience are not the methods that will lead to progress. H. Vierordt has* given us a noble example; let us hope that others will follow in his footsteps.

Accentuation of the second aortic sound is found in cases of increased blood-pressure in the aorta, especially in acute and chronic nephritis and in many cases of arteriosclerosis. As in chronic nephritis the pressure is often increased in both the large arteries and both ventricles hypertrophy, the second sound in such cases is, as a matter of fact, frequently accentuated on both sides.

The *quality of the second heart-sound* is also quite variable in disease. It loses its indefinite, noise-like character and becomes more like a pure tone; to adopt the customary terminology, it becomes more "ringing"—the pitch is easier to determine. This phenomenon, in my opinion, is found more frequently in changes in the vessel-wall, especially in the aorta, that is, chiefly in arteriosclerosis and diseases of the kidneys associated with vascular changes. This change in the quality of the sound does not appear to me to be influenced by the absolute height of the blood-pressure, although in actual practice one often meets a combination of a ringing and accentuated second aortic sound; for arteriosclerosis not infrequently leads to heightened arterial tension. It follows, therefore, that one should exercise great caution in deducing an increase of arterial tension from a change in the second aortic sound. A ringing sound from sclerosis of the aorta may quite easily simulate an accentuation.‡

Weakening of the second heart-sound, as has been mentioned, is due to lowering of the arterial tension, and may be unilateral. The conditions due to diseases in the semilunar valves will not be discussed here for reasons that have already been given.

Splitting or reduplication of the second sound§ is quite frequently observed in healthy individuals, but is then only a temporary phenomenon, heard at the height of inspiration. The phenomenon is undoubtedly due to a temporary abnormal difference in the tension of the aortic and

* H. Vierordt, "Die Messung der Intensität der Herztöne," Tübingen, 1885.

† Compare Bucquoy and Marfan, "Revue de médecine," 1888, p. 857.

‡ Compare Schmaltz, "Archiv für klinische Medicin," xlvi, p. 79.

§ Dehio, "Petersburger medicinische Wochenschrift," 1891, No. 32. Landgraf, "Deutsche militärärtzliche Zeitschrift," 1895, p. 1. Neukirch, "Zeitschrift für klinische Medicin," ii, p. 313. Petrezzani, "Sperimentale," 1887; ref. "Centralblatt für klinische Medicin," 1888, p. 611.

[Consult also Henry Sewall, "The Clinical Significance of Reduplication of the Heart-sounds," "American Journal of the Medical Sciences," June and July, 1898; and "On the Clinical Relations of the Papillary Muscles of the Heart," "The Philadelphia Monthly Medical Journal," September, 1899.—Ed.]

pulmonary valves, and apparently it is the second sound that, as a rule, corresponds to the pulmonary artery. The phenomenon would, therefore, point to delayed contraction of the right ventricle. The sign is not infrequently seen when pressure conditions in the large vessels are altered. It has no diagnostic significance. The conditions that exist in mitral stenosis will not be discussed.

The *first heart-sound* is accentuated when the contraction of the ventricles is rapid, as was mentioned in the beginning, and especially in some forms of mitral stenosis.* The sound is *weakened* whenever the cardiac contractions are weak; it may disappear altogether, as has been observed in typhus and typhoid fever. Curiously enough, however, the sound sometimes presents the same characteristics in cases of dilatation and hypertrophy of the left ventricle even when the contractions are vigorous, as, for example, in simple hypertrophy or in insufficiency of the aortic valve, even when compensation is good. For the present the explanation of this phenomenon seems to us impossible. The attempts found in the literature are absolutely unsatisfactory. The theories which assume a slight increase in the systolic tension at the atrioventricular valves—on the ground that their tension is already increased at the end of diastole on account of the overfilling of the ventricles—are based on incorrect premises in regard to the pressure conditions in the cavities of the heart, and do not take sufficient account of the significance of the muscular component of the first sound.

That it is due partly to delayed contraction of the ventricular musculature seems to me quite possible. The fact demontrated by Bayer† that diseased valves are not resonant is probably not without significance in a good many cases.

Reduplication of the first heart-sound, like the similar phenomenon in the second, is not infrequently observed in healthy individuals, and is then temporary and heard only during certain phases of the respiration. It is also observed in heart patients, and is probably attributable in every instance to differences in the contraction time of the two ventricles, but we are still far from having any precise knowledge of the true causes.

Systolic murmurs frequently, but by no means always, cover up the first sound, but as to the way in which this occurs, and how constantly it is present, we are still very much in the dark.

Sometimes the rhythm of the heart-sounds becomes so altered that the pauses between the two are equal. The first and second sound, under such circumstances, cannot be distinguished by loudness or quality, and the heart's action is always accelerated.‡ This condition, which was observed§ by Stokes in typhus fever, and studied chiefly by Huchard, who gave it the name of embryocardia, is found chiefly in infectious diseases, such as typhoid fever, typhus fever, and scarlet fever; but it is also observed in many chronic diseases of the heart muscle. It is undoubtedly a sign of cardiac weakness. The distinguished French clinician goes so far as to say that the prognosis is quite unfavorable whenever this pendulum rhythm is heard for any length of time. I would not venture to indorse this statement because I am on principle opposed to

* See v. Jürgensen, "Valvular Diseases."

† Bayer, "Archiv der Heilkunde," x, pp. 1 and 270.

‡ Huchard, "Maladies du coeur," Paris, 1889, p. 49. Pawinski, "Deutsche medicinische Wochenschrift," 1891, No. 4.

§ "Diseases of the Heart," German translation by Lindwurm, Würzburg, 1855, p. 311.

the practice of basing the clinical diagnosis or prognosis on one single phenomenon. However, the symptom is of grave significance. With our present understanding of the origin of the cardiac rhythm it is impossible to say how it is produced. As acceleration of the heart's action is always associated with shortening of the pause, and embryocardia, at least according to Huchard, is always accompanied by tachycardia, the latter would assist in the development of the former; but the two conditions are not identical.

The occurrence of three heart-sounds, a phenomenon described as *gallop rhythm*,* calls for a more detailed discussion. In this condition three sounds are heard over the heart, and I may say, in agreement with most physicians, that the third sound, which is pathologic, is heard, as a rule, at the end of diastole, shortly before the first ventricular sound, although distinctly separate from the latter; at least, I do not remember of ever having seen a case where the conditions were not as I have said. The accent, when listening at the apex, is usually on the second, and when listening at the base more on the third, of the three sounds; in this respect, however, some differences are observed. For example, at the apex, too, the accent is occasionally heard on the third sound, which corresponds to what is usually the second ventricular sound. The descriptions of gallop rhythm found in the literature exhibit some extraordinary discrepancies, and it is possible that many of them refer merely to simple splitting or reduplication of the heart-sounds;† thus, in my opinion, the so-called *bruit de rappel* does not belong to this category at all. I believe the above description is correct, at least in the majority of cases.

If the adventitious sound is heard shortly before the first ventricular sound, it must be produced in one of two ways: either the ventricles do not contract at the same time, or the contraction of the auricles precedes that of the ventricles by a sufficient interval to make it possible to hear the sound produced by both portions of the heart, which is not the case under normal conditions. Nothing can be brought forward in support of the former view; in my opinion, it is directly refuted by the fact that the second heart-sound remains absolutely single (not duplicated).

Against the assumption of the additional sound originating in contraction of the auricles there is practically nothing to be said. On the contrary, the cardiograms obtained by Kriege and Schmall undoubtedly support this view, as must be admitted by even the most skeptical detractor of the value of apex-beat curves. Hence we may say that the adventitious sound in gallop rhythm is the muscle tone‡ of the left auricle, which is heard abnormally early. Why the cardiac rhythm is disturbed in this way is, to be sure, quite unknown.

Gallop rhythm is found most frequently in cardiac hypertrophy secondary to nephritis, but is not very rare in other conditions, as, for example, in arteriosclerosis, myocarditis, acute infectious diseases, and, as O. Vierordt observed,§ in perfectly healthy individuals when the heart is excited. In many cases the phenomenon is a serious evidence of cardiac weakness, but its significance appears to vary with the conditions under

* See references in: Kriege and Schmall, "Zeitschrift für klinische Medicin," xviii, p. 261. Lépine, "Revue de médecine," 1882, p. 243.

† Compare Cuffer et Guinon, "Revue de médecine," 1886, p. 562.

‡ Compare Lépine, "Revue de médecine," 1882, p. 239. Krehl, "Du Bois' Archiv," 1889, p. 253.

§ O. Vierordt, "Diagnostik innerer Krankheiten," fifth edition, p. 210.

which it is observed. For example, although it is a grave sign in diphtheria, it may be present for a long time in chronic nephritis and be relatively harmless.

Heart Murmurs.—I shall not attempt an exhaustive discussion of the origin and significance of heart murmurs. But one thing should be emphasized in connection with our subject; that is, that it always requires two factors for the production of valvular insufficiency and heart murmurs: structural changes in the valves and functional anomalies in the muscles necessary to effect the closure of the valves. It cannot be emphasized too often that the auriculoventricular valves are unable to effect perfect closure of the ventricles unless the orifices are properly constricted during systole by the circular fibers at the base of the ventricles. The same is true of the arterial orifices; but in the case of the former, another factor comes into play, namely, the correct position of the valvular leaflets, and especially of their insertions in the papillary muscles.

These things at once become clear when one studies the pictures illustrating the changes in the shape of the chambers of the heart, and their different parts during systole and diastole. Unfortunately, the most peculiar ideas still prevail in regard to the rôle played by muscular contraction in the closure of the valves. A great many men seem to find it difficult to rid themselves of the notion that the sole duty of the papillary muscles is to prevent the valve from "flapping back" into the auricle.

There is no doubt that the action of the circular muscles surrounding the venous orifices, and the traction exerted by the papillary muscles on definite points in the interior of the chamber, are absolutely indispensable to valvular closure, and it is equally certain that any anomaly in these two functions may be, and undoubtedly very frequently is, the sole cause of valvular insufficiency. Even in endocarditic processes they undoubtedly often have a greater influence on the valvular disturbances than the direct lesion of the leaflets. For example, when a slight marginal endocarditis of the mitral leaflets is associated with insufficiency of the valves, deficient muscular contraction is undoubtedly the most important factor in the pathology of the case.*

These muscular insufficiencies are distinctly more frequent in the auricular valves than in the semilunars. The production of these insufficiencies becomes perfectly clear and intelligible as soon as we make a careful study of the arrangement of the fibers of the heart muscle and the changes that occur in them during systole and diastole.† The older conception that this insufficiency is "relative" in the sense that the valvular leaflets are not large enough to close the dilated orifice, is assuredly applicable only to a very small number of cases. At all events any one who is familiar with the mechanism of valvular closure will find it difficult to reconcile the theory with the facts.

In most cases it is exceedingly difficult to differentiate between this form of ventricular insufficiency, and that due to structural changes in the leaflets, that it to say, to endocarditis and arteriosclerosis.‡ The differential diagnosis cannot be based on the character of the sound itself; the determining factors must be sought in concomitant conditions and

* Romberg, "Archiv für klinische Medicin," liii, p. 141.

† Krehl, "Beitrag zur Kenntniss der Füllung and Entleerung des Herzens," "Abhandlungen der Sächsischen Gesellschaft der Wissenschaften," xvii, No. 5. Compare Emthoven and de Lint, "Pflüger's Archiv," lxxx, p. 139.

‡ Compare Leube, "Archiv für klinische Medicin," lvii, p. 225.

elements of a more general nature, such as the causes of the disease, and, especially, a variability in the appearance and intensity of the murmurs and the like. In short, the question is decided on the general principles which obtain in a differentiation between muscular and valvular diseases in the heart, which will be discussed in connection with the diagnosis of myocarditis.

The origin of so-called *accidental or anemic heart murmurs* is much more difficult to explain, and it is by no means easy to determine their diagnostic value. These murmurs are almost exclusively systolic, are heard more frequently in the second intercostal space on the left side, and next in order of frequency at the apex. They have nothing to do with structural changes in the heart valves, as the definition alone implies. But one cannot by any means be equally positive in excluding insufficient muscular contraction, that is to say, muscular insufficiency of the valves, from the pathogenesis. On the contrary, when all the factors in the case are duly taken into account, it is unquestionably justifiable in many cases to assume that muscular insufficiency is the chief factor. I shall merely recall the association of accidental murmurs with dilatation of the heart, which is so frequent in anemia, and the simultaneous disappearance of both phenomena.

But this does not altogether dispose of the question. Systolic murmurs are heard quite often without obvious or even distinct signs of anemia, and without any cause to justify the assumption of a functional valvular disturbance.

In such cases we are altogether in the dark in regard to the origin of the murmurs. There is no doubt that, in many cases, the phenomena have nothing whatever to do with the ventricles or arteries, but arise in the large veins. Sahli* emphasizes this in the case of accidental diastolic murmurs, and personally I have often seen how easily a confusion may arise between a venous hum and a heart murmur. But even this explanation is applicable only to individual cases.

Th. Weber† has shown that a moving column of fluid confined in a tube gives forth a sound when the velocity exceeds a certain critical point, and the ease with which the sound is produced is proportionate to the mobility of the fluid. Whether, as Sahli believes, this factor has any influence on the question may be doubted. At all events, there are no accurate investigations either for or against it. Personally, it seems to me very doubtful whether such a difference in velocity as that would imply ever occurs in disease, or if it does, that the velocity of the blood-current is accelerated in individuals suffering from anemia or in patients generally.

It is often exceedingly difficult to differentiate these accidental murmurs from the murmurs of true insufficiency. The character of the auscultatory phenomena is in itself of very little value. It is true that hemic murmurs are usually very soft, but the same thing is occasionally observed in cases of true insufficiency. A consideration of all the factors in the individual case is far more important, and in the end must decide the diagnosis; such factors are enlargement of the various portions of the heart, character of the second pulmonary sound, etiology, etc.

It is well known that respiratory sounds are not rarely heard in the

* Sahli, "Lehrbuch der klinischen Untersuchungsmethoden," second edition, p. 243. Geigel, "Münchener medicinische Wochenschrift," 1896, No. 15.

† Th. Weber, "Archiv für physiologische Heilkunde," 1855, xiv, p. 40.

precordial region, the rhythm of which depends on the action of the heart. These sounds are unmistakable to any one listening attentively, and have no significance whatever. They hardly deserve the amount of attention accorded to them especially in Potain's communications.*

FUNCTIONAL EXAMINATION OF THE HEART.

All the above-described methods of examination have as their ultimate object to furnish information in regard to the functional capacity of the heart. We wish to know, and indeed we must know, to what extent the heart in an individual case is capable of satisfying the demands that are made on it. In actual practice the greatest imaginable contrasts are encountered: sometimes the organ is quite incapable of adequately supplying the circulation even during absolute rest; at others, only very slight functional disturbances are observed even during the greatest exertions.

How, then, is the physician to recognize whether and to what extent the heart in a given case is doing enough work under varying conditions?

In order to answer this question, especially for the milder cases of functional disturbance, a *knowledge of the history* is necessary.

By judiciously varying our questions we can at least find out how the patient feels subjectively when he does certain things which we know from experience influence the circulation. The patient's habits are often quite characteristic. Many persons give up certain things which they find they cannot endure, nor is it always at once manifest how much they are influenced by any conscious purpose. At all events, it will not do to assume that it exists, for its influence is very frequently forgotten or denied by the patient. The physician must, therefore, endeavor to gain a comprehensive idea of the patient's entire mode of life, and to do so is, of course, easier in proportion to the intimacy of his acquaintance with the patient. The general attitude of the physician toward his patient is most important in a case of this kind.

Much valuable information is obtained in this way, but the data, of course, have all the disadvantages of statements with a subjective coloring. In weighing such data the physician must bring all his acumen and his knowledge of men to bear, and take the quality of his patient's mental makeup into account. Above all, he must note any changes that have occurred in the individual, just as we do when we wish to form an opinion of a patient's mental condition.

In order to determine from the subjective symptoms whether a process deserving the appellation morbid is present or not, it is also necessary to take into account the individual's antecedents and the degree of exertion which, according to his statements, brings on a disturbance of the heart. For example, if an elderly man whose custom has been to sit quietly at his writing table or in the office expresses surprise at becoming short-winded during the first days of his attempts to ride a bicycle, especially in going up hill, the condition can hardly be called abnormal; and even less so, if the individual in question is a woman. Finally, all those things which are set forth in the general discussion of the etiology must be duly considered. Thus, for example, convalescence from acute disease, the existence of a nutritional disturbance, care and worry, and other things that injure the general strength of the organism affect the strength of the heart unfavorably, although there may be no local disease in the organ.

Next in order are the results of the objective examination of the entire circulatory apparatus, which are important in two different ways: In the first place, signs are often found during this examination showing that the heart is insufficient even during rest; I shall merely refer to the significance of edema, dilatation, and similar things. In the second place,

* Potain, "Clinique médicale."

the examination, if it does not yield direct signs, may elicit premonitory symptoms that at least make one think of the possibility of alarm, such as anomalies in the action of the heart, in the kidneys, and the like.

A good deal is learned in this way, but all the facts in the case are not by any means exhausted. For example, the greatest discrepancy is sometimes discovered between the results of the objective (physical) examination and the patient's statements. The patients complain of all kinds of functional disturbances, and yet nothing is found to account for them; that is, while the heart action is not absolutely normal, no physical signs can be discovered, at least during rest.

One cannot help wishing it were possible to determine the functional power of the organ by objective means in the same way as we are in the habit of determining the functional conditions of other systems. Who among us would in this day care to give an opinion or undertake the treatment of a diabetic case before accurately determining by quantitative examination the patient's power of burning up sugar? And even in diabetes we have by no means reached our ideal; there is still much for us to learn—for example, the variations in the assimilation of sugar in health. Nevertheless, the ability to make a quantitative estimate is a distinct progress, and a similar advance is desirable in the functional examination of the heart.

The method to be adopted will probably be to give the muscles, and through them the heart, a definite amount of work to do, and to observe what effect it has on the circulation. In experiments of this kind, which have been performed for some time in patients with valvular lesions, either the position of the body was changed or the patient was directed to make certain movements. Jaquet* and Gärtner† constructed very serviceable contrivances (ergostats) by means of which muscular work in graded quantities can be prescribed. Jaquet utilized the muscles employed during climbing, while with Gärtner's instrument the arms are used. Subsequent observation must decide whether these apparatus and the special kind of muscular movement which they require will suffice, or whether it will be necessary for certain special purposes to afford an opportunity for other kinds of innervation. We are very far from having attained the goal with this method, but in my opinion it is so important and promises to be useful in so many ways that it had to be mentioned here.

But how shall we recognize the functional power of the heart objectively? As a rule, by noting the changes in the pulse‡ and in the respiration. Both are unquestionably valuable, both are closely related to the power of the heart as well as to that of the blood-vessels. It is quite possible that future experience may discover other signs, but for the present we must content ourselves with these. The points to be noted in the pulse are frequency, rhythm, form, and tension.

Some things are positively known. Increased muscular work is followed by an increase in the frequency of the heart-beat and, to a certain extent, the two things are in proportion. It is true that our knowledge of the exact degree and limits of this parallelism and of the degree of acceleration of the heart-beat is far from adequate, especially in health.§

* Jaquet, "Schweizer Correspondenzblatt," xxiv (1894).

† Gärtner, "Allgemeine Wiener medicinische Zeitung," 1887, No. 49, 50.

‡ Compare Spengler, "Dissertation," Zürich, 1887. Christ, "Archiv für klinische Medicin," liii, p. 102. Minassian, "Dissertation," Basel, 1895.

§ Compare, Christ, *loc. cit.*

In the case of patients with changes in the myocardium or endocardium clinical experience has long ago taught us that the frequency of the heart-beat is much more labile than in healthy individuals; it fluctuates much more readily in both directions in response to all kinds of influences. In such patients the pulse is frequently rendered irregular by muscular movement, while in many healthy individuals and during convalescence from infectious diseases the pulse is more regular when it is accelerated than when beating at its usual rate.*

Generally speaking, the blood-pressure is raised by muscular movement in health,† but the influence of individual conditions still remains to be explained. Is there a constant, unalterable relation between the height of the blood-pressure and the degree of muscular exertion? What is the influence of sustained muscular exertion? Evidently we here have to deal with peculiar and very important conditions. There are grounds for believing that if the individual is accustomed to a certain muscular movement, even though it be severe, an increase in blood-pressure does not take place with the same regularity. The question will be referred to again in connection with diseases of the heart following excessive muscular exertion.

The ingestion of fluid, especially when it contains chemically or mechanically active substances, such as alcohol, carbon dioxid, and the like, is capable of raising the blood-pressure; ‡ and the effect is increased when the introduction of fluid is associated with muscular movement.

There is, therefore, no lack of building material, and with proper care it ought to be possible to erect a useful structure, but the undertaking calls for perseverance.

It has already been stated that certain isolated pathologic facts have already been gained by experience; for example, in almost all heart patients the pulse becomes more irregular and less uniform after muscular exertion. It is also known that when the heart is not quite equal to the demands made upon it, a trifling cause suffices to bring on an unusual acceleration of its action; any cyclist in mountainous regions will have ample opportunity of convincing himself of this fact.

Rieder found that occasionally in cases of heart disease the effect of muscular exertion on the blood-pressure is different from the effect in health; instead of being increased, the pressure remains the same or may even fall.

[A very useful contribution to this unsettled subject, with references to recent literature, is given by Dr. Geo. W. Norris, "The Functional Capacity of the Heart," "International Clinics," vol. i, seventeenth series, 1907.—Ed.]

Cardiac Rhythm and Its Disturbances.—The heart belongs to the so-called automatically working organs, that is, organs in which occur periodic stimulations to activity, *i. e.*, contraction of four hollow muscles, the two auricles and the two ventricles.

Until quite recently these stimuli were supposed to originate in the ganglion-cells of the heart. But it may now be regarded as positively established that the ability to originate rhythmic stimuli resides in the

* Hüsler, "Archiv für klinische Medicin," liv, p. 229.

† Oertel, "Allgemeine Therapie der Kreislaufstörungen," fourth edition, p. 170 *et seq.* v. Maximowitsch und Rieder, "Archiv fur klinische Medicin," xlvi, p. 329.

‡ v. Maximowitsch and Rieder, *loc. cit.*

muscle itself;* a large proportion of the investigators accordingly attribute the automatism of the heart to this property of the myocardium. It is obviously not my duty to furnish the proof of this view, which I fully share, since it would require an extensive physiologic discussion.

Nor do I care to express an opinion in regard to the relation of the ganglia to this automatism of the muscle. A great many theories are possible, and one idea in particular, namely, that the activity of the nerve-cells is associated with numerous fine adjustments of the heart's action, naturally suggests itself.

The contraction begins at the point where the veins empty into the auricles, and is propagated to the auricles, and, by means of the muscular connecting bridges, to the ventricles. Frequency, rhythm, force, and velocity of the contractions exhibit an extraordinary degree of variation and are subject to a great number of influences from various quarters.†

[The statement as to the connection between auricles and ventricles does not quite correspond to the present state of our knowledge. Such a bridge was first discovered by Stanley Kent, and independently by His, Jr., in 1893, and its existence, location, and size described more accurately in 1904 by Retzer and Braeunig. But Tawara, in Aschoff's laboratory, has shown that the connection is more complicated, and his work goes far to clear up many obscure points in cardiac physiology and pathology. According to Tawara, the "auriculoventricular" or His' bundle is only part of a complicated system of muscular fibers, which begin in the auricular septum, pass through the auriculoventricular septum as a bundle which divides into two limbs, traverse the ventricles in the trabeculæ or "false tendons," and finally communicate with the heart muscle-fibers in the papillary muscles and walls of the ventricles. The terminal fibers are identical with those described by and called after Purkinje. In some animals (ungulates) the fibers show distinct differences from true heart muscle, the sarcoplasm being more extensive and the fibrillæ less well developed. They have been described as transition forms from smooth to striated muscle fibers. In man the fibers are not so characteristic, but in general structure and general arrangement the atrioventricular bundle and its terminal branches are fairly uniform in different classes of animals. As regards function, all who have investigated it consider the system as a conducting one, through which the coördinate action of the various parts of the heart is brought about.‡—Ed.]

The central nervous system influences the heart through two nerves, the sympathetic and pneumogastric, the nervous impulses being transmitted in a variety of ways and through a great number of different fibers. The central terminations of these two nerve-trunks connect in their turn with numerous sensory nerve-paths of the entire body, and are, therefore, subject to a great many reflexes.

* For a résumé of the entire literature and original investigations see Engelmann, "Pflüger's Archiv," lxv, p. 535. See also the report of the general session of the medical section at the Düsseldorf Naturforscherversammlung, 1898, abstracted by v. Frey. Compare the opposite views of Kronecker, "Zeitschrift für Biologie," xxxiv, p. 529. [See also, Howell, "Text-book of Physiology," and Mills, "Journal of the American Medical Association," January 19, 1907, p. 190.—Ed.]

† The literature bearing on this subject is quite extensive, and an excellent résumé is given by Tigerstedt, "Physiologie des Kreislaufes," Leipzig, 1893, and in his "Lehrbuch der Physiologie," English translation by Murlin, Appleton, 1906.

‡ See "Das Reizleitungs System des Saeugetierherzens," by Dr. S. Tawara, Jena, 1906. "Physiology of Heart Block in Mammals," etc., Joseph Erlanger, "Journal of Experimental Medicine," 1906, vol. viii, p. 8.

From the heart itself fibers pass to the central nervous system and influence the circulation partly by reflexes acting on the heart itself and partly by reflexes acting on the cells.

It is probable that the ganglion-cells of the heart play an important part in these reflexes, although we, unfortunately, as yet know nothing about the details of the function of these ganglia. Finally the heart-wall itself is sensitive to a great many different influences, as, for example, poisons, the degree of distention of the cavities, and blood-pressure, and under such influences is directly capable of altering the rhythm.

The nervous mechanism of the heart is, therefore, most complicated, and we have barely taken more than the first step in the work of unraveling this mechanism in man. The only method by which we can gradually hope to progress is true experimental pathology, which must coördinate the phenomena observed in man with animal experiments in an absolutely uniform and impartial manner. The goal will never be reached by a single method alone; for in man a certain definite process may be caused by so many different things that it is impossible, by observation alone, to solve the question, and animal experiments alone do not admit of sufficiently great variations in the terms of the problem to be solved, besides which it will probably never be possible to realize certain pathologic conditions in animals. Arguing from the animal, we must constantly strive to gain an understanding of the individual phenomena and then try to approach nearer to the complicated conditions in man.

We are as yet far from having a clear understanding of the phenomena, and it is our duty openly to acknowledge our ignorance instead of keeping up the state which has been so aptly called by Martius "sham-physiologism" (Scheinphysiologismus), as is done too frequently in this field, especially in the clinical literature.

In this connection we, of course, have to do only with the rhythmic anomalies that occur in muscular and nervous diseases of the heart. Influences acting upon the heart from without interest us only in so far as they exhibit some peculiar features in the above-mentioned conditions.

The greatest difficulty in diagnosis is always encountered in slight or beginning alterations of the cardiac action, because phenomena which in some individuals may be regarded as entirely normal in others may be the expression of a morbid change. In other words, the individual peculiarities must be accurately known and duly taken into account, which is easy enough in general discussions, but most difficult in the individual cases.

In judging from the *frequency of the heart-beat* it is well to remind ourselves of normal conditions; the relatively high frequency of the pulse in children diminishes rapidly with advancing age. In old persons the frequency of the heart-beat is, generally speaking, not lowered, as is frequently stated, but is at least as high or even higher than in middle-aged persons.*

In the prime of life individual variations are quite marked, but I believe that even during this period a pulse varying from 56 to 78 during complete rest must be regarded as normal. At least it is certain that a pulse below 64 is not frequent in health; it does occur, however, and it would, therefore, be wrong to pronounce such a pulse pathologic in every case.

* Canstatt, "Die Krankheiten des höheren Alters," Erlangen, 1839. Geist "Klinik der Greisenkrankheiten," Erlangen, 1860.

In heart patients the frequency of the pulse is also subject to very great variations. A heart with a diseased muscle or abnormal nervous system is abnormally sensitive to most external influences, and especially to influences which, in the healthy individual, usually produce an acceleration. For example, the frequency of the pulse in heart patients is disproportionately increased after the slightest muscular exertion, after a rise of temperature or comparatively insignificant psychic excitement, and not infrequently the individual contractions are extremely irregular and unequal. Whether retardation is also apt to occur more readily and in a more pronounced degree in a diseased heart, as, for example, from central irritation of the vagus, I am unable to say.

The cause of the increased irritability in a myocarditic heart is probably some change in the muscle. One naturally thinks of the auricles because the automatic innervation begins in the muscle of these cavities; and one might also argue by analogy since all inflamed tissues elsewhere in the body also exhibit heightened irritability to all kinds of influences. But these are very feeble arguments, and, in fact, not much more than surmises.

Acceleration of the heart-beat in the absence of any of the causes that produce the condition in health is found very frequently in diseased hearts. In fact, it may be said that the action of such hearts is abnormally frequent most of the time, both in diseases of the muscle and in diseases of the nervous system. But we have by no means advanced so far in our knowledge of these matters as to be able, for example, to deduce, from a definite change in the muscle, what the character of the pulse will be. Such a thing is, for the present, altogether out of the question and will remain so for some time. All that we know at present is that, according to medical experience, most diseases of the heart muscle are attended by acceleration of the pulse and only a few, which will be discussed presently, exhibit retardation. It is never possible to give a definite reason for the acceleration of the pulse, at least so far as influences emanating directly from the heart are concerned. The most that can be said is that inflammation of the myocardium probably intensifies the automatic stimuli or renders the muscle more receptive to such stimuli. Again, however, this is mere conjecture and is true only within limitations.

The conditions are practically the same in the so-called nervous diseases of the heart. In the majority of cases these also exhibit acceleration; but, again, we are far from possessing a clear understanding of the causal connection, and let no one be deceived in this respect by the numerous pseudophysiologic explanations found in clinical literature.

The usual number of heart-beats in the minute is very variable. In general the acceleration is only moderate, but attains a greater height as the result of some unusual influences. Such great variations are observed in individual cases that it is better to discuss this matter in connection with the various clinical conditions.

In a great variety of cardiac diseases peculiar attacks of very marked acceleration of the heart-beat, known as *tachycardia*, are observed. We usually distinguish between symptomatic and genuine tachycardia; that is to say, acceleration of the heart-beats occurring paroxysmally is a symptom which is found in a great variety of cardiac disturbances, such as valvular lesions, diseases of the muscle and of the coronary arteries. The causal connection between these diseases and the attacks is not clear. The exciting causes given are the same as we know influence the heart action in healthy individuals; for example, psychic emotion, violent

muscular exertion, and the like; the direct cause of their action, which is so peculiar in these cases, is still shrouded in obscurity.

But this is not all. There are forms of tachycardia that we cannot, with our present knowledge, refer to any known morbid condition of the heart—so-called essential tachycardia. In these cases the acceleration is usually regarded as the primary, essential phenomenon; hence the peculiar classification.

The direct consequences of accelerated heart action can hardly be said to be the same in all cases. The conditions in the arteries depend on the completeness with which the accelerated heart becomes filled during diastole and the thoroughness and rapidity with which it empties itself; and these factors again are influenced by the nutrition of the muscle itself. It is evident, therefore, that the conditions are extremely complicated, and the factors which influence the rhythm of the heart are often capable, at the same time, of affecting the strength of its contractions; hence no one will be inclined to lay down a law that is applicable to the individual case.

So far as the frequency of the heart-beat alone is concerned, it may be assumed that at first acceleration causes an increase in the arterial pressure and the velocity of the blood-stream; for so long as the ventricles fill and empty properly,—as they continue to do until the duration of the pause is reduced below a certain minimum,—the arteries must become more completely filled. But when the acceleration is very great, the ventricles do not fill properly, the ventricular musculature is unable to obtain the necessary quantity of nutritive material, and the contractions become weaker. The arterial pressure then diminishes, the quantity of blood in the arteries lessens, while that in the veins increases. Hence, when the heart action is very greatly accelerated, signs of retarded circulation are not infrequently observed. It is, however, practically impossible to say positively to what extent these conditions obtain in any individual case; for we have seen that a number of special factors, the significance of which we are still unable to estimate, also come into play.

What is the diagnostic and prognostic significance of acceleration of the heart-beat? Experience teaches that diseased hearts frequently exhibit very great frequency of contractions before death, and that loss of strength in the heart is accompanied by acceleration of its rhythm. This is not a rule without exceptions, however; since, as will presently be mentioned, feeble hearts quite often exhibit retardation; but this is distinctly less frequent than the opposite condition; of that there can be no doubt.

It is not very easy to decide how far the standpoint of the theoretic pathologist coincides in this matter with that of the physician; in other words, to what extent one is justified in saying that heart weakness as such is the cause of acceleration of the pulse. In view of the great number of influences capable of affecting the frequency of the heart-beat, the acceleration may be secondary, as, for example, due to a reduction of the blood-pressure, while the change in the heart itself would influence its rhythm in quite a different manner.

The boundary-line where *pathologic retardation of the heart-beat or bradycardia** may be said to begin has already been mentioned. Cases of retardation of the heart-beat due to extracardial causes and to general diseases are not included in this discussion; we are interested only in bradycardia due to changes in the heart muscle or its nervous apparatus.

Such cases are unquestionably uncommon. This form of bradycardia is observed chiefly under two conditions: in a number of infectious

* Riegel, "Zeitschrift für klinische Medicin," xvii, p. 221. Grob, "Archiv für klinische Medicin," xlii, p. 574. Strübing, "Deutsche medicinische Wochenschrift," 1893, No. 4 and 5. Schütze, "Zur Casuistik der Bradycardia," Dissertation, Greifswald, 1891.

diseases, as, for example, typhoid fever, but chiefly diphtheria; and especially when the heart is so weak that the patient's life is in danger. We now know that inflammation of the myocardium frequently develops during these infections, and a causal connection between the myocarditis and bradycardia naturally suggests itself. However, one must always think of the possibility of a toxic influence.

The most persistent cases of bradycardia occur in embolism of the coronary arteries or disease of the vessel-walls (sclerosis, arteritis), and occasionally in chronic myocarditis. It is in such cases also that the highest degrees of bradycardia are observed: twenty to thirty cardiac contractions in a minute is not so very rare, and even twelve and eight have been observed. The first thing that naturally suggests itself in such a case is the existence of irritative or paralytic phenomena on the part of the diseased musculature itself. Dromotropic disturbances (disturbances of conductivity) are also possible. The bradycardia unquestionably does not depend on the influence of the vagus, for Dehio* found that it did not disappear after the exhibition of atropin. However, we are still far from a proper comprehension of the true connection between the various phenomena.

Finally, bradycardia occasionally occurs in nervous diseases of the heart. In many cases of this kind cerebral irritation of the vagus is not at all improbable.

Retardation of the heart-beat, especially when it is marked, causes a diminution of the blood-pressure on account of insufficient filling of the aorta. At first it is possible that the anemia of the aorta, due to the increase in the length of the pauses, may be compensated by an increase of the pulse volume [systolic output—Ed.]. But when the bradycardia becomes extreme, this is impossible and the blood-pressure falls. Besides, the condition which is responsible for the change in the frequency is often, at the same time, the cause of the diminution in the strength of the contractions.

The prognostic significance of bradycardia depends chiefly on its cause. It is extremely grave in infectious myocarditis; in sclerosis of the coronary arteries it may be well borne for years, but is always a positive sign that some grave process is at work in the heart.

[It is unfortunate that the same term (slow) should be used for an infrequent pulse (pulsus rarus) and a pulse with a slow wave (pulsus tardus). The word infrequent is better for the former. The terms infrequent pulse and bradycardia are not exactly synonymous. The heart may beat infrequently, but heart-beat and pulse correspond. Or the heart may beat "at the normal or even at an increased rate, and yet a 'rare' pulse is found at the radial." Wenckebach classifies these in groups according to their dependence upon—(1) "extra-systoles, which may not be perceptible in the peripheral arteries, and when they occur regularly may even produce a pulse with half the frequency of that of the heart. (2) True bigeminy of the heart. (3) Ventricular systoles which drop out or cannot be felt at the periphery. In such cases the systoles drop out as the result of grave disturbance of the stimulus conduction or the excitability: $\frac{1}{2}$, $\frac{1}{3}$ or $\frac{1}{4}$ frequency can then occur (heart-block)." In alternating action "the weaker systole may not be perceptible at the periphery, and there may be produced a half frequency of the pulse."

"In all cases of pulsus rarus, therefore, we have to decide whether

* Dehio, "Petersburger medicinische Wochenschrift," 1892, No. 1.

bradycardia is really present, or some process obscuring the normal frequency. Extra-systoles can be easily recognized. In a case where the frequency is reduced in a definite proportion, we would first think of heart-block."*—Ed.]

Disturbances of the Cardiac Rhythm, Irregularity and Inequality of the Heart Action.†—The cardiac contractions in a healthy, middle-aged individual follow one another approximately at regular intervals and are of uniform strength, or, at any rate, appear so to an observer who bases his judgment on his sense of touch and on auditory impressions. A cardiographic record of the movements of the heart, taken either at the apex-beat or over some artery, shows, when the individual periods are measured, that even in a healthy individual the fluctuations in the duration of the pulse‡ are not inconsiderable; that is to say, the time that intervenes between the beginning of one beat and the beginning of the next is quite variable.

Since it is inconceivable that the length of contraction should be subject to marked variations under identical conditions, the variations in the duration of the pause may be accepted as a positive fact. These variations not infrequently amount to several tenths of a second and represent such a considerable percentage of the entire "pulse-duration" that it seems remarkable they should escape one's sense of touch. Perhaps, however, this is partly due to our ingrained views in regard to the regularity of the heart-beat in healthy individuals; for who can deny the powerful influence of firmly rooted convictions even in the keenest observer?

These "normal" fluctuations in the duration of diastole persist under a great variety of conditions; even when the cardiac activity is profoundly affected—so much so that the organ does absolutely less work—the irregularity need not necessarily become greater. In fact, we know of some conditions, such as muscular movements, which distinctly diminish the irregularity. Perhaps the latter act by diminishing the tone of the vagus; for this nerve, in addition to its retarding influence, also causes irregularity, and if, therefore, its irritability is diminished, the pulse may evidently become accelerated and more regular.

A healthy child presents sometimes during rest, and quite frequently under the influence of a variety of bodily and mental emotions, marked irregularities of the heart-beat concerning the cause of which we are absolutely in the dark.§ The same phenomenon is not rare in older individuals, in whom, however, sclerosis of the coronary arteries may possibly play a part.

The rhythm of the heart-beat may be disturbed in a great variety of

* Wenckebach, "Arhythmia of the Heart," English translation, p. 178.

† Riegel, "Die Lehre von der Herzirregularität," etc., Wiesbaden, 1891. *Ibid.*, "Archiv für klinische Medicin," vol. xviii, p. 94; vol. xx, p. 465. Nothnagel, *ibid.*, vol. xvii, p. 192. Sommerbrodt, *ibid.*, vol. xix, p. 392; vol. xxiii, p. 542. Schreiber, "Archiv für experimentelle Pathologie," vol. vii, p. 317. Heidenhain, "Pflüger's Archiv," vol. v, p. 143. Knoll, "Wiener Sitzungsberichte," 66, vol. iii, p. 195. Heubner, "Zeitschrift für klinische Medicin," vol. xxvi, p. 493. Ebstein, "Archiv für klinische Medicin," vol. lxv, p. 81. Dehio, "Petersburger medicinische Wochenschrift," 1890.

‡ K. Vierordt, "Die Lehre vom Arterienpuls," Braunschweig, 1855. von der Mühll, "Archiv für klinische Medicin," vol. xlix, p. 348. Hüsler, *ibid.*, vol. liv, p. 229. Hürthle, "Deutsche medicinische Wochenschrift," 1892, No. 4. François-Franck, "Gaz. des hôp.," 1877, No. 68, Abstract Virchow-Hirsch, 1877, ii, p. 188.

§ See Heubner, in "Zeitschrift für klinische Medicin," vol. xxvi, p. 493.

ways. First of all, periods of rapid heart-beats alternate with periods when the pulsations are slower, and sometimes the alternation depends on the phases of the respiration.

Or the pauses between the beats are lengthened or shortened, as the case may be, in comparison with their duration in health. This irregularity may be observed in a great many different forms; for example, every ten or twelve beats, or at even longer intervals, a systolic contraction occurs ahead of time or is omitted altogether. On the other hand, the irregularity may be so marked that even to the sense of touch no one pause appears to be equal to any other (*delirium cordis*).

In this matter of irregularity there are certain most interesting groups which result in the production of a kind of system in the irregularity [allorrhythmia, or regular irregularity—Ed.]. Thus, for example, there may be two systolic contractions in rapid succession, followed by a longer pause (*pulsus bigeminus*); or the longer pause occurs after three systolic contractions in regular succession (*pulsus trigeminus*). Strong and weak cardiac contractions may follow one another in regular alternation, producing the so-called *pulsus alternans*.

Wenckebach, in an interesting treatise, showed that in numerous cases the irregularity of the heart's action consists in the appearance of (premature) extra-systoles. If the next succeeding normal stimulus to contraction coincides with the refractory phase of this extra-systole, it is unable to produce a contraction of the muscle; the normal systole then fails to take place, and the time that elapses between two regular contractions corresponds with tolerable accuracy to two cardiac cycles. The strength of the extra-systole itself varies according to the phase of cardiac activity with which it coincides; that is, according to the irritability of the heart. A combination of several of these extra-systoles may give rise to very marked disturbances of the rhythm.

Wenckebach* suggests that this phenomenon, due to the occurrence of extra-systoles with maintenance of the original rhythm of the heart, should be distinguished from genuine arhythmia, and proposes the term "pararhythmia." He believes the distinction to have some prognostic significance. My own urgent advice in matters of this kind is to leave the decision entirely to bedside experience; as far as I can see, the latter does not as yet permit us to draw any positive conclusions in regard to diagnosis and prognosis in disease from these finer differences in the type of the irregularity.

The important results obtained by Engelmann† in his excellent work in regard to the physiologic conditions of cardiac rhythm promise to throw an interesting light on many of these questions and are destined to be of much value in helping us to understand these disturbances. To Wenckebach we are indebted for many beautiful observations.‡ But before we can think of discussing these matters generally, our data and our experience will have to be considerably enlarged.

Even in these conditions simple irregularity of the heart-beat cannot be strictly separated from fluctuations in the volume of the heart-beat.

* Wenckebach, "Zeitschrift für klinische Medicin," vol. xxxvi, p. 181.

† Engelmann, "Pflüger's Archiv," lii, p. 357; xvl, p. 149; lix, p. 309; lxi, p. 275; lxii, pp. 400 and 543; lxv, pp. 109 and 535. "Untersuchungen aus dem physiologischen Laboratorium in Utrecht," iv, 1896.

‡ Wenckebach, "Zeitschrift für klinische Medicin," xxxvii, p. 475; xxxix, p. 293. "Congress für innere Medicin," 1900, p. 182.

For during a longer pause the ventricles generally fill more completely than during a shorter pause, and then, of course, the succeeding contraction forces more blood out of the heart; besides, as has already been mentioned, extra-systoles vary in the results they produce according to the phase of irritability with which they coincide.

However, the inequality in the size of the individual systolic contractions is not always ascribable to fluctuations in the duration of the pause; for there is no doubt that a diseased heart not infrequently presents a primary irregularity in its various systoles. Such an irregularity may occur independently; but more frequently irregularity and inequality of heart action go hand in hand and may obviously give rise to the greatest variety of clinical pictures.

Marked irregularity in the cardiac contractions, so great as to be unmistakable even to the palpating finger, is particularly common in the diseases with which we are here concerned. In the first place, such irregularity occurs in the wake of disease of the muscle. Here again the conditions are extremely complicated in so far as we have not as yet attained an accurate knowledge of the causal connection.

There is no doubt that recent inflammatory changes in the heart muscle and the related condition of loss of individual muscle-fibers from sclerosis of the coronary arteries very frequently produce irregularity of the heart-beat. But even under these circumstances the irregularity may not be present, just as it may be absent in simple degenerative changes of the muscle-fibers.

After what has been said above in regard to the automatism of the heart muscle, it is not difficult to understand that changes in the muscle should have a considerable influence on the heart-beat; and as inflammatory processes cause irritative phenomena in the same way as the infarcts which occur in the wake of coronary sclerosis, there is nothing peculiar about their exerting a special influence. The disturbance of the rhythm which occurs in simple stenosis of the coronary vessels may also be included in this category; for cellular anemia produces irritative conditions.

We must try to understand why these factors, which have the general faculty of disturbing the cardiac action, produce such a result in one patient and do not produce it in another under apparently identical conditions. A number of possibilities must be taken into account. It is well known that poisons have a considerable influence on the rhythm of the heart. Now toxic material is not only produced during infectious processes, but also results from the death of body tissues, in this case of muscle-fibers; and the differences in the toxic material thus produced may possibly be responsible for the differences observed in the symptoms. While this is only a surmise, it is not beyond the possibilities. Another point must also be borne in mind: the rhythm of the heart-beat is determined at the orifices of the large veins. We ought, therefore, to find out accurately to what extent the muscles at these orifices take part in the inflammatory processes—*i. e.*, the irregularity possibly depends on the disease focus being localized at a definite point. We are not without some observations in support of such a hypothesis. Radasewsky,* under Dehio's guidance, observed in certain hearts which exhibited marked arhythmia that the disease chiefly affected the musculature of the auricles. This was observed in cases of chronic myocarditis. Our investigations

* Radasewsky, "Zeitschrift für klinische Medicin," xxvii, p. 381.

in this line should be still further extended for the purpose of proving that the same thing applies to other changes in the muscle.

In many cases the occurrence of irregular contractions is due to a marked increase in the pressure within the arteries and the cavities of the heart. Knoll* has shown that in animal experiments increase of the arterial pressure produces an irregularity and an inequality of the heart-beat quite independently of the vagus and its terminal apparatus, solely by its direct influence on the heart. This fact has often been utilized to explain the various forms of arhythmia occurring during disease—and not without reason, if abnormal filling of the heart is assumed as the intermediate factor, but not so if the latter is sought in the increased pressure. For, while most cases of arhythmia observed at the bedside occur when the cavities of the heart are overfilled, they are not associated with vigorous cardiac contractions and a high arterial pressure, but, on the contrary, with a small and soft pulse. In fact, the overfilling of the heart is the result of this condition of the pulse.

There is no doubt that this offers a fruitful field for accurate observation and keen inductive reasoning. Henschen's† explanation of *pulsus bigeminus* in a case of insufficiency and stenosis of the mitral valve I hail as a most interesting attempt.

Whether, and to what extent, the nervous apparatus of the heart in diseases of the muscle takes a part in the production of the irregular rhythm we are not as yet in a position to say. It is quite possible, and various causal relations are conceivable. Thus, for example, an inflammatory process may attack ganglion-cells and nerves as well as muscles. For the present, however, it is impossible to set up any elaborate theory, because we have no physiologic or pathologic data on which to base such a theory. This candid admission, however, is not to be mistaken for an expression of the foolish view that the ganglion-cells have no significance.

In cases of cardiac disturbances which follow excessive exertion or liberal indulgence in wine and beer, or both of these factors together, the conditions are very much more complicated and very obscure, as we shall have to show later on. A great many different kinds of disturbances of the heart action are observed which we are as yet unable to understand.

In the so-called nervous diseases of the heart, also, disturbances of the rhythm are not infrequently observed which again are most difficult of interpretation. Later I shall attempt to explain at some length what we mean by a nervous disturbance of the heart. I may, however, state at once that we have to deal with subjective and objective cardiac disturbances due to some nervous influence. Now, it is well known that nervous influences are capable of markedly affecting the heart-beat—not only its frequency, but also its rhythm. I need only refer, for example, to the influence of the vagus or to the significance of psychic emotion. The individual ways in which this influence exerts itself in disease are quite beyond our ken at present, and nothing whatever is gained by indulging in hypotheses in regard to the question because there is absolutely no foundation for them. We cannot help asking, however: What is the form of these rhythmic disturbances? Have they any special characteristics?

* Knoll, "Wiener Sitzungsberichte. Mathematisch-physikalische Classe," lxv, iii, 5, p. 221; and "Archiv für experimentelle Pathologie," ix, p. 408.

† Henschen, "Arbeiten aus der medicinischen Klinik in Upsala," Jena, 1898, p. 14.

There is no doubt that disturbances of the cardiac rhythm manifest themselves in a large number of nervous individuals ranging from such as may be still considered healthy to the most pronounced neurasthenics. These disturbances very frequently take the form of periodic fluctuations in the frequency of the pulse, a series of rapid beats following a series of slower pulsations in more or less rapid succession. This is not all, however. Fluctuations in the duration of individual pauses, as well as fluctuations in the volume of the pulse, also occur from purely nervous causes.

This point requires careful investigation. It is of the highest theoretic importance, but, above all, it is of importance practically, that is to say, in a diagnostic sense. It will be discussed in greater detail in the section devoted to Nervous Disturbances of the Heart.

The above observations and considerations refer chiefly to simple disturbances of rhythm, but these cannot be strictly separated from fluctuations in the volume of the pulse. For, if the periodic stimulus to contraction (systole) begins in the muscles around the venous orifices, and if the individual contractions are of uniform strength in health, it must be assumed that these stimuli not only follow one another at uniform intervals, but also that they are of uniform strength. In other words, inequality of the heart action may quite easily be due to disease of the auricular musculature. It remains to be investigated what it is that causes either abnormal excitation or altered irritability at these points.

A special form of disturbance of the heart-beat exists in the so-called *hemisystole*. In this condition either the ventricles are supposed to contract alternately (true hemisystole) or a contraction of the entire heart is followed by contraction of one ventricle only (alternating systole).

In my opinion the ventricles are capable of contracting independently of one another; at least the arrangement of the musculature makes it possible, and I believe I have often observed in the animal that it actually occurs. Frank and F. Voit,* it is true, are very skeptical on this point; but the divergence of opinions is probably due to the fact that these two investigators were unable to produce the phenomenon artificially and, therefore, regarded its occurrence as impossible. I have frequently observed it accidentally in animal experiments, although I confess I am unable to give any reasons or state the conditions necessary for its production.

Observations have been made on man which can be interpreted in the same way. But it is hardly possible in an individual case to prove that the interpretation given is the only possible and the only correct one. For, as Riegel† very correctly points out, a bigeminal pulse in which a weak systole rapidly follows a vigorous contraction must yield very similar or identical signs.‡ Nor does it seem altogether right to attempt a sharp distinction between the various conditions, as it is probable that a great many transitional forms exist. Information in regard to all these points will be found in the works referred to. It does not seem right for us to enter into the subject more fully at this point, for we do not believe that in the present state of our knowledge the significance of these matters is definite enough to make them of value in interpreting a morbid condition. For the present all they can be said to indicate is some profound disturbance of the heart action.

* Frank and F. Voit, "Archiv für klinische Medicin," xv, p. 580.
† Riegel, *loc. cit.*
‡ Unverricht "Berliner klinische Wochenschrift," 1890, No. 26.

EXAMINATION OF THE BLOOD-VESSELS.

The examination of the *arteries* is of the very greatest importance. From the nature and condition of the vessels and the processes taking place in them we can derive a great deal of positive information in regard to the condition of the circulation in the individual organs. For the circulation in the various parenchymatous tissues may be influenced by a variety of causes, and especially by diseases of the blood-vessels. However, it is not our province to enter into this question; we have to do only with cases in which disturbances of the heart represent the peccant element. But it must be borne in mind that our interest in diseases of the heart in the last analysis depends on their significance in the blood-supply of parenchymatous cells. Hence, although we are chiefly interested in the disturbances of the motor organ, we must, nevertheless, devote our attention to the arteries also. In the first place, their condition influences the heart and, therefore, affords information in regard to it; and, in the second place, examination of the arteries at the present time affords us the best means of judging the functional power of the heart.

The condition of the *arterial walls* is determined by palpation, not only one, but many different vessels being examined: the temporal, carotid, subclavian, brachial, radial, femoral, popliteal, and pedal arteries must be most carefully examined by inspection and palpation. We must see whether the vessel is tortuous; whether the walls are hard and tense, as a whole, or contain calcareous plates at irregular intervals.

Before drawing his conclusions the observer should distinguish as accurately as possible between rigidity of the vessel-wall as such and the degree of tension due to the height of the blood-pressure. To determine this point the artery under observation is compressed and the wall of the peripheral portion is examined by palpation before the recurrent wave has entered it. The blood-pressure is then examined according to the principles to be laid down presently. In this way, by observing accurately and duly weighing the findings, it will be possible, in many cases, to make this distinction; but there will always remain a large number in which it will be impossible, or at least inconclusive. This explains various diversities of opinion in regard to arteriosclerosis.

Diffuse hardening of the arterial wall occurs under circumstances the nature and significance of which we do not as yet know (compare the section devoted to the Circulation During the Period of Development), and, in addition, in chronic nephritis and arteriosclerosis. The latter is undoubtedly by far the most frequent cause, and it is not infrequently possible in this disease to feel the individual calcareous deposits distinctly in the vessel-wall.

Tortuosity of the arteries in itself has not the same significance; for it may be due solely to an increase of the blood-pressure. The condition may also be due to abnormal softness and want of resistance of the blood-vessel wall, although this cause need hardly be considered in actual practice; for, as a matter of fact, diminished elasticity of the vessel-wall, as a rule, represents the result of a former overdistention from heightened blood-pressure or of arteriosclerosis, or both.

We next note the *character of the blood-current within the arteries.*

The nature of the current within the arteries depends, as every one knows, on a number of factors: the filling and emptying of the heart; the condition of the arteries, especially their resistance; and, finally, the influences exerted on the

veins (as, for example, the negative pressure of the heart and lungs, muscular movements, and the like). But of all these factors, the heart action is the most important and probably also the most variable, and under very many circumstances we are perfectly justified in basing our judgment of the functional activity of the heart on peculiarities observed in the arteries.

In the arteries we note, first of all, the height of the blood-pressure.

In practice the blood-pressure is determined by feeling the tension of the radial artery. The use of this vessel for examination is sanctioned by time-honored custom, and there are many good reasons for it, especially the great accessibility of the vessel, which makes it possible for any physician to perfect himself in the practice of feeling the pulse; and practice in this matter is most necessary.

The surest method of gauging arterial tension consists in determining how much muscular strength the palpating finger must exert in compressing the radial artery against the bone so as to cause disappearance of the pulse beyond the part compressed. A good way is to compress the radial artery against the styloid process with one index-finger while feeling for the disappearance of the pulse with the other. [Still better, to use three fingers, and compress alternately with one, while feeling the vessel with the others.—Ed.] In practice, however, this is not often done, as the same finger is generally used to compress the artery and feel the pulse.

It is true that we can only *estimate* the blood-pressure in this way. The accuracy of estimates thus obtained, however, is quite sufficient for the needs of ordinary practice, provided the examination is always made with due care and by a practised finger.

In this way it is, of course, the maximal pressure that is found, but this is not an important point; for one gains at the same time a sufficiently accurate idea of the mean pressure, as the pathologic fluctuations of the latter are much greater than the relatively small difference between maximum and mean pressure.

[Much can be discovered by a cultivated pulse palpation, but certain possibilities of error must be recognized at the outset. Maximum pressure can be estimated with great accuracy in some cases, and in these the greater the experience, the more accurate the guess. In some other cases the expert realizes that he cannot make a guess that satisfies himself; and in still others, in general the most important cases on account of the pathologic conditions, the guess even of an expert may be wide of the mark —sometimes as much as 80 to 100 mm. These statements are based upon several years' experience in which several clinicians of varying length of training have recorded guesses made after careful palpation, and compared the record with the results of various blood-pressure apparatus. Careful training, comparing palpatory impressions with sphygmomanometer findings and sphygmograms, will make one more accurate, but also more reserved, in the interpretation of the pulse by the touch.—Ed.]

For determining the mean and maximum blood-pressure in animals more accurate methods are available, as it is, of course, permissible to open an animal's arteries. After many experiments and much effort expended in this direction we now have instruments of precision for man which can be used without causing any injury whatever. I will mention two methods—that of v. Basch* and that of Riva-Rocci.† The principle of both of these instruments consists in measuring the pressure necessary to cause complete compression of an artery (in the case of v. Basch's instrument the radial or temporal; in the case of Riva-Rocci's, the brachial). By compression is meant obliteration so that the pulse disappears at the periphery. In both cases, therefore, we measure the maximum of the arterial pressure plus the pressure necessary to overcome the rigidity of the vessel-wall

*v. Basch, "Zeitschrift für klinische Medicin," vol. ii, p. 79. *Ibid.*, "Berliner klinische Wochenschrift," 1887, No. 11 and 12.

†Gumprecht, "Zeitschrift für klinische Medicin," vol. xxxix, p. 337. Hensen, "Archiv für klinische Medicin," vol. lxvii, p. 436.

and the covering soft parts. The sum of these two factors is unquestionably very inconstant, and under certain circumstances, such as arteriosclerosis, for example, is probably much greater than is generally stated. In Riva-Rocci's method it seems to me the influence of the muscle tone in the upper arm should not be underestimated, and I am sure that this factor has not as yet been sufficiently investigated.

Hence, in comparing the blood-pressure in different individuals, both methods must be used with a certain caution; but the fluctuations in the blood-pressure in the same individual can be quite accurately determined by means of either.

[Instruments of the von Basch type are now superseded by those of the Riva-Rocci type. Of these, the modifications of Cook, Stanton, and Janeway are all useful, but should be supplied with the wide cuff (10 to 15 cm., usually 12 cm.), as recommended by von Recklinghausen. Such instruments readily give the systolic pressure, and this alone is of great value, since it shows "the strain to which the arteries are subjected" (Erlanger) and furnishes therapeutic indications in diseases with high arterial tension. But from a broader standpoint, and in many clinical conditions, it is essential to know the diastolic pressure, since this is nearer to the mean pressure. The most accurate apparatus is that of Erlanger, but its use must be restricted to the clinic and consulting-room. For medical purposes it is fortunate that it is sometimes possible to estimate the diastolic pressure with considerable accuracy by methods applicable to the simple blood-pressure machines mentioned above. One of these depends upon the fact that, as the mercury in the manometer nears the point of minimum pressure, it shows wide oscillations—usually they should be not less than 5 mm. They are more frequently seen in cases with large pulses. Strasburger's method consists in determining when the radial pulse suddenly becomes distinctly smaller as the pressure rises in the tube, or, conversely, when the pulse becomes larger as the mercury falls. Both methods require practice, and in many cases fail. Sahli's method, by noting the change in the height of the sphygmogram at different stages of pressure on the artery above, is not really simpler than Erlanger's, and not more easily applied. The details of the important matter of sphygmomanometry cannot be included here. There is a large and rapidly growing special literature. See especially Janeway, T. C., "The Clinical Study of Blood-pressure," New York, 1904. Morris and Edmunds, "Observations on the Blood-pressure in Disease," "The Medical News," January 14, 1905. A. D. Hirschfelder, "Some Observations upon Blood-pressure and Pulse Form," "The Johns Hopkins Hospital Bulletin," June-July, 1907, p. 262.—Ed.]

The magnitude of the blood-pressure or, as the physician expresses himself, the tension (hardness) of the pulse, depends on the relation existing between the quantity and velocity of the column of blood that enters the root of the aorta and the arterial resistance. We know that diminution of the latter alone may produce considerable lowering of the blood-pressure; but more frequently abnormal softness of the pulse must be ascribed to insufficient contraction or filling of the left ventricle. Hence the tension of the pulse is the most important criterion of the activity of the left ventricle that we possess. We have no better. That is why it is so very important for the physician to test the blood-pressure.

Another point that interests us in the arteries is *the degree of distention;* this also depends on the relation between influx and efflux—that is to say, if we regard the latter as uniform, it depends, above all, on the volume of blood expelled from the left ventricle at each pulsation. Accordingly, we also find arteries containing a relatively small quantity of blood,

"empty" arteries, chiefly when the contractions of the left ventricle are incomplete, a condition present in so many diseases of the heart. It is obvious that an empty pulse is practically always a soft pulse.

In order to recognize the degree of distention of the arteries the periphery or diameter of a known vessel is determined. By long practice the physician must learn whether the vessel corresponds to the individual's age and general conditions of life or not. In making this observation the character of the vessel-wall must, of course, be carefully taken into account; if the wall is abnormally yielding, the pulse may well be abnormally full, but there is usually no difficulty in recognizing and distinguishing this condition.

By the *size of the pulse* in an artery we mean the increment in the size of the vessel that is produced by the passage of the pulse-wave. This increment depends partly on the relation existing between the pulse-volume [systolic outflow—Ed.] and the time in which the left ventricle empties itself, and partly on the distensibility of the arteries; in addition, there is the behavior of the secondary (reflected) waves. With certain reservations, therefore, a large pulse may be regarded as an index that copious quantities of blood are being expelled from the left ventricle, and a small pulse is in many cases—especially when the condition of the vessels is known—a direct and most important sign of incomplete emptying of the left ventricle. Here, again, it is obvious that a small pulse is soft and frequently also empty.

It is quite possible to determine differences of size with the palpating finger with sufficient accuracy for practical work. Here, again, the importance of practice cannot be too strongly insisted upon; it is much easier to learn to determine the size of the pulse with considerable accuracy than it is to become proficient in judging the tension of the pulse and in recognizing the various types of pulse, which will be mentioned presently.

An exact quantitative method for determining the size of the pulse consists in taking the volume curve with a graduated plethysmograph.* We merely mention this for the benefit of the investigator who wishes to discover new facts in regard to the physiology or pathology of the circulation. The *physician* may dispense with the procedure at present because it is not yet established on a practical foundation. From the volume curve† the *velocity of the blood-current* in the arteries of the extremities in man can be calculated, or it can be more conveniently determined directly by v. Kries'‡ method. In the hands of expert pathologists this method is no doubt destined to produce much valuable information. We shall not enter into a discussion of it, however, because it is not sufficiently well founded to be of any use to the physician.

Certain phenomena of the arterial pulse, namely, its frequency, regularity, and uniformity—I take for granted that it is not necessary to define these expressions—are directly and without reservation dependent upon the activity of the heart.

Here, again, the results obtained by palpation are quite sufficient for practical purposes; but, if many of the pulses are small, the examination must be made on an artery situated more centrally than the radial. Under certain circumstances the frequency is best determined by palpation or auscultation of the heart, a method

* See v. Frey, "Die Untersuchung des Pulses," p. 60.

† Fick, "Untersuchungen aus dem physiologischen Laboratorium in Zürich," Wien, 1869. *Ibid.*, "Verhandlungen der physikalisch-medicinischen Gesellschaft zu Würzburg," 1886.

‡ v Kries, "Du Bois' Archiv," 1887, p. 254. Compare v. Frey, "Untersuchung des Pulses," p. 64.

which becomes necessary whenever some of the contractions of the left ventricle are so weak that they fail to produce any pulse in the accessible vessels. In determining the frequency of the pulse, a watch with a good second-hand should be used and the full quarter or half minute [not less than a full minute in important cases—Ed.] should be counted; the first beat, which corresponds with the zero-point of the second-hand, is counted as zero as in determining any other kind of measurements. The pulse-count must be used with caution if the individual is in a state of bodily or mental excitement, particularly in heart patients, as under such circumstances one might easily obtain an entirely erroneous idea of the frequency of the heart-beat.

There remains to be considered the examination of the *type of pulse*, that is, the exact time relations of the fluctuations of pressure in the artery. Here, again, palpation with the finger suffices for all practical purposes, providing the observer possesses sufficient practice and the examination is carried out carefully. The shape of the pulse-wave can, however, be determined very much more easily with the eye than by the pressure sense, and we have in many of the modern sphygmographs, as, for example, the one designed by M. v. Frey, excellent contrivances for obtaining a graphic record of the pulse.

[For general medical work the Jaquet chronosphygmograph is the best. The time recorder (in this instrument giving one-fifth seconds at two speeds) is essential for determining the rhythm as well as the character of the pulse. The instrument has been made additionally useful by the cardiograph and polygraph attachments, so that one or two tracings may be made besides the radial, *e. g.*, apex, jugular, liver pulse, etc.—Ed.]

But it must be distinctly stated that, so far as the diagnostic value of the sphygmograph is concerned, our hopes are all in the future. This statement is apparently contradicted by the results reported in the literature on the subject, which is quite extensive; but it cannot be successfully denied that the sphygmogram shows merely the shape of the pulse.* Conclusions as to the size of the pulse and the blood-pressure drawn from the shape of the sphygmogram are indirect conclusions and must as yet be accepted with the greatest caution. For my part I am inclined to give a most favorable prognosis for sphygmography, both for diagnostic purposes and for the theoretic interpretation of a great many processes in the circulation. But before this hope can be fulfilled the shape of the pulse-curve in the individual arteries in healthy animals and human beings must be more accurately analyzed, and we must make ourselves acquainted with the many extremely complicated conditions that influence the shape of the curve. It is, unfortunately, true that almost all that has been learned so far is uncertain, and we must insist upon the greatest skepticism on the part of physicians. Old, convenient dogmas, such, for example, as the belief in a direct parallelism between diminution of the arterial tension and pronounced dicrotism of the radial pulse, which have become deeply rooted, are of no value. Such a parallelism may exist; but the two processes cannot by any means be regarded as absolutely linked together without any reservation, for a great many different factors influence the position, size, and number of the individual secondary waves, and these things all require more accurate study before any definite conclusion can be reached.

When the pulse-wave rises abruptly and rapidly falls again, the pulse is described as "bounding" or "jumping" ("celer," "quick"); this type of pulse occurs most typically in marked cases of aortic insufficiency for reasons which need not be explained here.

The rapidity with which the circumference of the artery increases depends partly on the systolic output and the time occupied by the left ventricle in emptying itself: thus, in cases of dilatation and hypertrophy, a rapidly rising pulse is often observed. The tension of the arterial walls, of course, also exerts a considerable influence. The shape of the depres-

*In regard to these matters consult the excellent monograph by v. Frey, "Die Untersuchung des Pulses," Berlin, 1892.

sion between waves is determined chiefly by the character of the flow toward the periphery.

The systolic rise of the artery is abnormally gradual when the left ventricle contracts slowly and the so-called "pulsus tardus" or slow pulse is produced. This type is also observed in its most typical form in a valvular lesion, namely, aortic stenosis; but occurs also when the contraction of the left ventricle is slow for other reasons.

To what extent the combined use of tactile and visual impressions has confused our ideas on this subject is shown by the fact that the pulse in lead colic or arteriosclerosis is frequently described as "slow" (tardus), although the distention of the artery is by no means always effected slowly. It is true that the transition from the smallest to the largest diameter is slow to the touch; but this is not infrequently due to the presence of secondary waves, resembling anacrotic waves, so high on the descending (postsystolic) leg of the pulse-curve that they in reality produce the greatest distention of the vessel. In other words, the conditions are altogether different from those which obtain in the first-named variety of "pulsus tardus"

Clinical medicine has been enriched by the acquisition of a number of concepts and technical terms corresponding to various types of pulse the recognition of which is based partly on the sense of touch and partly on the sphygmogram, and the combination of the latter with a great many other peculiarities. It is now high time to examine these complicated conditions seriously, according to the principles of hydraulics and with the aid of rigidly accurate methods. Excellent physiologists have preceded us. Fick, v. Kries, v. Frey, Hürthle, have pointed the way. Let us follow them!

In every case one should observe carefully whether the pulse in symmetric vessels on both sides of the body is the same in every respect. If any differences are discovered, we must determine whether they can be explained by local anomalies in the arteries or by functional conditions, such as diminished muscular development or palsy. If that is not the case, the cause must be sought in the aorta. Although we are not directly concerned with diseases of the aorta in this discussion, arteriosclerosis is such an important factor in heart disease that asymmetry of the pulse may be useful as an indication of the cardiac condition.

In most healthy individuals* two sounds are heard over the *carotid* and *subclavian arteries;* the first is identical with ventricular systole and, as compared with the character of the heart-sounds, is impure. Its origin is still obscure. I shall refrain from discussing the question because additional observations are indispensable before such a discussion is possible. One thing, however, may be mentioned: The sound is generally referred to vibrations taking place in the walls of the artery during its distention; it is probable that the first sound of the heart takes part in this. But the conditions are evidently complicated, and for the present difficult to understand.†

The second sound heard over these two vessels is unquestionably due to the closure of the semilunar valves. It is distinctly more pure and higher in pitch than the first. Both sounds are influenced in a great variety of ways by the sounds produced in the heart. For our purposes it should be mentioned chiefly that systolic murmurs at the base of the aorta due to arteriosclerosis are usually propagated very distinctly into these two large vessels. This is an important point; but it must be remembered, at the same time, that murmurs are frequently heard, especially in the subclavian artery, synchronous with its pulse, and having

*Weil, "Die Auscultation der Arterien und Venen," Leipzig, 1875.

†Compare v. Frey, "Die Untersuchung des Pulses," p. 6.

no connection whatever with any changes at the base of the aorta. It is at present impossible, in my opinion, to give a common cause for these well-known phenomena; for even in the same individual they vary quite a good deal. As a rule, there is no difficulty, so far as I can see, in distinguishing them positively from murmurs due to sclerosis at the base of the aorta. In the first place, the latter are usually much rougher and more irregular and are always audible both in the carotid and subclavian, or even chiefly in the former, while the peculiar phenomena of arteriosclerosis predominantly affect the subclavian artery.

In the remaining arteries of the body auscultatory phenomena are much more rare, and as they have no direct bearing on the diseases with which we are here concerned, we need not discuss them further.

Examination of the Veins.—In the veins the blood flows slowly under low pressure and with slight velocity. The column of blood is kept in motion by a feeble remainder of pressure from the left ventricle and by the movement of the muscles, aided by certain special contrivances and the suction exerted by the thoracic cavity and the right ventricle. Owing to their great softness, the walls of the veins are easily compressed by a great many different causes, and thus various local venous stases are produced. Diminution of the negative pressure within the thorax may cause general venous stasis; but these things do not concern us here as we have to deal only with distention of the veins due to anomalies of the heart. Such a condition always indicates weakness of the right ventricle; the congestion, it is needless to say, affects all the veins, and under certain conditions leads to the production of edema, as will be explained later.

Marked swelling of the veins of the body is not by any means always an indication of weakness of the right ventricle. We all know that the development of the cutaneous veins varies greatly in different individuals, and that it is greatly favored by constant muscular exercise of certain portions of the body. Thus there are persons in whom the cutaneous veins of all the extremities and of the trunk appear to be abnormally distended.

The severer grades of pathologic venous stasis are quite unmistakable, and their diagnosis need not be discussed; but slighter grades of congestion may easily cause diagnostic difficulties. In such cases the veins near the heart which are accessible to observation, especially the external jugulars, are first examined. These veins also exhibit different degrees of development in different individuals, even in health. Movements are sometimes observed in these veins which are either quite irregular or are connected with respiration: the vessels swell during expiration and collapse during inspiration. In cases of cardiac stasis the jugular veins are greatly distended; as a rule, the respiratory fluctuations are increased, and very frequently regular variations, or "pulsations," which are dependent on the action of the heart, are observed. Two kinds of pulsations are distinguished.*

In cases of venous stasis the valves beginning above the bulb of the jugular vein may become insufficient, and, as a result, the natural movements of the large venæ cavæ are propagated to the external jugular veins. The greatest distention of the venæ cavæ is synchronous with the contraction of the auricles, after which the vessels empty their contents into the auricles and ventricles and collapse. I purposely refrain from giving a minute description of the peculiarities of the changes of pressure that

* An important paper by Riegel, "Archiv für klinische Medicin," vol. xxxi, p. 1. For the literature see D. Gerhardt, "Archiv für experimentelle Pathologie," vol. xxxiv, p. 402.

have been rendered familiar by the graphic method because, as yet, they have no significance for the physician.† This "normal" pulse of the large venæ cavæ is called *negative* because its greatest distention is not coincident with ventricular systole, but precedes it, and this *negative* pulse is propagated to the external jugular veins whenever the valves of these veins do not prevent it—that is, whenever the vessels are sufficiently dilated. At the instant of ventricular systole veins with a negative pulse, therefore, collapse. Hence O. Vierordt's expression, "systolic" venous collapse, is eminently appropriate. Occasionally this venous pulse is also observed in healthy individuals.

For purposes of diagnosis it is important to recognize whether the jugular veins attain their greatest circumference immediately before or coincidentally with the apex-beat. Hence the latter is palpated with the finger while the movements of the venæ cavæ are observed. It may be quite easy, it may be difficult, or it may even be impossible to make this distinction, depending partly on the rapidity and rhythm of the cardiac movement and partly on the respiration. During systole the vein in such cases, therefore, collapses *quickly;* and that also, as O. Vierordt quite correctly pointed out, may have diagnostic significance.

In a few cases a tracing of the venous pulse and of the apex-beat may assist in diagnosis, but such rare cases, in which this is possible, need scarcely be taken into consideration in practice. On the other hand, it may be very useful to ask the patient to hold his breath during inspection of the veins.

A "*positive*" *venous pulse,* that is, maximum distention of the veins of the neck coincident with ventricular systole, is always a pathologic condition and is characteristic of tricuspid insufficiency. This valvular lesion interests us because it is frequently due to insufficient contraction of the right ventricle and, therefore, occurs not infrequently in the conditions with which we are dealing. The distinction from the negative pulse has already been discussed; it should now be mentioned, in addition, that a positive venous pulse may be simulated by pulsations transmitted to the veins from the carotids. When that is the case, if the vein is compressed with a finger or a pleximeter, pulsation ceases in the central segment because the latter is empty.

This positive venous pulse may also be propagated to the smaller and more distant vessels, and sometimes sounds synchronous with the ventricular systole may be heard in the cervical vessels or even in the femoral veins. Aside from this, auscultation of the veins practically yields nothing of importance for our purposes. It might be well to utter a caution in regard to the danger of mistaking an anemic murmur for one derived from the heart.

GENERAL ETIOLOGY OF MUSCULAR DISEASES OF THE HEART.

The causes that lead to pathologic conditions in the myocardium may be divided into two main groups: The first includes a variety of chemic or morphologic bodies circulating in the blood—that is to say, poisons and parasites; the second group consists of certain mechanic factors which are brought into play whenever an excessive demand is made on the heart.

*Compare D. Gerhardt, *loc. cit.*

Substances circulating in the blood may attack the heart through the endocardium. In this way the so-called endocarditic processes are brought about. These do not fall within our province. For the myocardium the coronary circulation appears to be more important than the endocardium as a portal of entry for injurious substances in the circulation.

Hence the myocardium is exposed to injury in all conditions associated with the circulation of noxious bodies in the blood. Of these, the infectious diseases are the most important and very frequently produce changes in the myocardium which can be distinctly recognized, and the pathologic significance of which is not difficult to estimate; these changes consist in true inflammation and parenchymatous degenerations. The heart in this respect does not differ essentially from other organs; in many parenchymatous tissues inflammatory and degenerative processes develop under the action of infections and, for reasons that are not always quite evident, certain organs are chiefly attacked in different processes. The heart, like the kidneys, does not always escape; but, like the kidneys, the heart is specially liable to injury in certain infectious diseases; and, what is still more curious, certain infectious diseases chiefly attack certain portions of the heart.

The disease may affect the endocardium, the myocardium, or the pericardium. As a matter of fact, all three structures are usually attacked, at least in the anatomic sense; but to the physician the symptoms of one portion of the heart usually appear so much more pronounced that it has become the custom to speak of disease of such and such a portion.

In this discussion we shall describe only the abnormal conditions of the heart muscle, that is, cases in which inflammation of the muscle is distinctly the chief thing, both anatomically and clinically.

Acute myocarditis, like acute nephritis, may be caused by any one of the infectious diseases; but the most important, so far as the heart is concerned, are rheumatic polyarthritis, diphtheria, typhoid fever, scarlet fever, malaria, syphilis, streptococcus, staphylococcus, and gonococcus infections, smallpox, and erysipelas.

In polyarthritis the endocarditis clinically appears to be the most important condition. But a careful study of the concomitant phenomena will convince any one that many of the symptoms must be charged to a deficiency of the muscle, and pathologic investigations have also taught us that, as a rule, not only the endocardium, but the muscle also, is profoundly involved.* The combination of endocarditis and myocarditis occurs in the other infectious diseases also, but myocarditis alone is much more often, both pathologically and clinically, the essential morbid process.

The nature of the pathologic changes will form the subject of a special section. We have no positive knowledge about the true nature of the relationship between infectious disease and muscle lesion. It is probable that the poisons generated by the microbes represent the active agent, and future observations may possibly show why these poisons are produced or reach the muscle-fibers in sufficient concentration to cause disease in certain cases only, or, finally, why the fibers are only at times vulnerable.

It is possible that inflammation of the arteries of the heart forms part of the pathologic process in infectious diseases. The inflammation in

*Krehl, "Archiv für klinische Medicin," vol. xlvi, S. 484. Romberg, *ibid.*, vol. liii, p. 141. Both contain recent observations and the latest literature.

that case would affect the function of the heart in the same way as sclerotic processes in the coronary vessels.

In addition to the poisons generated by bacteria, those imported from without may injure the heart muscle; and this suggests an important point in the pathology of diseases of the heart, which we encounter again and again, namely, the combination of several injurious agents, especially the association of intoxication with mechanic influences. By far the most important of these injurious substances is *beer; wine*, and especially *brandy*, are of distinctly secondary importance in this respect.*

It is possible that alcohol is the injurious agent, for there is no lack of analogy. We know its injurious effect on other tissues, such as the kidneys, the liver, vessel-walls, and nerve-fibers. It is true that alcohol, as a rule, injures the heart only when taken in large quantities and during a considerable length of time, although it must be admitted that the injurious dose varies greatly for different individuals. To what extent other chemic substances besides alcohol must be incriminated it is impossible at the present time to say, and in any case it must be remembered that the whole question is extremely complicated. If the ingestion of alcohol alone were the important factor, diseases of the heart would be most common and most severe among whiskey [orig.: "Schnaps"—stronger alcoholic beverages in general—Ed.] drinkers, which is certainly not the case. But other factors besides the ingestion of alcohol come into play when beer, and also to a certain extent when wine is taken, such as the introduction of large quantities of fluid, the abundant ingestion of nutritive material, and the obesity which beer-drinkers so often develop as a result of their mode of life. There is another consideration. Not a few drinkers are heavy smokers; not a few commit sexual excesses; and, most important of all, many of them, in addition to indulging in alcohol, are engaged in work that calls for great muscular exertion. These things will be discussed again later.

Still less clear is the significance of *smoking*. It may be confidently asserted that in many individuals smoking produces cardiac disturbances which can be recognized both subjectively and objectively, and consist in more active contraction of the heart, with a feeling of palpitation and anxiety.

Many of us remember these symptoms from our first attempts at smoking; once one has become accustomed to the habit, they usually disappear altogether, even when large quantities of tobacco are consumed. They may, however, occur in seasoned smokers if they transgress their limit, which, to be sure, varies greatly in different individuals, or if the heart is suffering from some other injury—this has already been referred to. The cardiac symptoms are particularly apt to be brought on by the free use of so-called imported Havana cigars. Wherein the difference consists we are as yet unable to say, but no experienced physician can doubt the truth of the assertion; everybody is agreed that there is an enormous difference between the effect of imported cigars and that of the cheaper, domestic grades.

Some maintain, while others deny, that nicotin is the only active poison. Vas has shown that nicotin is the only active substance in tobacco smoke; but in my opinion this does not prove that the intoxication is caused solely by the smoke. Some one ought to investigate why the imported varieties of cigars, which are said to contain very little of the alkaloid, are so injurious and what the peculiarities are that render the heart especially sensitive.

The effect of strong *coffee* on the heart is quite similar. In this case caffein is unquestionably the active agent. The effect of this alkaloid

* The literature bearing on this point will be found in the section on the Diseases of the Heart Caused by the Abuse of Alcoholic Beverages.

† Vas, "Archiv für experimentelle Pathologie," vol. xxxiii, p. 141.

on the circulation is given in the section devoted to the discussion of drugs, and it is quite in line with what is said in that connection that coffee-poisoning is followed by an increased contraction of the ventricles, palpitation, compression, and anginoid attacks. Two factors contribute to the production of these conditions: the direct stimulation of the heart by the poison and the increased strength of the contractions in response to vasomotor irritation. The latter is unquestionably due to caffein. That the excessive use of coffee ultimately leads to symptoms of cardiac insufficiency is quite possible, although I do not know that it is the case.

Again it should be borne in mind that in our social gatherings the copious drinking of good, strong coffee is frequently combined with indulgence in large quantities of wine and beer and the smoking of expensive, and therefore imported, cigars, so that here again the combination of several different injurious agents unquestionably affects the result. In the case of coffee, as in the case of cigars, the dearer preparations are most injurious to the heart. Not only is a much larger quantity of the drug used in making the decoction, but the kind of coffee selected is "stronger," that is, it probably contains more caffein. Another possibility that cannot be altogether excluded is that some other active substances may be present. And, finally, there is the individual sensitiveness to coffee, which is also extremely variable, but regarding which there is no possibility of agreement.

Finally, the significance of *mechanic influences*, especially overfilling of the ventricles and increased resistance to them, should be mentioned.

Here, also, there is much that is obscure. If a large quantity of blood enters the cavities of the heart during diastole,—assuming, for simplicity's sake, that the resistance is unchanged,—the latter, in emptying their contents, use their power of accommodation and the work of the corresponding portion of the muscle is increased (see the following section). This is true within certain limits, which vary in many respects: for one thing, with the individual portions of the heart. It is true that, as a rule, the entire heart is called into action; it must be remembered, however, that the auricles cannot cope with as much blood as the ventricles, and that the right ventricle is weaker than the left. Moreover, the heart's power of accommodation varies greatly in different individuals, and, finally, the structural condition of the heart muscle has an enormous influence; for it may be accepted as a fact, confirmed by physiologic experience, that the so-called reserve strength of the heart is directly dependent on certain qualities of the musculature.

When the resistance in the circulation is great, the demand on the heart is also greatly increased; this calls out all the power of accommodation of the organ for the management of which the same factors are concerned that have just been mentioned.

In fact, abnormal and excessive diastolic filling of the ventricles usually combines with a good deal of resistance in the arterial system, and the effect on the heart is thereby greatly increased. This is always the case when violent muscular movements coincide with forced expirations—that is, during severe bodily exertion.

Owing to the excessive pressure and differences in pressure, the mitral, and more frequently the aortic, valves in such cases not so very rarely suffer laceration, or a portion of the valve may even be torn off. This leads to the production of valvular lesions, which, as a rule, are severe, and the suddenness of their development makes a heavy demand on the power of accommodation of the muscle.

This point need not be discussed further.

What is the condition of the myocardium on such occasions? That is the question that interests us most. The combined effect of large quantities of blood in the ventricles and of the systolic pressure on the heart-wall may produce a condition analogous to overdistention of an elastic body.

Such a condition is specially favored by a number of qualities inherent in the muscle. E. Weber* has shown that the elasticity of the

*Article on "Muskelbewegung" in R. Wagner's "Handwörterbuch der Physiologie," vol. iii, pt. ii, p. 100.

muscle is diminished during contraction, and that the muscle itself is, therefore, more extensible. This explains the occurrence of these cases of hyperdistention under definite conditions, that is, when the ventricles strive to propel an unusually large pulse-volume against an extraordinary resistance and thus tax the elasticity of the contracted muscle to its utmost.

Whether, in an individual case, a single severe muscular strain is followed by overdistention of the heart probably depends on a number of factors—to begin with, on the severity of the strain. It is in the nature of things, and is fully borne out by clinical experience as recorded in the literature, that in most cases of this kind the sudden exertion is made under the influence of some powerful emotion, which is also the reason why the individual forgets to consider the limits of his strength as he would under ordinary circumstances. Every healthy, active man occasionally approaches the limit of his strength; we all know the peculiar sensations that occur when the overfilled heart fails to contract properly: a sense of oppression and constriction, dyspnea, vertigo, and faintness. These symptoms are due to a beginning insufficiency of the heart, such as ultimately develops after violent muscular exertion in every man—in some a little earlier, in others later, according to the strength of the heart and its functional capacity. At the first glance this picture exhibits a great similarity to the conditions found in overdistention of the heart. The subsequent course, however, presents certain characteristic differences in the two cases.

The cardiac insufficiency, which every healthy man ultimately experiences after bodily exertion of a certain degree of intensity and duration, undoubtedly is due, first of all, to fatigue.

The frequent repetition of violent cardiac contractions prevents the organ from recovering completely during diastole; the heart becomes temporarily dilated;* but as the individual is unwilling or unable to keep up the same muscular exertion, fatigue is followed by rest and recuperation, and, after a certain time, the original functional capacity of the heart is restored. These facts also are familiar to all of us.

The result is quite different, however, if the individual continues to exert himself and refuses to yield to the sense of fatigue. Such a course may lead to overdistention and, as will be explained more fully later, a heart lesion not infrequently persists for some time or even becomes permanent. This question will be discussed in detail in connection with diseases following severe muscular exertion.

Here we have to do with the general and significant fact that severe bodily exertion under certain circumstances works the greatest injury to the heart muscle. If it is already diseased, even mild degrees of fatigue are often injurious, and there is, obviously, special danger of overdistention. That is why violent bodily exertion is particularly dangerous for heart patients and may convert a relatively harmless condition, which hardly causes any symptoms and certainly does not in the least interfere with the patient's attending to his work, into the gravest disease.

It is perhaps not too much to say that bodily exertion is such an important factor in heart disease because it is so very apt to injure an organ that has ceased to be entirely sound. This fact has not always been suffi-

*Compare Th. Schott, "Die Ueberanstrengung des Herzens," Wiesbaden, 1898, third edition.

ciently appreciated in the past in treating heart patients by means of exercise.

A few words are necessary in regard to the *kind of exercise* that is especially dangerous to the heart. It is, of course, obvious that we cannot exclude any class of muscular innervation if it has the effect of driving large quantities of blood toward the heart and producing increased resistance in the arterial system. But it is also obvious that in many cases the muscular exertion may be excessively severe, especially when the effort is executed with passion, when the individual has not been sufficiently trained, and in athletic contests.

In these days when athletic feats are stamped as heroic deeds, when the ambition of so many men and women does not mount higher than the wish to distinguish themselves in some form of muscular exercise, when the untrained denizen of the city boldly undertakes the most difficult mountain-climbing, there is plenty of opportunity for indulging in excessive and unwisely executed muscular exercise. I have seen a great deal of injury done to the heart, especially by thoughtless bicycle riding. Every one knows that the exercise requires severe muscular exertion, and that many bicyclists carry themselves so badly that thorax and lungs are subjected to the most unfavorable conditions. Especially when going up hill or against the wind and when ambition and rivalry come into play, the danger-signals are apt to be disregarded and the cyclist very easily overexerts himself. It is my impression that bicycling, except in moderation, does a vast amount of harm even when the heart is sound, and especially when the organ is not quite normal.*

All these factors, which are capable of impairing the function of the heart either suddenly or gradually, have a special interest for the physician because they are capable of causing in the hearts of healthy individuals lesions of all varieties, up to complete abolition of function. The abnormal condition may be brief or long; it may be curable, susceptible of improvement, or hopeless. The conditions in an individual case can only be discussed in connection with the various clinical pictures.

Attention should here be called to a matter which has a very important bearing on the pathology of the heart, and which has already been touched upon here and there; namely, *that the coincidence of several injurious factors plays an important part in the production of heart disease.*

This manifests itself in a variety of ways. It may be, as has just been shown, that one cause prepares the field for the attack of another—an occurrence that frequently happens in pathology, as, for example, in the infectious diseases. Or the simultaneous action of several factors, any one of which by itself is insignificant, may produce an effect which one of them alone would be incapable of bringing about. Other possibilities are conceivable and unquestionably occur, but much remains to be discovered by future investigation in this field. This much may be said, however: there is hardly another department of clinical medicine in which it is so important for the physician to consider a great number of things which apparently are quite trivial in connection with the etiology of the disease, providing they occur in combination. By doing so one not infrequently gains an insight into the occurrence of cardiac diseases which one could not obtain in any other way.

It is, in addition, important to consider *under what circumstances the heart is exposed to certain influences;* for the organ is in a very marked degree dependent upon the general condition of the body.

In this connection deficient nutrition, as Romberg has quite correctly pointed out, is one of the first things to be considered. It is evidently

* Mendelsohn, "Der Einfluss des Radfahrens auf den menschlichen Organismus," Berlin, 1896. Leo Zuntz, "Ueber Gaswechsel und Energieumsatz des Radfahrers," Berlin, 1898.

much more important than actual hunger, because it is much more frequently encountered in disease; and it is not impossible that malnutrition has a much more unfavorable effect on the volume and function of the heart than mere hunger. Analogous conditions obtain with regard to the blood.* It is evident that individuals suffering from chronic malnutrition, which, unfortunately, is not seldom due to social conditions, are constantly exposed to a great variety of influences that affect the heart or throw an extra demand on it, and although they are in themselves quite harmless and would have absolutely no influence in health, these influences may lead to functional disturbances.

This is often seen, for example, in young persons, especially during the period of development, among domestic servants, factory girls, and apprentices. The degree of malnutrition and the amount of work performed in spite of it are subject to very great variation in different individuals, and that is why this etiologic factor is of such great and far-reaching importance.

This is a very complicated question. It is possible that the diminution in the volume of the muscle due to malnutrition may be the direct cause; but it is difficult to obtain any positive data bearing on this point. There is no doubt, however, that the *functional* dependence of the heart on the general condition of the organism is of far greater importance. Malnutrition, when present, is not, as a rule, the only defect in the individual. How often is it not associated with lack of sleep, vexation, excitement, worry, or grief? Any one of these things is capable by itself of reducing the power of the skeletal muscles and probably that of the heart also.

Disturbances of the strength of the heart in every respect similar to those which occur after violent muscular exertion and in conditions of malnutrition are also observed *after severe illness*, a fact which is even more difficult to estimate. For in this case it is necessary, before the points to be investigated can be classified, to show that the disease in question does not directly cause heart disease.

After *diseases of the gastro-intestinal canal* anomalies in the action of the heart are observed which are directly dependent on the affection of the intestinal tract (see the corresponding section). It appears, however, that intestinal disease is also eminently capable of indirectly menacing the functional power of the heart by impairing the general nutrition. This point has been emphasized by Romberg.

Occasionally we encounter cardiac weakness after *infectious diseases*, as, for example, typhoid fever, diphtheria, and especially influenza—sometimes long after the acute symptoms have subsided. During the illness or immediately afterward the heart not infrequently develops a form of genuine inflammation or intoxication (see the section on Acute Myocarditis), and in any disturbances occurring after one of these diseases such morbid processes should be thought of first. Occasionally, however, diminished functional power is observed after an illness of this kind; without any manifest signs of actual disease. Romberg, who has given an excellent exposition of these matters, brought direct proof of this in a case of influenza.†

The facts are, therefore, indubitable, but for all that we must be extremely cautious in venturing an interpretation. For a great many well-known clinical facts connected with the kidneys, the peripheral nerves, and especially the nervous system tend to prove that intoxication is capable of exerting its baneful influence long

* See Krehl, "Pathologische Physiologie," p. 136.
† Romberg, "Herzkrankheiten," p. 766.

after the acute infectious disease has subsided. This is not at all contradictory to our theoretic views, for the processes associated with the production and maintenance of immunity prove that the chemic change in the body substances brought about by the infectious disease persists for some time.

Chronic debilitating and exhausting diseases or any process that affects the organism in a similar way and reduces its power as a whole—our very defective knowledge of these things is my excuse for using this expression, which practically means little—are next in order. In this category we may perhaps include diabetes mellitus and gout. Perhaps! For here again great caution is necessary in assuming the presence of a general "debilitating" influence on account of the close connection between these two conditions and arteriosclerosis.

Romberg also mentions *obesity cures,* and I fully agree with him. I also have seen very bad results from the practice, even when it was not pushed to extremes and no special folly or even violence was indulged in. All such methods of treatment require the minutest care and accuracy; the effect of the treatment on the individual especially must be narrowly watched. In like manner much harm has been done by injudicious *treatment at mineral springs* with strong purgative waters, and the harm cannot be explained by the accompanying exercise alone. There is no doubt that the essence of the trouble resides in certain influences which directly weaken the organism. I need only mention the distinctly injurious effect of an apparently harmless course of Carlsbad water on phthisical patients. What happens in all these cases is that the heart becomes abnormally sensitive not only to injurious influences, but also to the ordinary functions of life. Thus, slight muscular exertion, which is at first perfectly well borne by the same individual, may produce the symptoms of overexertion.

The exact causes appear to me quite unfathomable, nor do I believe that anything will be learned about them in the near future; for the question is connected with the most intimate processes of cellular life, of which at the present time we have not even a glimmering. In the same way we are still altogether in the dark as to why the heart should be the organ that suffers under these circumstances in some individuals. Life shows the most curious things, and the *locus minoris resistentiæ* also enters into the question. For example, a man who four years previously had suffered from acute infectious myocarditis, had completely recovered, as was proved by a number of examinations, and had become capable of any kind of exertion, developed a slight apical cararrh and at once began to suffer anew from marked cardiac symptoms: great acceleration of the pulse and dyspnea during walking, which could not possibly be explained by the pulmonary process.

The question of general nutritional disturbances and their effect on the strength of the heart naturally leads to the question of the *direct nutrition of the muscle-fibers.*

The heart, being an organ endowed with great and almost ceaseless activity, requires an abundant supply of blood, as the facts of physiology prove; for anything that interferes with perfect respiration and nutrition rapidly leads to structural and functional lesions of the heart. Hence the extraordinary influence on the heart muscle of all diseases of the coronary arteries; by reducing the amount of blood going to the heart they diminish its functional power and lead to nutritional disturbances and necrosis of the muscle-fibers. Much, of course, depends on the extent, location, and intensity of the coronary affection; it is well known that stenosis at the orifices of the arteries, for example, may bring about the gravest symptoms.

We now come to the *influence of violent psychic processes* on the circulation, and especially on the heart. Every one knows from his own personal experience how much the heart is influenced by one's mental condition. Psychic impressions especially affect the heart, and in this respect different individuals exhibit different degrees of sensitiveness.

Various kinds of psychic processes, constant worry and care, anger, annoyance, and fear are capable of directly causing heart disease, as the older physicians have believed from time immemorial. It is probable that they are even capable of directly producing death; at least there are some examples that seem to prove such a thing to be possible (see the section on Nervous Diseases of the Heart).

Much more frequently the effect manifests itself on the entire nervous system, and incidentally on the innervation of the heart. In this way some of the so-called nervous disturbances of the heart originate. In discussing sclerosis of the coronary arteries it will have to be shown that this process also may owe its origin to some psychic injury.

Violent mental emotion has an absolutely devastating effect on hearts that are not quite normal; of this I have seen some very sad examples.

HYPERTROPHY OF THE HEART MUSCLE.

The volume of a striated muscle, whether it be a skeletal muscle or the heart, depends on three factors: the natural disposition (Anlage), the peculiar conditions of individual development, and the amount of work performed. The mere ingestion of nutriment in itself is not, so far as we know, capable of increasing the size of normal muscles, or at most only to a very limited extent, unless the parenchyma at the same time develops an increase of its peculiar property; for otherwise the cells cannot assimilate a large quantity of nutritive material. The actual weight of the heart, that is, the mass of its muscle, therefore, bears a direct relation to the body-weight,* or, to be more exact, probably to the bulk of functionating parenchymatous cells, and, since the muscles make up more than half of these cells, especially to the mass of the muscle. The very interesting investigation of Bollinger and his pupils regarding the relative size of the heart in different animals agrees approximately with the view here expressed.† They show that in animals which lead a quiet life and become fat,—sheep, pigs, cows, and oxen, for example,—the weight of the heart as compared to the mass of the entire body is small; while animals that are allowed to run at large and use their muscles actively in running and jumping have relatively heavy hearts. Thus, for example, the weight of the heart in the deer is proportionately twice as great as in man and in the horse, and in birds still greater values are found. With our present knowledge this interesting fact is not altogether easy to explain; there are still several unknown quantities in the equation. The explanations that suggest themselves are the relatively large mass of muscle in wild, running animals, the small quantity of fat in their bodies, the large extent of the surface, and the wide fluctuations of tem-

* W. Müller, "Die Massenverhältnisse des menschlichen Herzens," Hamburg, 1884. Bergmann, "Dissertation," Munich, 1884.

† Bergmann, "Ueber die Grösse des Herzens bei Menschen und Thieren," dissertation, Munich, 1884. Parrot, in Sprengel's "Zoologische Jahrbücher," 1893, vii, p. 496.

perature to which they are exposed; that is, there is an increased demand on heat regulation, which in turn is intimately associated with the increased amount of work performed by the circulation. Peculiarities in the mode of life may also affect the work to be performed by the heart, a point on which our knowledge is still incomplete.

Hence the observations at our disposal are not yet quite sufficient to enable us to draw any final conclusions about the factors which determine the weight of the heart in the individual animal or, to be more exact, in the individual species. The path pointed out by W. Müller and Bollinger must be trodden by other investigators, who will undertake the equally difficult and time-consuming task of finding out to what extent the weight of the heart alone bears a definite relation to the mass of certain tissues, such as the muscles and the large glands, for example, and their relation to definite visceral functions necessarily associated with the mode of life of the individual species.

There is no doubt that definite relations exist. In the general sense the question must be unreservedly answered in the affirmative, simply because it cannot be otherwise. If the assumption that the volume of the muscle is determined chiefly by the extent of its function is correct, the sum-total of the work performed by all the organs in the body must find its expression in the absolute weight of the heart.

In order to obtain a basis for estimating the size of the heart, it must be compared with the entire work of the organism, or, as the latter is practically not mensurable, with the weight of the functionating parenchymatous cells, assuming that the weight is determined by the degree of function. This assumption is unquestionably correct in the case of muscles, and, as the latter make up by far the greater half of all parenchymatous tissue, the above-mentioned theory of a relation between the weight of the heart and that of the skeletal muscles is undoubtedly justified. Parrot's tables, however, contain some very considerable gaps, showing that still unknown factors are at work.

The function of the glands calls for still more accurate investigation. Investigations of the human kidney teach us that the weight of the organ also grows in proportion to its function, and as the latter is possible only in the presence of a sufficient blood-supply, a definite relation probably exists between the weight of the glands and that of the heart.

In man these things must be so; they cannot be otherwise, for these important primary truths, judging by what we know, apply at least to the entire series of vertebrates. In addition, the excellent systematic investigations of W. Müller on a large number of cadavers and the very interesting investigations carried on by Hirsch* in the same way have yielded accurate averages for the relation between the weight of the heart and that of the body. These figures show that individuals who have to do hard manual work also have heavy hearts; but, so far as we are at present able to judge, the weight of the heart in these individuals bears about the same relation to the weight of the body with its well-developed muscles and freedom from fat as in the case of individuals in a moderate state of nutrition. It seems to me, however, that it would be desirable in this connection also to examine a number of healthy individuals engaged in some definite occupation, for instance, men who have to do heavy physical work every day, in order to find out whether the weight of the heart in such individuals exhibits any special peculiarities. Bergmann's† observations on the deer and the observations of veterinary surgeons on race horses, although the bulk of the skeletal muscles has not been de-

* Hirsch, "Archiv für klinische Medicin," lxiv, p. 597.

† Friedberger and Fröhner, "Lehrbuch der speciellen Pathologie und Therapie der Hausthiere," 4th edition, Stuttgart, 1896, vol. i, p. 503.

termined, give one the impression that in these animals, which exercise so violently, the weight of the heart increases at a much more rapid rate than does that of the voluntary muscles. In the case of man, we have no convincing observations. Bollinger's* investigations justify the supposition, however, that, under certain circumstances, very severe muscular exertion in man also causes a more rapid and more marked increase in the size of the heart as compared with the development of the skeletal muscles. In such a case the possibility of hypertrophy due to overwork would naturally suggest itself.

In healthy men who are engaged in very severe muscular work it cannot, at least in the great majority of cases, be proved that the size of the heart is greater than would naturally be expected.

My attention has been given to these matters for the past twelve years, and I have examined a number of men who do the heaviest kind of work every day from morning till night. So long as these men kept healthy I could find no sign of abnormal enlargement of the heart. C. Gerhardt and Romberg had similar experiences. While this is important from a medical standpoint, observations carried on in this way can be applied to questions of biology only with the greatest caution, for it is exceedingly difficult to recognize a slight or even moderate increase in the size of the heart in the living subject. In going over the literature I found one exception to the above observations. Henschen† saw in several Laplanders who were prize skee-runners, hypertrophy of the entire heart or, at all events, an undoubted hypertrophy of the left ventricle, with perfect or even greatly increased functional power.

I must repeat, however, that what we need, above all, is accurate weighings; the results of examinations of living subjects are much too ambiguous to be correctly interpreted. For individuals who do hard work also have well-developed lungs and thorax, and since the size of the heart as determined in the living subject is after all only relative, the boundaries of the heart need not necessarily change in such individuals, even if the absolute weight of the organ has increased.

The physiology of the question is still quite obscure in many respects. During severe muscular exertion there is no doubt that the work of the heart is very materially increased. The work performed by the heart may, with tolerable accuracy, be represented as the product of the mass of blood propelled by the ventricle and the resistance encountered. We shall confine ourselves here to the left ventricle, because the conditions in the right are not sufficiently well known for purposes of discussion. The arterial tension during muscular exercise appears to vary in different cases. In man it is often found—with Basch's sphygmomanometer—to be increased,‡ nor do I believe that the result is due to any faultiness in the method; the same observation was made in the dog,§ while in the horse the arterial tension, in the opinion of all observers, is diminished.‖ The cause of these differences, whether they depend on the kind of move-

* Gocke, "Dissertation," Munich, 1883.

† Henschen, "Mittheilungen aus der medicinischen Klinik zu Upsala," Jena, 1899, p. 53.

‡ Maximowitsch and Rieder, "Archiv für klinische Medicin," vol. xlvi, S. 329. Oertel, "Allgemeine Therapie der Kreislaufstörungen," fourth edition, p. 170. Tschlenoff, "Zeitschrift für diätetische und physikalische Therapie," vol. i.

§ Tangl and Zuntz, "Pflüger's Archiv," vol. lxx, p. 544.

‖ Kaufmann, "Arch. de physiologie," 1892. Zuntz, cited from Tschlenoff, *loc. cit.*

ment, the animal's condition as regards training, or habituation, still remains to be discovered.

It would be useful to know the effect on the blood-pressure of the work which an individual is used to doing every day, as that is the essential point. But even that information might not be decisive.

What we wish to know is the total amount of work performed by the heart. But as the degree of distention and the output of the heart are materially increased during muscular work, as appears probable, at least on general grounds, and has been specifically proved by Zuntz,* the arterial tension ought to undergo a marked fall, even if the work of the heart were merely to remain the same, which is certainly not the case. So far as I can see now, I am inclined to believe that the work of the heart is certainly increased during muscular exertion, although there is no doubt a tendency for the vessels to dilate in order to regulate the action of the heart.†

Under such circumstances it would be remarkable if the size of the heart, as a whole, were not altered at the same time. In my opinion this very positively indicates that, on the whole,—say during twenty-four hours,—the work of the heart is not materially increased as compared with the amount of work performed during rest. The details of this process of equalization need not be discussed.

There is one point I should like to call attention to, however, which is that heart and vessels form a functional unit. We do not know as yet what individual processes regulate the coöperation of the various structures, but there is no doubt that it is of vital importance for the tissues that their functionating cells should receive a plentiful supply of blood under a certain degree of pressure. They are capable of attracting blood to themselves by some unknown forces, causing dilatation of the afferent vessels.‡ The work of the heart during this process need not necessarily be modified; for under certain conditions one system of organs may be almost bloodless, while under different conditions the same system may receive large quantities of blood under high pressure and moving at considerable velocity,—that is, at a time when other vascular regions are correspondingly depleted,—and the work of the heart may remain the same in both conditions.

This procedure on the part of the organism is well known, but we do not know to what extent it is employed.

As in order to preserve life the blood-supply of numerous organs must not fall below a certain minimum, it is obvious that an abundant blood-supply cannot be furnished to a large number of cells at the same time without an *increase in the velocity of the circulation*, and such an increase must, as has already been mentioned, so far as the volume of the ventricular beat increases and the blood-pressure does not fall in the same degree, be accompanied by an increase in the work of the heart.

For our purposes it is important to know to what extent active muscles can receive the large quantities of blood which they need by abstracting it from other organs, that is, without affecting the work of the heart. This depends in part on the strength of the muscular contractions—the intensity of their need for blood. In the second place it depends on the number of muscles engaged. As a smaller number of muscles with a weaker innervation suffices to perform a certain amount of accustomed work than is required to perform the same amount of unaccustomed work, it follows that the work of the heart diminishes with training. Finally, it depends on the need for blood (activity) of other groups of cells. The problem is, therefore, quite complicated, and it will, no doubt, cost much labor to solve it.

The influence of *pregnancy* on the heart is merely the influence of a general increase in the volume of the body. On this point the observa-

* Zuntz, "Deutsche medicinische Wochenschrift," 1892, No. 6 and 13.

† Compare Sommerbrodt, "Ueber eine bisher nicht gekannte wichtige Einrichtung des Organismus," Tübingen, 1882. *Ibid.*, "Berliner klinische Wochenschrift," 1894, No. 5. Oertel, *loc. cit.*

‡ See Bier, "Virchow's Archiv," vol. cxlvii and cliii.

tions of W. Müller,* Bollinger-Dreysel,† and Hirsch‡ are in perfect accord.

Among the variable factors that influence the volume of the myocardium the one that is undoubtedly of the greatest importance is the amount of work performed by it and by the skeletal muscles. The heart possesses the power, by changing its tonicity and its contractility, of adapting itself to a great variety of conditions; within wide limits it is capable of taking up and expelling large quantities of blood and of overcoming great resistance. The heart does this almost in an instant, as soon as the demand is made upon it, and this wonderful faculty of the heart, which alone makes life possible, is designated its *power of accommodation*.§ What this faculty really is we, of course, do not know; but whatever it may be, it is certainly an inherent property of the heart muscle.

The power of the heart to meet the constantly changing demands of the circulation within such wide limits forms part of the wonderful mechanism for the maintenance of the circulation. It goes hand in hand with the changes in the vessel-walls, and, reasoning by analogy, with the physical and chemic regulation of heat, it is probable that the increase in the activity of the heart, which is associated with increased metabolism, is capable of effecting that which fluctuations in the width of the vessels are not able to accomplish.

It has been shown with absolute certainty by v. Frey‖ that the healthy heart, when accommodating itself to altered conditions, also performs more work; that is, whenever the heart adapts itself to an increase in the resistance or to higher degrees of distention (fulness), its work is always increased.** It is true that the evacuation of the ventricles is neither so complete nor so rapid when the resistance is greatly increased and when larger quantities of blood are to be expelled as under normal conditions; but the deficit, both as regards quantity of blood and velocity, is not by any means so large as the increase in the velocity in the former case and the increase in the volume of the output in the latter. Hence it must be accepted that the accommodation of the heart keeps pace with the increase in the amount of work performed.

The power of the muscle-cells to assimilate nutritive material and to enlarge increases at the same time, the necessary material being supplied by a copious flow of blood. The muscle-fibers become thicker than normal.†† The process, which in the normal individual begins at birth and continues until late in life, here exhibits a more rapid evolution. The thickening of the muscle-fibers is undoubtedly the most important result of their increased function. In addition, there seems to be a numeric increase‡‡ as compared with the first process; however, this is doubtless secondary.

* W. Müller, *loc. cit.*, p. 218. For the older literature see Löhlein, "Zeitschrift für Geburtshilfe und Frauenkrankheiten," vol. i, p. 482.

† Dreysel, "Münchener medicinische Abhandlungen," i, p. 3, 1891.

‡ Hirsch, *loc. cit.*, p. 5.

§ The theory underlying these processes is discussed by Krehl, "Pathologische Physiologie," pp. 3 *et seq.*

‖ v. Frey, "Archiv für klinische Medicin," vol. xlvi, p. 398. Compare O. Frank, "Zeitschrift für Biologie," vol. xxxii, p. 370.

** For an accurate exposition of the principles involved see Krehl, "Pathologische Physiologie," pp. 7 *et seq.*

†† For the literature see Goldenberg, "Virchow's Archiv," vol. ciii, p. 88. Original observations in Goldenberg, *loc. cit.* Facilides, "Dissertation," Leipzig, 1870; Tangl, "Virchow's Archiv," vol. cxvi, p. 432.

‡‡ See Zielonko, "Virchow's Archiv," lxii, p. 29, where the literature will also be found. Goldenberg, *loc. cit.* Schalk, "Dissertation," Würzburg, 1882.

The above-described change in the muscle-fibers, therefore, brings about a thickening of the musculature in those portions of the heart which perform an increased amount of work. If the weight of these portions exceeds the mean (as determined by W. Müller's method), we speak of hypertrophy of the corresponding portion of the muscle, and this is the only condition that is called hypertrophy. If a muscular individual has an enlarged ("erstarkt") heart, corresponding in weight to the body-weight, such a heart cannot be called hypertrophic* (see the remarks on p. 485).

The arrangement and function of the cardiac musculature is such that the auricles and the individual ventricles may hypertrophy independently. It is simply absurd to doubt this proposition, as is still so often done; hypertrophy of one chamber does not necessarily induce hypertrophy of the other. This has been proved both by pathologic observations and by animal experiments† whenever attention has been directed to the question, and the phenomenon is in entire accord with our views in regard to the course of the muscle-fibers in the heart.‡

The relation existing between increased work of the heart and the development of hypertrophy of the muscle is so close that the former can be positively deduced from the latter, provided only the diagnosis of hypertrophy is assured.

This is often quite difficult, and, in fact, the only method that is absolutely above criticism is that of W. Müller, which consists in determining the weight. Mere inspection frequently yields doubtful results; for there are two factors with reciprocal relations to be considered: the thickness of the muscular wall and the width of the cavities. There is no need here to enter into further detail, but it was necessary to point out the difficulties that exist, because it is only by taking them into account that we can understand why so many of the statements are uncertain and why we are so little acquainted with apparently the simplest things.

The degree of hypertrophy of a given portion of the heart must be regarded as a direct measure of the degree of increase in the work, which cannot be determined by the duration§ of the hypertrophy nor, above all, by the general condition of the organism. Again and again we meet with the assertion that cachexia may prevent the development of hypertrophy. But this is inadmissible on theoretic grounds alone; for the muscle either performs the increased amount of work demanded of it, and in that case must hypertrophy, or hypertrophy fails to take place under conditions that call for its development, and in that case it is always a sign either that the work demanded of the muscle is not increased or is not being performed. In the latter event cardiac weakness (failure) is the inevitable result.

Experience is unanimous on that point: cardiac hypertrophy is observed in persons in the most depraved state of health, and in the animal also the condition develops even when it is enormously emaciated.‖

The growth of the muscle-fibers, when the work of the heart is increased, harmonizes with our knowledge of cellular physiology.** If the cell is acted upon by stimuli to assimilation, and sufficient nutritive material is supplied, growth takes

* Bauer, in Bauer and Bollinger, "Festschrift für M. v. Pettenkofer," Munich, 1893.

† Compare Romberg and Hasenfeld, "Archiv für experimentelle Pathologie," xxxix, p. 341.

‡ Krehl, "Abhandlungen der Sächsischen Gesellschaft der Wissenschaften. Mathematisch-physikalische Classe," vol. xxvii, No. 5.

§ See Romberg and Hasenfeld, *loc. cit.*

‖ Tangl, "Virchow's Archiv," vol. cxvi. Romberg and Hasenfeld, *loc. cit.*

** Compare Verworn, "Allgemeine Physiologie," second edition, p. 534.

place. The blood-supply of the muscle always increases in response to heightened function, and in the same way, if the cell is stimulated to greater activity, it acquires the power not only to make good the loss of substance, but also to grow—that is, to bring about numeric and volumetric increase of the individual cells. Both processes are limited by the changes in metabolism associated with enlargement of size. That is why, when the extra demands on the heart exceed a certain limit, cardiac insufficiency instead of additional hypertrophy is the result.

The *cause* of cardiac hypertrophy may be any one of the processes that increase the work of a muscular part. The individual conditions will be found discussed at length in connection with the different diseases. Essentially, cardiac hypertrophy results from the necessity of propelling larger quantities of blood, of overcoming increased resistance, or a combination of both factors; and each of these may be due to a variety of causes in the individual case.

I should like to call attention to a matter which seems to me to possess peculiar importance. Any etiologic factor, whether it be known—as in the case of valvular lesions—or unknown—as in the case of diseases of the kidneys, which are positively known to produce hypertrophy—acts continuously, day and night, with each contraction of the heart. It is perhaps more than an accident that other factors, on the contrary, which are given as occasional causes for the development of cardiac hypertrophy, but are not by any means generally recognized as such, only act at times and, as compared with the entire day of twenty-four hours, often for only a comparatively short time. To what extent the condition can be neutralized during the intervals we cannot as yet state positively, as this again depends on certain factors that have been mentioned above: thus, severe bodily exertion causes an increase in the weight of the heart in healthy individuals; but, so far as we know at present, the increase is not greater in proportion than that of the skeletal muscles, and, of course, work of this kind is only performed at long intervals.

A very important point, therefore, urgently demands further investigation, *i. e.*, the amount of work done by the heart in the intervals between excessively hard work. For the purely mechanic explanation of these forms of cardiac hypertrophy, in which the increase in the work of the heart is never persistent and uniform, hinges on the question whether the excessive amount of work performed by the organ at certain times is or is not completely recovered from (equalized) at other times (during rest). Does a diseased heart or the heart of an individual exposed to certain definite influences differ from that of a healthy individual? This, therefore, is another point that may possibly have considerable influence on the development of hypertrophy, and there is at least one more that deserves consideration. Accommodation of the heart follows increase of its work. Although it is not known what determines the measure of accommodation, the view that a diseased parenchyma, or one that is exposed to the action of substances of any kind, is abnormally irritable, and therefore responds even to a slight stimulus by an abnormally increased activity, is not to be altogether rejected. There are many analogies, as, for example, in the nervous system. Under such circumstances it is evident that cardiac hypertrophy must easily develop.

That portions of the heart which actually perform an increased amount of work for any length of time undergo hypertrophy can no longer be doubted. But it is not necessarily true that the stimulation incident to the performance of function is the only cause of increase in the number and size of the muscular fibers. Processes associated with inflammation have again and again vindicated their claim to the possession of the same faculty.* There is no doubt that an inflammatory process is capable of inciting abnormal proliferation of parenchyma cells, as in the case of epithelium. Whether the same process takes place in the heart does not as yet appear from the available material. The assertions advanced

* Bard and Philippe say, for example, in their excellent treatise on chronic myocarditis ("Revue de médecine," 1891, p. 351), that hypertrophy of the muscle is a constant phenomenon in chronic myocarditis.

by Buhl* can no longer be utilized, but I have myself frequently gained the impression that a myocarditic heart was hypertrophic in cases in which I could not attribute the condition to any one of the known causes of hypertrophy. I will not venture to call it more than an impression, because I, unfortunately, did not carry out any careful weighing. It is possible that this question, which is so vitally important, might be decided by W. Müller's method, and until it is decided, speculations about the nature of the process may as well be abandoned. There are, in fact, several possibilities, none of which are very remote.

The *importance of hypertrophy* for the functional capacity of the heart is unquestionably very great. While at first it may only be a sign that the organ is responding to an increased demand, after a time it is not too much to say that, although a muscle of abnormal volume may be able to perform a considerably greater amount of work, such a thing is only possible through increased exertion of the muscle, which, of course, lessens its power to respond to still further demands. After hypertrophy has developed, on the other hand, the muscle again enters a state of equilibrium, which enables it to respond to an increased demand for work without any special exertion. Hence the faculty of accommodation is also increased, and by virtue of this faculty the muscle is able to propel more blood and overcome greater resistance in response to increased demand nearly or quite as in the case of a healthy heart.

This view of the importance of hypertrophy is confirmed by numerous clinical observations. One often encounters persons with cardiac hypertrophy in whom the capacity of the organ, especially when the original trouble which is responsible for the hypertrophy is taken into account, is actually the same as in a healthy person, and continues so for years or even decades. Other physicians besides myself have frequently made this observation.†

In general we believe that cardiac hypertrophy, after a certain time, gives way to insufficiency, and in many respects the view is quite correct.

Everybody envies a man with strong skeletal muscles, and no one predicts a gloomy future for him on that account; but in the case of the heart, such a prognosis is, to a certain extent, justified, not on account of the muscular hypertrophy as such, but either because the cause of the cardiac change is a morbid process and shows a progressive tendency, so that in the end the demands on the heart will exceed its functional power and accommodation, and insufficiency be the result, or because the development of hypertrophy is from the very beginning accompanied by the causes of injury to the heart muscle, which in turn cause deficient heart action.

It is more difficult to treat these matters as a whole than to take them up separately, because special factors come into play in every individual case; they will, therefore, be discussed in connection with the various forms of diseases of the heart muscle.

For the present we shall merely answer the question which has already been asked: how are we to regard the importance of hypertrophy in general? Is it a condition in which the heart muscle is overstrained and but little room is allowed for still further accommodation? This is the

* v. Buhl, "Mittheilungen aus dem pathologischen Institute zu München," Stuttgart, 1878, p. 38.

† Compare Andrew Clark, "British Medical Journal," 1887, pp. 259, 315, 370.

view of Martius,* as set forth by him in an interesting treatise. As has been mentioned, Romberg and I do not share this view. I have already given the arguments derived from clinical observations, which appear to me to contradict it. In addition, the theory is directly refuted by animal experiments performed by Romberg and Hasenfeld.† In rabbits with hypertrophy, the result of insufficiency of the aortic valves, "the heart muscle has a supply of available strength for external work equal to the normal. The reserve strength of the normal and that of the hypertrophied heart muscle are equally great." I, therefore, incline to regard the question as definitely settled,‡ especially as my own view is supported by every general consideration. For in my opinion the development of hypertrophy and hyperplasia as such is difficult to understand unless we assume that a new state of equilibrium is created. But if increase in the size of the fibers progresses *pari passu* not only with an increase in their strength, but also with an increase in the power of accommodation, it is an extremely wise provision of nature, and what has been said above in regard to the production of hypertrophy also harmonizes fully with this view.

The difficulties encountered in making the *diagnosis of cardiac hypertrophy* vary greatly, depending as they do on the conditions under which the hypertrophy develops. Accentuation of the second pulmonic sound is regarded as a positive sign of hypertrophy of the *right ventricle*. In itself it indicates merely an increase of pressure in the pulmonary artery (see p. 450), but we know that if the increase of pressure in this vessel persists for some time, hypertrophy of the right ventricle results; hence that condition may be assumed to be present if accentuation of the second pulmonic is persistently heard. Even if the sound is heard once, the conclusion is, as a rule, justified, because temporary increase of pressure in the pulmonary artery is extremely rare.

Accentuation of the second aortic sound has a similar significance for the *left ventricle*. But it must be remembered that the inexperienced is in danger of being deceived by the presence of sclerosis of the ascending portion of the aorta. Another indication of hypertrophy of the left ventricle is found in the apex-beat: increased resistance and heaving of the apex-beat are regarded as conclusive evidence of the existence of cardiac hypertrophy when found in association with a normal pulse or one with abnormal tension (for the details, see p. 431). It has already been explained that these findings in themselves merely indicate increased activity on the part of the ventricles, and it is only when they persist that they indicate hypertrophy of the muscle.

CARDIAC INSUFFICIENCY. THE VARIOUS TYPES OF CARDIAC DILATATION.

When the heart is not able to satisfy all the demands made upon it, the condition is described as cardiac insufficiency.

In this condition also the pathologic merges into the normal, and it

* Martius, "Ergebnisse der allgemeinen pathologischen Anatomie und Physiologie," 1895, p. 45.

† Romberg and Hasenfeld, "Archiv für experimentelle Pathologie," vol. xxxix, p. 362.

‡ Compare the excellent presentation of Romberg, "Congress für innere Medicin," 1899, p. 115.

is impossible to draw a sharp line of separation. Even within the limits of good health not every heart is capable of meeting every demand that is made upon it; for the functional capacity of the organ in every individual has its limits, determined partly by the greatness of the demands made upon it. There are some demands which are beyond the strength of even the strongest. This point will be discussed more fully in connection with so-called overexertion of the heart.

The limits of functional capacity are also in part determined by individual variations in the working capacity of the heart, which is influenced by a great variety of factors. Training as well as natural disposition plays a very important rôle. It is owing to these two factors that what is very well borne, or performed without any difficulty, by one individual is impossible, or at least harmful, to another, and that the amount of work which the same individual's heart is capable of varies greatly at different times, depending partly on how much work has preceded and partly on the general condition. These things must all be taken into consideration in forming an opinion of the functional capacity of the heart.

The essential fact in cardiac insufficiency is that the heart is unable either to receive the blood which flows into it or to propel the mass with the necessary velocity as required by the condition of the organism at the time. In other words, the trouble consists in deficient power of distention and contraction, and the question is very much complicated by the fact that either the entire heart or only individual portions may be affected.

Insufficiency of a portion of the heart manifests itself, first of all, by a diminution of its power of accommodation. An amount of work which the muscle had previously been able to do easily and without any distress either becomes altogether impossible or its performance is attended by palpitation and dyspnea. In cases of this kind a moderate amount of work is usually performed without any trouble—that is to say, moderate muscular movements cause the patient no distress, and in a certain sense he is well. In other cases he is comfortable only when the body is completely at rest.

At another stage of the trouble the organ is not even capable of taking care of the small quantities of blood required by the circulation during muscular rest, or to overcome the slight resistance in the arterial system. This separation into different stages is, of course, quite arbitrary, and each group embraces a great many different degrees of cardiac disturbance. Just as in diabetes sugar assimilation is reduced in varying degrees,—as the existence of the trouble in the mildest cases is discovered only after very careful observation and only occasionally causes the patient any discomfort, while, on the other hand, the picture of grave diabetes makes an indelible impression even on the inexperienced,—so it is with the various grades of cardiac insufficiency. In both conditions the disease develops from a normal state by almost imperceptible degrees.

In reality there are innumerable transitional forms from the mildest grade of cardiac insufficiency, in which the condition is barely noticeable, to the most profound states of cardiac weakness; the latter frequently develop from the former; and, finally, the disease may become arrested at any stage and may begin at any stage. There are many reasons, chiefly connected with the question of treatment, which, in my opinion, make it desirable to distinguish cases in which the work of the muscle is insufficient even during rest, and the deficiency is, therefore, permanent

and necessarily attended by the phenomena of abnormal distribution of blood, presently to be described, from cases in which the disturbance manifests itself only when a greater demand is made on the heart. In the latter class the disturbance of the blood distribution only lasts a short time, and for that reason certain pathologic processes which follow in the wake of cardiac insufficiency do not develop. In my opinion it would be advisable to use different terms to describe the two conditions.

The *immediate effect** *of insufficiency of a portion of the heart,* whether it be the power of dilating or the power of contracting that has suffered, is a diminution of the quantity of blood in the arterial, and an accumulation of blood in the venous, portions of the vascular system. It follows, from the nature of the causes which lead to cardiac insufficiency, that both parts are, as a rule, affected (one auricle and the corresponding ventricle are counted as one part). Quite frequently, however, the left and the right heart are affected in such different degrees that, in order to understand the clinical symptoms, it is necessary to discuss separately the functional disturbances produced by each.

Weakness affecting chiefly the left ventricle leads to diminished filling of the systemic arteries, and later of the veins, with stagnation in the left ventricle, pulmonary veins, and pulmonary artery. If the right ventricle still retains sufficient power to overcome the abnormal resistance—in reality it is not, as a rule, able to do so altogether, although quite often to a considerable extent—the absolute blood-pressure in the lungs is abnormally high, but the "head" remains approximately the same. The resulting phenomena in the respiratory apparatus will be discussed in the next section. Theoretically, there is no congestion in the systemic veins. But, since the functional capacity of the right ventricle is not, as a rule, perfectly intact, more or less venous congestion is observed in most cases. After a time, of course, the condition becomes stationary: the right ventricle expels no more blood than the left because it does not receive any more. The velocity of the circulation is diminished, the distribution of blood modified, and the lung overfilled.

When the right heart is not quite able to perform its work, the blood collects in the auricle and in the venæ cavæ; when the insufficiency of the right ventricle is marked, the blood stagnates in all the organs and, of course, chiefly in the more inelastic organs. The phenomena produced by this congestion in the various systems of the body will be discussed at length in the next section. What interests us chiefly here is the way in which the symptoms are combined.

In severe cases symptoms of congestion manifest themselves in all the organs of the body, and the well-known clinical picture results. Even in these severe cases great variations, often apparently without any reason, are observed in the degree to which the various tissues are implicated in the morbid process; but in the milder grades of congestion these variations are very much more marked. In these cases only a single organ is sometimes involved, at least so far as gross observation during life is able to determine; or only two may be involved—in fact, the most remarkable variations are seen.

The reasons for this curious behavior are, for the most part, obscure. I fully share the opinion of Romberg that individual peculiarities have

* Compare v. Basch, "Allgemeine Physiologie und Pathologie des Kreislaufes," Vienna, 1892. Moritz, "Archiv für klinische Medicin," vol. lxvi, p. 349. Krehl, "Pathologische Physiologie."

much more influence on the character of the disease than have the causes of cardiac weakness.

When the right ventricle is weak, the arteries, as a rule, contain less blood and the tension is lower than normal, even when the work of the left heart is comparatively good. This is due to the fact that the left ventricle cannot propel as much blood as usual, because it receives less. The arteries contract, all the other signs of disturbance of the cardiac action make their appearance, and the pulse becomes smaller, less full, and—if the contraction of the vessels is not sufficient to compensate for the diminished quantity of blood—also weaker. Hence weakness of the right ventricle is always followed by disturbances in the arterial side of the vascular system and slowing of the circulation.

Clinically, isolated weakness of one portion of the heart only, without involvement of any other is never seen; for the poisons which enter the heart from the blood-current affect all portions of the organ. Muscular exertion, with the demand it makes for dealing with a sudden inflow of excessive quantities of blood and the rapidly developing abnormal resistance, always influence both sides of the heart, although not always in the same degree; and all anatomic processes in the heart muscle are of such a nature that they *usually* involve the entire organ.

Nevertheless, there are sufficient reasons for the existence of unequal disturbances of the individual parts. Even inflammations may in some cases attack one portion of the heart more severely than another; this is particularly the case in coronary sclerosis (*q. v.*): the left coronary artery is attacked distinctly more often than the right. It is, therefore, easy to understand why dyspnea is observed so frequently in this particular condition. A familiarity with the circulation, when the disturbance in an individual portion of the heart is chiefly functional, is, therefore, most desirable, if not indispensable. I have not mentioned the auricles separately because in disturbances of the circulation, so far as is known, they act essentially as appendages to the veins. If, as is usually the case in life, the strength of both ventricles is impaired, a combination of secondary phenomena results—diminished velocity, altered distribution of blood, with lessened quantity of blood in the arteries and engorgement of the veins.*

When one portion of the heart is unable to contract completely, a larger quantity of blood remains in that portion at the end of systole, more blood flows in, and the cavity dilates and exceeds the mean volume in diastole and systole—in short, a condition is produced which, in general, is called *dilatation*.

It would be a great mistake, however, to regard every dilatation of individual portions of the heart as pathologic.

It has been mentioned repeatedly that the volume of the auricles, as well as that of the ventricles, undergoes rapid and marked changes, depending on the pressure and quantity of the inflowing blood. The accuracy with which the heart is able to adapt both its diastolic size and the strength of its contractions to the quantity of blood that flows into it is one of the chief attributes of this wonderful organ—in other words, the tonicity and the contractile power of the heart muscle depend on the demands that are made upon it. Thus, for example, during muscular movements marked diastolic dilatation may be associated with unusually extensive and vigorous systoles.

* For the theory of these processes see Krehl, "Pathologische Physiologie," p. 65.

It would be entirely wrong to regard such a dilatation of the cavities of the heart as pathologic. *When we use the term dilatation of the heart loosely to describe a pathologic condition,* we have in mind a condition that is entirely and fundamentally different from the above, namely, *dilatation of the heart the result of insufficient contraction.*

This form has been called congestive dilatation (Stauungsdilatation) to distinguish it from the first-mentioned or compensatory form; the distinction has also been made between "active" and "passive" dilatation. These terms ought not to be discarded because the rigid distinction which must be made between these two concepts is more readily retained if definite terms are used.

Passive (congestive) dilatation causes an enlargement of the boundaries of the heart beyond the average for the individual's period of life, and, if the dilatation affects the left ventricle, diminution in the force of the apex-beat, while in the case of either ventricle the corresponding second sound at the base is diminished in intensity. The mode of arriving at a diagnosis with the means at present at our disposal has been described. In addition, the effects of cardiac weakness on the blood-vessels and other organs is also taken into account.

Whenever the quantity of blood that enters the arteries is diminished,—it has been explained under what circumstances this takes place,—changes in the arterial pulse make their appearance. It becomes smaller, less full, softer, and may exhibit the changes in rhythm described on another page.

The symptoms dependent upon congestion of blood in the lungs will be discussed in connection with the respiratory apparatus. Engorgement of the systemic veins manifests itself by swelling and in peculiar movements in the visible veins (see p. 475), as well as by characteristic phenomena in certain organs, especially the liver, kidneys, gastro-intestinal canal, and skin. The resulting symptoms will be discussed in the following section.

In order to make a *diagnosis of cardiac insufficiency* it is necessary not only to be perfectly familiar with the symptoms of the condition, but also to inquire into every detail of the history and make a most searching examination of the patient; for the functional or structural disturbances in the various organs are often so slight as to be readily overlooked by a superficial observer.

No one has any difficulty in recognizing pronounced insufficiency which is constantly present under all circumstances, and with a certain knowledge of physiologic conditions it is usually possible to determine which part of the heart is chiefly affected.

The problem is a much more difficult one, however, in slight and very insignificant degrees of cardiac weakness, when the organ, under ordinary conditions,—for example, during complete bodily and mental rest,—apparently responds fully to all the requirements and the disturbance shows itself only when greater demands are made on the functional capacity. In such cases objective examination is often absolutely disappointing; during rest there is not a symptom either in the circulation or in the other organs. Objective examination of the functional capacity of the heart is, no doubt, destined in future to give the best results; for the present we have to depend for information chiefly on the patient's statements in regard to subjective disturbances of the various organic functions, and the latter will, no doubt, always play an important part because they are noticed early, especially by observant persons.

In determining the significance of objective disturbances in organs, which will be described later, even the most insignificant details must be taken into account—such, for example, as a slight swelling of the liver or an obstinate, although quite moderate, bronchitis. Here, again, it

is important to find out whether there has been a change from normal conditions and whether the existing functional disturbance, whatever it may be, cannot be explained in some other way—by the patient's diet, mode of life, and the like.

Dilatation of this kind may be due to any of the causes that diminish the contractile power of the heart; the existence of such a cardiac dilatation is always a direct sign that certain portions of the organ are not performing their functions perfectly.

There is no doubt whatever that the great majority of cases of cardiac dilatation observed during life are due either to the fact that the heart muscle receives excessively large quantities of blood into its cavities and expels them again, or that the contractions are insufficient, so that the blood is retained in the chambers of the heart, with a resultant loss of tone and dilatation. There is, however, another possible mechanism which must not be altogether disregarded, and which, in fact, I regard as probable. It is possible that, as Bauer* maintains, the symptoms hinge on diminution of the muscular tonicity during diastole. The existence of dilatation of the heart with unimpaired power of contraction has been referred to. This condition exists when, with a healthy heart muscle, abnormal quantities of blood enter the cavities of the heart during diastole and are again expelled. Insufficiency of the aortic valves is a classic example of this condition. It is also true that we sometimes see dilatation of the heart associated with apparently unimpaired contractile power of the muscle and without finding anything that might cause the entrance of excessive quantities of blood during diastole. These forms of dilatations are frequently associated with hypertrophy of the muscle.

I can think of no other explanation for this condition than a supposed primary diminution of the muscle tone. This would be followed by overfilling of the ventricles; for we know from Hesse's investigations that, under normal conditions, the filling of the cavity is arrested by the rapidly increasing tension of the wall. If the contractile power is preserved and the entire (increased) contents are expelled, the circulation will be accelerated and more blood will again enter the heart. Thus the organ does more work and must, therefore, undergo hypertrophy. The question involves matters which are in urgent need of further investigation, and to take it for granted that they are really well known, would only act as a bar to further investigation of these questions and put off their final solution.†

Clinicians have ever maintained that hypertrophy develops from dilatation. This assertion is generally accepted for the small number of cases so far reported of active dilatation with an increased amount of blood; but those who are not content with anything short of a full and complete understanding of the processes of the circulation look upon the development of hypertrophy from dilatation with profound skepticism in all other cases. If, as has just been shown, it is assumed that the muscular tonus may be primarily diminished in certain cardiac conditions, while the contractile power of the organ remains intact or practically intact, it is not difficult to understand that hypertrophy of the heart in

* Bauer, "Festschrift der Münchener medicinischen Facultät für M. von Pettenkofer," Munich, 1893, p. 10.

† Compare Henschen, "Arbeiten aus der medicinischen Klinik zu Upsala," Jena, 1898. H. Herz, "Ueber die active Dilatation des Herzens," "Deutsche medicinische Wochenschrift," 1900, No. 8, 9.

such a case also may develop from dilatation. Bauer believes that this diminution of the tone plays an important part in the pathogenesis of so-called idiopathic hypertrophy, especially the forms which develop after excessive beer-drinking. For my part I am also convinced that the muscle tone deserves very careful investigation, particularly in its relations to contractility. The question is exceedingly complicated. There is no doubt that hypertrophy may develop in a heart in which the contractions are relatively deficient, namely, when a greater demand is made on the affected portions of the heart and the demand is partially satisfied. Under such conditions the heart muscle is in the peculiar state that it performs more work than normally, although not enough to achieve quite all that is required of it. It hypertrophies because it is doing more work, but at the same time it undergoes passive dilatation because it is unable to do as much work as it ought. In such a case, therefore, hypertrophy develops, although dilatation is present at the same time. This condition plays an important part in the development of valvular defects and in other conditions also, as, for example, arteriosclerosis. Theoretically, therefore, the conditions are altogether different from the conditions in the above-described cases.

Theoretically, there should be a second type of cardiac insufficiency due to insufficient dilatation of the cavities during diastole. The capacity of the organ to take up varying quantities of blood without much increase in the tension of its walls is as natural as its power of accommodation during contraction. The disturbance in this case would have to consist in premature stiffening of the walls and would lead to passive congestion and lowering of the pressure beyond the diseased portion of the heart. I am not aware that there is any known clinical picture that could be identified with this form of functional anomaly, and it is, therefore, not worth while to enter any farther into this question.

The Functional and Structural Disturbances in the Muscle Responsible for Cardiac Insufficiency. A Few Remarks on the Pathologic Anatomy.—When the heart fails to respond to the demands made upon it, it is either because the impulse to do the necessary amount of work is not sufficient, or because the work is not completely performed. For the present, however, it is impossible to separate these two conceptions in this discussion. Both depend on certain peculiar properties of the heart muscle; hence disturbances of this function must also be due to changes in the myocardium. The part played by the ganglia and nerves of the heart remains to be discovered by future investigation. At present our knowledge in regard to their function is indefinite, and we are, therefore, unable to explain functional anomalies by any structural changes found in them.*

Our ignorance of these matters is the only reason why we do not enter into them more fully. For the present they baffle us completely; no one who has any knowledge of the subject will conclude that we wish to underestimate or even to suppress the importance of the cardiac ganglia; but there is nothing gained by talking at length about hypothetic matters of the future.

For the present, therefore, we are concerned with the heart muscle. Even in health processes take place in the heart muscle after increased function which are closely related to insufficiency; I refer to fatigue.

* The literature on this subject will be found in the section on Diseases of the Cardiac Nervous System.

Fatigue leads to certain changes in striated muscles which have been very carefully examined by physiologists, although we are still quite in the dark in regard to the structural and chemic conditions which determine these changes.

By fatigue we mean a condition of diminished functional capacity which follows severe exertion. Processes in the heart and in the skeletal muscles are obviously very similar; only we are not as yet able to separate fatigue of the heart from fatigue of the muscles generally. During the period of rest which follows excessive activity the functional capacity is fully restored (see p. 480).

It is natural to assume that when the heart muscle has been damaged in any way, it is especially apt to become fatigued; but we have no positive facts to prove this, because every attempt to obtain more accurate knowledge is frustrated by the extremely complicated conditions encountered.

I must, at least, protest against the freedom with which pathologists explain the production of cardiac insufficiency by fatigue. It only acts as a bar to any further investigation into the nature and causes of the process, for premature and inadequate explanations partially satisfy the mental discontent indispensable to scientific advance.

In the interest of further progress I would urge that the term fatigue be limited to the class of cases for which it has always been used generally, namely, a condition of diminished functional capacity after increased exertion. As Romberg and myself have shown elsewhere,* fatigue in general presupposes recuperation; the latter repairs the damages caused by the former. It is, therefore, in my opinion, improper to speak of permanent fatigue. There may possibly be such a condition, but to assume its existence would, for the present, unquestionably retard progress and prevent a further amplification of our knowledge.

If an individual disregards fatigue and continues to throw a severe strain on the heart in spite of it, the injury to the muscle is often much greater than in ordinary fatigue; for recovery takes place much more slowly, and in some cases perhaps not at all. This process is properly termed *overexertion, and the condition of the muscle which results therefrom, overdistention of the heart.* Whenever this condition develops, it is because the heart has performed or tried to perform work far in excess of what it can do without injury. The important point is the disproportion between the strength of the heart and the demand made upon it.

An attempt was made in the section on Etiology to give a detailed explanation of these matters, and it was seen that overexertion plays an important part as a cause of disease. The muscular fibers as they contract in their effort to overcome the overwhelming distention with blood undergo abnormal distention. The same process in the skeletal muscles is well known to be harmful; but we do not know what chemic and structural disturbances lie at the bottom of the condition, any more than we do in the case of fatigue, and for the present there is no immediate prospect of any addition to our knowledge. It will be difficult to acquire this knowledge in proportion to the frequency with which existing pathologic processes in the heart are responsible for the overdistention. Their picture will, as a rule, first meet the microscopist's eye and make it more difficult for him to recognize the manifestations of the other process.

In cases of *intoxication* the conditions are very similar. Neither chemic nor microscopic analysis renders us any assistance in the diagnosis, especially during the first stages, although even now conscientious work

* Krehl and Romberg, "Archiv für experimentelle Pathologie," vol. xxx, p. 157.

in this field is not without hope of success, for we are approaching familiar grounds. We know that certain definite abnormalities in cellular structure—so-called *parenchymatous degenerations*—are chiefly attributable to the action of poisons.

In the heart the most important changes in the muscle-cells consist in *granular, hyaline,* and *fatty degeneration.* The general principles that underlie the production, appearance, and distribution of these degenerations in the heart will not be considered here, as some of these things must be mentioned in the various sections.

In regard to the relations existing between these structural changes and the functional capacity of the heart, a sharp distinction must be made, so far as I can see, between the damage caused by fatty degeneration—which offers the best opportunities for studying the condition—through the destruction of contractile substance, and its significance as a sign of the action of certain poisons. Like the majority of pathologists, we take the ground that cellular albumin in certain forms of degeneration is partly converted into fat and that the fat may remain within the cells. Fatty degeneration is, therefore, a sign of diminution of albumin in the parenchyma cells. Theoretically, this must affect the contractile power of the muscle-fibers unfavorably; to what degree, depends chiefly on the intensity and extent of the fatty degeneration. Actual experience is in accord with this theoretic assumption. There are hearts in which the great majority of the muscle-fibers are degenerated,* in which a large percentage of fat is present in the muscle,† and which have thereby suffered a serious loss of function.

These cases are not very frequent, and it may be said positively that a considerable degree of fatty degeneration (following phosphorus-poisoning) is not incompatible with a fair degree of functional capacity.‡ This, in my opinion, is positively proved by the fact that pure fatty degeneration as such, so long as it does not exceed certain limits,—as is usually the case,—has a relatively slight effect on the strength of the heart. This view is supported by direct observation. For a time fatty degeneration of the myocardium was regarded by pathologists as the most important cause of insufficiency; but it was found from microscopic§ as well as chemic‖ investigations that in those cases in which it was chiefly to be expected the degeneration actually proved to be of but small extent.

There is no doubt that our views in regard to fatty degeneration are in a transition stage. In addition to the local production of fat from cellular albumin, the question of immigration of fat plays an important part not only in the case of the liver, but in other organs as well. This renders the question more complicated than was formerly believed, but, on the other hand, the association of an accumulation of fat in the cells with preserved function is more comprehensible.

The whole question is still further complicated by another factor. There is no doubt that under certain circumstances fatty degeneration occurs, so to speak, in an uncomplicated form, that is to say, there are cases

* See, for example, Curschmann, "Archiv für klinische Medicin," vol. xii, p. 193.

† Krehl, "Archiv für klinische Medicin," vol. li, p. 446.

‡ Welch, "Medical News," 1888. Quoted from Virchow-Hirsch's "Jahresbericht." Balint, "Deutsche medicinische Wochenschrift," 1898, No. 1, 2. Hasenfeld and Fenyvessy, "Berliner klinische Wochenschrift," 1899, No. 4.

§ Krehl, "Archiv für klinische Medicin," vol. xlvi and xlviii. Romberg, *ibid.*, vol. xlviii and xlix.

‖ Krehl, "Archiv für klinische Medicin," vol. li, p. 416.

in which the cause sinks into insignificance beside the effect produced by the fatty degeneration on the function of the heart—this is not by any means always the case. In a great many cases of fatty degeneration a severe intoxication is the primary cause, and in such cases it is, above all, important to know what influence the intoxication has on the cardiac function. It seems to me that the effect of the intoxication varies very greatly in different cases. It is evidently not very great in phosphorus poisoning, although here also it is not altogether wanting; in this condition dilatation of the heart and disturbances of the rhythm are also observed.

The conditions are, I believe, quite different in cases of poisoning associated with bacterial infection. So far as we know, these forms are exceedingly dangerous to the circulation; it is not the fatty degeneration so much as the toxic injury to the muscle that is responsible, and the degeneration must be regarded as an expression and concomitant of the injury to the muscle.

What has been said applies, in my opinion, also to the functional significance of granular and of hyaline degeneration, and it is immaterial what views in regard to their relations to fatty degeneration are adopted.

The *protoplasmic spindle surrounding the nucleus of the muscle-fiber* is, in many cases, greatly increased both in width and in length; in the latter case the nuclei of two adjacent fibers may be united by a strip of protoplasm. Hardened preparations sometimes convey the impression of gaps in the protoplasm. The functional significance of these changes is as obscure as their origin, but experience teaches that they occur in conditions associated with diminished heart power.* As they represent a destruction of the living substance, it appears safe to assume that they bear a causal relation to the diminution of functional capacity.

I shall not venture to render a judgment in regard to the significance of *fragmentation of the heart muscle.*† If the process becomes extensive, it cannot possibly be without any effect on the heart. The great question is, when does the change develop? Is it an agonal change‡ and, therefore, merely the expression of approaching death?

Next we have the *changes in the muscle nuclei.* Sometimes they exhibit a pathologic increase, but this is assuredly not a frequent occurrence, and caution must be observed in giving an opinion, because the number of nuclei varies with the patient's period of life, being normally large in children, for example.

Much more frequently the *shape of the nucleus is altered.*§ Large, swollen, flat, badly staining specimens are quite frequently encountered, or the nucleus may be several times its normal length, or constricted, or may stain very deeply. In other cases the nuclei are reduced in size and shrunken. Again, a copious brown pigmentation often accumulates in the protoplasmic spindle surrounding the nucleus. These changes in

* Compare Lépine and Molard, "Archives de médecine expérimentale," 1891, vol. iii, p. 776.

† For the literature see Karcher, "Archiv für klinische Medicin," vol. lx, p. 67. Compare v. Recklinghausen, "Verhandlungen des internationalen Congresses in Berlin," vol. iii, p. 67. [Hektoen, "Segmentation and Fragmentation of the Myocardium," "American Journal of the Medical Sciences," November, 1897.—Ed.]

‡ v. Recklinghausen, *loc. cit.* [For an accidental fragmentation in some cases, see Buhlig, "Journal of Medical Research," vol. vii, p. 428.—Ed.]

§ An excellent review of the nuclear changes in diseased heart muscle is given by Romberg, "Archiv für klinische Medicin," vol. xlviii, p. 383. The most important treatises on the subject are also cited.

the nuclei impress me as signs of degeneration, and I, therefore, assume at once that they are not indifferent, but have some effect on the function of the muscle-fibers. The more wide-spread the degeneration, the more far-reaching will be its injurious effect. So much no one will doubt; but for the present we have no more accurate means of estimating quantitatively the injury produced by these granular degenerations.

Degenerations may undergo involution; at least we know positively that many of them do. But for involution to take place, the vitality of the muscle-fiber must be preserved. Dead muscle-fibers are not replaced by living fibers, or at best only to a very limited extent, and there is always danger that these degenerations may lead to atrophy of the fibrils. In what way this atrophy develops and what are the intermediate stages will depend on the intensity and kind of the exciting cause. An excellent example of this is found in anemia. If a portion of the heart is deprived of blood, even for a short time only, the fibers rapidly die; on the other hand, if there is merely a perceptible anemia, various forms of degeneration develop, and if the injury persists or increases, the degeneration ultimately goes on to necrosis.

Simple atrophy and brown atrophy do not introduce any new factor into the functional disturbance; their injurious effect depends on the reduction in the size and number of fibers and the damage to their structure. This is always the salient point in estimating the importance of a pathologic process and its effect on the cardiac function, provided no special factors are introduced into the problem by intoxication.

The significance of *necrosis of the muscle-fibers*, characterized by loss of nuclei and transverse striation, need not detain us. It indicates that function has entirely ceased in the affected part. The degree of functional loss arising therefrom depends, of course, mainly on the extent of the process; but it is often astonishing how an extensive necrosis may exist with unimpaired cardiac function and, therefore, with continued life. In the future we may possibly learn that this depends on the seat of the foci; for the present there is nothing to be said on this point.

Diseases of the blood-vessels of the heart, especially of the arteries, are exceedingly important. These diseases usually represent processes which belong to arteriosclerosis, although inflammation of the arterial walls also occurs in consequence of infectious processes. The significance of these processes lies in the fact that, as soon as the arterial disease causes a diminution of the lumen, the blood-supply of the portion of the heart supplied by the respective vessels suffers in exact proportion to the interference with the flow of blood. All grades, from complete necrosis of individual portions of the heart such as follow thrombotic or embolic occlusion of the supplying artery to the mildest form of fatty degeneration, such as occurs after low grades of anemia, are observed.

All that has been said in regard to the effect of these degenerations on function applies, of course, equally to the effects of arterial disease.

And exactly the same principles—all that has been explained in the preceding paragraphs—must also be applied in estimating the functional disturbance due to *inflammation of the muscle*. Inflammation is of inestimable value in allaying our craving for etiologic knowledge, because experience dating from the earliest times and confirmed again and again, as well as diversified in a number of ways, shows us its harmful influence on the function of the tissues. More specifically inflammation profoundly affects the contractile power of the muscle.

The fact is well established; the explanation may vary slightly, depending on one's general ideas in regard to the significance and relative importance of parenchymatous and interstitial processes in inflammation. Here we shall have to confine the discussion to the heart. Microscopic observation shows beyond a doubt that changes in the muscle-fibers and in the interstitial tissue may be highly independent of one another, but may also be combined in a great variety of ways. In cases in which degenerations of the muscle-fibers are pronounced, what has been said above applies with special force, and even he who will not acknowledge the truth of what has been said, will have to admit that the injury which attacks vessel-walls and connective-tissue cells is not likely to leave the function of the parenchyma intact. On the other hand, if the argument is used that the structure, as is frequently the case, remains intact,—an objection which I acknowledge is a weighty one,—it will be said that the interstitial disease constitutes a mechanic or chemic disturbance which interferes with the contraction of the muscle-fibers.

The nature of the injury can be partly learned by the course of the inflammation; after brief (acute) interstitial processes the muscular fibers may entirely regain their normal condition. But even in processes of short duration this is not always the case; and if the process is protracted, the muscle-fibers always fall a prey to the various forms and grades of atrophy and degeneration.

Whether the inflammation is to be acute or chronic depends, in my estimation, mainly, or even exclusively, on its cause. Perhaps one might go even farther and say that it depends on whether some injury acts for a length of time or even permanently. It is quite possible that the functional injury resulting from inflammation, aside from its extent, depends in a great measure on its cause. It is not possible to do more than offer a hypothesis, but clinical experience, especially in heart diseases, is decidedly in favor of such a view.

Acute inflammation manifests itself by a more or less abundant accumulation of round-cells in the interstitial tissue. This infiltration is frequently more marked underneath the endocardium and pericardium, and spreads from there to the muscle; the intimate relation existing between endocarditis, pericarditis, and myocarditis has already been mentioned. At a later stage delicate embryonal connective tissue containing an abundance of cells is found in the intervals between the muscle-fibers. This connective tissue gradually becomes denser, and its cellular constituents fewer; the muscle-fibers contained within it degenerate and atrophy in a variety of ways, sometimes by a gradual diminution of the transversely striated substance, so that at last nothing remains but the pigmented, protoplasmic spindle and the nucleus, and, finally, the entire fiber is destroyed. Thus the recent inflammatory focus goes through a long series of intermediate stages and finally becomes the well-known hard cicatrix containing but very few cells.

The connection between inflammatory phenomena and the formation of embryonal tissue, which is such an important pathologic question, I shall purposely disregard because it has no immediate bearing on our subject. What we chiefly want to know as physicians is the amount of functional injury to the heart arising from these interstitial processes.

The significance of a recent inflammatory irritation has already been discussed. How about the chronic processes? They manifest themselves in two forms: either in the form of more or less circumscribed

foci, or diffuse, causing a certain thickening of the connective tissue already existing in the heart (Dehio's myofibrosis).*

All the changes produced in the muscle-fibers during a recent inflammation occur also in the chronic conditions so long as they represent true inflammatory processes. The effect may possibly be less intense, but it always shows itself in a varying degree. On the other hand, the effect is unquestionably absent whenever the foci consist of nothing but the remains of an inflammation—fibroid patches, in other words. For the latter do harm only through the loss of parenchyma cells, which they have displaced, and it is possible that the weakening of the muscle interferes with function more at one point than at another. Like Romberg,† I cannot bring myself to accept the theory which finds in diffuse thickening of the connective tissue a protection for the heart-wall.

According to universal belief, circumscribed scars derive their origin from inflammatory foci. The development of diffuse increase of the connective tissue or myofibrosis, according to Dehio, is due to purely mechanic conditions. The muscle-fibers are supposed to be partially destroyed by certain influences associated with dilatation and later to be replaced by connective tissue in the usual way. Although the demonstration of diffuse connective-tissue proliferation appears to be interesting and to have an important bearing on our understanding of functional disturbance (I have myself observed it not infrequently after fatal cardiac dilatation, especially when associated with valvular lesions), I am, nevertheless, unable to accept without reservation Dehio's explanation of the pathogenesis and still incline to the belief that functional disturbance is very frequently directly due to inflammation.‡

In the paper just referred to, and in a number of other excellent monographs from Dehio's laboratory,§ it is shown that the formation of connective tissue is often very marked in the auricles of the heart. This seems to me an important point. It is in the musculature of the auricles at the point of origin of the large veins that the stimulus to cardiac contraction originates. It is, therefore, possible that disease and atrophy of the auricular musculature have a special influence in producing severe disturbances of the contractions.

FUNCTIONAL AND STRUCTURAL DISTURBANCES OF ORGANS FREQUENTLY ENCOUNTERED IN ASSOCIATION WITH CIRCULATORY DISTURBANCES.

RESPIRATORY APPARATUS.

Any disturbance of the circulation that has any influence at all on the blood-stream must necessarily affect the respiration, for respiration and circulation reciprocally influence one another in a great variety of ways.

Respiratory sensations and respiratory movements depend upon the interchange of gas between certain cells in the central nervous system, on their ability to take up oxygen and give off carbon dioxid. The latter

* See Dehio, "Archiv für klinische Medicin," vol. lxii, p. 1.

† Romberg, "Congress für innere Medicin," 1899, p. 115.

‡ Compare Krehl, "Archiv für klinische Medicin," vol. xlvii, p. 414.

§ Radasewsky, "Zeitschrift für klinische Medicin," vol. xxvii, p. 381. Sack, "Ueber difuse fibröse Degeneration der Vorhöfe des Herzens," Dissertation. Dorpat, 1894.

again depends partly on the condition of the cells and partly on the kind, quantity, and tension of the gases that are carried past the cells. Composition and velocity of the blood are, therefore, the most important factors, and both are, as we know, very much dependent upon one another.

So far as its effect on respiration is concerned, the passage of the current of blood must be considered chiefly as it affects two different parts of the body: the sensitive points in the central nervous system and the lungs. At both points the velocity of the blood-current is the essential thing. In the lung, since the width of the pulmonary vessels fluctuates but little, the velocity may be directly compared to the quantity of blood passing through the organ in a unit of time,* which again undoubtedly depends on the difference in pressure between the arterial and venous sides of the pulmonary circulation, that is, on the difference between the pressure in the pulmonary artery and that in the left auricle. When the left ventricle and its valves are normal, the pressure in the left auricle is extraordinarily low—either negative or equal to only a few millimeters of mercury. Hence the velocity of the blood-stream in the lung is greatly dependent upon the filling and emptying of the right ventricle, and in the same way everything depends on the right ventricle's power of accommodation whenever, owing to some functional anomaly in the left ventricle, the pressure in the auricle and pulmonary veins is raised. In that case, if the right ventricle can adapt itself fully to the increased resistance, the blood in the lung will be subjected to an absolutely higher pressure, although the "head" remains the same. It is probable, however, that other factors then become operative and affect the respiration in spite of the fact that the velocity is preserved. The point will be discussed again.

Although this explanation is somewhat lengthy, it seems to me necessary, in order to render the complicated relations between circulation and respiration more intelligible. That the velocity and quantity of blood passing through the lungs in a unit of time depend mainly on the activity of both sides of the heart appears to be self-evident.

But the same thing is true also of the quantity and condition of its gases. Whatever theory be adopted with regard to the nature of the process taking place in the lungs, whether the interchange of gases be regarded as a result of differences in tension or as the product of a secretory activity—in any case the nature of the blood-current in the lungs must have an important influence on this process. Here, again, the most important factors appear to be the velocity and the quantity of blood passing through the lungs in a unit of time. He who believes that the interchange of gases in the alveoli is due to diffusion will have to remember how very small is the difference in pressure, which undoubtedly implies the persistence of a certain degree of velocity in the blood-current, as otherwise the escape of carbon dioxid from the blood and the entrance of oxygen into the blood would soon suffer materially. But he who adopts Bohr's view of a glandular activity must also grant that the velocity of the blood is an important factor; for we know that all glandular functions are greatly dependent upon the velocity of the blood.

The conditions are, however, much more complicated. In the first place, a copious supply of blood is indispensable for the maintenance of adequate nutrition in the respiratory epithelial cells, which is so important for the preservation of function, and, finally, there is the ventila-

* Compare Romberg, in Ebstein-Schwalbe's "Handbuch," vol. i, p. 699.

tion of the lungs. The latter also seems to be greatly dependent upon circulatory conditions.

It is very difficult as yet to give anything like a final opinion on this point because—although it seems incredible—our knowledge of the mechanism by which the air is carried to the alveoli is unquestionably quite inadequate. How far does direct interchange of air extend? To what extent does the passage of air from one part of the bronchial tree to another depend on diffusion? By what mechanism does the air gain access to the alveoli? What part do the bronchial muscles take in respiration? All these questions must be answered before we can quite understand the mechanism of respiration in heart patients. One thing, however, we can say: It seems that the accumulation of blood in the lungs can interfere with the interchange of gases even in the absence of any loss in velocity (or in the quantity passing through). This is the case, for example, when an obstruction at the mitral orifices (weakness of the left ventricle must have the same effect) is overcome by increased action on the part of the right ventricle and the current of blood in the lungs has approximately the same "head," although the pressure is absolutely higher. It is probable that this affects the vital properties of the alveolar epithelium, a view that finds support in a number of things, especially in the fact that disease of the trachea and lungs is common in these conditions. According to general opinion, however, the supply of air to the alveoli is directly diminished, because the engorged blood-vessels are supposed to bulge into the alveolar space and diminish its size. Basch believes that the abnormally plethoric lung swells and becomes rigid and immovable. This view has always appealed to me very strongly; the experiments on which it is based seem to me good, and the entire hypothesis a useful one for the explanation of the phenomenon in man. In the course of years, however, distinct doubts have assailed me; for it must be generally admitted* that the lungs in man during passive congestion do not swell to a degree which can be appreciated by the physician. I have always kept a special lookout for this, but have never been able to observe it, nor have I ever seen the lung become rigid, and the fault cannot be laid to methods of examination in the living subject, as they are quite adequate for that purpose.

I cannot, therefore, bring myself to share v. Basch's view in its present form, especially since the beautiful observations and interesting interpretations of Kraus† prove that during dyspnea, produced by violent muscular exertion in heart patients, the respiratory volume of the lungs is not restricted.

This does not exhaust the question, however, for we still have to reckon with the effect of the pneumogastric on the lungs. Unfortunately, our knowledge of the extent to which respiratory sensations or modifications of the respiratory movements can be initiated through the agency of the nerve-endings in the lungs is very limited. There is no reason why this should not be the case; only we are not aware as yet of any physiologic facts that can be used as arguments in its support. It is a theory, however, that would render many pathologic phenomena distinctly more intelligible.

At least there is no doubt that there are abundant opportunities for

* For the literature and personal observations see Krehl, "Pathologische Physiologie." Romberg, "Herzkrankheiten."

† Kraus, "Die Ermüdung als Mass der Constitution," "Bibliotheca medica."

influencing the respiration: the causative factors attack both brain and lungs, each in a different way. Subjectively, a feeling of air-hunger is experienced during disturbance of the respiration; objectively, the respiratory movements are reinforced and accelerated.* Unfortunately, the term *dyspnea* is used in clinical terminology to describe both processes, although it would be better if we could agree on a definition of the word which should be free from ambiguity.

It follows, from the foregoing explanations, that, so far as the blood-current is concerned, the condition of the respiration is influenced mainly by diminution of its velocity (which is important both in the brain and in the lungs) and increased pressure in the pulmonary vessels. Accordingly, respiratory disturbances occur in all anomalies of the cardiac function which produce one of these sequelæ. The respiratory disturbance is proportionate to the need of gas on the part of the organism: a patient, other things being equal, always suffers less from dyspnea when at rest than during exercise. For, in the first place, muscular action demands a considerably greater interchange of gases, and a diseased heart, in order to perform an equal amount of extrinsic work, has to make much greater exertions than a sound heart,† and this increased exertion not only increases the demands made by the body-cells on the gaseous interchange, but in addition, of course, aggravates the circulatory disturbance which is responsible for the dyspnea. Hence bodily activity plays a very important part. A person who feels quite comfortable when at rest may get out of breath with the slightest exertion.

Thus, respiratory disturbances occur when there is weakness of the right ventricle, because in that condition the velocity of the blood in the lungs and brain is always diminished. The degree of dyspnea depends on the disproportion between the velocity that is required and that which actually exists—that is to say, on the degree of cardiac weakness. If there is unusual resistance to the blood-current through the lungs, as, for example, in kyphoscoliosis and emphysema, and the velocity can be kept up only by constant exertion on the part of the right ventricle, any weakness of that chamber will make itself doubly felt. Hence insufficiency of the right ventricle is always associated with extreme dyspnea.

In moderate grades of cardiac weakness the symptoms are much milder. The patients are often without subjective symptoms during rest, and it would not occur to any one to think that they suffered from dyspnea; but they state positively that they get out of breath after the slightest muscular exertion. In other cases there is more or less cyanosis even during rest. The observer is struck by a certain acceleration and deepening of the respiration and jerky speech. When the patient begins to eat or performs a few movements, the picture changes completely. Violent respiratory movements appear, the face assumes the expression of air-hunger, and the cyanosis increases. Still, however, the conditions return nearly or quite to normal as soon as the patient rests. But in many cases the patients are not comfortable even during absolute rest; the dyspnea does not disappear, but persists in varying degrees. The condition of some patients is, at least, bearable, especially if it has existed for some time, because the sufferer learns to adapt himself

* See Krehl, "Pathologische Physiologie."

† Compare B. Lewy, "Zeitschrift für klinische Medicin," vol. xxxi, pp. 321 and 520.

to his disability in a variety of ways. But the slightest complication, which would not produce any respiratory disturbance in a healthy person, may completely upset the "calm before the storm"; for example, a small pleuritic exudate, a mild attack of bronchitis, moderate elevation of the diaphragm, overloading of the stomach—any one of these factors may suffice to bring on dyspnea. It is frequently necessary to take into account a combination of various causes.

In the severest cases there is no need of any special cause to produce the dyspnea, which is always present because there is a constant disproportion between the needs of the organism for gaseous interchange and the amount of such interchange which the circulation is able to effect even during rest. In these frightful conditions the cause, as a rule, is not merely retardation of the blood-current.

Engorgement of the pulmonary vessels may, as has been stated, produce dyspnea even when the velocity is not diminished. Peculiar changes in the pulmonary tissue are thus effected, and the condition is of practical importance in those cases in which an obstruction in the left ventricle is neutralized by increased action on the part of the right. Under such circumstances a disturbance similar to what has been referred to as occurring in the lower grades of diminished velocity may be present even during rest; only the feeling of anxiety is more intense and becomes a more prominent symptom. The latter is a sign of some value, and another characteristic factor of this condition is the paroxysmal occurrence of the respiratory disturbances. Sometimes during the day, more frequently at night, the patient is seized with a sensation of anxiety and a feeling as if he were unable to get air in the usual way. Patients vary greatly in their descriptions of symptoms, but they all agree that there is dyspnea associated with fear. While the attacks in this form are bearable and are chiefly important in a symptomatic sense, they change altogether as soon as diminished velocity of the blood is superadded to the pulmonary congestion. This must, obviously, produce a very peculiar combination; that is, the right ventricle necessarily does more work than the left, although not quite enough to keep up the original difference between arterial and venous pressure in the lungs.

This condition develops whenever the two sides of the heart are unequally involved in the morbid process. The respiration is then accelerated, deepened, and forced in the extreme; all the auxiliary muscles are called into action; the patient assumes the oddest attitudes in order to obtain supports for more muscles to aid in respiration and relieve the thorax as much as possible. The patients, as a rule, cannot lie down, and if they stay in bed at all, must have their bodies raised; or they are unable to remain in bed and have to sit up or even stand.* The air-hunger is great and may attain an extreme degree; intense anxiety develops. When one considers the forced position assumed by the patient, the insomnia, the edema, and all the other effects of a severe circulatory disturbance that are often present at the same time, the full horror of this condition and the agony of the poor sufferer may be imagined. There is no doubt that the severest grades of this frightful dyspnea are produced by a combination of pulmonary congestion and retardation of the blood-current. Relief may sometimes be obtained if one of the two factors is removed or at least improved. For example,

*See the excellent description by Hope, "Diseases of the Heart and Great Vessels."

paradoxic though it may seem, the dyspnea improves materially as the force of the right ventricle diminishes. This is not infrequently observed, as v. Basch quite correctly points out, and the theoretic significance of the fact must not be overlooked. [See J. H. Musser's interesting paper, "Angina Pectoris: Its Relation to Dilatation of the Heart," "American Journal of the Medical Sciences," September, 1897.—Ed.] It is a direct proof that high blood-pressure in the lungs interferes with respiration. This view of the matter may have an important bearing on the administration of digitalis in many cases. If the use of the drug is merely followed by some improvement in the contractions of the ventricle and more complete filling of the lung with blood, without materially increasing the velocity of the circulation, what advantage is gained?

There are diseases, among which I may mention chronic myocarditis, nephritis, and sclerosis of the coronary arteries, in which this frightful condition of dyspnea persists for weeks or even months. Fortunately, this is not frequent. More often the severest type of dyspnea occurs only in paroxysms lasting from one-quarter to several hours. This *cardiac asthma* * is no doubt due to the same peculiar conditions in the action of both sides of the heart, and may occur in any disease of the heart muscle. It is most frequent, however, in sclerosis of the coronary arteries, especially when it involves chiefly the vessels of the left heart, and in contracted kidney.

I believe that a great many asthmatic attacks can be explained in this way without doing violence to the facts; at least, I have often succeeded in so interpreting the symptoms. However, the question whether the disease may not originate in some other way must still remain open. A great variety of causes has been suggested;† toxic influences in particular, such as are assumed to act in uremic and diabetic asthma, have been considered. But as we have not progressed beyond the stage of surmise in this subject, it need not be discussed at greater length. François-Franck,‡ by a series of experiments, revealed the existence of special relations between the heart and the respiration: stimulation of the endothelium at the base of the aorta and of the endocardium at the semilunar valves causes a disturbance of the respiration and possibly also modifies the width of the bronchioles. By accurate clinical observation these facts might possibly be turned to account in human pathology also.

A better knowledge concerning early and mild conditions of nocturnal dyspnea, of the origin of which nothing definite is known, seems to me particularly desirable. The subjects attacked are also, for the most part, affected with arteriosclerosis, and we know that sclerosis of the coronary arteries produces the most peculiar effects on the heart and its individual parts. Nevertheless, it is as curious as it is incomprehensible that other diseases of the muscle, as, for example, myocarditis of the left ventricle with or without valvular lesion, much more rarely produce these conditions, notwithstanding considerable pulmonary congestion. It would appear that anything in the way of a "paroxysmal" symptom is preëminently characteristic of coronary sclerosis. The same is true of stenocardia which is frequently associated with cardiac asthma.

* Compare A. Fränkel, "Berliner klinische Wochenschrift," 1888, No. 15, 16.

† See the excellent thesis by Fournier, "La Dyspnée cardiaque," "Thesis," Paris, 1892.

‡ François-Franck, "Archives de physiologie," 1890.

Fournier * may be right when he says that the German theories of cardiac asthma are one-sided because they attempt to discover one and the same cause under all circumstances. A number of factors have already been pointed out in this discussion. For the present we have no other positive data than retardation of the blood-current and pulmonary congestion; at all events, I, for my part, am unable to follow him when he boldly erects nervous and toxic forms of dyspnea, in spite of the fact that they are defended by so excellent a clinician as Huchard. Diminution of the so-called urotoxic coëfficient in the urine,† as a proof of intoxication, does not impress me as much as it appears to have impressed French investigators; and, besides, it would be necessary to show that the hypothetic poison also influences the respiration. The theory according to which these asthmatic attacks in heart patients are compared with similar attacks in uremia, or the two are even regarded as identical, is open to a number of objections. In the first place, all the other signs of uremic intoxication which usually accompany uremic asthma are absent; in the second place, the nature of uremic asthma itself is by no means clearly established. One thing is certain, however, and we owe our French colleagues a debt of gratitude for starting investigations which have shown that there exist peculiar relations between the phenomena produced by arteriosclerosis and uremic phenomena. The matter will be referred to again in connection with uremia.

Sometimes these asthmatic attacks apparently come like a bolt from the blue; that is, they are the first sign of disease. The attacks begin either during some muscular exertion after a meal, after psychic excitement, or with special frequency at night. I do not believe it possible as yet to offer any well-founded opinion as to the cause of its occurrence in the latter case (during the night);‡ in the former the increased demand on the circulation is probably the chief cause.

During the attacks the patients manifest the severest symptoms of dyspnea and anxiety, the pulse is usually small, soft, accelerated, and very often irregular and unequal. Attacks also occur, however,—for example, in arteriosclerosis,—when the weakness of the left ventricle is comparatively slight, and in these cases the arterial pressure may be higher than normal. Indeed, the occurrence of the attacks is partly associated with rise of pressure. After the attack the patient usually feels fatigued and prostrated for some time. Some recover completely; others never recover from the horror of the event, as happens sometimes after a severe attack of angina pectoris.

Not every attack of paroxysmal dyspnea, however, attains this degree of severity. Like more protracted respiratory disturbances, the paroxysmal dyspnea exhibits a great many gradations, depending on the severity of the circulatory disturbance which is the basal condition. In many cases the patient merely complains of a slight feeling of air-hunger, coming on either without any known cause or due to one of the above-mentioned factors; the patient awakes at night and cannot "get his breath properly." This condition lasts half an hour or an hour, after which the patient goes to sleep again. In more severe attacks the patient has to leave his bed and hasten to the window.

In many cases of cardiac asthma no objective sign is discovered in the

* Fournier, *loc. cit.*, p. 48.

† See Fournier, *loc. cit.*, p. 81. Ducamp, "Montpellier médical," 1891, No. 7.

‡ [See pt. I, p. 116.—Ed.]

respiratory apparatus except the change in the respiratory movements. Quite frequently, however, the signs of exudation into the alveoli are present in the form of medium-sized and small mucous râles, stridor, and the expectoration of serosanguineous, meat-juice-colored or clear sputum—in other words, all the signs of pulmonary edema.

The exudation may be uniformly distributed over both lungs, in which case the condition probably represents true congestive edema, such as Cohnheim observed in his famous animal experiments, and v. Basch later made an accurate study of with his pupils.* In the animal this edema develops after a severe injury to the left heart when the action of the right is good. Qualitatively, therefore, the same factors come into play as those which are operative in the production of cardiac asthma; if these factors reach their highest development, they produce not only a functional respiratory disturbance, but congestive edema in addition. The symptoms of that condition become pronounced whenever the injury to the left heart is sufficiently severe. Complete paralysis of the left heart creates the most favorable condition for its production. In muscarin poisoning spasm produces the same result.

As I have already remarked elsewhere,† the theory that pulmonary edema is due to stasis or passive congestion cannot be altogether discarded. In those cases in which cardiac asthma leads to exudation of serous fluid into the alveoli which disappears completely after one or two hours one's natural pathologic feeling, it seems to me, is distinctly in favor of such an origin. It is equally certain, however, that the details of the pathogenesis of this form of edema are by no means clear. For animal experiments prove nothing except that, under certain conditions of extreme violence, such as are realized very rarely in sick persons, congestive edema, may take place; no pathologist who is at the same time a physician will, for the present at least, dream of applying these experiments to man. Any attempt in that direction would be met by the numerous objections that have been made by excellent physicians.‡

Since that time, however, our views in regard to the value of animal experiments in clearing up pathologic questions have, unless I am mistaken, undergone a material change. No intelligent man would attempt to apply data obtained from healthy animals directly to the conditions existing in human beings in disease. These data merely enlarge our knowledge of the behavior of the organism in general, and in that way furnish some assistance in the enormous and difficult interpretation of phenomena observed at the bedside. Congestive edema is also possible in the lung, as animal experiments have shown beyond the possibility of a doubt. The fact that in the healthy animal complete paralysis or severe spasm of the left ventricle is necessary to produce the condition does not prove that an equally severe cause is necessary in the case of man whose blood-vessels have surely been altered by long-continued congestion. So far as I can see, there is no reason why, in the latter case, a mere disproportion, more or less marked, between the functional capac-

* Grossmann, "Zeitschrift für klinische Medicin," vol. xii, p. 550. *Ibid.*, vol. xvi, pp. 161 and 270. *Ibid.*, vol. xxvii, p. 151. Cf. Löwit, Ziegler's "Beiträge," vol. xxvii, p. 151.

† Krehl, "Pathologische Physiologie," p. 120.

‡ Sahli, "Archiv für experimentelle Pathologie," vol. xix, p. 433. *Ibid.*, "Zeitschrift für klinische Medicin," vol. xiii, p. 482. Jürgensen, v. Ziemssen's "Handbuch der allgemeinen Therapie," vol. i, ii, p. 228. A. Fränkel, "Diagnostik der Lungenkrankheiten," Vienna, 1890, p. 187.

ity of the two sides of the heart, should not suffice to produce the same effect. I merely say there is nothing against such a view—it remains for future investigation to reveal the true conditions.

Frequently the attacks of dyspnea with exudation into the alveoli manifest themselves by quite a different clinical picture.* The abundant moist râles are heard only over a part of the lungs, not infrequently, for example, over one of the upper lobes. The percussion-note may be slightly impaired here and there. The above-described sputum is produced, or the sputum is red from the beginning and cannot be distinguished from the rusty sputum of pneumonia. Sometimes there is low fever, or the temperature may be quite elevated; collapse and coldness of the peripheral portions are not infrequently present. The attacks often last some time—from twelve hours to one or two days. Accordingly there are numerous signs pointing directly to an inflammatory process in the lungs, and this view is fully borne out by pathologic anatomy.

At all events the inflammation in these cases is peculiar both anatomically and etiologically. Traube † designated the condition *serous pneumonia* and emphasized the great importance of venous stasis in its production. The congestion prepares the soil for an infectious inflammation.‡ The latter leads to the production of a watery exudate with little tendency to coagulate, and may involve individual lobes or only parts of lobes, or, in other cases, attack large portions of the lungs. So far as I can see and the present state of our knowledge permits us to judge, we have to deal with an intermediate form between a chiefly transudative and a purely inflammatory process. It is possible that the development of this inflammation bears some relation to the bronchitis, which is present as a rule.

Many points in this problem still require elucidation; as in the case of edema and inflammations in the course of nephritis, it is evident that certain special factors are at work. The foregoing view may assist in explaining the variability of the clinical symptoms—the fluctuation between transudative and exudative processes, between conditions of shorter and of longer duration.

In the course of the foregoing discussion we have seen that anomalies of the circulation may lead to pathologic-anatomic processes in the lungs as well as to functional disturbances of the respiration. Such processes are, in fact, quite frequent, and it is evident that the interference with the proper renewal of the blood favors the deposition of the micro-organisms of inflammation and enables them to obtain a permanent foothold. The observations of F. Müller§ help us to understand this even more clearly.

Heart patients with congestion in the lesser circulation very frequently suffer from obstinate *bronchitis*. The bronchitis is often very mild, and its only significance is that it is present, *i. e.*, its presence shows the existence

*Compare Sahli, "Archiv für experimentelle Pathologie," vol. xix, p. 433. *Ibid.*, "Zeitschrift für klinische Medicin," vol. xiii, p. 482. Huchard, "Maladies de cœur," p. 227. A. Fränkel, "Diagnostik der Lungenkrankheiten," Vienna, 1890, p. 187. J. Honnorat, "Processus histologique de l'œdème pulmonaire," "Thesis, Lyons," 1887. Marfan, "Traité de médecine," Paris, 1893, vol. v, p. 412.

† Traube, "Beiträge," vol. iii, pp. 152, 307, and 313.

‡ Kockel, "Naturforscherversammlung, Frankfort," 1896, p. 30.

§ F. Müller, "Sitzungsberichte zur Beförderung der gesammten Naturwissenschaft zu Marburg," 1896, No. 6. *Ibid.*, "Münchener medicinische Wochenschrift," 1897, No. 49.

of a pathologic process and not infrequently serves to suggest a careful observation of the heart to the expert clinician. Hence, from the standpoint of diagnosis, the bronchitis is always very important, and sometimes it is of clinical importance also, especially in patients with a good deal of dyspnea, in whom the slightest obstacle to the entrance of air into the alveoli, such as results from bronchitis under all circumstances, may constitute a serious disturbance. How much more so when the catarrh involves the finer branches of the bronchial tree.

Heart patients present the most various forms of dry and moist catarrh. To employ a modern terminology, the bronchial mucous membrane may become infected in a variety of ways and the inflammation may assume a great many different forms, depending on the condition of the circulation and of the mucous membrane. The patients always cough and often expectorate. The sputum will be discussed presently.

The lungs themselves are always affected by passive congestion if it is at all protracted, and develop the condition known as *brown induration*. The development of connective tissue produces a true cirrhosis;* the walls of the blood-vessels are frequently the seats of endarteritis. Extravasations of blood lead to the accumulation of brown pigment. In the form of hemosiderin this is taken up by large cells, expelled with these cells by the ciliary movement and coughing in the form of sputum. The finding of these heart-failure cells is a positive sign of the existence of brown induration,† at least for one who is familiar with the cells and is certain not to confound them with carbon-laden cells. Orth's observations‡ have shown that large numbers of vessels may be occluded if the accumulation of pigment is abundant. Orth's findings, in my opinion, also settle the question of the origin of these heart-failure cells. There has been a great divergence of opinion as to whether they represent epithelium or leukocytes. Orth found masses of connected cells and thereby recognized them as alveolar epithelium.

In addition, there are a great many varieties of peculiar, *secondary pneumonia* due partly to circulatory anomalies and partly to the greater liability to infection incident thereto. These processes usually develop in the most dependent portions of the lower lobes. Dulness, bronchial or diminished vesicular breathing, crepitant, all kinds of moist, sonorous, and non-sonorous râles, are found in the most irregular order and grouping. Fever may or may not be present. There is no rule for anything, and he who expects to find typical conditions will be disappointed.

As thrombosis of the veins or of the auricles is quite frequently observed in heart patients, the symptoms of pulmonary embolism or infarct and of embolic pneumonia and pleurisy are by no means rare. I mention this merely for the sake of completeness, as there is no occasion to go into a detailed description of the symptoms.

Hydrothorax is very apt to cause a disturbance of the respiration quite apart from its causes, which in themselves produce dyspnea, because it is usually bilateral and cannot help diminishing the volume and the excursions of the lungs. It is evident that the disturbance is proportionate to the quantity of the accumulated fluid.

* Boy, "Du poumon cardiaque," "Thesis," Lyons, 1883.

† E. Wagner, verbal communication. F. A. H. Hoffmann, "Archiv für klinische Medicin," vol. xlv, p. 252. Lenhartz, "Deutsche medicinische Wochenschrift," 1889, No. 51. Cohn, "Ueber Herzfehlerzellen," Dissertation, Würzburg, 1890. Krönig, "Charité-Annalen," vol. xv, 1890, p. 227.

‡ Orth, "Virchow's Archiv," vol. lviii, p. 126.

Cardiac patients very frequently have *pleurisy*, both dry and exudative. The exudate is either unilateral, or, if it is associated with hydrothorax, at least greater in one side of the chest than in the other. The fluid is serous, but rich in albumin, often somewhat hemorrhagic, and immovable, or does not change its level so easily as a simple transudate.

The exudate may persist for a long time—months and even years.

Romberg states that the size of the exudate increases and diminishes with the patient's condition. I have observed the same thing in arteriosclerotic and in nephritic persons. The temperature may be normal; not infrequently exacerbations of the process go hand-in-hand with evening rises of temperature.

These exudates frequently owe their origin to an infarct or to an atypical pneumonia. But these conditions cannot by any means always be demonstrated, and in such cases the process in the pleura is, therefore, primary.

Careful examination is absolutely necessary in order to recognize the pulmonary symptoms; in this way, and by carefully noting the sputum, bronchitis and, as a rule, pneumonic processes will rarely be overlooked. It is also possible to determine whether the dyspnea is pulmonary or cardiac. But it is by no means always possible to make a diagnosis. The patients are often so gravely ill that one naturally shrinks from subjecting them to the additional discomfort of a thorough examination, and sometimes the patient's condition is such as to render examination absolutely impossible. But any one who is familiar with these conditions and will bear them in mind will, nevertheless, usually succeed in discovering them. It is not unimportant to do so, for the prognosis in every case is greatly affected by these complications, which are frequently the direct cause of death.

Our judgment of the "attacks" must be, to a very large extent, based on the history. As the disturbance is comparatively mild, it is most important to interrogate the patient minutely, and, in forming an opinion, to correlate the patient's statements with the objective findings, which, so to speak, illustrate his story. In deciding the question whether a nocturnal attack of dyspnea is due to bronchial or to cardiac asthma the objective findings are extremely important. Emphysema and bronchitis with expiratory dyspnea suggest bronchial asthma, while signs of arteriosclerosis point to a cardiac origin. If the physical signs are absolutely negative, as may frequently be the case in the beginning, the decision of this question may be exceedingly difficult or even impossible until the patient has been under observation for some time.

The bearing of congestive bronchitis, the signs of cardiac lesion in the lungs, and the various forms of pneumonia on the diagnosis of the case, as a whole, is sufficiently evident from the foregoing discussion. Attacks of cardiac asthma with or without one of the forms of pulmonary edema always indicate a very grave process, for they occur chiefly in sclerosis of the coronary arteries and are always a sign of serious lesion of the left ventricle. The effect of these lesions on the prognosis in an individual case will be discussed in connection with arteriosclerosis.

In the **treatment** of these respiratory complications we must, of course, first of all, remove the cause of the disturbance, that is to say, the insufficiency either of the entire heart or of individual portions of the viscus. This point is discussed at length in the section devoted to Treatment. In the attacks of asthma or pulmonary edema with a bad pulse,

in addition to giving camphor, ether, coffee, and wine, cutaneous stimulation should be tried in various ways: by applying a large mustard-plaster to the chest, by means of hot foot-baths, etc. Brilliant results are often obtained in pulmonary edema by venesection and the withdrawal of 200 to 300 c.c. of blood from the cephalic vein. I advise this procedure in all severe cases, especially in robust individuals with high blood-pressure. But even in weakly persons and when the pulse is bad, one ought not to be too timid. How venesection acts is not easy to understand; it must be assumed that the diseased portion of the heart (left ventricle) and the abnormal vessels are in some way benefited by relieving the engorgement in the blood-channels.

In severe attacks of cardiac dyspnea morphin in the dose of 0.01 to 0.02 gm. ($\frac{1}{6}$ to $\frac{1}{3}$ gr.) should always be administered hypodermically; it does no harm and alleviates the patient's terrible suffering. Whatever therapeutic measures are adopted to combat these attacks must be energetic. Bad as the conditions may seem to be at the first glance, they may be only transitory, and the patient may continue in good health for a long time. In all these conditions the physician must be politic and prudent in his choice of methods, as well as in the order of their application. It is hardly necessary to add that all the other general measures which are indicated in the treatment of abnormal conditions of the bronchi and lungs, such as fresh air with a suitable degree of moisture, hydrotherapeutic and other similar procedures, must be utilized to their fullest extent as far as the condition of the circulation permits. In cases of exudative pleurisy I first try sodium salicylate if the drug is not counterindicated by the condition of the heart. If it fails and the exudate becomes alarming on account of its size alone, or because of its interference with respiration, thoracocentesis must be performed without delay.

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THE SKIN.

The skin and mucous membranes in heart patients often have a bluish color (*cyanosis*). The chief cause of the phenomenon is the presence, in the affected portions, of large quantities of reduced hemoglobin, but it is not so easy to say positively just what peculiar conditions of the circulation and respiration are necessary to fulfil the above condition. There is no doubt that disturbance of the circulation alone is capable of producing a marked cyanosis; both animal experiments and anesthetization in man furnish abundant proof of this. But it is evident that in these cases the giving off of oxygen must be very greatly diminished—at all events, marked cyanosis may be present without any appreciable diminution in the oxygen-content of the venous blood. On the other hand, venous congestion alone may produce cyanosis. So much is clear. Difficulties, however, arise in cases of cyanosis in which neither the venous congestion nor the interference with respiration is at all marked, and such

cases are quite frequent. Much additional detailed observation will be necessary before we shall be able to understand such cases. The condition of the vessels in the skin and mucous membrane ought, above all, to be thoroughly examined in heart patients, for it is, unfortunately, a fact that we know practically nothing about the behavior of the arteries or of the veins in heart disease. So far as I can see, cyanosis depends, in the first place, on the quantity of reduced hemoglobin present in the affected portion of skin; the quantity may be increased when the interchange of gases in the lungs is insufficient, but the increase may also be due to dilatation of small veins in the skin and consequent overfilling with venous blood. Hence venous congestion, by retarding the flow of blood and at the same time withdrawing larger quantities of oxygen, may also contribute to the production of cyanosis. Circulatory disturbances may easily exist in the skin even when there is no general venous congestion, and, finally, the association of circulatory and respiratory anomalies is much more apt to produce cyanosis than either respiratory or circulatory disturbance by itself. The question is, therefore, a difficult one, and its elucidation much to be desired in the interest of practice. For the present we regard cyanosis as a sign of venous stasis and of deficient respiration. No doubt we are right in doing so; but the diagnostic value of this phenomenon would be greatly enhanced if we had a clearer insight into its origin.

The bluish discoloration is seen at first most distinctly in the lips, the tip of the nose, the cheeks, and the ears. In other words, the peripheral portions, where the skin exhibits the greatest degree of curvature, furnish the most favorable conditions for the production of cyanosis. One is forced to assume that the circulation is most interfered with at these points. Venous stasis and retardation of the blood-stream most rapidly produce the discoloration at these points. These same parts of the body become blue after exposure to cold. In that case the flow of blood in the skin is very scanty and the blood probably flows more slowly in the cutaneous vessels, so that a larger proportion of the oxygen is used up. This leaves a comparatively large quantity of reduced hemoglobin and thus fulfils the chief requirement for the production of cyanosis. It is obvious, therefore, that the phenomenon may appear under a variety of circumstances.

The skin often feels cold to the touch in heart patients, especially at the peripheral portions of the body. This phenomenon must be attributed directly to the retardation of the blood-current and the insufficient supply of oxygenated blood, which also explains why those portions of the body which most readily become cyanotic are also the first to feel cold to the touch.

For the occurrence of *icterus* see under Liver, p. 521.

BLOOD AND LYMPH.

The quantity of water contained in the blood and in the tissues in circulatory anomalies must be considered here because the views that obtain on the subject and which do not by any means rest on a sure foundation have been at once utilized as a basis for therapeutic efforts.

We are chiefly concerned with the effect on the circulation of the composition of the blood. What effect has the quantity of the blood? Cer-

tain well-known investigations performed in Ludwig's laboratory* have shown that within wide limits the blood-pressure is independent of fluctuations in the quantity of the blood. From this fact it has been inferred that the work of the heart is also independent of that factor. Nothing could be farther from the truth. For an increased quantity of blood causes more complete filling of the heart, and, since the blood-pressure does not fall, also increases the demands on the organ. There is no doubt, therefore, that an increase in the quantity of blood is capable of materially increasing the work of the heart. But is there such a thing as an increase of the entire quantity of blood? That is the great question. It has never been definitely settled, for we are absolutely without any suitable methods to solve the question in the living subject, particularly in man. On the other hand, it cannot be denied with the same confidence as was frequently done in the past that the entire quantity of blood may be increased; for the negative result obtained in animal experiments is by no means decisive in questions of this kind, and in many conditions occurring in man there is, in my opinion, presumptive evidence of the existence of true plethora.†

It is very probable that an increase in the quantity of blood may take place in heart patients in the form of a serous plethora, that is, an increase of the quantity of water contained in the serum. For there is no doubt that in many patients suffering from cardiac weakness, especially when the power of the right ventricle‡ is diminished, the albumin, or rather the specific gravity of the blood and of the serum, is reduced.§ As the strength of the heart improves and the venous pressure falls, the arterial pressure rises; as the velocity of the circulation increases the blood regains its normal composition. This observation was made in a number of cases by excellent observers with the aid of reliable methods. Stintzing and Gumprecht determined the solid substances contained in the blood, using a method the slight fallacies of which are accurately known, and Hammerschlag's method is also quite reliable, provided the work is performed rapidly.‖ Askanazy employed the same or similar methods.

A diminution of the solids in the blood as a whole or in the serum may be brought about by a considerable reduction in the number of red blood-cells, as in the case of anemia, or by the destruction of serum-albumin, such as is observed in cachexia following malignant tumors. Neither of these possibilities is admissible in cases of uncompensated cardiac lesions and, as Stintzing and Gumprecht quite correctly remark, the less so as the

* z. B. Worm-Müller, "Berichte der sächsischen Gesellschaft der Wissenschaften," vol. xxv, 1873, p. 573.

For this question of the composition of the blood the famous treatises of Becquerel and Rodier, "Recherches sur la composition du sang," etc., Paris, 1844 and 1846, or German translation, 1845; "Nouvelles recherches d'hématologie," etc., Paris, 1852; C. Schmidt, "Charakteristik der epidemischen Cholera," Leipzig, 1850; Andral and Gavarret, "Recherches sur la modification," etc., Paris, 1840, German translation, Nördlingen, 1842, are still authoritative. See also the newer publications quoted below.

† Tigerstedt, "Skandinavisches Archiv für Physiologie," vol. iv, p. 241.

‡ See Stintzing and Gumprecht, "Archiv für klinische Medicin," vol. liii, p. 265.

§ See, for example, Hammerschlag, "Zeitschrift für klinische Medicin," vol. xxi, p. 475. Stintzing and Gumprecht, "Archiv für klinische Medicin," vol. liii, p. 265. Grawitz, "Archiv für klinische Medicin," vol. liv, p. 588. Askanazy, "Archiv für klinische Medicin," vol. lix, p. 385. This paper contains a complete bibliography.

‖ Compare Zuntz, "Pflüger's Archiv," vol. lxv, p. 539.

change in the findings was positively observed both during compensation and during failure of compensation.

On the other hand, swelling of the blood as a whole—a kind of edema of the blood—offers an accurate explanation of the findings; for it would indicate, at the same time, an increase in the entire mass of blood, a serous plethora, and I cannot see what objection there can be to this theory in the cases described. There is no good argument against it. Experiments on healthy animals, which have been utilized in the attempt to solve the question, appear to me quite unsuitable because, as v. Recklinghausen quite correctly states,* such experiments do not afford a convincing theory in regard to the nature of gradual processes taking place in the human subject during disease.

I believe, therefore, that we are justified in saying that in many patients with uncompensated cardiac lesions—the variety of cardiac disease is immaterial—the existence of a serous plethora may be assumed with much show of probability. It must not be thought, however, that the conditions, generally speaking, are in any sense clear. On examining the question critically it is found that practically everything still remains to be investigated. Above all, observations are needed to determine by some reliable method the different properties of the same kind of blood at the same time in a large number of patients, and it would seem feasible to make a comparison which should be above criticism between the arterial and the venous blood in animals, as, for example, dogs with uncompensated valvular lesions. For it seems probable that demonstrable differences† often exist between the arterial and the venous blood in disease; but with the observations at present available I will not venture to express a definite opinion in regard to their nature or their cause. Most of the blood examinations in man were performed with blood obtained by making a puncture in the tip of the finger or the lobe of the ear, and it seems to me that by such a method it is hardly possible to obtain any positive knowledge of conditions in the large vessels. But in animals with chronic disease the entire question could be solved in a manner to defy criticism, because blood certainly derived from known vessels can be examined.

The difference in the results obtained in the various examinations is the best proof that the question is still surrounded by obscurity. There is no doubt that in many patients with disturbances of compensation the blood has been found abnormally concentrated, although the kind of cardiac disturbances failed to give any explanation of the fact.‡ This must be taken into account, and it is the more curious because concentration of the blood, as a whole, is often associated with an increase in the quantity of water contained in the serum.§ For the present, however, I consider it quite impossible to obtain a clear insight into this question. Owing to differences in the methods employed and the sources from which the blood was obtained, the individual observations cannot be strictly compared one with another, nor has the problem been approached from a sufficient number of different directions. For example, to obtain a clearer

* v. Recklinghausen, "Allgemeine Pathologie," p. 110.

† Zuntz and Cohnstein, "Pflüger's Archiv," vol. xlii, p. 303. Oertel, "Archiv für klinische Medicin," vol. l, p. 293. Grawitz, *ibid.*, vol. liv, p. 588. Compare the contrary opinion expressed in his excellent treatise by Askanazy.

‡ See Naunyn, "Schweizer Correspondenzblatt," 1872, p. 300. Penzoldt and v. Bamberger, "Wiener klinische Wochenschrift," 1888, No. 1. Schwendtner, "Dissertation," Bern, 1888. Schneider, "Dissertation," Berlin, 1888.

§ Askanazy, *loc. cit.*

insight it will be indispensable in the future to determine the volume of the red corpuscles and of the salts contained in the body and in the serum. Satisfactory methods for this purpose are available.* It does not, therefore, seem to me that it would be a difficult matter to throw some light on these questions by means of additional exhaustive and painstaking observations. A comparison of the blood obtained from different kinds of vessels would be of the greatest value.

How and why hydremia develops, on the one hand, and increased concentration of blood, on the other, are questions that admit of a great many surmises. Dyspnea increases the evaporation of water from the lungs; low arterial and high venous pressure diminish the quantity of urine. There is, besides, as we know, a very close relation between lymph- and blood-vessels. Differences in pressure and concentration of the fluid are at once followed by the passage of saline solution from one system into the other. But how much better it would be if, instead of surmises, we could base our opinions on accurate observation! And how many things there are to be examined! First of all, one would need to know the quantity of water contained in the individual tissues in different cases and to be able to compare it with that of the blood; we shall advert to this point again in connection with edema. There is also urgent need of some real knowledge of water metabolism. Of what use are repeated comparisons between the quantity of urine and the food when we do not know how much water is excreted by the skin and lungs? Surely this is not beyond the scope of inquiry.

A large clinic equipped with a Pettenkofer-Voit's apparatus and with sufficient clinical material could settle all these questions; for the methods have been worked out. We must not rest content with the assumption that the quantity of water which escapes from the body in so-called insensible ways bears the same percentage-relation to the entire excretion in disease as in health. The proposition still remains to be proved. It is the irony of fate that the first work on "difference-determinations," that is, a comparison of the quantity of water in food with that in the urine, was done by a savant of the same university to which we owe our most important knowledge of the water metabolism in the body through such investigators as Pettenkofer, Voit, and Rubner. The large respiratory apparatus in Voit's laboratory would, I presume, have been equally available for heart patients.

EDEMA AND DROPSY.

As we have seen, a more complete knowledge of the quantity of water contained in the tissues in circulatory disturbances would be most desirable. We know most about the accumulation of fluid in the subcutaneous tissue, where the condition is known as *edema* and is readily recognized by clinical methods. Whenever a large quantity of lymph accumulates in the loose meshes of the subcutaneous tissue, a swelling is produced. The external form of the affected part of the body undergoes a change; it becomes less slender; normal depressions disappear. In heart patients in whom, as will be presently shown, venous stasis must be regarded as the chief cause, the accumulation of lymph always shows itself in the dependent portions of the body because the effect of gravity

*Compare M. and L. Bleibtreu, "Pflüger's Archiv," vol. li, p. 151. Kossler, "Centralblatt für innere Medicin," 1897, No. 26 ff.

is greatest in those parts. Bed-patients accordingly exhibit edema first in the buttocks and scrotum and in the heels; those who are going about, in the ankles and legs. The skin is swollen and glistening in proportion to the degree of swelling. When a part overlying a hard surface is indented with the finger, the pit remains for some time (the parts "pit on pressure"); the crepitation characteristic of cutaneous emphysema is absent. A positive diagnosis is accordingly quite simple and easily made.

The degree of edema varies greatly in different cases from the slightest swelling, which can be discovered only by careful examination, to the most ungainly deformity of the entire body.

The intensity of the swelling depends on the degree of stasis, for the latter is the chief factor in the production of edema. When the right heart is weak, the pressure in all the veins of the body increases and, as the lymph-vessels empty into the veins, they are also affected by the increase in venous pressure. In the small veins it is probable that the endothelium suffers some functional deterioration owing to the combined effect of the increase in pressure and the diminution of velocity; the endothelium becomes permeable to the blood-serum, and a lymph collects outside the blood-vessels, which contains less albumin and leukocytes and more erythrocytes than normal lymph. Owing to the persistent increase of pressure in the vessels, the elasticity of the tissues is impaired; for the tissues, as we know, bear a considerable portion of the blood-pressure.* This again leads to the loss or impairment of forces which propel the tissue-juices from the lymph-spaces into the true lymph-vessels, and hence also into the heart. The lymphatic circulation is specially retarded by the fact that the pressure in the subclavian veins, into which the lymph flows, increases so that the "fall" of the blood is diminished in every way. In short, it is not difficult to understand the entrance of large quantities of blood into the tissue-spaces nor the interference with its movement—in other words, the development of edema. It is needless to say that edema affects not only the meshes of the subcutaneous tissue, but that of all the organs as well, particularly in tissues that are not naturally endowed with much elasticity and in which the elasticity is easily diminished by a slight degree of pressure.

Hence the degree of edema varies in different organs; but, on the other hand, the time of its appearance also varies considerably in the same tissue in different individuals, even when it may be assumed that the degree of venous stasis is approximately the same. These differences depend on individual variations in the sensitiveness of the blood-vessel walls and of the tissues.

Dropsy of any severity at all is always a disturbing symptom, because it renders movement difficult. Edema of the prepuce interferes with urination; hydrothorax and ascites embarrass the breathing.

When the swelling becomes extreme, the skin may give way in places. This is followed by oozing of fluid. It is, to a certain extent, a conservative event, but one that is fraught with considerable danger because most unpleasant infectious, inflammatory processes often start in such regions.

THE DIGESTIVE APPARATUS.

The organs that make up the digestive apparatus are affected by disturbances of the general circulation chiefly when stasis develops.

* See Landerer, "Die Gewebespannung," Leipzig, 1884.

The symptoms referable to the *gastrointestinal canal* are distinguished by their indefinite character. The patients complain chiefly of anorexia, nausea, and frequent belching. Vomiting is rare; constipation is sometimes present. Hemorrhoids sometimes become more swollen as the result of stasis, and not rarely bleeding occurs from them.

The patients very frequently complain of a sense of pressure, oppression, and a feeling of fulness after eating. These symptoms may, of course, be due to disturbance of gastric motility, but it is possible that the mere filling of the stomach suffices to produce disturbances in patients who can breathe freely only when the diaphragm is absolutely unhampered in its movements and when no special demand is made on the respiration.

That gastrointestinal symptoms develop chiefly as the result of stasis cannot be doubted, but we are still quite ignorant of their direct causation. In some cases the cause is probably a functional disturbance of the cells resulting from the circulatory anomaly. But the latter sometimes also gives rise to catarrhal conditions in the mucous membranes of the digestive tract, especially when there is marked involvement of the liver. Under such conditions stomatitis also quite frequently develops and the tongue in many cases is heavily coated. But on this point also much still remains to be said; even the older observers* were struck by the discrepancy between clinical symptoms and pathologic findings.

There is no doubt, at least, that gastric symptoms occur more easily and more quickly in some persons than in others. They may be the very first symptoms. The patients seek the physician's advice solely for their stomach troubles, and in these cases error can be guarded against only by carefully considering all the factors in the problem.

The special conditions in stomach digestion have been repeatedly investigated and with varying results.† It is evident that the secretory and motor activity of the stomach varies greatly in different cases. In the intestine the absorption of fat ‡ is frequently diminished in circulatory disturbances that produce a slowing of the blood-current; in other cases it may be normal. The absorption of nitrogenous substances does not, as a rule, exceed the average.

Circulatory disturbances exert an important influence on the *liver*. In this connection, again, all that we know anything about is passive hyperemia, and our knowledge is limited in the main to semiology and the subjective symptoms complained of by the patient. Whether, and if so how, the hepatic function is modified as a result of circulatory anomalies is, for the present, beyond our ken.

The liver is exceedingly rich in veins, belonging to the vena cava and the portal vein; it contains enormous quantities of blood; the hepatic veins are very near the right auricle and are not separated from that by valves—all these things help us to understand that venous congestion has a marked effect on the liver, the more so as the tissue of a living, healthy liver is exceedingly soft and the organ itself is very distensible.

* Z. B. Frerichs, "Leberkrankheiten," vol. i, S. 377.

† Z. B. Hüfler, "Münchener medicinische Wochenschrift," 1889, No. 33. Adler and Stern, "Berliner klinische Wochenschrift," 1889, No. 49. Hautecoeur, "Étude sur les troubles et les lésions de l'estomac chez les cardiaques," Thesis, Paris, 1891. Jorns, "Ueber das Verhalten der Magensecretion bei Herzkranken," Dissertation, Würzburg, 1893.

‡ F. Müller, "Congress für innere Medicin," 1887, p. 404. *Ibid.*, "Zeitschrift für klinische Medicin," vol. xii, p. 45. Grossmann, *ibid.*, vol. xv, p. 183. Husche, "Zeitschrift für klinische Medicin," vol. xxvi, p. 44.

Owing to the softness and vascularity of the organ it has frequently been asserted that the function of the liver is to take up large quantities of blood in order to protect the heart, and especially the right ventricle, against excessive engorgement; it was thought to do this especially in a number of animals, such as dive under the water, for instance. This is not the place to enter into this question, nor are the observations on this point by any means complete.

In venous stasis, therefore, the liver becomes enlarged. The lower border occupies a lower position, as can be readily demonstrated by percussion; if the congestion is very great, the upper border may even be displaced upward. Dulness is not infrequently present over the lower half of the lower lobe of the lung on the right side, which must be attributed solely to the swelling of the liver. Soon the outline and form of the organ can be felt; the surface is at first smooth, hard, and sensitive to pressure; the edge is rounded and displaced downward some distance. In women swelling of a lobe due to lacing may give rise to the most remarkable tumors, lying far down in the abdomen and often quite movable. [Twice I have known this to be mistaken for ovarian tumor. One patient died suddenly (mitral stenosis) the day before she was to undergo laparotomy for tumor; in the other a few doses of digitalis caused the globular tumor to withdraw from the pelvis to the liver region, continuous with the liver. —Ed.] The enlargement of the gland affects all its dimensions, including those of the left lobe, and causes enlargement of the upper portions of the abdomen far into the left hypochondrium. This encroachment on the intraperitoneal space is undoubtedly one of the reasons why even moderate overfilling of the stomach is apt to produce a feeling of fulness and pressure.

The capsule of the liver contains sensory nerves, which are irritated as the tension of the peritoneal covering increases. A feeling of pressure and pain, therefore, belongs to the clinical picture of hepatic congestion. Not infrequently pain is the very first sign—that is, the pain may develop even in very mild grades of hepatic enlargement. Often the pain becomes severe and it always renders the patient uncomfortable.

Like all the other phenomena due to stasis, the enlargement of the liver is irregular in its occurrence and cannot be determined beforehand. According to French physicians, its development is affected by the previous condition of the organ.* While it is practically never absent when there is a marked interference with the flow of blood toward the right heart,—although the cause of it cannot be said to be the intensity of stasis,—it, nevertheless, varies greatly in different individuals, just as is the case, for example, with edema and albuminuria. It is probable that the elasticity of the hepatic tissue is normally subject to individual variations and, in addition, it may perhaps be influenced by pathologic processes.

Pathologically the process begins as a simple passive hyperemia, which readily causes swelling of the distensible gland.† If the hyperemia continues, the liver-cells at the center of the acini between the dilated blood-vessels undergo atrophy. In a transverse section taken from the center of a lobule the vessels are the most prominent features, while the periphery presents a more brownish or yellowish appearance, depending on the amount of pigment and fat in the cells. In combination with the

* Faure-Miller, "Gaz. de Paris," 1891, No. 2 and 3; ref. "Virchow-Hirsch," vol. ii, p. 122.

† Compare Frerichs' excellent exposition of the subject with illustrations, in his book on "Diseases of the Liver," vol. i, p. 372.

red center of the acini this produces the picture of the so-called nutmeg liver. Around the hepatic vein the liver-cells are replaced by connective tissue which, at first soft and rich in cells, later shrinks and thus ultimately leads to a peculiar kind of cirrhosis which gives the surface of the organ an unequal and nodular character. This can often be felt in the living subject; the liver appears hard and irregular. Not very infrequently patients with marked hepatic congestion develop a variable degree of *jaundice*, the milder forms being by far the most common. The jaundice is always due to the deposition of true bilirubin in the cells, never to derivatives of bilirubin. The absorption of bile is undoubtedly caused in some cases by catarrh of the small intestine due to the congestion; but intestinal catarrh is by no means the only cause of jaundice in heart patients, for the condition may also be due to intrahepatic changes which we do not as yet understand. Liebermeister and Minkowski's theory of biliary secretion in an abnormal direction is quite plausible.

It should be mentioned in connection with diagnosis that hepatic congestion is often the first symptom to be noticed by the patient and for which he consults the physician.* In such a case a *thorough examination of the entire body* is the only safeguard against painful surprises, which, however, are always avoidable if that precaution is faithfully observed.

The large, smooth, hard liver of hepatic congestion may be mistaken for a number of other diseases of the liver which produce similar changes in the shape of the organ. I shall not discuss the differential diagnosis in detail, because to do so I should have to describe a large part of liver pathology. The essential thing in these cases also is to demonstrate the existence of a primary cardiac disease. When this has been done, two other questions remain to be decided. First, is the cardiac anomaly sufficient to explain the presence of hepatic congestion? In cases of general stasis this presents no difficulties, but it must be remembered that swelling of the liver may be the only symptom of venous stasis, particularly in many forms of arteriosclerosis.

The second question is whether congestion is the only pathologic condition in the liver or, in other words, whether the heart patient is not also suffering from some other liver disease. This question can only be decided by carefully weighing everything that points to a special involvement of the liver in the morbid process. Sometimes it appears to me to be very difficult to decide whether a heart patient suffering from venous congestion also has cirrhosis of the liver.

French physicians are of the opinion† that in persons who are predisposed to diseases of the liver and in whom the conditions for their development are fulfilled a simple congestion not infrequently develops into some other anomaly of the organ. I myself have known heart patients in whom the edema and albuminuria disappeared completely, while swelling of the liver with granular surface, enlargement of the spleen, and ascites persisted until death. At the autopsy no signs of diseases of the peritoneum were found, the liver was large, hard, and granular from the presence of contracted connective tissue which had its origin in the central portions of the lobules. Such cases give one the impression that the secondary disease

* Compare Parmentier, "Gaz. des hôp.," 1891, vol. lxiv, p. 229; ref. "Schmidt's Jahrbücher," vol. ccxxxiii, p. 191.

† Tapret, "Union médicale," 1889, No. 87 and 90. Quoted from "Virchow-Hirsch," vol. ii, p, 162. Hartle, "Gaz. méd. de Paris," 1891, vol. lxii, No. 2 and 3; ref. "Schmidt's Jahrbücher," vol. ccxxxiii, p. 191.

of the liver may cause additional symptoms; for these symptoms closely resemble those of Laennec's cirrhosis. Alcoholism was not present in the cases referred to.

It does not seem probable to me that the condition was simply one of general stasis; the chief argument against such a view is the marked enlargement of the spleen. It is true that the spleen enlarges under the influence of congestion; but, so far as I remember, the enlargement is not great enough to make the organ accessible to palpation during life unless some other causes are also present. At least, the splenic enlargement is not out of proportion to the enlargement of all the other organs. But in the cases that I have just cited there were never, at any time, any signs of stasis. There was only cirrhosis of the liver with ascites and splenic enlargement, and it seems most logical to attribute these symptoms directly to the disease of the liver which, in turn, was dependent on the stasis. Practically the same thing is true in the case of the kidneys.

The question to what extent cirrhosis may develop from hepatic congestion has been thoroughly discussed in German, and especially in French, literature. French physicians particularly insist that marked splenic enlargement and ascites develop after *foie cardiaque*, although the congestion elsewhere in the body by no means shows even an approximately proportionate development. The connection is by no means clear, for severe disease of the abdominal organs is observed very frequently in association with pericardial adhesions. In the section on the Sequelæ of Mediastinopericarditis will be found the literature on the subject, and in that section I have pointed out that *inflammatory* processes of the serous membranes probably play an important part in these very conditions.

THE KIDNEYS.

In a large number of cases we observe renal and cardiac symptoms side by side. The two sets of symptoms may be interrelated in a variety of ways. In the first place, there is no doubt that in a large group of patients the changes in the heart depend on changes in the kidney, *i. e.*, they are only secondary to the latter. The relation between the heart and kidneys will form the subject of a special section.

Again, cardiac and renal disease may coëxist as coördinate results of a common cause. This is very frequently the case—much more frequently, no doubt, than has heretofore been believed. The reason is that blood-vessels and kidneys are in many respects sensitive to similar influences and exhibit irregularity and inequality in the order in which they succumb to such influences. This question will have to be touched upon in almost every section.

In a third group of cases the cardiac affection causes functional changes in the kidneys. In these cases the causal factor always resides in stasis of the venous blood and the lowered pressure in the arterial blood. The effect of circulatory disturbances on the secretion of urine* has been determined by a series of beautiful investigations, especially some carried out under C. Ludwig's guidance in the Vienna Josephinum. A rise in the venous pressure, accompanied by a fall in the velocity of the blood-current, diminishes the excretion of urine, the diminution affecting the watery, more than the solid, constituents; at the same time the cells of the kidney become permeable to the albumin in the blood-serum.

* See Heidenhain's excellent treatment of the subject in Hermann's "Handbuch," fifth edition, vol. i, p. 314; also Cohnheim, "Allgemeine Pathologie," second edition, vol. ii, p. 313.

The quantity of urine is diminished usually to less than one liter (quart), and often much more. The amount of fluid ingested by the patient must, of course, be taken into account, and this statement is based on the assumption that the patient takes in an average quantity of fluid which would correspond to the secretion of one and one-half to two liters (quarts). The urine becomes more concentrated and the specific gravity rises. A sediment is usually precipitated. Soon albumin makes its appearance, being the usual albumin and globulin of the blood-serum; the quantity is often quite small—about 0.5 to 2.0 in 1000 by the inaccurate method of Esbach. I believe, however, that many authors lay too much stress on the low degree of albuminuria in passive congestion of the kidney ("Stauungsniere") as compared with nephritis. I have seen very much larger quantities of albumin in the urine in cases of uncomplicated passive congestion.

The sediment is usually quite abundant and contains chiefly urates and uric-acid crystals, leukocytes, a few red blood-cells, occasionally renal epithelium, and very frequently tube-casts. All authorities agree that hyaline casts are never absent, but I have often seen granular and epithelial casts in cases in which I felt perfectly justified in assuming that the condition was purely one of passive congestion.

The anatomic changes at first consist simply in swelling of all the veins and capillaries within the gland. This causes swelling of the entire viscus, which becomes large and of a dark bluish red, especially in the medullary portion. As the renal tissue is naturally much firmer than that of the liver, the degrees of enlargement observed are, as a rule, relatively less than in the liver.

If the circulatory disturbance ceases, the passive hyperemia of the kidney disappears altogether. The quantity of urine is increased and is often greatly in excess of the normal because an opportunity is now afforded for the excretion of the water that has accumulated in the body. The urine becomes pale, the specific gravity falls, the albumin disappears, and normal conditions are speedily restored.

Sometimes the congestion persists for weeks and months and then the condition becomes much more complicated. At first the kidney remains large and hard; now, however, the enlargement is due not only to the increased quantity of blood, but also to an increase in the scanty connective tissue of the gland and to round-cell infiltration, especially in the cortex, around the glomeruli and the blood-vessels. Degeneration of the epithelium is superadded. The walls of the veins and arteries become thicker. Gradually the newly formed connective tissue begins to contract at definite points on the surface, obliterating urinary tubules and glomeruli, and the kidney becomes smaller. In severe cases the volume may be reduced to one-half or even one-third. There develops a true contracted kidney with numerous depressions (scars), especially on the surface. The histologic changes of contracted kidney have been very carefully investigated, especially by Schmaus.

The severer grade of this disease, which Bollinger has designated "cyanotic contracted kidney" (Stauungsschrumpfniere), occurs as a result of a great variety of conditions that lead to venous stasis, but is more frequent in the forms of cardiac disease which are associated with excessive beer-drinking than in any other. Schmaus questions that the venous stasis is the sole cause of the condition and suggests that arterial processes may also contribute to its production. This question will be

taken up again; for the present we must consider the character of the urine in *chronic passive hyperemia.*

In chronic passive hyperemia the urine is concentrated and diminished in quantity; it contains albumin, white and red corpuscles, and always hyaline, quite frequently granular, but rarely epithelial, casts. I have repeatedly convinced myself that the last two varieties of casts also occur in this condition. The interpretation of the urinary findings may be quite easy and again may present enormous difficulties, especially in cases of myocardial disease. According to the books, it is a very simple matter, but in my opinion this is far from being so. As a rule, one has to decide whether the condition is passive hyperemia of the kidney secondary to dilatation of the heart due to one cause or another, or Bright's disease with secondary disease of the heart. In cases of typical valvular lesions the problem is much simpler because in those cases alterations of the kidney are much more frequently, not to say always, secondary.* If in a case of myocardial disease that is probably primary there is found, in addition to other typical symptoms of congestion in the liver and skin, for example, scanty, concentrated urine containing but little albumin, a few formed constituents, and hyalin, and possibly a few granular casts, one is justified from the beginning in saying that in all probability the renal processes are secondary; and if, on the administration of digitalis or diuretic remedies, the cardiac insufficiency and renal symptoms disappear, one may be fairly sure of one's diagnosis.

It is obviously most desirable to know something about the course of the disease in order to be able to give a positive opinion; and until such knowledge has been obtained, I would advise great caution and reserve in giving a diagnosis. Without this precaution one is easily led into error, because even in true nephritis the formed constituents in the urine may be very few in number, and the whole urinary picture is not infrequently identical with that of passive hyperemia.

The decision is most difficult when the urine, in addition to albumin and hyaline casts, contains numerous granular and epithelial casts, as well as red and white blood-corpuscles. In such cases I would advise the physician first to determine by careful examination whether there is cardiac insufficiency and whether signs of congestion are present in the other organs. If such signs are found, as, for example, a thickened liver or edema and weakness of the right ventricle, passive renal hyperemia is quite probably present. At least I would not, in making a diagnosis, place too much dependence upon the statement that only a few hyaline casts are found in passive hyperemia to the exclusion of granular, epithelial, and blood-casts; for I have but too frequently seen the opposite. I consider it very important in every case to be on the lookout for any other features in the case that would render the presence of renal inflammation probable. For example, albuminuric retinitis is almost a direct proof; uremic symptoms, to say the least, point strongly in favor of inflammation, and the kind and distribution of the edema may be characteristic.

If there are no signs of congestion in other organs, an extremely guarded opinion must be given; for it is to be borne in mind, as I have already stated, that the individual organs in different persons vary in their susceptibility to a general rise of venous pressure and that the kidney alone may be affected by it. In my opinion this marks the limit of our present diagnostic powers. The old pathologists held that passive hyperemia

* Compare Traube, "Beiträge," vol. ii, p. 433.

was one of the causes of Bright's disease. It is true that Traube undertook to distinguish clinically between passive hyperemia and nephritis, and he was unquestionably right in doing so in many cases, especially during the beginning of venous stasis. It seems to me doubtful, however, whether the distinction can be made in every case. The pathologic conditions have already been referred to. They show that long-continued, passive hyperemia almost regularly leads to hyperplastic processes in the kidneys which very closely resemble the processes of chronic inflammation.

There is another point that still further complicates the question. Unless I am altogether mistaken, the symptomatology of nephritis is also in a transitional stage or, to be more accurate, we are beginning to recall statements which were first made by the classic authors, but which, in the course of time, have been, to say the least, neglected. I do not think any one can doubt that there are cases of nephritis in which albumin is totally absent for long periods at a time. Frerichs* mentions this fact and states that it was known to the older investigators. E. Wagner† also mentions it, and so does Leyden.‡ It is a well-known fact that in typical contracted kidney, in which the quantity of urine is increased and the specific gravity is low, albumin is frequently absent; but that is not what I refer to. There are patients with nephritis who excrete an average quantity of urine, of average specific gravity, and absolutely free from albumin. The heart shows hypertrophy and dilatation of both ventricles, without valvular lesions. Suddenly traces of albumin make their appearance and the patients become uremic. The intoxication may pass away and the albumin disappear from the urine again for months. I have seen these things in cases of old arteriosclerotics (see the description of that disease). It is probable that in these cases, as in true nephritis in general, for example, in contracted kidney, albumin will appear after exercise. If a patient of this kind, when examined, has albuminuria with hyaline, granular, and epithelial casts in addition to cardiac hypertrophy, how is it possible to make a differential diagnosis between secondary and primary renal disease? And if albumin is entirely absent from the urine, who will dare to diagnose primary nephritis? Yet it cannot be excluded!

I cannot go into any more detail on these matters because the anatomic data available are still too inaccurate and too unreliable; various other factors besides tend to make the diagnosis of these conditions still more difficult. For example, the presence of granular and epithelial casts in itself by no means justifies a positive diagnosis of inflammation in the kidneys, as the urinary findings in bicyclists appear to me to show. Again, the differences existing between the so-called congested arteriosclerotic and true contracted kidney, both as regards the pathologic anatomy and the clinical symptoms, are still far from being perfectly known.

It has already been stated that many cases of so-called congested and contracted kidney may owe their origin not to passive congestion, but to arterial processes—that is to say, are not dependent upon cardiac insufficiency. The arteriosclerotic contracted kidney has, in my opinion, received much too little attention as a clinical entity. Owing to its imperceptible onset and irregular course it is evident that, in many cases,

* Frerichs, "Die Bright'sche Nierenkrankheit," p. 61.
† E. Wagner, "Der Morbus Brightii," p. 268.
‡ Leyden, "Zeitschrift für klinische Medicin," vol. ii, p. 156.

the disease produces very faint, and at the same time variable and intermittent, symptoms, which are readily overlooked unless the patient is under observation for a long time, and in any case are difficult to interpret. The greatest caution and reserve in diagnosis are, therefore, indicated in these cases. At all events I have an impression that insidious nephritis plays an important part in the production of changes in the size of the heart and that it must be thought of even in cases in which no suspicion of its presence would arise during a routine examination.

Examination of the eye-grounds is of the greatest importance. A typical radiating figure with hemorrhages in a patient presenting albuminuria is, generally speaking, a positive sign of nephritis. I know that the phenomenon may also occur in other conditions, and that in a few cases it has been observed in patients who did not present the slightest signs of kidney disease. But such a thing is extraordinarily rare, and it is safe to say that the combination of a radiating figure with albuminuria is practically a positive sign of the existence of nephritis.

Granting the possibility of embolism taking place in the case of heart disease, it is obvious that the accident may befall the renal arteries as well as any others. Such an accident gives rise to anemic, or much more rarely hemorrhagic, infarcts in the kidneys, the anatomy of which need not be described here. Infarcts probably produce no clinical symptoms, at least I cannot remember ever to have seen any. Trustworthy authors, however, connect the appearance of blood in the urine and pain in the region of the kidneys with the development of infarcts.

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GENITAL ORGANS.

There is nothing special to be said about the condition of the genital organs in myocardial disease. Women with chronic circulatory disturbances not infrequently suffer from anomalies of menstruation, and the heart sometimes fails to respond to the extra demand thrown upon it during pregnancy, and particularly during parturition; so that, as we all know is the case in valvular lesions, child-bearing may become the cause of cardiac insufficiency in the morbid conditions with which we are dealing.

THE NERVOUS SYSTEM.

The nervous system is affected by heart disease in a variety of ways. I shall, of course, disregard all accidental combinations, which must necessarily occur in the case of two such large and frequent clinical groups.

The fact that in grave circulatory disturbances thrombi may form at many points in the body and may send out emboli affords an opportunity

for the development of a great variety of focal diseases of the central nervous system. On the other hand, the interference with respiration by a change in the composition of the blood is also effected through the medium of the nervous system.

The *phenomenon of Cheyne-Stokes breathing* is not infrequently observed in heart patients. Its clinical features are as well known as its mode of origin is obscure.* At least there is no doubt that it always indicates grave alterations in the character of the blood and of the brain-cells, and, therefore, develops whenever the blood-supply of the latter is impaired; for the present, however, we are unable to explain fully the original cause of the phenomenon. It indicates a change in the brain-cells—therein lies its importance; it is a grave symptom. But in this matter also I would sound a warning against the tendency to go by hard-and-fast rules. In fact, one should never give an unfavorable prognosis based on a single symptom, not even on Cheyne-Stokes breathing.

Headache, vertigo, tinnitus aurium, flashes before the eyes, and syncopal attacks are quite frequently observed either singly or in combination. The headache may be very variable, both as regards severity and duration; it may be permanent or paroxysmal, and may be referred to different parts of the head. Sometimes the headache is a concomitant of general nervousness, but quite often it is unaccompanied by any neurasthenic symptoms, and in such cases is generally as obscure in its origin as the other above-mentioned symptoms, unless it can be attributed to a profound cerebral anemia. In many cases, however, that is impossible.

The condition of the heart exerts a considerable influence on the entire psychic behavior of the patient. The symptoms of heart disease, and still more the knowledge of its existence, very frequently produce general depression; this was noticed by the older authors, for example, by Kreysig. The patients cease to enjoy life, become discouraged, and lose confidence in themselves, especially when they have never learned self-control. The most enthusiastic eulogist of the present time will hardly be able to deny that the inordinate desire for personal happiness which, as appears both from a large portion of our literature and from the views of large numbers of people, especially among the so-called educated class, is regarded as the only justifiable and desirable aim in life, is not calculated to strengthen the character. We find but few among our patients nowadays who even take the trouble to try to control themselves, and still fewer ever learn to do so.

Thus the symptoms of hypochondriac neurasthenia of every grade, from the mildest to the most severe, very frequently develop in heart patients, and it is undoubtedly the knowledge of being afflicted with heart disease that forms the connecting link. I shall not go into detail; for practically all the symptoms observed in neurasthenia are exhibited by patients of this type. As a rule, it is not difficult to recognize the symptoms as neurasthenic, but it is not always easy to gain a clear idea of the connection between the cardiac and the nervous symptoms. Quite frequently the latter are dependent on the former, as has already been stated. On the other hand, there are numerous cases in which neurasthenia is the primary condition and causes symptoms referable to the heart; for nervous cardiac symptoms, as we shall presently have to explain, usually mean simply that the neurasthenia manifests itself first in a disturbance of the heart. Nevertheless, it may be exceedingly difficult to decide whether

* Krehl, "Pathologische Physiologie," p. 210.

the heart symptoms are nervous or muscular, or, to put it differently, how much of the cardiac disturbance must be attributed to general nervousness and how much to disease of the heart. To what extent this is possible will be explained in discussing nervous conditions.

While neurasthenia often develops secondarily to heart disease, hysteria, in a similar relation, is comparatively rare. There is no doubt that the two conditions are sometimes associated, as might be expected in the case of two such common diseases. But I cannot remember, during recent years, ever to have seen hysteric phenomena develop as the result of a myocardial disease, and I attach very much importance to the fact that Romberg has had the same experience. It is true that French physicians hold an opposite opinion; but I do not believe that in this particular point we can compare their people with ours; the national differences are too great.

There is nothing special to be said about the relations existing between epilepsy and heart disease; it is now known that the association of the two diseases is usually accidental.* I have occasionally seen in heart patients conditions of intense excitement with hallucinations. The attack would occur either without any cause or as the result of some occurrence which would hardly disturb a normal individual. If nothing else, this shows the importance of bearing in mind that heart patients are always extremely irritable and are subject to unexpected nervous explosions. This fact must be borne in mind in connection with the treatment.

When no special cause can be demonstrated for one of these attacks, the case is usually found to be an attack of cardiac weakness lasting for several days or even weeks, and attended by the usual unpleasant symptoms such as dyspnea, anxiety, inability to assume a comfortable position, and insomnia. Hence it seems probable that in these cases a condition resembling exhaustion may be at the bottom of the trouble; the patients are both fatigued in the extreme and excited at the same time, intensely irritable, and sometimes even inclined to violence. They try to get out of bed; to leave the room; they are slow to recognize people whom they know perfectly well or fail to recognize them altogether; give wrong orders and then fly into passion if they are not obeyed. The condition is not infrequently complicated by auditory, and especially visual, hallucinations.

After a few days, especially if improvement takes place in the circulation, or even without it, the attack, as a rule, passes away. Heart disease does not specially predispose to the development of chronic mental disturbances.†

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SUBJECTIVE DISTURBANCES OF THE CARDIAC FUNCTION.

DYSPNEA.

Subjective dyspnea occurs in association with anomalies of the cardiac function in one of two ways: First, disease of the circulatory apparatus may be the cause of the respiratory disturbance. An attempt has already been made, in discussing the influence of cardiac disease on the respiratory apparatus (p. 504), to explain the pathology, which is exceedingly complicated.

Second, dyspnea and abnormalities of the cardiac function may be "nervous," in which case they must be regarded as due to a common cause, namely, some disease of the central nervous system. This point will be discussed in the section on Nervous Diseases of the Heart.

CARDIAC FEAR, CARDIAC PAIN, ANGINA PECTORIS.

Some heart patients are subjectively quite unaware of their disease, although this is exceptional, particularly in the conditions with which we are here concerned. As a rule, some abnormal sensations are present, at least at times. The character of these sensations varies very greatly. We have already mentioned the sensation of dyspnea, and have attempted to show how it may be explained. It is usually, but not invariably, associated with a sense of fear; severe dyspnea, in the beginning at least, always produces fear. Sometimes, however, the patients become so used to the condition of dyspnea that they lose their dread of it, so to speak, and the sensation of fear becomes less and less prominent. On the other hand, fear may be disproportionately great in mild degrees of dyspnea. It is evident, therefore, that the two conditions are, to a great extent, independent of one another, and there is no doubt that the patient's personality plays an important rôle, although we do not as yet understand the subject in all its details.

Fear occurs in heart patients even when there is no dyspnea whatever; it is often the only sensory symptom. I, of course, disregard the conditions of "heart fear" occurring in melancholic and paralytic individuals; for we are dealing only with heart patients. In the former class the fear is of a purely psychic origin, and changes in the cardiac function, if present, are only secondary. It is well known that fear not infrequently influences the action of the heart and of the blood-vessels in a variety of ways, and if the patient accidentally is also a sufferer from arteriosclerotic changes in the heart,—which in this case need not necessarily have anything to do with the production of the symptoms,—it is readily seen that it may be difficult and require much penetration to arrive at a correct understanding of the origin of the various disturbances. It is possible that in these cases also there may be a deeper connection than one is inclined to think. Thus, it is possible that the heart is more apt to be affected by fear in patients whose physical condition, owing, for example, to the existence of arteriosclerosis, predisposes them to such disturbances.

The sensations of fear that occur in nervous, neurasthenic, and hysteric subjects have a close pathogenetic relation to the condition just described. The fear in these cases rests on a basis of psychic abnormality which in many cases manifests itself chiefly by cardiac phenomena. This point will be discussed more fully and an attempt will be made to show to what

extent we can recognize implication of the heart. At the same time an accurate description of these symptoms will be given. (See the section on Nervous Diseases of the Heart.)

For the present we are interested in the occurrence of fear dependent upon diseases of the heart. Viewed from this standpoint, the sensation is, therefore, a symptom which may be produced by any functional anomaly of the heart. It is extremely frequent not only in sclerosis of the coronary arteries, but also in myocarditis and the various other forms of muscular disease. Careful interrogation of the patient hardly ever fails to elicit complaints of this kind.

The symptom occurs in every conceivable degree. The patients complain of a feeling of anxiety and constriction or oppression; sometimes the sensation has an element of pain.

Pain may be the most prominent feature; it may even be present alone, without any trace of fear. That, however, is very much more rare. The painful sensations vary exceedingly in different cases; but, so far as I can observe, and so far as I have been able to ascertain from patients, they have a certain peculiarity in common. Patients who are careful observers can readily distinguish heart pain from other sensations localized in the cardiac region. For the present we do not know the precise conditions of its development. We are only aware that pain is much more frequent in diseases of the heart muscle, especially in diseases of the coronary arteries and at the base of the aorta, than when the disease is located at the venous orifices. Nothnagel lays particular stress on this point.*

Even at this stage not a few patients complain of the indefinable sensation which is so terribly feared by sufferers from pronounced stenocardia, and which is described as that of impending annihilation. Not that the symptom is observed in this severe form, but the patients, nevertheless, complain of a good deal of pain. Finally, paresthesiæ and pain radiating to the left arm are also present.

On the whole, these sensations exhibit a great variety of different shades, both as regards the severity of the individual impression and the association with anxiety, oppression, and pain. It is impossible to give a description applicable to all cases, as the statements of individual patients vary widely. It is quite natural that it should be so; for what we are told is, of course, greatly influenced by the patient's own personality.

As a rule, palpitation is also present. This peculiar symptom will be discussed presently in its proper connection, and I shall, therefore, say nothing more about it here. (See the next section.)

The exciting cause of the sensations is likewise variable. In some cases the symptom makes its appearance whenever a special demand is made on the heart, as, for example, during muscular movements accompanied by exertion. Many individuals suffer terrible disability from the symptom, and in some cases are entirely incapacitated. Not infrequently, in the latter class of cases, physical examination of the heart would indicate that the functional capacity is still quite good; but the patients insist, nevertheless, that the oppressive symptoms are brought on by any attempt at work and absolutely force them to desist. Those who are engaged in manual work, of course, suffer the most.

Or the sensations are complained of at night, and are then associated

* Nothnagel, "Zeitschrift für klinische Medicin," vol. xix, p. 209.

with a certain degree of dyspnea. It is my impression that these milder conditions are particularly frequent at night, although the same may be true of angina pectoris. But the latter is very apt to occur during the day, mostly after overexertion.

In other words, we are dealing with the mildest forms of cardiac asthma and stenocardia. In arteriosclerosis these conditions are very frequent; they either precede the severe attacks or persist for many years with the same intensity. The patients awake at night after several hours of sound sleep with a certain sense of dyspnea or with a pain in the chest, and at once become anxious. Orthopnea is the rule. Some feel better if they walk up and down the room; others open the window and seek relief by inhaling the cool air. The attack lasts from one-half to two hours, after which the sense of oppression and the anxiety disappear. Sometimes the patients break out in perspiration, after which they are able to sleep again. While some patients feel quite well the next morning, others complain of more or less weakness.

These attacks belong neither to typical cardiac asthma nor to pure angina pectoris. They represent a mixed form and not infrequently a prodromal stage of the former. As I have already remarked, nature does not adhere to fixed schemes. The severe forms of angina pectoris are the most impressive and have always arrested the attention of observers. That is why they have received the most extensive description in the literature. Fortunately, however, the above-described milder phenomena are much more frequent in practice and, therefore, much more important from a practical standpoint, as Laennec pointed out. The conditions under which they occur indicate their similarity to the severe attacks and, as a matter of fact, there is no sharp line of separation between the two forms. While it is possible to differentiate by means of clear definitions and there is some advantage in doing so, especially for purposes of diagnosis and treatment, such definitions do not correspond to the actual morbid processes, which do not present any sharp boundary-lines. This should be borne in mind. For it is only by doing so that the very great frequency of sensory disturbances and their significance in the diagnosis of diseases of the heart can be properly appreciated.

A proper appreciation of the frequency of these milder forms of angina pectoris is also important if one would understand the pathogenesis of the disease. The severe attacks, as appears from what we have already said about cardiac asthma and from what we shall have to say about stenocardia, occur preëminently with sclerosis of the coronary arteries. This is also true of the milder forms, and the reason they are so frequent is that arteriosclerosis of the heart is a common condition. This, therefore, disposes of an objection that is so often made against the theory of a causal connection between these two conditions, namely, that sclerosis of the coronary arteries is quite common, while severe attacks of angina pectoris are rare. The explanation is that, for the production of severe attacks, certain special or especially marked changes are required, which, fortunately, are but seldom present. In the great majority of cases the attacks are mild and might perhaps appropriately be called *anginoid*.

It is needless to say that we are far from having a clear understanding of the pathologic process. We do not even know to what morbid process the sense of oppression, anxiety, and pain is due in those quite common cases in which we are unable to explain these symptoms on the ground of actual dyspnea.

The conditions known as *angina pectoris* or *stenocardia*,* in the narrower sense of the term, are characterized by an unspeakable and indescribable feeling of annihilation of a painful character, and associated with most intense fear.

The pain is seated behind the sternum, often a little to the left or to the right of the bone, much more rarely behind the apex of the heart. From this point of origin the pain may radiate to other regions, but this is not by any means a constant feature. Most frequently the pain radiates to the left arm, either over the entire distribution of the brachial plexus or only to some of its individual portions. Frequently the patients have a sense of weight and formication in the left arm. Occasionally the entire extremity feels as if paralyzed for hours after the attack, or must be kept absolutely immovable on account of the great pain. Vasomotor disturbances—pallor of the skin or, as described by Huchard, weakening of the pulse—are not infrequently observed in the arm and hand of the left side.

The pain may radiate to any other portion of the body, as the right arm, back, epigastrium, legs, and testicles.

Sometimes the pain attains its highest degree of severity in these remote portions of the body; or the latter represent the original, and in some cases the only seat of the pain. In such cases the diagnosis of stenocardia may be extremely difficult and, in order to identify the condition, great stress must be laid upon the accompanying feeling of anxiety and impending annihilation.

The pain is always very severe, and is usually described as constricting and oppressive. Although individual patients vary in their statements, such expressions as the feeling of a tight ring around the chest, an enormous weight on the chest, or a feeling as if the chest were compressed by an iron hand or crushed between two plates of steel are encountered again and again.

In addition to the pain there is always, except in a very few cases, a feeling of terrible oppression, a horrible fear, and a sense of annihilation. It often seems to me that this fear and sensation of impending destruction are the most terrible features of the condition and explain the patient's extraordinary dread of its occurrence. In the patient's mind, at least, the remembrance of these sensations is far worse than that of the pain. This is also mentioned by Latham and Huchard. The patients feel as if they were facing death, and the vision takes such a horrible form that many declare their willingness to bear anything rather than go through such terror again. As an old English and German description has it, there is "a general pause in the vital functions" so far as the patient's sensations are concerned.

The heart action itself is usually disturbed during the attack; the pulse is small, soft, often irregular and unequal, usually accelerated, and sometimes abnormally slow. Not infrequently embryocardia is present.

Hence *cardiac weakness is unquestionably the rule*, but according to the literature it does not invariably accompany the attacks. This fact has been positively stated by excellent observers, in fact, by the classic writers, on the subject, among whom I need only mention Heberden and Huchard.

*I omit the numerous other synonyms which have come up since this disease has become known. The two expressions used in the text may be regarded as having been generally adopted.

Some authorities even contend that the action of the heart may be stronger during the attack. There is no doubt that a large number of detailed and careful descriptions of the circulation during the attack would be most desirable. For it would be difficult to find a condition in which the statements of experienced and reliable observers exhibit such marked divergence, as any one may convince himself by examining the statements collected in the various text-books. Nothing is gained by citing the various theories, as every conceivable view has the support of physicians of the first rank. It only shows the necessity of further investigation. It is quite true that these investigations are not easy to carry out, for severe stenocardia is very rare and the patient's condition is so grave that it is not easy to make observations. But, at all events, the pulse [heart-beat or sounds—Ed.] can be counted and its properties, as determined by palpation, can be described. One hundred detailed clinical histories written by good observers would be of great value. That the pulse may be very bad during the attack is proved by direct statements to that effect found in the literature. Eichwald describes a very curious change during the attacks; but I have not been able to find any convincing case histories to prove that the action of the heart may remain unchanged during the paroxysm. In this connection, however, the reader is reminded of the very positive and explicit statements by O. Vierordt and A. Fraenkel.

A number of patients complain of palpitation during the attack, that is, they are aware of violent and irregular contractions. If cardiac insufficiency is present, the symptoms of acute cardiac dilatation may develop rapidly.

The respiration during true stenocardia is absolutely unaffected, which positively distinguishes the attacks from cardiac asthma. Typical cases of this kind do occur. The patients, in their effort to remain as quiet as possible, even hold their breath, which is certainly the best proof that there is no interference with respiration. As usual, however, uncomplicated cases are not the rule. Mixed forms* are quite often observed in which the attacks are accompanied by more or less severe dyspnea; that is to say, the patient first feels anxious and complains of pain, after which cardiac asthma is superadded, probably due to weakness of the heart.

The appearance of the patient during the attack is quite characteristic; the features are sunken, the face is pale and cold and wears an expression of agony, and of the most intense anxiety and distress.

The patients try, if possible, to avoid every form of muscular movement. If the attack occurs while the patient is in bed, he sits up and grasps the sides of the bed. If it comes on while the patient is walking, he grasps the nearest object, such as a fence or wall, and clings to it for support or braces some part of the body—the chest or the back—against it; but if he is unable to obtain some such support, he stops short, or crouches, or even sits down on the ground. The patient's whole demeanor, and the fact, finally, that he even holds his breath, indicate his extreme anxiety to remain perfectly quiet.

As a rule, and it must be added unfortunately, consciousness is preserved during the entire attack; the poor sufferers are forced to taste the full horror of fear. Occasionally, however, syncope takes place either during, or at the beginning of, the attack. As Vierordt has quite correctly

* See also Leyden's case histories, "Zeitschrift für klinische Medicin," vol. vii.

pointed out, such an event has diagnostic importance, as the stenocardia in such cases may be masked by an apparently benign process.

In organs the seat of abnormal sensations, and in others as well, the most peculiar functional and secretory disturbances, too numerous to be enumerated here, sometimes occur. For example, insufficiency of the sphincters, the excretion of a large quantity of pale urine, persistent belching, and a number of other phenomena are observed. The attack is quite commonly either accompanied or followed by profuse general perspiration.

The duration of the attack is variously given. In most cases it lasts only a few seconds or minutes, which, in view of the horrible nature of the symptoms, is most fortunate. But attacks lasting half an hour also occur, and some lasting even much longer have been reported. I believe attacks of such duration undoubtedly occur. In the cases of long duration that I have seen there were a number of short attacks in succession, during the intervals between which the patients did not return to their normal condition, but continued to complain of a sense of severe pressure on the chest and a certain degree of pain and anxiety, lasting many hours, and from time to time increased by the frightful sensations of a typical attack.

The *exciting cause of the attack* is found sometimes in muscular exertion. The degree of the latter which is necessary to bring on an attack varies with the individual. It is impossible to predict in a given case what particular form of muscular exertion will prove harmful, as a great many factors enter into the problem. On the other hand, some patients know perfectly well that if they perform such and such a movement they will get an attack. In these cases the above-described milder conditions of heart fear and oppression are present at the beginning and later give place to true stenocardia. Violent psychic emotion, a copious meal, or coitus may be the direct cause of the attack, as a large number of examples show. The peculiar cases described by Nothnagel in which a wide-spread spasm of the arteries appeared to be responsible for the occurrence of anginoid conditions will be discussed later. But all these causal conditions have in common that they make a greater demand on the heart.

But the attacks quite often occur while the patient is absolutely quiet. Not infrequently the attack occurs first at night, and may at once assume the most frightful severity; in these cases the direct cause of the attacks is absolutely unknown.

With regard to the number of severe attacks that may occur in the same patient, it is impossible to make a general statement. Some are only called upon to go through the horrible torment once, twice, or three times, either because death occurs in one of the first attacks or because the attacks do also actually disappear. On the other hand, the attacks may be very frequently repeated; in some cases several times or even frequently during the day, after every severe movement.

In other cases there may be intervals of freedom lasting months or years. It is impossible, therefore, to lay down a rule. One thing is positive, however: true stenocardia is always an exceedingly grave condition. Many of those who are subject to it die during the attack, and their manner of death is frequently more rapid and sudden than in any other fatal condition that I am acquainted with.

Death may be preceded by the well-known signs of stenocardia, but quite often there is not even time for their development; the patient

collapses while engaged in some occupation, and life is extinct. Stenocardia is probably the most important cause of sudden death in old people.

It follows, therefore, that a guarded prognosis should be given in every case; but it is impossible to tell beforehand how often the attack will be repeated nor which attack will prove fatal.

The incidence and distribution of well-marked angina pectoris are explained by the fact that arteriosclerosis, as will be presently shown, plays an important part in its production, particularly, as it seems, that form of arteriosclerosis which is associated with increase of the arterial tension.* The condition is undoubtedly quite common among the well-to-do, and particularly among people who are given to dietetic excesses. It is observed chiefly after the thirty-fifth year of life, and more frequently in men than in women. The same statements apply to stenocardia; at least such is my very positive impression, gained both from the literature and from personal experience. Blanc† observed, among 3813 private patients (with 380 deaths), 21 instances of palpitation and angina pectoris (4 deaths), and among 3835 patients in St. Thomas' Hospital in London (with 389 deaths) he observed palpitation only in 4 cases and angina pectoris not at all. It is true that even such statistics as these are not above criticism; for it may be objected, on the one hand, that well-to-do people formerly were in the habit of consulting a physician earlier and more frequently, and, on the other hand, the diseases represented in hospital and in private practice are quite different. There is no doubt that the only way to acquire reliable knowledge is by extensive observations in different kinds of private practice. Nevertheless, Blanc's statistics seem to me to be of some value because they are so large, because in both cases the clinical material is uniform, and because the patients were seen by the same physician.

Angina pectoris is a symptom. In attempting to understand its true nature one is naturally led to inquire whether the circumstances under which it occurs do not throw some light on the problem. Of one thing I am certain, both from the study of the literature and from my own experience: the conditions necessary for the production of stenocardia are most frequently produced by changes in the coronary arteries. Our interest naturally centers in arteriosclerosis, as most attacks of angina develop in its train. The seat and extent of the vascular disease vary much in different cases; sometimes only a few branches of the second and third order are affected. Other cases exhibit disease of the main trunks, or the arteriosclerosis is confined to the points of origin, that is, the first part of the aorta.

The fact that aneurysm of the aorta and disease of the semilunar valves are frequently associated with stenocardia harmonizes perfectly with this theory, for we know that these conditions are frequently produced by arteriosclerosis. Whether endocarditic insufficiency and stenosis of the aortic valves ever become the cause of anginoid conditions, and if so, whether they are as frequent a cause as arteriosclerosis, I have been unable to gather from the literature, and my own experience does not enable me to decide the point.

Psychic excitement, especially long-continued worry and exhausting

* Compare Nothnagel, "Zeitschrift für klinische Medicin," vol. xix, p. 214.

† Blanc, "Medico-chirurgical Transactions," vol. iv, p. 133. Quoted from Wunderlich, "Pathologie und Therapie," vol. iii, i, p. 54. I was unable to procure the original.

care, is not only a possible cause of an individual attack, but unquestionably also produces a predisposition to the occurrence of attacks. This also harmonizes with what has been said above, for again arteriosclerosis represents the intermediary agent. Mental distress undoubtedly favors the development of sclerosis of the coronary arteries.

The relation of syphilis to angina pectoris can also be explained through the medium of arteriosclerosis. During the secondary stage stenocardia is rarely observed, and when it does occur, always develops on a neurotic soil. The severe form of angina pectoris which is produced by syphilis and yields to antisyphilitic treatment is due to syphilitic aortitis or coronary arteritis* (see Syphilis of the Heart).

The various diseases in the course of which attacks of angina are occasionally observed, such as gout, diabetes mellitus, tabes dorsalis, and lead-poisoning, all favor the development of arteriosclerosis, and I cannot see what valid objection can be brought against Huchard's comprehensive view that the occurrence of stenocardia in these diseases is due to arteriosclerosis.

Finally, the occurrence of stenocardia in thrombosis or embolism of the coronary vessels is perfectly compatible with the etiologic significance of coronary sclerosis, since these forms of occlusion are practically always associated with that condition.

In earlier works, especially those of the great English physicians, fatty disease of the heart occupies a prominent place as the basis for the development of stenocardia. Nor does this view militate against this theory of ours in regard to the importance of arteriosclerosis; for many, at least of the English observations, would now, as will be shown later, be included in the section on Coronary Sclerosis.

Finally, angina has been observed in very rare cases as an accompaniment of chronic and acute myocarditis following infectious diseases. Such cases as these require minute observation of their every detail, especially for the purpose of determining whether arteritic processes, such as are often observed after the same causes, are present in these rare cases also. One of the things that should, in my opinion, be done in the near future, is to obtain accurate clinical histories and postmortem reports of a large number of persons suffering from angina pectoris. A problem of this kind can only be solved by the coöperation of practitioner and pathologist, and such coöperation is absolutely indispensable; for it must decide whether severe, life-threatening stenocardia can exist without arterial changes. With the material available at the present time I would not venture to answer this question, for many of the older observations are useless because but few are confirmed by postmortem reports.

Whatever else may be true, sclerosis at the root of the aorta and in the coronary arteries is unquestionably the chief factor in the problem. But how does arteriosclerosis produce this effect? It is evident that some special factor must also be present when one remembers the extraordinary frequency of coronary sclerosis and the comparative rarity of well-marked cases of stenocardia. Here I must interpolate a remark, not to say an objection. Well-marked cases of stenocardia unquestionably are rare; but it is equally certain that the above-described conditions of heart pain, heart fear, and oppression in varying degrees of severity are very frequently observed in arteriosclerotic subjects. It is purely a matter of taste where the line of distinction from true angina is drawn

* Huchard, p. 397.

or whether these conditions and true angina pectoris are included in the same category. For if the latter view is adopted, the question at once assumes a different aspect. Peculiar subjective disturbances are frequently observed in association with coronary sclerosis, and in rare cases these disturbances present the picture of a severe stenocardia. The question is: in what class of cases does this occur? What is the nature of the anatomic process that lies at the bottom of it? Certainly not sclerosis of the coronary arteries alone.* French investigators and Curschmann have called attention to the significance of mere stenosis of the large arteries, either at the orifice of the vessel or along its course, and this seems to me an important point. They observed angina pectoris of the severest type in cases of stenosis limited to the beginning of the large vessels and without extensive disease involving a number of arterial branches. I myself have frequently seen the same thing (see the section on Arteriosclerosis). Evidently this means that whenever the blood-supply of a portion of the heart is diminished or abolished, the essential conditions for the occurrence of stenocardia are created. A final factor is the disproportion existing between what the heart needs and what it gets, which is borne out by the well-known fact that severe attacks of angina preferably follow certain definite causes, such as muscular movements, coitus, and the like.

While stenosis of the arteries is a most important etiologic factor, it cannot be regarded as the only, nor even as the predominant, cause of the origin of the attack. On the contrary, stenocardia has also been observed in cases of occlusion of branches of the second and third order.† But this point unquestionably requires still further investigation, and, above all, it ought to be determined to what extent occlusion may take place without leading to stenocardia. There is a wide field before us, but the question will not prove an easy one to decide, because coronary embolism is not infrequently followed by sudden death, and the observer cannot learn in such cases whether anginoid symptoms were present; hence the investigation would have to include only those cases in which stenocardiac attacks were positively known to have occurred.

In my opinion, we are not justified in saying more than that the conditions for the occurrence of stenocardia in heart disease are probably brought about by the sudden development of a disproportion between what the heart needs and what it gets.

This disproportion, in many cases, leads to insufficiency of the heart muscle as well as to stenocardia. It is possible that the insufficiency is sometimes dependent on the anginoid condition, but in general Vierordt's view‡ that cardiac weakness and stenocardia are the effects of a common cause is probably correct. On the other hand, it would be absolutely wrong to regard the insufficiency as the cause of the angina, and any theory based on such an assumption should be finally discarded. It has already been stated that excellent observers have seen stenocardia associated with good heart action; and severe cardiac insufficiency, in contradistinction to cardiac asthma, is very rarely a precursor of stenocardiac attacks. But if we separate angina and cardiac weakness, I cannot see how the experiments of ligating the cardiac vessels, which have been per-

* This was distinctly stated even by the older physicians, as, for example, Testa-Sprengel, "Die Krankheiten des Herzens," p. 328.

† See, for example, Curschmann, "Congress für innere Medicin," 1891, p. 275.

‡ See his excellent report, "Congress für innere Medicin," 1891.

formed on animals, can help to clear up the nature of stenocardia, as is so frequently mentioned; although, of course, their importance for the solution of other questions is not to be denied. In my opinion, these experiments have nothing whatever to do with the question of stenocardia. Will any one believe that attacks of angina have been produced by these experiments? What they produced was cardiac weakness, and it is positively known that the nature of anginoid attacks is quite independent of that condition.

How are we to imagine that the terrible sensations of pain and fear are produced in the heart? They are brought about, of course, by the nervous system, which may suffer either directly or through the muscle. The question might, perhaps, be asked whether the point of attack is actually situated in that portion of the nervous system contained within the heart. Heberden, the first observer of angina pectoris, did not regard it as a symptom of a disease of the heart, [though he thought it spasmodic rather than inflammatory—Ed.], and even since its connection with anomalies of the heart began to be appreciated, repeated attempts have been made to locate the disease focus in every conceivable portion of the nervous system. But I believe that its primary seat in the heart is now established beyond the possibility of a doubt. The patient refers the abnormal sensation to the heart; in true angina the heart exhibits changes, and the attack is accompanied by functional disturbance. It would be like splitting hairs to deny the existence of a connection between the attack and the heart in favor of some remote and altogether unfounded localization.

But how shall we imagine the nature of the process in the heart? As we have seen, the important cause is a sudden lack of blood in some portion of the heart. If this theory is accepted, the analogy to similar processes in the voluntary muscles, pointed out by Potain, G. Sée, and A. Fraenkel, appears quite accurate. They refer to the phenomena of so-called intermittent claudication, a condition which has long been known in animals and which was described by Charcot [1856] in the case of man. Erb has pointed out, in his excellent recent work on the subject, that this condition also rests on an arteriosclerotic base; the narrowing of the vessels supplying voluntary muscles causes no symptoms during rest, but produces violent pain and paralysis as soon as the action of the muscle is increased and a greater quantity of blood is, therefore, required. [Space does not permit more than a reference to the valuable discussion by Ortner, "Zur Klinik der Angiosklerose der Darmarterien nebst einem Beitrag zur Klinik des intermittirenden Hinkens und des Stokes-Adam'sche Symptomenkomplexes," "Volkmann's Sammlung klin. Vorträge," No. 347, 1903.—Ed.]

What goes on in the myocardium itself? The violence of the phenomenon, its sudden onset and equally sudden disappearance, suggest a spasm. Indeed, the theories of heart-cramp play an important part in the explanation of stenocardia. By analogy with the painful cramps in the muscles of the intestines or of the calf of the leg and the resulting compression of nerve-fibers, this theory would render the occurrence of the frightful pain and of the fear quite intelligible. But the fact that the pulse is occasionally good and strong during the attacks, and that it is sometimes accelerated, is a strong argument against the assumption of heart-cramp; the only symptom that would fit this theory is the so-called "fluttering of the heart," which is not by any means present in

every case. These are all additional reasons why more accurate statements in regard to the behavior of apex-beat, heart-sounds, and heart action during the attack would be so desirable. In the curious case described by Heine the contractions of the ventricles were apparently abolished completely for a period which, under normal conditions, would correspond to from four to six cardiac revolutions. In this case spasm may have been present, and this patient had anginoid symptoms. [John Hunter, in one of his attacks, could feel no pulse in either arm for forty-five minutes. In one of Adams' cases of angina pectoris with Stokes-Adams symptoms, the pulse could not be felt in any artery of the body in the last six weeks of life.—Ed.] The theory of inflammation of the cardiac nerves advanced by certain French authors* shall be mentioned only to be dismissed, for I consider it as unfounded† as it is improbable.

In discussing the pathogenesis of stenocardia it must always be borne in mind that, although in heart patients changes in the coronary arteries create the conditions necessary for the production of an attack, the latter are by no means always associated with anomalies of the vessels. Epileptic attacks occur with a number of anatomic changes in the brain; they are found as the result of some forms of poisoning, in nervous women, and as the expression of epilepsy, a disease as curious as it is obscure. The same is true of stenocardiac conditions; they also develop in other conditions besides heart disease (in the latter case chiefly on a foundation of sclerosis of the coronary arteries), in cases of poisoning and of general nervousness. In other words, the excitation which produces an attack of stenocardia may begin in certain nerves of the heart in a variety of ways, of which one, and the most important, is a sudden stoppage of the blood-supply from disease of the vessels. This fact must be rigorously kept in mind in a theoretic discussion. In these other cases the stenocardia is, therefore, not a symptom of heart disease, but of some other condition, which sometimes lends to it a peculiar character.

Toxic angina pectoris occurs after the *immoderate use of tobacco*, especially after excessive smoking of imported cigars and cigarettes, a habit that is considerably more common in France, Russia, and Hungary than it is in Germany. But taking snuff and chewing tobacco to excess also seems to be harmful at times.

In association with other symptoms of tobacco-poisoning we sometimes observe attacks which absolutely resemble those that have just been described and which are sometimes due to the same exciting causes; for nicotinism favors the development of arteriosclerosis because men who smoke to excess, as a rule, are in the habit of indulging in other habits that tend to produce the same pathologic condition.

Tobacco poisoning, however, is capable of causing stenocardia directly, without the intervention of arteriosclerosis. How, I will not venture to say; for I cannot agree with Huchard that what we at present know of the pharmacology of tobacco holds out certain possibilities in this respect. But it is a well-known fact that after smoking has been given up, this form of stenocardia may disappear either at once or after a few months, never to appear again.

The attacks due to mere intoxication do not, as a rule, exhibit the

* Z. B. Lancereaux, "Bullet. de l'académie," vol. xxxii, No. 29. Grocco, "Settimana medic.," 1896, No. 1, 2.

† Hérard ("Bullet. de l'académie de méd.," 1883, No. 52) examined a heart after angina pectoris and found nothing abnormal in the cardiac plexus.

most severe type; pain and fear do not attain the same severity. On the other hand, the attacks are frequently much protracted and occasionally last several hours. Often they are accompanied by vasomotor disturbances and other symptoms of tobacco-poisoning: headache, vertigo, general lassitude, and gastric disturbances. As a rule, the heart action is extremely irregular and unequal, accelerated or retarded, and the attack generally comes on spontaneously, much more rarely after severe muscular exertion.

It is evident, therefore, that tobacco-poisoning is a possible cause of angina pectoris. In many cases, by carefully considering all the features of the case, the attack may be suspected to be due to nicotinism, and in such a case a very definite opinion can be expressed in regard to prognosis and treatment. If the patient will rigidly abstain from all use of tobacco, an absolutely favorable prognosis can be given.

In stenocardia due to disease of the gastrointestinal canal it is not definitely known how much must be attributed to the toxic influence and how much to reflex causes. That the symptoms of angina pectoris may be due to purely nervous forms of excitement is proved by the behavior of neurasthenic and hysteric individuals. Either spontaneously or under the influence of psychic excitement these patients develop conditions which very closely resemble the above-described attacks, from which, however, they can frequently be differentiated by certain characteristic features. This phase of the question will not be discussed here because it will have to be brought up again in the discussion of so-called nervous disturbances of the heart.

The attacks of vasomotor angina pectoris first described by Nothnagel,* and later by other observers, represent border-line cases. In these cases the attack is ushered in by partial or general spasms of the cutaneous arteries. It is possible that there may be a simultaneous increase in the demand made on the heart, but in my opinion this should not be assumed unless it is proved by an increase in the arterial tension. For blood-pressure is determined by the splanchnic, rather than by the cutaneous, vessels, if we may be allowed to apply the experience gained in animals to the case of man, which we have a certain right to do in this question especially. The cutaneous phenomena are followed by the attack of stenocardia, and in some of the cases the attack strongly suggests a severe form of true angina pectoris. It is a well-known fact that spasmodic fluctuations in the diameter of the blood-vessels occur with special frequency in arteriosclerotic subjects, as Wagenmann observed in the arteries of the eyeground.†

In other cases, however, anginoid conditions accompanied by vascular phenomena are probably of the same character as nervous attacks, which, as we know, are frequently characterized by a combination of cardiac and vascular symptoms (see section on Nervous Disturbances of the Heart). But it must be carefully borne in mind that the boundaries can never be sharply drawn. Severe forms of angina pectoris may develop in neurotic individuals, and it is not always possible to be absolutely certain that they are of a purely nervous character and, therefore, harmless.‡

* Nothnagel, "Archiv für klinische Medicin," vol. iii, p. 309.

† Wagenmann, "v. Graefe's Archiv," vol. xliv, p. 219.

‡ Compare the curious reports by Morel-Lavallée, "Revue de médecine," 1899, p. 753.

Theoretically, we must, therefore, say that the peculiar conditions within the heart which are the cause of angina pectoris may be started by sudden local anemia or by intoxication with nicotin and possibly some other poisons, but may also be due to nervous irritation localized in the brain or in peripheral portions of the pneumogastric nerve.

Diagnosis.—The diagnostic determination of the sensory heart anomalies will first attempt to arrange them according to the usual classification. While in a purely pathologic sense angina pectoris develops gradually from other symptoms, without a sharp line of separation, it is worth while, from the standpoint of diagnosis, to adhere to a definite scheme; for our ultimate purpose is to find out what process lies at the bottom of any one sensory symptom. First of all, let it be remembered that anginoid conditions are chiefly, although not by any means exclusively, associated with sclerosis of the coronary arteries. Severe attacks of stenocardia also develop chiefly on a basis of coronary sclerosis; in fact, the cardinal question to be answered in any case of angina pectoris is whether the attack is the expression of a coronary sclerosis, because therein lies its chief danger. In order to recognize this primary cause the observer must bear in mind not only what has been described as constituting the characteristic features of severe angina, but also all other possible indications of disease of the coronary arteries (see the section on Arteriosclerosis).

If coronary angina is unlikely, the other disturbances of the cardiac action which are capable of producing stenocardia must be thought of, and, finally, if the latter can also be excluded, tobacco-poisoning, diseases of the gastrointestinal canal, and the various forms of nervousness must be considered as possible causes of the angina. The diagnosis depends partly on the character of the stenocardiac phenomena, partly on the history, and finally on the results of a minute examination of the entire organism. I would advise that least importance be attached to the particular variety of angina present.

As a rule, a positive diagnosis is possible. It is sometimes difficult to decide in patients beyond the age of thirty-five or forty whether mild anginoid conditions are purely nervous in origin or indicate a beginning sclerosis of the coronary arteries. I am very much inclined to agree with Vierordt on this point, and advise the greatest caution in assuming that the angina is of a purely nervous form under such circumstances. The diagnosis may also present great difficulties in case a patient who is known to have arteriosclerosis becomes neurasthenic or hysteric. In such a case a conclusion can be arrived at only after long observation and with only a certain degree of probability. In every case the possible existence of arteriosclerosis must be borne in mind.

Treatment.—It is needless to say that the primary cause of the symptom must be treated as soon as possible, and to the full extent of our therapeutic powers. The rule, therefore, is: first make the correct diagnosis and then follow out the principles laid down in the individual sections. Vierordt's advice, always to think of a possible syphilitic cause for the arterial disease, should be emphasized. And I may add the urgent advice always to administer potassium iodid for some time whenever arteriosclerosis is present. The French clinicians, especially Huchard, speak very highly of the remedy. O. Vierordt* saw very good results from its use, and my personal experience has been that anginoid symp-

* Vierordt, "Congress für innere Medicin," 1897, p. 277.

toms in persons with arteriosclerosis are frequently very much improved by a systematic course of potassium iodid. The remainder of the treatment of arteriosclerosis with angina is reserved for the section on Coronary Sclerosis.

The indications afforded by tobacco-poisoning, gastro-intestinal affections, and general nervousness must, of course, receive due attention, and the patients must be made to avoid whatever they have themselves observed favors the occurrence of an attack.

This is not enough, however. The attack itself is so terrible that it absolutely demands direct treatment. But this is not possible in every case; for, aside from the attacks which end in sudden death before the physician has been able to do anything, many of the frequently recurring attacks are so brief that therapeutic interference is out of the question. But in the attacks of longer duration I believe the administration of some remedy to mitigate the pain and fear is, generally speaking, absolutely indispensable, if not in all cases, at least whenever simple nervousness can be excluded with absolute certainty from the etiology. Even in the purely nervous form of angina morphin cannot always be dispensed with; but I prefer to reserve the full discussion of this question for the section on the Treatment of Nervous Diseases of the Heart.

In all other cases, if seen before the attack is over, I advise injecting 0.01 gm. ($\frac{1}{6}$ gr.), or, if that proves insufficient, 0.02 gm. ($\frac{1}{3}$ gr.) of morphin under the skin of the breast, near the precordial region. In addition, a hot foot-bath at 45° to 50° C. (113° to 122° F.) may be ordered, or a large mustard poultice, 30 by 40 cm., applied to the back or thighs. The latter probably acts not only as a "derivative," but also as a stimulant to the heart action and, therefore, directly combats the cardiac weakness, as we are justified in concluding from Grossmann's important observations. The cardiac weakness must always be kept in mind; nor should treatment be deferred until it is well marked, for the possibility of its development at any time must be remembered. O. Vierordt's advice, always to have the sedative ready in one hand and the stimulant in the other, seems to me to hit the nail on the head. A rapid effect being called for, camphor and ether, on the one hand, and morphin, on the other, are the most useful drugs, because they can be given by subcutaneous or intramuscular injection. I am well aware that some eminent authorities deprecate the use of morphin up to a certain degree, but this caution applies only to large doses. The harmlessness of moderate doses of morphin, let us say 0.01 to 0.02 gm. ($\frac{1}{6}$ to $\frac{1}{3}$ gr.), seems to me to be well demonstrated, especially in heart patients, and I am firmly convinced that no physician can dispense with morphin in any protracted case of stenocardia.

From a study of the literature I have gained the impression that morphin is, as a matter of fact, given almost universally, and the warnings refer only to excessive doses. Hence we shall undoubtedly continue to use this wonderful remedy. I recommend it for the purpose of alleviating the frightful pain; although I may remind the reader that French investigators, as, for example, Huchard, in agreement with older physicians, attribute to morphin the property of relieving the strain on the heart. If for practical reasons morphin cannot be given hypodermically, either opium or morphin may be given internally.

Additional remedies for the purpose of stimulating the action of the heart are black coffee, strong wine, and cognac, because they are probably

oftener at hand than camphor and ether. Spirit [compound—Ed.] of ether is also a satisfactory remedy, especially for patients who go about and wish to carry a stimulant with them.

I wish to add a few words in regard to the use of certain substances which have been advised by prominent physicians, especially in England and France, for cases of stenocardia, both such as are due to coronary sclerosis and other forms. I refer chiefly to amyl nitrite, sodium nitrite, and nitroglycerin. One or two drops of a 1 per cent. watery solution is the dose of nitroglycerin; 0.1 to 0.3 gm. (2 to 5 gr.) of the sodium nitrite, dissolved in water; and two or three drops of amyl nitrite, by inhalation. As large quantities of amyl nitrite are not altogether free from danger, it is well to prescribe the required dose put up in glass capillary tubes or pearls. The glass is broken in the handkerchief and the contents inhaled.

There are, no doubt, some individuals in whom the attack of stenocardia can be mitigated, avoided, or suppressed by means of these preparations, and for that reason they should be *cautiously* tried in cases of frequently occurring attacks that have proved refractory to any other form of treatment. But it must be remembered that these preparations are not free from danger and that, in many cases, they absolutely increase the patient's distress. Special caution is necessary in cases of coronary sclerosis and when there is danger of heart failure.

These therapeutic measures will, I believe, be found to suffice. I wish to warn the physician explicitly against anything in the form of experimental therapeutics in this dangerous condition, as it might result in a great deal of harm. Thus, for example, the inhalation of chloroform is to be condemned, and when operations are necessary in anginoid patients, the advisability of performing them without chloroform must be seriously considered, because such patients bear anesthesia very badly.

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THE SENSATION OF ABNORMAL CARDIAC ACTION. PALPITATION.

A healthy individual is unconscious of the action of his heart so long as it remains within certain bounds, probably because the sensory nerves are fully accustomed to certain definite contractions of the organ. This, at least, is borne out by analogous conditions in other tissues and by the fact that when, as is exceptionally the case, the heart is overfilled and contracting vigorously and at short intervals, even most healthy individuals are conscious of its action. Under such circumstances the rhythm is appreciated and one experiences the sensation of palpitation, which will be discussed later.

Whether some pathologic alteration of the heart action is necessary to bring it within the scope of the patient's consciousness, and if so, how profound an alteration is required, are questions which it is as yet impossible for us to decide. Many people, perhaps the majority, are absolutely unaware of any change in the cardiac rhythm, whether it be retardation, acceleration, irregularity, or inequality. On the other hand, cessation of the heart-beat is felt as such by not a few persons, and the sensation is accompanied by a certain sense of oppression and anxiety. It might be suggested that this sensation is also due to the contraction of a heart that has become overfilled during a long pause; but this is hardly correct,

for the sensation is completely absent in other cases in which the ventricles are unquestionably overfilled, and, on the other hand, there are individuals in whom rapid contractions of the heart at short intervals and in large numbers produce a peculiar sensation which they describe as a "fluttering" of the heart.

So far as I can see, the only possible explanation is that these particular sensations are produced whenever the sensory nerves are irritated in some peculiar way by certain peculiar processes in the heart (probably special forms of contraction), the nature of which is as yet unknown.

The sensations due to the consciousness of the action of one's own heart may be combined in a variety of ways with sensations of pain, anxiety, and palpitation. As a matter of fact, every conceivable combination of sensations occurs.

The difficulties encountered in attempting to determine the nature of palpitation are exactly the same as in the case of stenocardia, that is to say, it is purely a question of subjective sensations; *palpitation is characterized solely by a feeling of increased cardiac action.* The sensation may occur alone or may be combined with other feelings, especially anxiety and oppression, also pain or the sensation of cessation of the heart-beat (intermittence). I must refer to what has been said in the preceding section, and shall confine myself to what applies only to simple palpitation.

Some people do not know the feeling of palpitation, and in such cases it may be difficult to find out from the patient's statements whether he has it or not. Such cases, however, are extremely rare. As a rule, the patient can give a very accurate description, and, besides, most healthy persons have already experienced the feeling in moments of violent bodily exertion.

The results of an objective examination, if the opportunity to observe the patient during an attack of palpitation is afforded, are quite variable.

As a rule, the action of the heart is altered. It may be increased in force, and the apex-beat may be high and heaving. In other cases the action is more or less accelerated or retarded; not infrequently it is somewhat irregular. Any one of these changes in the rhythm of the heart may be the only accompaniment or cause of the palpitation. On the other hand, a great many combinations may occur. The pulse may be large and full or small and soft, depending chiefly on the frequency of the cardiac contractions, because the latter determine the time of diastole and, therefore, the degree of distention of the ventricles. The second sounds at the base are often strikingly accentuated. The first sound also is not infrequently stronger than normal, and sometimes has even a ringing character.

These are not infrequent findings in cases of palpitation. Under such circumstances, when the cardiac contractions are vigorous, and especially when the cavities are well filled, a sense of palpitation is experienced by many perfectly healthy individuals, and patients, especially those with valvular lesions, are very apt to have the sensation under such circumstances. But I do not here refer to that class. In the same way patients with dilatation and hypertrophy of the ventricles, whatever may be the cause, suffer from the same symptom, especially when the heart is trying to respond to an increased demand. Some individuals develop the symptom more easily than others. All of which goes to show that something special is required to produce this form of palpitation. It occurs not only when the heart is contracting vigorously; for the conditions for its occurrences are vigorous contraction of the heart at the limit of its functional capacity. At this point the ventricles begin to dilate, and we may,

therefore, say that this form of palpitation is produced by the tumultuous contraction of a dilated heart, and that the phenomenon is a danger-signal to the individual. For, if the heart should continue its exertions, there would be danger of so-called overdistention (see p. 479). We may, therefore, assume that palpitation is the expression of sensory irritation due to tension in the walls of the ventricles.

It seems to me that this view is in accord with clinical experience. It is by no means true that every case of cardiac dilatation is associated with palpitation, and we also know that dilatation is by no means always associated with an increase in the tension of the walls; and, finally, the effect of habituation on the sensory nerves of the heart must not be underestimated. In the healthy subject palpitation occurs during excessive muscular exertion, when the functional capacity of the organ is beginning to be exhausted; and as this point is reached much earlier in persons with valvular lesions or disease of the myocardium, and the time of its occurrence varies in different individuals, we can readily understand why the symptom is so variable in its appearance. Sometimes a dilated heart contracts too vigorously even during rest, and in such a case the patient is never without the sense of palpitation.

This form of palpitation is especially frequent in the subject of so-called plethoric obesity, in whom it is often induced by the slightest muscular movement and is probably due to the rapid entrance of large quantities of blood into the right auricle in a heart whose function is below par. The symptom is also observed not infrequently in anemic individuals.

But we do not, by any means, always find a dilated heart when we examine patients who are suffering from palpitation at the time; and even when dilatation is present, the distention of the ventricle, or rather the abnormal tension of the wall, is not the cause of the palpitation. Not infrequently only one of the above-mentioned alterations in the action of the heart is present, namely, an increase in the force of the contractions, either without disturbance of the rhythm or with retardation or acceleration of the rate. Acceleration alone may produce the feeling of palpitation, and there is no doubt that a more careful analysis of the heart action in such conditions would yield some additional information. In these cases the heart is, therefore, of normal size or possibly enlarged; at any rate—and this is the important point—dilatation is not specially concerned in the pathogenesis of this form of palpitation.

Palpitation of this character occurs in heart patients, especially after acute inflammations, and much more frequently in individuals suffering from a great variety of nervous disturbances, chief among which are disturbances of the circulation. This condition also is illustrated in health by the occurrence of palpitation after psychic emotion, such as shame, grief, or joy. It is also observed after the ingestion of heart poisons, tobacco, tea, coffee, wine, and possibly also after reflex excitation, especially such as involves the sensory branches of the pneumogastrics, that is to say, in diseases of the abdomen and of the lungs. The palpitation which occurs in Basedow's disease probably also belongs to this category.

While in cases of dilatation with vigorous systolic contractions the irritation of the sensory nerve elements is readily explained by the tension of the heart-wall, the cause of this irritation in the second variety of palpitation is much less clear.

It is probable that in this variety also the sensory fibers are irritated

by some abnormality of the systole. It is possible that any disturbance of the cardiac action may result in palpitation; but etiologically the most important, undoubtedly, is increased vigor of the contractions, not due to an increased demand on the organ, but to a morbid irritation of the heart itself. The violent systolic contractions which accompany the palpitation can often be recognized at once, as, for example, in coffee-poisoning, in arteriosclerosis, and after psychic excitement in nervous individuals.

In some cases of palpitation in nervous individuals F. Müller* observed an increase in the rapidity of the systole over the normal. This is an interesting observation and needs to be further investigated. It seems to me to explain a special irritation of sensory nerves, although this question also requires further elucidation because there may possibly be an interesting relation between the duration of systole and stimulation of the accelerators; for we know, from investigations pursued in Ludwig's Institute, that such irritation is always accompanied by shortening of the systole. This fact is probably especially important in paroxysmal tachycardia, in which the acceleration of the heart is extreme.

At any rate, our next task should be to make a minute examination of the differences in the cardiac cycle in the etiologically different forms of palpitation belonging to this group.

But in many cases another factor undoubtedly comes into play: the *abnormal irritability of sensory nerves,* possibly of the chest-walls, but much more probably of the heart itself. We know absolutely nothing about this; but without some such hypothesis we can hardly explain the condition even in the above-named cases, and much less in the third group of patients. For there are undoubtedly patients with palpitation in whom no sign whatever of any change in the action of the heart is demonstrable, that is to say, heart action, apex-beat, sounds, pulse, everything is absolutely normal, but the patient, nevertheless, says that he feels palpitation. At least I have seen this frequently in nervous and anemic girls. What the frequency of the condition in general is has not, so far as I know, been investigated. Unfortunately for the solution of such questions the palpitations are, as a rule, only paroxysmal, and it is by no means always possible to examine the patient during the attack.

Taking it altogether, our knowledge of palpitation is deplorably slight as soon as we attempt to go beyond the mere enumeration of symptoms in association with which it occurs, or attempt to make any progress toward an understanding of the conditions involved. The above-mentioned etiologic factors of palpitation afford but scanty data, and it is as yet impossible to formulate a definite and satisfactory classification of the various clinical forms of palpitation, partly on account of the scarcity of observations and partly because the methods of determining the action of the heart are still inadequate. It is possible, and indeed probable, that many of the clinical forms are due to a multiplicity of causes. For example, I am inclined to believe that in sclerosis of the coronary arteries and in anemia the feeling of palpitation may be due to a great variety of causes.

At all events, the problem is extremely complicated, not only on account of the confused medley of sensory and motor phenomena, but also because the latter emanate from the heart muscle, the heart ganglia, and the central nervous system. The latter again affects the heart in

* F. Müller, "Berliner klinische Wochenschrift," 1895, No. 34.

a variety of ways through a number of different nerves, which explains the extensive complications that interfere with the study of the movements of the heart, complications which have already been referred to.

Palpitation is, therefore, purely a symptom, and what little we know of its diagnosis has already been stated. The important point is never to rest until it has been positively determined what morbid state the phenomenon is dependent upon. Before this is done, diagnosis, prognosis, and definite treatment are impossible.

Palpitation, as we have seen, is not infrequently observed in hearts which are either dilated or hypertrophied, or both. The question whether palpitation as such may lead to dilatation of the chambers of the heart or to thickening of their muscular walls has a great theoretic interest, for reasons explained on p. 490. The increased amount of work that is done in some cases of palpitation is only temporary, and if the entire duration is compared with the time occupied by rest, the latter will be found to exceed the former in length, at least in the majority of cases. The question is, does hypertrophy develop? It is a question of practical importance, for in the individual case it may be necessary to determine whether enlargement of the heart, if present with the palpitation, is solely due to the symptom and, therefore, likely to disappear as improvement takes place, or whether the feeling of palpitation is merely a symptom of the heart disease which is indicated by the enlargement. Passive dilatation (from stasis)—which is the only form that would be considered in such a case—cannot, in my opinion, be due to palpitation as such; and if the palpitation is not dependent upon the passive dilatation, it must be assumed that both conditions are due to a third factor, which has produced cardiac insufficiency.

Whether palpitation alone can become the cause of hypertrophy is a very difficult question to decide with absolute certainty, because positive proof can be obtained only by means of an autopsy, and, for obvious reasons, the opportunity of postmortem examination is rarely afforded in simple cases of palpitation not dependent on some actual disease of the heart. Hypertrophy of the ventricles unquestionably occurs in Basedow's disease and cannot well be attributed to anything but the increase in the frequency and strength of the cardiac action. But, so far as I am aware, no accurate observations have been made to determine how often in these cases with secondary hypertrophy the alteration of the heart action is permanent instead of merely temporary. Potain reports having seen hypertrophy of the heart after neuritis of the left brachial plexus. I myself have seen, in cases of onanism, phenomena which seemed to justify the assumption of cardiac hypertrophy. It seems to me, therefore, that in many cases severe palpitation may by itself be able to produce thickening of the cardiac musculature, although the fact has never been definitely proved.

It is evident that there can be no question of **prognosis** so far as the palpitation as such is concerned, as it depends altogether on the primary disease. If the latter persists and the question is asked what prospects are there that the palpitation, at least, will cease, the only answer that can be given is: the same prospects as those with which we combat the individual attack and attempt to remove its causes if the primary trouble persists.

The **treatment** of palpitation, therefore, consists principally in combating the primary disease on which it depends and the causes that lead

to an individual attack. For the first indication we must refer the reader to the special sections, and as for the second, I have only a few remarks to offer. It is true that in individual cases the direct cause of the palpitation is known. If some psychic emotion, muscular exercise, or the ingestion of some special substances produces palpitation, these causes must be avoided as much as possible until the patient's heart and general constitution have become strong enough to enable him to withstand these influences without any injury.

The attack itself is in most cases best combated by complete bodily and mental rest; quite often the horizontal position may be necessary. But there may be exceptions to this rule. Many cases of palpitation due to some disturbance of the digestive or nervous system are improved by exercise. If the heart action is increased in force, repeated cold compresses to the precordia often do good; while retardation of the heart, provided the action is vigorous, is not a contraindication to their use. The various methods of "derivation" are also to be commended, except that I would not employ any procedure that causes vasomotor or cardiac excitement. On the other hand, the latter are indicated whenever cardiac weakness lies at the bottom of the trouble. The remedies to be employed in such cases will be discussed in a succeeding section.

[All the irregularities of heart action need investigation with reference to the possibility of detecting an abnormality in one of the cardinal functions of heart muscle—automatic stimulus production, conductivity, contractility, and irritability, or by the exclusion of these, to trace the irregularity to some cause outside the heart. A beginning has been made, by the study of venous and arterial pulse-tracings, but much more must be done both by physiologists and by clinicians. Besides the works of Mackenzie and Wenckebach, see Cushny, "The Interpretation of Pulse-tracings," "Journal of Experimental Medicine," vol. iv. Cushny and Edmunds, "Paroxysmal Irregularity of the Heart and Auricular Fibrillation," "American Journal of the Medical Sciences," January, 1907. Hirschfelder, Arthur D., "Observations on a Case of Palpitation of the Heart," "Johns Hopkins Hospital Bulletin," vol. xvii, No. 186, p. 299, etc.—Ed.]

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GENERAL REMARKS ON THE DIAGNOSIS AND ANALYSIS OF DISEASES OF THE HEART.

In a case of heart disease the diagnosis is complete only if it enables us to form a sufficiently accurate idea of the functional capacity and structural condition of the heart to afford positive indications for the kind of treatment to be adopted and a definite idea of the probable course of the disease. But even in the most favorable case we are far, indeed, from being able to attain that object; only an approximately correct judgment, arrived at indirectly, is possible.

For the present the physician should not attempt to do more than

follow the classification given below. In classifying a given case he must take account of the symptoms the patient presents and which are obtained either from the history or the examination of the heart, the blood-vessels, and the circulation in the individual organs. I fully realize the disadvantages and inadequacy of this classification. As has been stated in the introduction, it is the result of a compromise between different kinds and possibilities of classification. Besides, as has also been stated, it has only a certain very limited value for the present state of our knowledge. But for the present we cannot do without it, and it has its advantages because the mere classification of a condition based on clinical experience affords certain indications for both diagnosis and treatment.

Only let the classification of an individual case be effected with due deliberation. Even a few days' observation often suffices to bring about a material change in one's original opinion. A special caution is necessary when the cardiac insufficiency is accompanied by alterations in the blood distribution. Valvular lesions due to endocarditis must, under all circumstances and in every individual case, be taken into account. Indeed, they are omitted here only because the distribution of subjects in this work renders the omission unavoidable. It is not too much to say that the conditions with which we are dealing do not enter into the question of diagnosis until after a valvular lesion and endocarditis have been excluded. Special difficulties are encountered in the differential diagnosis between endocarditic mitral insufficiency and symptomatic mitral insufficiency in chronic diseases of the heart muscle (compare p. 454 and the section on Chronic Myocarditis).

In every attempt to classify an individual case it must be remembered that any case of heart disease especially may have a multiple cause. Hence it is often difficult to decide what particular group has the greatest claim to an individual case. The various symptoms due to muscular exertion, obesity, alcoholism, as well as those due to myocarditis and coronary sclerosis, have a great many points in common, and the possibility of combinations must always be borne in mind. In such cases we must be on our guard against pedantry and resist the temptation to suppress certain facts for the sake of preserving the unity of a scheme, or under the mistaken impression that one is clearing up a doubtful point. Murder will out!

After the condition in a given case has been classified, the stage of the disease and the functional capacity of the organ remain to be determined. This determination is based mainly on the results of the objective examination: the degree of passive dilatation, the action of the heart, and the symptoms of disturbed blood distribution are the most important. After that a few special points in the history must receive consideration, and the methods of examination mentioned on page 456 resorted to.

The first thing to find out is whether the functional disturbances of the heart are merely due to some unfortunate reaction—to more or less violent and harmful influences, or to structural anomalies. If the former is the case and the physician has it in his power to protect the patient against such injurious influences, the prospect is brightest. The methods of determining the condition of the heart muscle and the difficulty, not to say impossibility, of arriving at a definite conclusion, have already been presented in the individual sections.

The significance of structural anomalies of the muscle depends on its cause, the mode of its development, and the patient's circumstances.

This subject has also been fully treated in the various sections; but here, again, the experience gained at the bedside must be our sole guide. Finally, the psychic personality of the patient, his bodily condition as a whole, his occupation, and his social condition enter into the problem in a given case, and it is often an exceedingly difficult and complicated matter to arrive at a conclusion. The entire course of the disease may ultimately depend on some apparently isolated conditions. This is as far as we can go in our general remarks; for in the final analysis all depends on a full knowledge of the individual case and of the patient's environment.

GENERAL PRINCIPLES TO BE OBSERVED IN THE TREATMENT OF HEART PATIENTS.

In heart disease, as in other pathologic conditions, it is most unwise to lay down dogmatic rules in regard to treatment, and it is, therefore, exceedingly difficult to discuss general principles. We may, however, venture to describe in a general way to what ends our treatment should be directed.

One of the first questions to be determined before treatment can be begun is *whether circulatory disturbances with altered blood distribution are present or not.*

If they are present, they must first of all be corrected. The means at our disposal are either rest and the drugs of the digitalis group, or exercising the heart by means of baths and bodily exercise, if necessary assisted by modifying the state of nutrition and the percentage of water in the body. In discussing the various methods it was shown how the indications for the individual case are obtained.

If circulatory disturbances in the sense just described are not present, our object must be to prevent or postpone their occurrence. This is accomplished chiefly by regulating the patient's entire mode of life in the widest sense of the term, and much can usually be accomplished by giving definite instructions in this respect.

Of course, these instructions also play a very important part when circulatory disturbances have already made their appearance.

All therapeutic directions must be adapted in every respect to the patient's individual circumstances, and must take in every detail of his mode of life. A good physician, after carefully diagnosing the disease in a given case, adopts a line of action appropriate to the patient's circumstances and personality. To do so the entire body and all its various functions must, of course, be taken into consideration. Heart disease, as has been shown, involves a great many different organs. In reality the number of possible and actually occurring cases in which cardiac disturbances are associated with disturbances in other organs is even greater than has been indicated above because only such morbid processes as are directly related to heart disease were described. Many more occur in disease, as the number of accidental and, therefore, possible combinations is unlimited. In other words, in order to treat one system in the body the physician must know and have thoroughly mastered the principles underlying the treatment of all the others, and must constantly apply these principles. As any complication in any one of the organs may profoundly influence the treatment of the heart disturbance itself,

the physician, in order to meet any emergency, must have the most varied knowledge at his disposal.

Such, I have no doubt, are the principles that have always determined the conduct of every good physician in every period of history. We are now told that therapeutics as a science was really invented during the past decad, and proudly refer to ours as the therapeutic era. It is quite true that the question of treatment was formerly somewhat neglected, and that too little was said about its details in medical lectures, but at the same time we must be very careful not to overestimate our therapeutic achievements and, above all, not to seek salvation in reiterated description of therapeutic directions in their most minute details. As I have already remarked, the physician must give his patients such detailed directions in every case. He deduces them from his knowledge of general principles and adapts them to the patient's individual circumstances. Precisely the same course should be pursued in clinical instruction; but such therapeutic directions can never be profitably discussed in print. A text-book, in my opinion, should contain merely the leading principles and the proper methods of applying them. Any attempt to give special prescriptions for individual cases and occurrences at the expense of a thorough discussion of principles and methods is sure to render the book commonplace. The author and his personal peculiarities are apt to rise into undue prominence; the dazzling diet-lists appear diagrammatic and exhibit a monotonous similarity in the most various diseases, or they are adapted only to a certain class of patients—very often only to wealthy people.

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REST AND EXERCISE.

The first and most important, and by far the most difficult, question is, how shall we regulate a heart patient's complete mode of life? Is he to stay in bed or may he sit up? Is he to rest a great deal, or is exercise desirable? Shall he be allowed to go on with his work, or does his condition demand that he should give it up and rest? In deciding these questions ordinary human considerations are frequently of vastly more importance than the medical aspects of the case. In my opinion it is the physician's duty not to give his instructions according to rigid principles, but rather to enter as far as possible into the patient's personal views.

As a rule, the physician will have to adopt the principle to preserve the patient's life as long as possible under all circumstances, even if the patient has to make everything else subservient to the necessity of giving his body entire rest. But on this fundamental point the patient may entertain altogether different views, and, in my opinion, he has a right to expect his physician to sympathize with him in these views. So long as men are not all weaklings, some will sacrifice health to duty and subordinate comfort to the ambition to carry out some great thought, even if it be great only in their own eyes. Individuals vary greatly in the value they place on life, and those who prefer to live a shorter time and keep at work, even if they must be ill, rather than keep their sick bodies alive in idleness, are not necessarily the least desirable members of society. It may be objected that all of this is no concern of the doctor's; that it is his business to preserve life, and a patient who strenuously resists his determined efforts to attain that end must do so on his own responsibility. Such is not my opinion. I believe that in some cases a physician ought to make it his chief object to keep up his patient's activity and not to preserve his life.

Under what circumstances must a heart patient be put to bed? As soon as the state of his circulation requires the most absolute rest attainable.

The amount of exercise performed by a person in bed is very small, and the movements are never active. Heat regulation and nutrition are relatively much diminished, and the demand on the various functions of the circulation correspondingly lessened. In bed the patient may assume whatever position he finds most comfortable and no special instructions on that point are required, at least for the present. It is quite true that sometimes, in the most severe cases of heart disease, rest in bed, which in such cases is so desirable, is impracticable. Sometimes the dyspnea is so great that the poor sufferers cannot even maintain a semirecumbent position in the most comfortable bed. In such cases they must be warmly dressed and wrapped up and provided with a well-padded arm-chair.

If the patient can stay in bed, it must be specially prepared, particularly when there is dyspnea. Heart patients cannot lie flat; the return flow of the venous blood from the cranium probably needs the assistance of gravity, and the pressure of the trunk must not bear on the posterior portions of the ribs. The patients maintain a semirecumbent position, or even a sitting posture, in bed, and this again calls for special attention to that part of the sacrum on which the weight of the body is supported. If the position is maintained for some time, the only way to prevent bed-sores is by means of a water-cushion. Other portions of the body that are in constant contact with the bed may also develop pressure necrosis on account of the edema, the retarded circulation, and the resulting interference with the nutrition of the tissues. The preparation of the bed is, therefore, a very important matter. If a water-cushion is not to be had, cotton, soft feathers, or pneumatic rings must be used; by the exercise of skill and experience much can be accomplished with these contrivances, although they cannot rival the water-cushion.

If this were an up-to-date work, a complete description of the shape and various kinds of water-cushions, pillows, and arm-chairs, with a few illustrations, would now be in order. (Nursing, I have recently learned, has now become a "science.") At the risk of being called old-fashioned I shall omit such detailed directions. The older physicians were not unsuccessful in looking after the comfort of their patients, and we of this generation also learned how it is to be done from our clinical teachers. I gratefully remember E. Wagner and H. Curschmann in this connection. We also make an effort to instruct our assistants and students in the technic of nursing. This is done as a matter of course, and I will not admit that our clinicians have been in the habit of grossly neglecting these things until quite recently.

Absolute rest in bed is imperative in cases of acute or chronic and severe cardiac insufficiency, at least during the beginning of an attack, and until it has been subjected to appropriate treatment. In recent cases especially a very marked improvement not infrequently takes place as soon as the patient begins to rest and the demands on the heart incident to everyday life are removed: the contractions of the heart become more vigorous and more regular, the blood-pressure rises, the quantity of urine increases, and the edema disappears. In many milder cases of cardiac insufficiency complete recovery can be obtained merely by putting the patient to bed, a fact which is as interesting theoretically as it is important practically.

On the other hand, it is much more difficult to decide what degree of rest is proper when a certain condition of cardiac insufficiency with abnormal blood distribution has become permanent or, so to speak, the rule. In such cases rest in bed usually does no good and methods of treatment which stimulate and thereby exercise the heart must be resorted to instead. Quite often these measures also fail, and in deciding the question whether rest is indicated or exercise may be allowed, only the patient's comfort and wishes need be considered.

Rest in bed is not always absolutely necessary when bodily rest is indicated for a time; in some cases the patient may be allowed to recline on a couch. But whenever it is desired to get the full benefit of the advantages that can be obtained by complete rest, the old rule to insist on rest in bed should be strictly observed.

It is hardly necessary to mention that rest in bed or on a couch does not mean that the patient is to be kept confined in an ill-ventilated room. General thera-

peutic principles will induce every physician to insist on ventilation of the room and on the patient being wheeled into the garden or on a porch when the weather permits.

The question whether complete rest in bed is to be ordered must be decided by the physician in the case of patients with a wrecked nervous system and greatly impaired nutrition. The indications for rest in bed are the same in these cases as in nervous patients generally if the patient is to be protected as much as possible against excitement and restlessness, if metabolism is to be restricted, and the nutrition improved; in other words, the indications are the same as those for the so-called "rest treatment" in nervous patients. No one will deny that the "rest treatment" has been greatly overdone recently; but for all that the method is extremely useful in many of the above-described cases, especially for the nervous heart symptoms.

It is often as important as it is difficult, in a case in which rest in bed has been indicated and actually carried out, to know when the time has come for allowing the patient to get up. It is practically impossible to give any general rule except not to let the patient get up too soon. He should be treated approximately like a convalescent from a severe attack of typhoid fever. The question is one that must be decided by medical experience.

At first the patient is allowed to be up only for a short time, and as convalescence progresses, the time is gradually lengthened. In this way he is gradually allowed to return to his customary mode of life and later resume his work.

If rest in bed is not necessary, the question whether the patient *may continue at work must be decided.* Some of the general principles which decide this question have already been mentioned. It depends, in the first place, on the character of the patient's work. It is often a question whether the individual's occupation need only be interrupted temporarily or whether an entire and permanent change of occupation is necessary. The latter, of course, implies a most important step, and a conscientious physician will not make such a proposal until he has carefully thought out all its consequences; but by offering him a choice of occupation he may save his patient from great misfortune. For there are cases in which one can almost positively predict speedy dissolution unless the former occupation is given up. Such patients must be advised to make a change if possible—but only if it is possible. In any case the physician must first carefully consider whether what he is offering is better than what the patient has to give up; for in these days, when all the professions are overcrowded, the attempt to change one's occupation often meets with unexpected difficulties.

Any occupation that throws an excessive demand on the heart is unsuitable for a heart patient. This, therefore, includes all occupations which necessarily imply violent and frequently repeated muscular movements. Again, in some callings a man is compelled, by our present customs, to drink large quantities of wine and beer; examples of this are found among brewers, beer-wagon drivers, waiters, and commercial travelers. And, finally, if even for a quiet man there are moments of great emotion, these are constant sources of danger to the heart.

In regard to all these matters the physician must carefully inquire into the work which the patient's occupation requires him to do. Everybody knows that a blacksmith's work is heavy and that a waiter's duties are about as unsuitable as possible

for a man with an abnormal heart muscle. In other callings, however, a correct judgment can be arrived at only by minute interrogation, and here again the patient's personality must be taken into account. While one man may find the duties of a certain office altogether too exciting, a more cold-blooded individual may be able to perform them with perfect equanimity.

If there is no practical obstacle to temporary interruption of the patient's work, I strongly recommend such a procedure whenever there is reasonable hope of achieving a real improvement or even cure by a certain period of rest. Not infrequently, however, it is wiser to allow the patient to go on with his work, especially when the heart cannot well get worse than it is.

Sometimes the solution of the question is taken out of the physician's hand by a higher power; the patients are simply unable to keep on with their work.

If a heart patient continues his work, he should "save himself," as the saying is, as much as possible; that is, he should do what he has to do with a minimum expenditure of psychic and bodily exertion—just enough to insure good work. Ambition, competition, and passion in the course of his work must be buried forever. But here, again, it is easy enough for the physician to give instructions, but very different for the patient to carry them out. Take away a man's passion for his calling: what is he likely to accomplish? As has already been stated, there are many heart patients who prefer to work rather than prolong their days in idleness. In such a case the physician is often at his wit's end. Much, however, may be accomplished by judicious admonition, and the patient's efforts to preserve his mental balance will be greatly aided by a strong moral and religious sense. In my opinion a physician in such cases should never alienate his patient's confidence by excessively rigorous instructions. He will accomplish most good by trying to understand and guide his inner life.

To what extent muscular exertion may be permitted has already been discussed in connection with the question of rest in bed, especially the differences in this respect between patients with recent cardiac insufficiency and those in whom the condition is of long standing.

Patients without any anomalies of blood distribution, but in whom a disease of the heart, nevertheless, reveals itself to the physician by the presence of symptoms under certain circumstances and by objective signs, must not by any means be ordered to abstain from exercise. This was expressly pointed out by Stokes.

Oertel unquestionably deserves credit for the fact that under his inspiration we are gradually losing our traditional dread of exercise in every kind of heart disease. Of course, the patient must always keep within the limit of his functional capacity (determined by the history and special tests); but within that limit he must take exercise in order to guard against the development of cardiac insufficiency from inaction. Regular constitutionals, if necessary, with graded climbing exercises, riding, and gymnastics of various kinds are indicated—but under supervision or according to the doctor's orders. It is impossible to give general rules because it is only by an accurate knowledge of the individual that the exercise can be adapted to his capacity.

A great many sensible persons may be allowed to pursue their customary mode of life provided it is not such as to throw a severe strain on the heart, a fact to be determined by the history. To other patients, who

cannot be trusted, the physician must give the most minute instructions: how long and how often they may go out, how high they may climb, and how long the exercises are to last. In such cases it becomes the physician's duty to be more or less dogmatic. In deciding whether any unusual and altogether unnecessary exercise, such as dancing, for example, may be permitted, the physician must be guided by the patient's age and personality and by what he has been able to make out in regard to the functional capacity of the patient's heart.

CARE OF THE SKIN. BATHS.

A *lukewarm* bath—32° to 35° C. (90° to 95° F.)—once or twice a week is as healthful for a heart patient as it is for every healthy man and is well borne in most cases. Cleanliness is a very important matter, especially in the severe cases, because the overstretched and edematous skin is very apt to become the seat of inflammatory processes which not infrequently assume serious proportions. Unfortunately, the dyspnea and cardiac weakness in such patients may be so severe that the movement necessarily associated with a bath, even when every precaution is taken to get the patient in and out of the bath without any exertion on his part and not to allow him to dry himself, become dangerous, or the bath itself distresses the patient. In such a case he must be sponged with lukewarm water—28° to 30° C. (82° to 86° F.)—or with dilute alcohol. Every part of the body that comes in contact with the bed must be protected against decubitus by every known method. If a bed-sore develops, it must be carefully treated—a matter of no small difficulty, particularly when there is edema.

There is no objection to a *cold rub* in the morning. Whenever there is no cardiac weakness with altered blood distribution, the exercise incident to a cold rub may be very beneficial to the heart, since it is associated with vasomotor stimulation and possibly also with stimulation of the heart itself. For this reason the procedure should be employed with caution, and the temperature of the water must be regulated according to the indications present in the heart; thus, moderately warm temperatures will be preferred for older people. In the case of individuals who have been used to taking a cold rub all their lives there is, of course, less need for anxiety, because in such cases the reflexes are much less active.

Whether *cold river-* or *sea-baths* may be allowed must be determined by the condition of the heart and largely also by the patient's strength and habits. There are some perfectly healthy persons even who cannot tolerate cold bathing. As a rule, cold baths may be permitted in the same cases in which exercise of the heart is possible and appears to be indicated.

I have no special instructions to give as regards clothing. Its selection is based on principles which everybody is in the habit of observing in every-day life. Heart patients with severe circulatory disturbances are very apt to be cold, especially to have cold feet, and this must, therefore, be remembered. They must be kept warm, but the clothing must not be too heavy.

DIET.

From the standpoint of diet, heart patients may be divided into two classes: those who go about or even attend to their work, and differ from

healthy individuals only by the fact that in some, or perhaps in a great many, respects the heart is unable to respond to the demands upon it; and the second class, consisting of patients suffering from profound circulatory disturbances.

As regards the first class, all that is required is to advise them to live sensibly; but as opinions differ in regard to the meaning of the word "sensible," the physician must go into details and in a given case must not hesitate to give his own opinion.

Even healthy persons vary a good deal in regard to the number of times they eat during the day and the quantity they eat at each meal. Some have the most peculiar habits, and in spite of them retain their bodily and mental efficiency in the highest degree. Heart patients must be absolutely forbidden to indulge in any such peculiarities.

Every meal makes a certain demand on the circulation, and when the meal is heavy, the demand is correspondingly greater. It is less injurious to a heart weakened by disease to exert itself a little at short intervals than to overexert itself at long intervals. Then there is another thing. After a copious meal the stomach and intestines enlarge and the diaphragm rises: oppression and a sense of pressure or constriction are apt to be the result, and it is possible that the overfilling of the intestinal canal may affect the heart in some special way through reflexes and that the disposal of numerous articles of food may also affect it for chemic reasons through the medium of the products of digestion. In the case of some patients this undoubtedly takes place, and when it does, must receive due consideration.

There is no reason why a heart patient's diet should be different from that of a healthy person. I mean that it is best in an individual case to allow the patient to eat what he has been accustomed to, again provided that he has not been accustomed to indulge in the most obvious eccentricities or absolute irregularities in diet. We know that, for a number of reasons, the amount of food required by different individuals varies greatly. It does not seem to me that our theoretic views are sufficiently definite to justify our subjecting persons whose habits of life have been widely different to the identical routine, and just because they are sick, and in the absence of any urgent reason, to prescribe an entirely different diet from what they have been accustomed.*

One thing, however, should be remembered: whatever tends to overfill the intestinal tract is injurious for the reasons given above. As a great many different articles of food may produce this effect in different individuals, and as patients vary greatly in the matter of personal habits, it is necessary to discuss the menu with every individual patient. I always discuss minutely with my patients what and how much they shall eat.

I shall not give any diet-lists because, in my estimation, they have only a very limited value in a text-book, except for such diseases as diabetes mellitus or obesity, in which the indications are very precise, for they apply, as a rule, only to one class of individuals or to the people living in one section of the country. For instance, what I order my patients in the Policlinic to eat is suitable only for Thuringian or Hessian peasants, and the diet-lists would be of little use to a physician in upper or lower Germany. Apparently precise instructions of this kind may do harm because the man who uses them is in constant danger of falling into routine methods, a tendency which is only too common among physicians in the matter of dietetics.

*Compare F. A. Hoffmann, "Allgemeine Therapie," Capitel: "Herz."

Certain special instructions are necessary in regard to a few articles of diet. Heart patients are quite as intolerant of large quantities of *wine*, *beer*, and *brandy*, or rather much more so, than healthy individuals, and it is not necessary to enter at length into the reasons for this. But just as a man of good sense and moderation need not give up wine and beer altogether merely because by doing so he may help to cure drunkards, in the same way heart patients, with whom we are here concerned, need not abstain altogether except when the physician finds that he must forbid the use of spirituous liquors altogether in order to prevent intemperance. I should not hesitate to allow one or two wineglasses of a thin white or red wine and a half liter of light beer in the evening. That is as much as sensible people in good health are in the habit of drinking. The "extra drinks" which the latter may allow themselves cannot be permitted in the case of heart patients of the type we are now discussing, nor can they be allowed to take brandy.

It is probably better to forbid the use of *coffee* altogether, at least in the case of patients who will not drink it weak. That strong coffee, when taken habitually, is injurious to heart patients I have often observed. As a rule, I would permit small quantities of weak coffee, but if that does not satisfy the patient and he demands a good beverage, it must be interdicted altogether. Malt coffee and "Kneipkaffee" ["postum cereal" and similar preparations—Ed.] have but feeble attractions for most people. Cocoa may usually be allowed, as may also carefully and properly prepared tea, that is, a half tablespoonful of good tea-leaves to two cups, allowing it to draw for only a few minutes, so that the beverage is light straw color, about like the color of Pilsner beer.

The *tobacco* habit in any form ought to be given up altogether by heart patients. At least, all the most experienced physicians are absolutely agreed that the smoking of so-called imported Havana cigars ought to be interdicted altogether. Cigarettes are harmful because the smoker, as a rule, smokes too many of them. If the patient is absolutely dependent on tobacco he would better smoke mild cigars, but at most two or three a day, and no extra smokes.

Chewing and probably also snuff-taking should be forbidden altogether.

The total quantity of the food must be strictly controlled; for as heart patients, as a rule, do not take as much, or as active, exercise as persons in good health, there is constant danger, if the food is too abundant, of their becoming fat, and obesity is anything but desirable, because it increases the demand on the heart during every kind of bodily movement. The patients should, therefore, be allowed to eat as much as they require, and especially should not be allowed to want for albumin, but, as a rule, they should not be put on fat.

Even the older physicians always preached moderation to heart patients. At present, when full feeding and gavage are so much in vogue and are believed to cure almost anything, such a theory almost causes surprise, and yet the reasons are clear enough, since most heart patients, owing to the fact that they take very little exercise, need considerably less nourishment than persons who are working. It is, therefore, well to calculate the total quantity of food for a heart patient according to the principles of metabolism and heat-production.

Some differences must be made in cases of *profound cardiac insufficiency*. A certain amount of individualization is necessary in these cases. There are some hopeless patients whose malady resists every attempt to bring about a real improvement; in such cases it seems to me reasonable to

forbid only articles of diet which are really harmful and to refrain from adding to the poor sufferer's discomfort by unnecessary restrictions.

The other class includes those who are still capable of improvement, in whom everything calculated to obstruct the progress toward recovery must be strictly avoided. In such cases I would not allow beer, brandy, or coffee as beverages, but only as medicines on prescription; the indications and mode of administration will be discussed later.

In selecting a diet the first consideration, aside from the patient's own inclinations, is that the quantity be just sufficient and that the food be of the proper composition and greater practicable concentration, as in that way overloading of the stomach is guarded against as much as possible.

Large quantities of food are neither necessary nor desirable; the patients do not need much because they are resting most of the time. It is true that the increased amount of work of the respiratory muscles in dyspneic individuals makes a certain extra demand on the nutrition, but I am quite unable to state with any accuracy how much this increase amounts to. The patient should not put on fat, and in order to relieve the circulation, it is even desirable that the food be relatively scanty.

As to the kinds of food that may be allowed, the choice must be based on the same principles as those which obtain in the feeding of very sick patients generally.

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SPECIAL DIETETIC TREATMENT.

It follows, from what has been said above, that, for our purposes, but little need be said under this head. Of course there may be individual cases in which heart patients need to have their nutrition improved, and in such cases the desired object will be attained by observing the well-known principles of dietetics. It is not necessary to go into detail because there are no indications peculiar to heart patients.

Much more frequently it becomes our duty, in the case of heart patients, to bring about a reduction in the amount of fat within the body; this point is discussed in the section on the Cardiac Symptoms in Obesity. But the indication for reducing fat is not, by any means, present only when the obesity is the cause of circulatory disturbances. The excessive accumulation of fat may be equally troublesome and disturbing in altogether different kinds of heart disease, and in such cases also a reduction of the fat is indicated.

The reduction must also follow the rules of dietetics (see Vol. VII of the German edition). But in withdrawing food the greatest caution must be observed; the withdrawal must never be effected rapidly, the amount of albumin contained in the body must be maintained and if possible increased, and all special instructions in regard to exercise, gymnastics, bathing, or drinking-cures must be directed toward the condition of the heart.

I am not much taken with the use of thyroid gland preparations in

heart disease. These remedies are not altogether free from danger, and ought, I think, to be avoided so long as we have other drugs at our disposal. Their use might be considered in those cases in which it is impossible to demonstrate any nutritional anomaly, that is to say, in the curious cases of constitutional obesity. But our experience with such cases is still altogether inadequate.

Treatment with Mineral Waters.—The drinking of laxative mineral waters for the purpose of reducing fat is, as we all know, habitually advised even in cases of heart trouble in the hope that, by diminishing the amount of fat in the body, the cardiac symptoms may be improved. When the theory of restricting fluid began to dominate the views of physicians, the profession began to lose faith in drinking-cures; indeed, some authorities went so far as to say that they are absolutely harmful. But experience has not altogether confirmed this view. Now, as heretofore, numerous patients suffering from obesity and cardiac symptoms go every year to Carlsbad, Neuenahr, and especially to Marienbad and Kissingen, and there find the relief that they had hoped for. We cannot help thinking that only a very small proportion of waters containing magnesium sulphate and bitter salts generally is absorbed; that the physicians practising at the above-mentioned resorts have had much experience in the treatment of the morbid conditions with which we are here dealing; and that instructions in regard to suitable diet and appropriate walking exercise make up a large part of their therapeusis. Hence the good results obtained in the above-mentioned health resorts cannot be denied, nor are they at all difficult to understand.

The indications for the use of the above-mentioned waters are found almost exclusively in obesity or, at most, in the first stages of various forms of arteriosclerosis. These indications will be discussed more at length in the respective sections, where it will also be shown what precautionary measures are necessary to prevent these drinking-cures from becoming harmful.

Milk-cures.—Milk-cures have been as enthusiastically recommended for diseases of the heart as for diseases of the kidney, in the hope that they would exert a favorable influence on the gastro-intestinal canal, on metabolism, and on the heart and kidneys. We are as yet absolutely unable to say how a milk-cure acts, and as F. A. Hoffmann* correctly says, at present our practical experience is not sufficient to enable us to form an accurate judgment, because we are still unable to deduce clear indications for the use of a milk-cure from our experience with the method. This statement applies to diseases of the heart just as much as to other diseases; some authorities warmly recommend the practice,† while others, on the contrary, restrict their patients considerably in the use of milk.‡

The truth probably lies between these two extremes, and in prescribing milk it must be remembered that if a man, even when he is at rest, is to be nourished entirely on milk, he must ingest a very large quantity of fluid—at least about three liters (quarts) a day. Hence an exclusive milk-diet is not to be thought of except in the case of heart patients who

*F. A. Hoffmann, "Zeitschrift für klinische Medicin," vol. vii, supplement, p. 8. *Ibid.*, "Allgemeine Therapie." Compare Högerstedt, "Zeitschrift für klinische Medicin," vol. xiv, p. 16. Koppel, "Petersburger medicinische Wochenschrift," 1892, p. 303.

†Karell, "Archives générales de médecine," 1866, vol. ii, p. 513.

‡Oertel, "Archiv für Hygiene," xvii.

have no tendency whatever to cardiac insufficiency, and even then I should not like to recommend it. I am aware of the fact that the best results with milk-cures in the case of heart patients with profound circulatory disturbances were obtained by giving small quantities—from 600 to 800 c.c.;* but this merely means a considerable abstraction of fluid combined with a reduction in the quantity of nourishment, the advantages of which have already been discussed in another place.

Milk is most useful for heart patients as a beverage and as an addition to other articles of food, and, aside from the question of the quantity of fluid to be allowed, will, therefore, always occupy a prominent place in the diet-list of any heart patient who is able to drink milk at all.

Many persons have, to a very large extent,—it is not too much to say to too great an extent,—lost the ability to drink milk. This is in part due to a peculiar system of education according to which the child's wishes are allowed to determine the choice of its food, and it is early allowed to indulge in highly seasoned dishes and alcoholic beverages. I cannot help mentioning this matter here because one can do a good deal with a child by perseverance and suggestion, and a child with heart disease can frequently be induced to take milk even if it refuses to do so at first.

In the case of adults, also, persuasion is not infrequently successful. But if the patient absolutely refuses to drink pure milk or really cannot tolerate it, the taste may be disguised by adding salt, sugar, cocoa, tea, coffee, cognac, or Nordhäuser [whiskey—Ed.].

I suppose a modern physician would not consider himself justified in ordering uncooked milk, and in the case of children who have been reared since bacterial days, and do not know the wonderful taste of good raw milk, it is a matter of no importance; but adults not infrequently declare that they will either drink their milk raw or not at all, and people with any idea of taste will not be surprised. For my part I always allow such patients to drink their milk raw. We physicians ought to make an effort toward having more clean dairies with non-tuberculous cows.

THE TREATMENT OF HEART PATIENTS BY MEANS OF MUSCULAR EXERCISES.

It is a well-known fact that the volume of a striated muscle increases in proportion to the work which it performs, and, as a result of the increase in size, the muscle's functional capacity is also augmented. Essentially the same thing is true of the heart, and the reasons why this similarity to the voluntary muscles is not more clearly apparent have already been explained. During muscular exertion the work of the heart is unquestionably also increased (see p. 486). It seems logical, therefore, to employ innervation of the skeletal muscles as a means of improving an ill-nourished, feeble heart, provided it is still capable of improvement.

This fact was known to Stokes; but although both Traube and his disciples, as, for example, Fräntzel, advised their heart patients to take a certain amount of exercise, Oertel was the first in recent times to adopt the procedure with a full consciousness of its significance, and will always deserve our thanks for this achievement.

It is not a question of *allowing* heart patients to exercise, but rather of *ordering* exercise for the definite purpose of imposing a certain amount of work on the muscle of the heart and thereby enabling the organ to improve its nutrition and increase its strength. Hence our object in ordering exercise is essentially different from that which we have in view when we

*Karell, *loc. cit.* Compare Hoffmann, "Allgemeine Therapie." Schnaubert, "Petersburger medicinische Wochenschrift," 1884, No. 5. Oertel, *loc. cit.*

give digitalis. The latter is a poison, and is given in order to call into action the latent strength of the heart muscle which has remained hidden and failed to make itself felt on account of the diseased condition of the organ. The stimulation incident to muscular exercise is, in addition, believed to give the heart muscle an opportunity to increase its functionating substance. The method, therefore, has a definite purpose. In order to accomplish that purpose we must have an accurate knowledge of the technic as well as of the kind of disturbances from which the patient who is to be subjected to treatment is suffering.

First of all, the patient's heart must be carefully examined in order to find out whether his case is a suitable one, a point which is determined by observing how the organ responds to certain definite demands, for the treatment cannot prove successful unless the heart is able to perform the increased amount of work induced by the artificial stimulation. This point must be determined beforehand—an important as well as a difficult task.

How can we determine that the heart still possesses functionating tissue capable of being educated? Some assistance is derived from clinical experience, which shows that many cases of obesity are suitable for treatment by exercise, it being immaterial whether circulatory disturbances have not as yet made their appearance or have already developed to a variable extent (see the section on the Cardiac Symptoms in Obesity). In fact, it may be said that patients of this class furnish by far the largest contingent of suitable cases.

Next in order are cases of cardiac disturbance in pulmonary patients. In this class of cases also very creditable results have been obtained. Finally, individual cases suitable for this mode of treatment will probably be found in any group of diseases. This will be referred to again in the special sections.

The question whether the method is likely to prove useful in an individual case may be determined partly by what the patient says about the behavior of his heart during muscular exercise and partly by making a cautious trial of the method, which yields more positive information. Thus the patient is made to walk on a slope of known grade or to go through a definite muscular exercise, and the physician observes whether the apex-beat becomes stronger, the radial pulse fuller and larger, or whether, on the contrary, acceleration of the heart action is accompanied by irregularity, inequality, and weakness, and whether the pulse becomes soft and small.

If the latter is the case, it is a positive indication that the heart will not tolerate the muscular exercise, and in that case the treatment is not only useless, but even dangerous, because of the possibility of acute overexertion of the heart. This so-called mechanotherapy is positively contraindicated by severe sclerosis of the coronary or other arteries in the body, advanced Bright's disease, and all acute processes (cardiac affections during and after infectious diseases, acute overexertion of the heart).

In some of these cases the organ is unable to secure a sufficient supply of blood for the increased demands made upon it; or it does not contain a sufficient quantity of tissue capable of increased work. In all acute processes the diseased muscle-fibers must be relieved from work as much as possible in order to carry them through the period during which their functional capacity is diminished.

In such conditions the treatment always involves danger of causing

overexertion of the heart muscle, and this has, in fact, frequently occurred. Great care is, therefore, urgently demanded, and in view of what has been said it is obviously sometimes exceedingly difficult to determine the indications for this method of treatment, because severer grades of arteriosclerosis of the heart must be excluded.

The method consists in prescribing systematized muscular movements.

Oertel recommends walking on a good road with known grades and distances, and this form of exercise seems to me also most satisfactory on account of the influence exerted simultaneously on the heart, the skin, and the lungs.

The exercises may be carried out either at a health resort or in the patient's home. While the latter plan is not always feasible, it can be carried out much more frequently than is generally thought. All that is necessary, in my opinion, is that the physician in charge of the case be familiar with the principles on which the method is based and be able to find a suitable road for the exercise. He should then personally try the stretch of road selected and carefully observe all its peculiarities. There is no doubt that in some cases, for reasons connected either with the patient or with the city in which he lives, it would be difficult, if not impossible, to get one's orders carried out. In such a case it is better to send the patient away, if possible, either to a well-managed private institution or to some suitable health resort. In some of the latter, for example Reichenhall, Schliersee, Meran, and Bozen, special walks for this so-called "Terraincur" have been laid out. These are undoubtedly of great advantage and simplify the treatment; but it should not be said that the treatment cannot be carried out without these walks because an exact dosage of movement cannot be obtained in any other way. That is a pure illusion, although masquerading in the garb of physiology, and cannot fail to excite the ridicule of any one who knows how complicated the conditions are.

There is no doubt that the treatment is simplified by means of these regularly laid-out walks, but it can be and in fact is carried out successfully in a number of places in Germany, in the Tirol, and in Switzerland. *An experienced physician is far more important than terrain walks*—at least for my own patients it is what determines my choice of health resorts or sanatoria. I send them to physicians who are specially well informed about the morbid conditions to be treated.

Others recommend for the exercise of the heart muscle carefully graded gymnastic exercises and the use of carbonated and saline baths.

The gymnastic exercises* are so designed as to offer a graded amount of resistance to the patient's movements for the purpose of causing an increase in the strength of the innervations up to a certain degree. The resistance to be overcome is supplied either by the hand of a physician or that of a trained instructor or by means of Zander's machines.

Whether in the treatment of heart patients the preference is to be given to the graded walks or to resistance exercises is a matter of opinion. For my part, I believe that either method may be employed in any case and for certain purposes; each has its special value. The demands made on the heart in resistance exercises are at first much smaller and more susceptible of being accurately regulated, and in many cases it may be wise to begin with a course of resistance exercises or simply to order the patient to go through certain exercises without any instruments or with light dumb-bells and wands, and, after the heart has had a certain amount of training, to follow this with hill-climbing.

Although hill-climbing has done a good deal of harm because pursued with unreasoning enthusiasm and employed in unsuitable cases, I cannot go so far as to exclude it altogether in any case in which exercise is useful at all. The effect of climbing on the respiration and the pleasure of being in the open air early in the morning ought not, in my opinion, to be neglected.

* Hughes, "Lehrbuch der schwedischen Heilgymnastik," Wiesbaden, Bergmann.

Heart patients, like all other kinds of patients, have been advised to ride a bicycle. Such advice in my opinion is absolutely dangerous. I do not believe that the patient ought to be allowed to indulge in such violent exercise for the purpose of strengthening his heart, especially as, for a variety of reasons, the patient is so apt to carry the exercise to excess. I, therefore, urgently advise against bicycle riding.

If the patient is so weak that active muscular exercise appears to be unsafe, massage of the extremities and of the trunk, if properly and carefully performed, is often well borne. This practice has been in vogue in Sweden for some time.*

In some cases massage has been known to be followed by direct improvement of the circulatory conditions and especially by the disappearance of edema. Gradually the patient is made to perform movements himself, and in this way the transition is gradually and cautiously made from passive to active exercises. So-called massage of the heart, as recommended by Oertel,† I have never dared to try.

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METHODS OF INFLUENCING THE METABOLISM OF WATER IN HEART PATIENTS.

There are many reasons why it is exceedingly difficult at present to give an opinion in regard to the advisability of *withholding fluid* in the treatment of heart patients, because both our practical experience with the method and our knowledge of its theoretic foundation are still, on the whole, rather limited.

That the theoretic foundation is insecure will surprise no one, for we have already referred to the many gaps in our knowledge of this subject (see p. 516). It is very difficult, in a given case of illness, to form any precise idea of the amount of water contained in the blood and tissues, and still more difficult to form an estimate of the exchange of water and the total quantity of blood. There are no good observations about the amount of water in the blood and tissues in heart patients, and yet we ought to be perfectly familiar with it before venturing on such active intervention as the restriction of fluid against the dictates of instinct and thirst on the part of the patient.

* Möller, "Ueber die manuelle Behandlung der Herzkrankheiten," Dissertation, Berlin, 1898. Compare also Zabludowski, "Berliner klinische Wochenschrift," 1896, No. 20.

† Oertel, "Münchener medicinische Wochenschrift," 1889, No. 37–39.

But, what is even more surprising, our practical experience in regard to the value of the method is also, on the whole, quite limited, although there is no lack of cases in which it has been tried. Indeed, the method has been tried more than enough. When Oertel first published his procedure and doctors, as well as the public, adopted it as a fashion, suitable and, of course, altogether unsuitable cases also began to be treated by restricting the ingestion of fluid. Some did well, others did badly. The former were remembered and, as usually happens, the failures were forgotten. They only began to be remembered again after the method had ceased to be a novelty. All that we now have left is the individual physician's custom. But when we look for the testimony of reliable observations, we find comparatively little of value.

The object of the treatment in certain cases of heart disease is to relieve the organism of excessive quantities of water that are supposed to have accumulated in the blood and in the tissues. The theoretic foundation for this procedure, as has been said, is extremely slender, and this lack of theoretic foundation renders the discussion of the method as a practical procedure exceedingly difficult.

Of course, there are patients with cardiac insufficiency who suffer from serous plethora, but in the present state of our knowledge it is hardly possible to make such a diagnosis in a given case; it certainly cannot be inferred from the kind of heart disease that is present—in short, there does not seem to be *a priori* any way to determine in a given case whether the abstraction of fluid will or will not prove beneficial. I shall discuss the methods before venturing to say what there is to be said on this point, because, after all, our chief guide must be the general results obtained.

A reduction in the amount of fluid contained in the organism may be accomplished by diminishing the ingestion and increasing the excretion through the kidneys, skin, and lungs. In the treatment of cardiac insufficiency it has always been customary to stimulate the excretion of urine. Evaporation of water from the skin can be increased by means of sweat-cures or the stimulation derived from exercise; evaporation from the lungs is no doubt effected chiefly by exercise.

Which of these two methods is to be adopted in a given case we are, unfortunately, unable to determine on rational grounds, for the question of water balance in heart patients has never been accurately investigated, and we do not know whether the various systems of the body contribute to the excretion of water in the same proportions as in health or whether the morbid condition causes a disturbance of these proportions. We have to be guided solely by practical experience, and it is, therefore, wiser to discard all "scientific" embroidery with theory as being mere pseudotheory. For example, numerous so-called differential determinations have been made between the amount of fluid ingested with the food and that excreted in the urine. I cannot bring myself to believe that such investigations afford any *positive* data in regard to the interchange of fluid in the body; for it is well known that the quantity of water excreted through the skin, lungs, and intestines is, for a variety of reasons, subject to marked fluctuations.

A discriminating critic will not conclude solely from a change in the relation between the quantity of water ingested and the quantity excreted in the urine that water has been retained in the body; but even the clinician cannot arbitrarily disregard an entire series of important physiologic factors bearing on the question. However, even this procedure may afford some information of value in the application of therapeutic measures.

Regulating the Ingestion of Liquid.—It has recently become the custom to regulate the amount of liquid ingested for two reasons: on

account of its effect on the circulation and because of its importance with reference to body-fat. The second reason I shall disregard, as I shall have occasion to speak about the body-fat in connection with so-called fatty heart. For the present we have to do only with the effect produced on the circulation by the ingestion of liquid.

The quality as well as the quantity of liquid introduced into the body is a matter of importance, and the effect varies considerably in different persons, depending not only on individual habits, but also on local or national customs. For, unless an individual suffers from a morbid thirst, he is not likely to take large quantities of liquid in the form of water. More likely it is coffee or tea—which I will not venture to decide, but I am under the impression that in some of the poorer regions of Thuringia the people are in the habit of indulging freely in an inferior kind of thin coffee.

But when large quantities of liquid are habitually ingested, it is most frequently in the form of beer or wine. In many parts of the world the people regularly drink large quantities of these liquors, and some individuals excel in the enormous quantities they are able to dispose of. This is attended by the entrance of excessive quantities of liquid into the circulation, and, in addition, the alcohol exerts an injurious influence on the vessels and probably also on the heart. In short, the result in this case is unquestionably effected by certain very special factors. It is not a mere coincidence that Oertel* lauded the good effects of limiting the quantity of liquid ingested in Munich, and that in the famous clinical history, the first one in his work, which illustrates the results of the procedure, what he had to forbid was beer in such quantities as any unprejudiced person will admit are much too large for the individual case, and not relatively insignificant quantities of some other, indifferent beverage.

The average quantity of liquid which a healthy man is in the habit of ingesting is usually given as 1½ to 2 liters (quarts). As a rule, the water contained in the food is not included in the calculation, although, in my opinion, disregarding the latter leads to considerable errors and, in fact, renders all the calculations illusory, for the quantity of water contained in the food is subject to extraordinary variations. Again, the character of the solid food must be taken into consideration, because it determines the quantity of water that the organism needs to excrete the waste-products, not to mention salts and their influence on thirst. It follows, therefore, that it is hardly correct to discuss the quantity of fluid only, independently of other factors; and any conclusions deduced from such a line of argument must be uttered with great reserve.

It is necessary, in the case of heart patients, to gain at least an approximate idea of the quantity ingested. If conditions which cause abnormal thirst are present, such, for example, as diabetes mellitus or insipidus or contracted kidney, they must receive due attention. If none of these special factors is present, it seems to me wise, even when the force of the heart is adequate, to limit the quantity of liquid to be taken during twenty-four hours to 1.5 liters (quarts), or, at least, to make an attempt to enforce such restriction. Of course, if abnormally large quantities of water are being excreted through the skin, the lungs, or the intestine, for example, the water-loss must be made good. In this respect also I wish to insist on the importance of individualization. Too much officiousness is unwise in any severe disease; if the patient does not bear

* Oertel, "Therapie, u.s.w." Feilchenfeld, "Zeitschrift für klinische Medicin," vol. xi, p. 463.

the reduction of liquid, it is better to give up. Our knowledge in regard to the quantity of water required by the processes of metabolism in an individual case is as yet so imperfect that I would not venture to enforce restriction in defiance of a rebellious organism. I believe, however, that it is wise in any case to get the patient out of the habit of drinking excessive quantities of liquid, provided it can be done without producing any grave disturbances.

In the presence of cardiac insufficiency, disturbance of compensation accompanying any form of muscular disease,—for we have nothing to do with valvular lesions,—the question of the withdrawal of fluid becomes much more urgent. In such cases, also, the patient must be restrained from taking large quantities of fluid, as explained above, it being particularly important to enforce these orders in such cases.

The indication for still further restricting the ingestion of liquid is found chiefly in hydremic plethora. Unfortunately, it is difficult to determine positively whether such a condition is present. As a rule, an approximate idea of the amount of water contained in the organism can be deduced from the presence of edema, which indicates a high percentage of water. If edema is present, the daily quantity of liquid should, if possible, be reduced to 1500 c.c. (1½ quarts) or even to 1 liter (quart), provided the patient shows no signs of marked weakness and does not expressly refuse to limit his ingestion of liquid after he has given the matter a fair trial. For not infrequently a marked reduction of the quantity of liquid ingested is attended by most unpleasant symptoms; aside from thirst, the patient complains of fatigue, depression, apathy, sometimes also of irritability, and as at best the indication for restricting fluid is uncertain, I do not think one is justified in causing the patient any actual distress.

The object of the whole procedure is, in the first place, to diminish the work of the heart and, in the second place, to stimulate the excretion of substances which are eliminated through the urine by attracting the excess of water from the blood and the tissues.

To what extent the work of the heart is actually influenced by such abstraction of liquid we are at present quite unable to determine.

The quantity of urine sometimes increases as the ingestion of liquid is restricted; sometimes it does not. An increase appears to take place when the tissues of the body contain such an excess of water that the blood is able to satisfy its need for liquid by absorbing it from the tissues as soon as the quantity of liquid supplied through the intestinal canal is diminished. But as soon as the water begins to drain from the tissues, the velocity of the circulation improves (the exact reasons for this are only partly known) and the quantity of urine increases more and more.

In practice, therefore, a comparison of the quantity of liquid introduced through the stomach with the quantity excreted in the urine is of some value in determining the treatment; but we have practically no knowledge of the interchange of water in circulatory disturbances.

The physician must experiment in every individual case, but even that is safer than running the risk of doing harm by a mistaken subservience to routine. I will cheerfully submit to the criticism that my indications are "indefinite" if I can induce every physician to experiment for the benefit of every patient. Is it for nothing the teachings of the sagacious Stokes are so generally respected?

Abstraction of Fluid by Increasing the Quantity of Urine.—The quantity of urine must be carefully regarded in the treatment of every heart patient; for, provided the above-mentioned precautionary measures are observed, the quantity of urine to a certain extent affords a measure of the functional capacity of the heart. Anything that tends to increase the pressure and velocity of the blood in the renal vessels at the same time causes an increase in the quantity of urine. Whenever a circulatory disturbance exists, the excretion of urine must, if possible, be increased by correcting the circulatory disturbance.

But we shall not speak of these matters; we are solely concerned with the drugs which, by acting on the kidneys, increase the quantity of urine excreted, that is, the so-called diuretics. In actual practice—as I wish to point out once more—these drugs are, as a rule, employed only after an effect has been produced on the heart or at least in combination with heart stimulants.

Any one who is in the habit of observing himself must have discovered that a number of substances taken with the ordinary food may, under certain circumstances, act as marked diuretics, such, for example, as tea, coffee, and cocoa. These and similar substances are destined to be used much more extensively in the future, although as yet observations in regard to their action are still inadequate.

Among *salts*, potassium acetate is now used most frequently as a diuretic.

The potassium salts, for reasons known to every one, are more active than the salts of sodium, and it is always well to select an acid which does not occur in the body or at most only in traces. The administration of salts is followed by the well-known changes explained by Bunge: the foreign salts are excreted, carry water with them, and probably also act as a stimulant to the renal epithelium.

Potassium acetate is given in the form of liquor potassii acetatis [German Pharm., 33⅓ per cent.—Ed.], usually with syrup and water, about 2 to 3 gm. (30 to 45 minims) every two hours. This is probably the mildest measure that can be employed to stimulate the excretion of urine.

A much greater effect can be produced on the cells of the kidney by various *substances belonging to the caffein group.** As, however, the renal arteries must have a certain width in order to allow a free secretion of urine, and many of these substances, like caffein, constrict the arteries, some additional means formerly had to be employed to secure dilatation of the vessels. But since v. Schroeder made known the peculiar qualities of *theobromin*, this difficulty has ceased to exist.† We now use the salts of theobromin; Knoll's "*diuretin*," which is the one most commonly used, is theobromin and sodium salicylate and contains about 50 per cent. of theobromin.

This preparation in animals does not exert any marked influence on the circulation,‡ its chief action being expended directly on the cells of the kidney. At the same time I purposely refrain from expressing any opinion as to what special parts of the kidney are acted upon by these substances.§

* v. Schroeder, "Archiv für experimentelle Pathologie," vol. xxii, p. 39, and vol. xxiv, p. 84.

† v. Schroeder, *ibid.*, vol. xxiv, p. 101.

‡ Cohnstein, "Berliner klinische Wochenschrift," 1893, No. 4.

§ Compare v. Sobieranski, "Archiv für experimentelle Pathologie," vol. xxxv, p. 144.

In disease it is not altogether impossible that theobromin may have some effect on the heart and vessels. Experience has frequently shown that great care is necessary in applying results obtained by the administration of drugs to animals, especially healthy animals, to conditions existing in man in the presence of disease. Many of these observations unquestionably indicate that the heart is also affected at the same time.* Of course, the question can only be decided by means of investigations carried out with equal accuracy as regards technic in man, such as the excellent observations of v. Schroeder. It is possible that some small part at least of the diuretic action is due to the sodium salicylate, but its influence in any case must be exceedingly slight.

The daily dose of diuretin is from 4 to 5 gm. (1 dr. to 75 gr.) [or even 8 gm. (2 dr.)—Ed.], administered preferably in a simple watery solution or at most with simple syrup, 1 gm. (15 gr.) every two hours. The drug should, in my opinion, be kept up for five consecutive days, and, if necessary, resumed after an interval of several days. Personally I have never seen any bad results of any severity, although collapse has occasionally been observed. It is, therefore, necessary never to exceed a daily dose of 5 gm. (75 gr.) [unless, as should always be the case, the patient is being carefully watched—Ed.]. Nausea and vomiting sometimes occur.

Whether or not theobromin exerts a certain influence on the heart and vessels, it should be used whenever the removal of dropsical effusions becomes necessary after direct heart remedies have been employed. Under such conditions the drug often proves most useful. The quantity of urine increases usually on the second or third day to from 1.5 to 5 liters (quarts). The factors which determine the efficacy of the drug and the reasons why it occasionally fails to act still remain to be discovered. Unfortunately, the preparation is very expensive.

In many cases *calomel* is an extremely useful drug for combating dropsy in heart disease.† My reason for advising its use after diuretin is not that it is less efficacious, but because it is unquestionably not quite free from danger. Adults are given 0.2 gm. (3 gr.) three times a day, and if diarrhea develops, 0.01 to 0.02 gm. ($\frac{1}{6}$ to $\frac{1}{3}$ gr.) of opium is added to each dose. Stomatitis not infrequently develops, especially when the condition of the mouth is bad. When the patient is gravely ill, the stomatitis alone may prove a serious discomfort on account of the pain and interference with eating and drinking, at least serious enough to merit attention; but, unfortunately, that is not all, for an enteritis ranging from a very mild grade to the most extensive ulceration may be superadded. These are the dangers that are always present when mercury is given in large doses; but the doses that have been mentioned, which are by no means small, are necessary if an effect is to be produced and, in fact, must be kept up for several days. For the increase in the excretion of urine, as a rule, does not appear until some time on the second to the fourth day, and occasionally even later. After the drug has begun to act, from 2 to 8 liters (quarts) of urine may be evacuated daily, and the effect of the remedy often outlasts its administration or may only begin after the administration has ceased. It is, therefore, well, if no effect shows itself within six days, to withhold the drug for a few days. It may be well to remind the reader that all the precautions ordinarily

* See B. Askanazy, "Archiv für klinische Medicin," vol. lvi, p. 209; contains the literature. Compare Pawinski, "Zeitschrift für klinische Medicin," vol. xxiv, p. 315.

† Jendrassik, "Archiv für klinische Medicin," vol. xxxviii, p. 499, and vol. xlvii, p. 226. Stintzing, *ibid.*, vol. xliii, p. 206. Stiller, "Wiener medicinische Blätter," 1886, No. 28. Fleiner, "Berliner klinische Wochenschrift," 1890, No. 48.

employed when mercury is given, such as thorough rinsing of the mouth and the like, must be rigorously observed.

With proper precautions in this respect, if the treatment is at once interrupted on the appearance of marked symptoms of intoxication, calomel proves one of the most efficient weapons against dropsy due to cardiac weakness. The reason why the remedy is not more highly thought of by some probably is that its use requires a certain degree of energy on the part of the patient as well as the physician. Small doses especially are, as a rule, ineffective, and the method is not altogether free from danger.

If the remedy proves successful, its administration may be continued for a longer time, say up to ten days, as in such a case there is little danger of intoxication because the mercury is evidently being rapidly eliminated. Repeated courses of calomel are justifiable and may prove very useful in such cases; for it must always be remembered that the remedy is a purely symptomatic one.

The indications for the use of calomel are practically the same as those for theobromin. So far as we know, the drug has no effect on the heart, the excretion of water being effected through its action on the intestine and the cells of the kidney. Hence it is in no sense comparable to cardiac stimulants proper; calomel is given solely for the purpose of removing dropsical effusions when their removal cannot be effected solely by increasing the action of the heart.

Calomel, in the opinion of most authorities, is contraindicated in the existence of any form of pronounced nephritis. In such cases experience shows that calomel is not well borne and also very uncertain in its action. There are some, however, who dissent from this view. Albuminuria, even if marked, in no sense contraindicates its use, since it is due chiefly to venous stasis.

Numerous other substances are credited with the property of influencing the excretion of urine; indeed, it would be very fortunate for us if their efficacy even remotely approached their number. But this is far from being the case. In fact, there is not a single remedy that I can recommend for diuretic purposes in comparison with those I have just mentioned.

The Excretion of Fluid Through the Skin.—Large quantities of fluid can be abstracted from the body through the sweat-glands, but profuse sweating is indubitably associated with marked changes in the circulation and is, therefore, anything but an indifferent procedure in cases of heart disease.

Of chemic substances that produce perspiration, *pilocarpin* is the only one that is reliable in its action, but the drug, unfortunately, exerts such an unfavorable influence on the heart that I cannot bring myself to recommend its use for heart patients.

The sweat-glands can also be stimulated through the agency of physical heat regulation by *heating the skin*. I would recommend that hot baths followed by sweating be employed only with great caution, for they are by no means free from danger in cases of circulatory disturbance. I prefer either a hot pack in bed, in which the vapor derived from an alcohol lamp is conducted under the blankets that surround the patient, or so-called cabinet vapor-baths. In either case the patient's head remains exposed to the air and may be still further protected with an ice-cap or a cold compress.

* Sklodowski, "Archiv für klinische Medicin," vol. lii, p. 300.

This procedure may be tried after it has been found impossible to remove any more fluid from the body by chemic means and the measures about to be mentioned either cannot be employed because the necessary appliances are not obtainable or because they are too risky. It is to be remembered, however, that sweat-baths are not well borne unless the heart muscle still possesses a certain strength and a certain power of accommodation.

Finally, sweating may be induced by *vigorous exercise* through the agency of physical heat regulation, especially in hot weather. This method has already been discussed in connection with procedures to strengthen the heart muscle, which act in part through the abstraction of water incident to the increased excretion of sweat, and it should be used for abstracting water from the organism whenever any other indication or justification for exercise exists.

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The Removal of Fluid from the Lymph-spaces by Means of Trocars.—Satisfactory methods are now available for the operative removal of fluid that has accumulated in the pleural cavities, the peritoneal cavity, and the lymph-spaces of the skin. But, as a rule, this procedure will not be thought of until every method has been tried to correct the disturbance by improving the force of the heart. I would even advise the adoption of measures to increase the excretion of urine and, if it is well borne, the secretion of the sweat-glands also before resorting to puncture for the removal of cutaneous edema, because the two former methods are distinctly less unpleasant to the patient.

Puncture of the abdomen can be quickly settled. It is indicated in the conditions with which we are dealing chiefly when a large quantity of fluid has accumulated in the abdominal cavity as a result of some special disease of the peritoneum or of the liver. The question of interference is then decided by the general principles that guide us in the management of such conditions, it being remembered that even a moderate upward forcing of the diaphragm often suffices to injure an enfeebled heart, so that the presence of only a small quantity of fluid may, in some cases, require paracentesis.

Even when the accumulation is due solely to the heart itself, puncture may, in rare cases, be required if the fluid becomes dangerous on account of its quantity alone. In such cases one must be prepared to see it reaccumulate in a short time, and hence the procedure in such cases, as a rule, has only a temporary value.

The case of the *pleural cavities* is quite similar. Puncture is not infrequently indicated for pleural exudates either because they are large or because they are obstinate. I take this opportunity once more to insist on the danger of delaying too long.

Puncture may also occasionally be required in hydrothorax, the indi-

cation being direct menace to the respiration; but in these cases again the fluid, as a rule, reaccumulates in a short time.

Puncture should be performed with one of the many instruments devised for the purpose, and the fluid withdrawn by means of a siphon. I am in the habit of using Curschmann's all-metal, flat, cutting trocars, made by Möcke in Leipzig (Universitätsstrasse). The instrument comes in three sizes, the smallest of which is very well adapted to our class of cases because its use is attended by very little discomfort to the patient. These trocars are, in my opinion, decidedly the best, because they are so readily disinfected and easy to handle.

Mechanic evacuation of the fluid in subcutaneous edema must be considered after repeated attempts to improve the strength of the heart or to stimulate the action of the glands have failed, and the edema is extensive and persistent. The edema in many cases is exceedingly unpleasant and annoying to the patient and for that reason alone its removal is desirable. Not infrequently the procedure is followed not only by the disappearance of the symptom, but also by a general improvement of the circulation. This point has been referred to before.

For removing the fluid I recommend Curschmann's flat, sharp, skin trocars. I have used both these instruments and Southey's tubes and consider that the former have a great advantage because they are easier to clean and do not become clogged so frequently.

From two to four of these trocars or needles, which must be previously boiled, are inserted in the cutis after the skin has been carefully cleansed, the sites of election being the outer aspect of the thigh and (or) the abdomen. The needles are fitted with small rubber tubes, the ends of which are immersed in a 0.7 per cent. saline solution. The rubber tubes, before the procedure is begun, must be filled and boiled with the same saline solution. The trocars will stay in the skin of their own accord, but it is better to secure them with silk ligatures and adhesive plaster. [Still better, to put a few turns of sterile gauze bandage around the part and under the head of the cannula.—Ed.] The puncture wound is dusted with iodoform or airol, or covered with a compress of aluminium acetate. The trocars are removed after from twenty-four to thirty-six hours, and may have to be introduced again in other places, depending on the patient's condition.

As a rule, the fluid drains off rapidly; sometimes many liters (quarts) are removed in a day. In a few cases, usually for no ascertainable reason, the method fails. If it were not for the danger of infection, the method could be employed much more generally; as it is, it is justifiable only under rather favorable external conditions. A disadvantage, which is sometimes very annoying, is the oozing of fluid from the wound, which may continue for some time after the trocar has been removed.

If the fluid cannot be removed by means of trocars, punctures or long incisions of the skin, as recently recommended by C. Gerhardt, Fürbringer, and Lenhartz, may be tried. The most scrupulous cleanliness is necessary because edematous tissue is particularly liable to infection. The parts where the incisions are to be made must be covered with large quantities of absorbent material to take up the fluid, in spite of which precaution large quantities of fluid often ooze through and wet the bed, which is, of course, very unpleasant. Nevertheless, the method is indispensable in some cases.

Curschmann has succeeded in constructing vessels with removable lids, which are first attached to the skin without the lid by means of adhesive plaster or collodion. The skin within the area covered by the vessels is then incised, the lid is applied, and the fluid escapes through

an opening in the lid as through a skin trocar. In this way wetting of the bed can be avoided.

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THE MEDICINAL TREATMENT OF CARDIAC INSUFFICIENCY.

It has been shown that retardation of the circulation, with lowering of the pressure in the arteries, may be due to weakness of the heart or to abnormal dilatation of extensive arterial regions, especially in the abdomen. Theoretically, it is most important that the cause be properly recognized before the condition is treated medicinally, because remedies which cause constriction of the vessels and thus increase the resistance would be absolutely dangerous to a weak heart and, on the other hand, but little is gained by improving the action of the heart when there is extensive paralysis of the vessels. We have by no means reached a point in practice where we can satisfactorily classify the remedies generally used in the treatment of circulatory weakness according to these rational indications; some attempts have, however, been made in this direction and are being constantly added to.*

In true cardiac insufficiency there is incomplete contraction of the musculature of the auricles, and especially of the ventricles, which, for the reasons explained above, is associated with dilatation of the respective cavities. As a rule, both ventricles are involved, although frequently the loss of function in one materially exceeds that in the other.

Among the remedies used in the treatment of cardiac insufficiency the leaves of **digitalis purpurea** indisputably hold the first place.† The physician must make himself thoroughly familiar with the properties and uses of this drug; for *it is not enough for him to know that digitalis is the remedy to give: he must also know how, when, and how much of it to administer.* It is my belief that an adequate knowledge of this one substance would enable a physician to dispense with all other "heart remedies"; but all the other remedies taken together, without digitalis, are inadequate.

In practice we are still in the habit of using the crude drug, which, of course, is a great disadvantage in the case of such an important and active substance; for the effect produced by the prescription is apt to depend on a great many different factors which it is difficult to estimate, such as the place where the plant is grown, the time of the year when the leaves were picked, the age of the leaves, the method used in keeping them, and other like factors. No one will deny that these are material disadvantages. But for all that we are not yet in a position to substitute the active agent for the crude drug in medical practice.‡ Some of the preparations are relatively dear, others are insoluble, and they all exhibit so many irregularities and peculiarities in regard to absorption and elimination that the possibility of the drug accumulating in the body

*Compare Pässler, "Archiv für klinische Medicin," vol. lxiv, p. 715.

†For a history of the pharmacology of digitalis see the excellent treatise by Schmiedeberg, "Archiv für experimentelle Pathologie," vol. xvi, p. 170.

‡See Schmiedeberg, "Archiv für experimentelle Pathologie," vol. xvi, p. 149. *Ibid.*, vol. iii, p. 16. *Ibid.*, "Grundriss der Arzneimittellehre," third edition, Leipzig, 1895. Kiliani, "Archiv der Pharmacie," 230, 233.

cannot be altogether excluded. In view of the great toxicity of these substances, such an accumulation would be extremely dangerous and, in fact, cases of poisoning of this kind have been observed. Good results have been reported with *digitalinum verum*,* but from what I have seen myself I would not venture to recommend its adoption in the place of the crude drug.† The only thing that would induce me to try the preparation would be some very definite contraindication to the administration of the leaves by the mouth or rectum, or some condition rendering such administration altogether impossible, or a desire to produce a more rapid effect than can be obtained through absorption from the mucous membranes. For instance, should it be necessary to get the heart under the influence of digitalis within a few hours, an injection of a solution of digitalinum might be recommended. But it must be remembered that the toxic effects manifest themselves more rapidly by that mode of administration, although their disappearance is also proportionately rapid.

Digitoxin has also been used ‡ both for administration by the mouth and per rectum. Unverricht has reported some cases in detail;§ he has never seen any bad results from the remedy, and has practically given up the use of the crude drug. I myself have repeatedly used the remedy to good advantage, but so far my experience has not been extensive.

There is no doubt that we are approaching the time when only the pure active principle, not only of digitalis, but of other plants as well, will be used in medicine; but before they can be recommended to the physician, their effect on every conceivable variety of cardiac disturbance must be determined by extensive clinical experience. At present there is always danger that, by giving a remedy the action of which is not accurately known, either harm may be done or, supposing the remedy to be free from danger, at least precious time may be lost by not at once giving the patient one of the approved and well-known preparations. And who would care to assume the responsibility of doing either of these things? [In the effort to get a preparation free from the disadvantages of others, Cloetta produced a substance which he named digalen, apparently chemically identical with crystalline digitoxin, but more soluble in water and less irritating to the tissues than any other preparation. It is sold in sterile glycerin solution, 1 c.c. of which contains 0.3 mg. of digalen (digitoxin solubile), having an effect equivalent to 0.1 gm. of digitalis leaves. My own brief experience has not led me to believe it has decided advantages over older preparations.‖—Ed.]

What then is the *effect of digitalis on the circulation?* When the heart is weak, the administration of digitalis increases the flow of blood in the arteries at the expense of the venous blood; hence the arterial pressure rises and the venous pressure falls. This effect is produced because the

*Pfaff, "Archiv für experimentelle Pathologie," vol. xxxii, p. 1. Stoitscheff, "Archiv für klinische Medicin," vol. lii, p. 475.

†Klingenberg is of the same opinion; "Archiv für experimentelle Pathologie," vol. xxxiii, p. 353. Deucher, "Archiv für klinische Medicin," vol. lvii, p. 1.

‡Wenzel (see Unverricht), "Centralblatt für innere Medicin," 1895, No. 19. v. Starck, "Münchener medicinische Wochenschrift," 1897, No. 4. Wellenhof, "Wiener klinische Wochenschrift," 1896, No. 42.

§Unverricht, "Congress für innere Medicin," 1899, p. 312.

‖See Cloetta, "Münchener medicinische Wochenschrift," 1904, No. 33. Kiliani has recently opposed the claims of Cloetta, "Münchener medicinische Wochenscrift," April 30, 1907.

contractions of the ventricles are more complete and, although their number at the same time diminishes, they, nevertheless, propel more blood in a unit of time from the veins into the arteries, so that the velocity of the blood-stream increases. The excretion of water in the urine is increased, while swelling of the liver and edema diminish. These things are readily explained by the changes that take place in the circulation.

A question which has always occupied the attention of clinicians is whether the substances contained in digitalis exert a direct influence on the vessels as well as on the heart. The vasomotor action, if it exists at all, assuredly plays a very minor rôle. This is shown by the very fact that the digitalis bodies improve the function of *diseased* hearts. If their action was chiefly on the vessels, they would only tend to increase the work of the heart and would, therefore, injure the diseased organ. This alone proves that the vasomotor action is at best exceedingly slight. But special observations on this point, in part at least, yield the same result.* It is true that other authorities† ascribe to the digitalis group a direct effect on the muscular tissue of the blood-vessels. If they really possess that property, it must be very slight, and in the light of clinical experience may be practically disregarded. It might be objected that, even granting the vasomotor action of digitalis, it might, nevertheless, strengthen the heart by exercising its power of accommodation; but in view of the notorious insufficiency of a feeble heart whenever a fresh demand is made upon it, this hardly seems likely.

Clinical experience, therefore, compels us to assume that the substances contained in digitalis influence the contractions of the muscle and render them more complete. The change in the character of the contractions, which become slower and more regular, may be regarded as coördinate with the above-mentioned (direct) action on the heart; for the retardation and greater regularity, like the more complete contraction of the heart, merely indicate that the conditions have returned to the normal—it is not at all necessary to reduce the rate below the average to obtain the desired effect, provided digitalis is used cautiously. It is possible, however, that, as Traube asserts, the rate of the heart is directly influenced by the action of digitalis,—whether the drug attacks the nervous system or the muscle,—and the central stimulation of the vagus, which is always associated with a rise of blood-pressure, may also have something to do with it.

Such is our incomplete conception of the mode of action of digitalis derived from observation of heart patients. The results of animal experiments are not altogether at variance with this conception, as the excellent work of Böhm,‡ for example, shows. The latter, with a technic above all criticism, observed strengthening of the contractions of the heart, even in a healthy animal, after the administration of digitalis and digitalin.

According to observations made in Schmiedeberg's laboratory, helleborein increases the amount of blood in the arteries of the healthy frog and mammals by increasing the systolic output without augmenting its frequency. The quantity of blood expelled by one systole is increased in a healthy and normally contracting

*Böhm, "Pflüger's Archiv," vol. v, p. 153. Williams, "Archiv für experimentelle Pathologie," vol. xiii, p. 9. Schmiedeberg, "Arzneimittellehre."

†B. Kobert, "Archiv für experimentelle Pathologie," vol. xxii, p. 102.

‡Böhm, "Pflüger's Archiv," vol. v, p. 153. See also Zerner, "Wiener klinische Wochenschrift," 1891, No. 37 and 38 (von Basch's laboratory). Fraenkel, "Archiv für experimentelle Pathologie," vol. xl, p. 40 (von Schroeder's institute).

heart by the reduction of the tonus during diastole. For, if the tone is diminished, the heart becomes more completely filled with blood; and since the muscle contracts normally and is, therefore, able to propel larger quantities of blood into the arteries without any difficulty, the quantity of blood and the blood-pressure in the latter rise. Williams* is, therefore, quite right when he describes this effect as a "greater utilization of the force of the heart."

The so-called "absolute strength of the heart," as defined by Schmiedeberg and his pupils and determined by the maximal pressure which the muscle is able to overcome, Williams found in the frog was not affected by digitalin and helleborein, while Böhm saw an increase in the same animal.

If, as we have seen, digitalis in many cases increases the quantity of blood in the arteries even when the heart is diseased and, therefore, less capable of doing its work, its action must evidently consist, in the main, in improving the contractions of the heart. A mere alteration of the diastolic tone, which increases the quantity of blood in a cavity, would have no effect in this direction for it is not, as in a healthy organ, necessarily associated with an increase in the systolic output. On the contrary, it is possible that the latter is diminished, since the disability of the diseased muscle manifests itself chiefly by the fact that it is unable to propel the usual quantity of blood. What, then, is to be gained by still more increasing the load?

Hence in the case of disease we must assume that an improvement always takes place in the contractility of the heart, and, this being admitted, the view naturally suggests itself that in a healthy organ also the improvement in the circulation is due to a direct influence on the contractions, that is to say, the strength of the heart is more fully utilized. The alteration of the diastolic tone and the increase in the contractions may be regarded as coördinate processes. Böhm, it must be remembered, directly demonstrated that the contractile force is increased in the healthy organ also. Romberg† is unquestionably right when he says that a diseased heart under the influence of digitalis learns to use a larger portion of its reserve strength than during insufficiency.

I purposely refrain from entering more deeply into a physiologic discussion of the action of digitalis on the heart and its individual portions, because our views on the subject are by no means clear and because fresh investigations by means of newly devised methods have recently been carried out, and while they offer a most favorable prospect for the future, these investigations are not as yet concluded (I refer, for example, to O. Frank and Jacobj‡). Many things can, of course, be decided only by experimentation. But it is to be hoped that in the future animal experimentation to determine such questions as the action of digitalis will not be confined to healthy animals, but will include diseased animals as well. It is probable that data obtained from the latter will yield much more valuable information, and it may quite possibly be found that some phenomena can only be observed in diseased animals. Perhaps also differences exist between the action of the substances derived from hellebore and the substances contained in digitalis, and these differences may possibly explain the lack of uniformity in the results obtained.

The above-mentioned changes in the cardiac action (the increase of the contractile power of the heart muscle) represent the *first* effects of the substances contained in digitalis. When given in large doses, they cause a further rise of the blood-pressure with, at first, a pathologic retardation of the pulse, followed by irregularity and inequality of the beat, later enormous acceleration of the pulse and a fall of the arterial tension, and, finally, arrest of the heart in systole. In these severe grades of intoxication other organs are also affected, especially the stomach, and nausea and vomiting occur. [For reference to recent studies in digitalis poisoning see the report of A. W. Hewlett, "Digitalis Heart Block," "Journal of the American Medical Association," January 5, 1907, p. 47.—Ed.]

*Williams, "Archiv für experimentelle Pathologie," vol. xiii, p. 1. Dreser, *ibid.*, vol. xxiv, p. 221. Compare the interesting experiments of Romberg, "Herzkrankheiten," p. 868.

† Romberg, "Herzkrankheiten," p. 868.

‡ Compare O. Frank, "Sitzungsberichte der Gesellschaft für Morphologie and Physiologie in München," 1897, vol. ii, p. 1. Jacobj, "Archiv für experimentelle Pathologie," vol. xliv, p. 368. Wegbauer, *ibid.*, p. 434.

These effects of the drug should all be avoided when digitalis is given in therapeutic doses, although they occasionally manifest themselves against the physician's wish, probably because the toxic substances sometimes accumulate in the body. They evidently accumulate because of their low solubility and resulting peculiar conditions of absorption and excretion. That this is more likely to occur in disease of the circulation than under normal conditions is self-evident, and the fact that much larger doses of digitalis are tolerated in health than in disease probably has the same explanation. There is one other point: digitalis, like a number of other substances, such as the antipyretics, for example, meets a greater sensibility in diseased than in healthy organs.

The physiology of the severe toxic symptoms has been repeatedly investigated. Since Traube made his famous animal experiments, investigators have been chiefly occupied in studying the relation of the pneumogastric nerve to the intoxication. We can only expect light on this subject from an entirely new series of experiments, performed with much more care than has previously been the case and in connection with clinical observation. But this question is, perhaps, by no means so vital as is sometimes supposed; it does not affect the size of the therapeutic dose of the remedy. It is quite true that the pulse-rate falls after the use of digitalis; that is, it approaches the normal in proportion as the force of the heart is restored. And it is not surprising that the rate falls slightly below the normal; the phenomenon is observed after any pronounced effect on the heart. Severe degrees of retardation undoubtedly indicate a more profound intoxication than it is desirable to produce in man; but in disease such a state of intoxication is sometimes brought about by small doses, probably because the sensitiveness of the organ is increased in disease.

The *indication for the administration of digitalis at the bedside* is the existence of profound cardiac insufficiency, the second stage of the condition in the sense explained on p. 494. The symptoms have already been given. They consist, in the main, of abnormal blood distribution, diminution in the quantity of the blood, lowering of the tension in the arteries, and passive congestion in the veins. These phenomena are due to peculiar functional disturbances of the heart muscle, which in many cases are capable of recovery. Sometimes rest in bed alone suffices to restore the muscle to its normal condition. In other cases this cannot be achieved without digitalis. These two facts are important and affect our judgment not only of digitalis, but of the nature of cardiac insufficiency also.

If a patient of this kind is given digitalis, a change soon takes place in his condition—at least in favorable cases; that is, after an interval, which varies according to circumstances, but is rarely more than twenty-four to thirty-six hours. The dyspnea and the oppression in the hepatic region diminish; the action of the heart becomes slower, more regular, and uniform; the quantity of urine increases; the edema begins to subside; and at night the patient regains his rest. All these symptoms continue to improve during the days immediately following, and so the patient's condition gradually returns to that of compensation. How long this lasts depends on a variety of circumstances: the severity of the disturbance, the gravity and stage of the cardiac lesion, and the efficacy of the preparation administered.

The success of digitalis depends, above all, on the state of the heart muscle, the most important condition being that it shall contain a sufficient quantity of substance capable of regeneration that can be influenced by the peculiar action of the remedy. The causes in a case of the disease practically do not affect the action of digitalis at all, except in

so far as the heart muscle is in some conditions more severely affected than in others.

A question of great importance and one that has been frequently investigated is the *comparative efficacy of digitalis in the different forms of cardiac disease.* We began with its classic and most frequent form, insufficiency of both ventricles, such as occurs in disturbance of compensation from mitral disease or in those processes which involve the entire heart. Unquestionably, the effect of digitalis is most certain in this form, provided the necessary conditions for its successful action are present in the myocardium. Also the drug is, at least, of great use in many cases in which the cardiac insufficiency is due predominantly, if not exclusively, to weakness of the right ventricle, as, for example, in pulmonary emphysema. It is true that this latter statement has been called in question by a most weighty authority.*

But the question probably assumes a somewhat different aspect in cases of insufficiency involving chiefly the left heart.

From time immemorial the prognosis of compensatory disturbance from aortic disease has been considered much more unfavorable than that of a mitral lesion. I am not called upon to enter into this question. There is no doubt that in these cases the injury from the dilatation of the arteries and the peculiar anomalies that obtain during diastole play an important part. When it is remembered that digitalis is often very unreliable in its action in cases of heart weakness due to arteriosclerosis, the thought naturally suggests itself that the drug is less efficient when the left ventricle is chiefly involved. This opinion has, in fact, frequently found expression. As was shown on p. 494, insufficiency of the left side of the heart not infrequently produces a characteristic picture: edema and hepatic congestion are not marked, but the dyspnea is a pronounced symptom and, as the weakness of the left ventricle is often only relative because the condition is partly due to arterial processes, the blood-pressure may be high.

In such cases digitalis, as a matter of fact, sometimes fails. But even in these cases I have occasionally seen brilliant results, and other physicians even go so far as to maintain that digitalis is of little or no use when the right heart is chiefly affected. The question whether the effect of the drug is less when individual portions of the heart are insufficient than when the contractions of the entire heart are inadequate is by no means decided. It is a very complicated question, and in approaching it in this way the greatest caution is necessary. In these cases of relative insufficiency of the left heart with high arterial pressure it is probably true, as Romberg quite correctly points out, that the heart, even when it exerts itself to the utmost, has reached the limit of its functional power. Besides, it must be remembered how often digitalis is improperly employed and the failure ascribed to the condition of the heart, although the fault lies in the way the drug was prescribed. To decide this point we need a large number of accurate observations instead of such indefinite data as the "impression" or the "personal experience" of individual physicians.

It is, nevertheless, interesting that such a physician and observer as Traube † advises that, in cases of high arterial pressure, digitalis be reinforced by the preliminary exhibition of laxatives.

He insisted on the curative effect of diminishing the quantity of blood. Perhaps he was right; but it is also possible that the dilatation of the intestinal vessels prevents too great a rise of the blood-pressure, so that the digitalis is able to exert a powerful influence on the heart muscle.

In these conditions of cardiac insufficiency with comparatively good tension in the pulse—mostly cases of arteriosclerosis and contracted kidney—some hesitation is felt in giving digitalis on the ground that a still further increase of the pressure might cause cerebral hemorrhage.

* Leyden, "Deutsche medicinische Wochenschrift," 1881, No. 25, 26.

† Traube, "Beiträge," vol. iii, p. 165.

It would, of course, be desirable to know whether cerebral hemorrhage really does occur with any frequency after the use of digitalis. Occasionally I myself have seen it occur. But further observations are necessary on this point.

There is no doubt that occasionally there may be some danger in improving the action of the heart. Hemorrhages have just been referred to. In addition, clots may be swept out of the heart and thus favor the production of emboli. If there is any reason to fear such a complication, the probabilities of the case must be weighed and due caution observed accordingly. Only I wish to warn the reader not to allow a groundless fear to prevent him from treating a case of cardiac insufficiency with digitalis. It is not an indifferent remedy. The physician's art consists in carefully weighing and correctly judging *all* the conditions present in an individual case. Arteriosclerosis is the source of many problems, and one of them is the use of digitalis. Next to the cases with high pressure are those with slow pulse, which, as we shall see, are usually cases of coronary sclerosis. Retardation of the pulse has been assigned such an important place in the action of digitalis, chiefly, no doubt, since Traube's work was published, that the question has been seriously asked whether in patients who, on account of their disease, already have an abnormally slow pulse, the remedy may be used at all. But the causes of this bradycardia are obscure and we know nothing positive about the connection between the action of digitalis and retardation of the pulse; hence we may as well dispense with theoretic discussion. Experience at the bedside undoubtedly teaches us that digitalis is useful in these cases also when the arteries are insufficiently filled and the veins are engorged. In fact, the pulse-rate sometimes increases in such cases.*

A few words about the *contraindications to the administration of digitalis* appear to be necessary. Unfortunately, digitalis has the undesirable property of producing disturbances of the gastric function, in some individuals even early in its administration. But occasionally a patient suffering from cardiac weakness and gastric symptoms is found who experiences relief from the latter when he is put on digitalis; that is, the function of the abdominal organ improves as the circulation is regulated. Other patients with gastric symptoms, however, especially when they occur independently of the circulatory disturbance, refuse to tolerate the remedy from the beginning; others have to give up its use after taking the smallest dose: nausea, belching, and vomiting of such severity develop that the physician is forced, at least, to cease giving the remedy by the stomach. Occasionally enemata with digitalis are tolerated for some little time longer, but not infrequently even enemata prove unbearable.

Dangerous rise of the blood-pressure has already been discussed.

It is not a good sign when the administration of digitalis is not followed by an improvement of the symptoms; but it would be wrong to lose courage at once. In such cases the remedy should be discontinued for a time,—two weeks, according to the present custom, although the experienced physician will often find it necessary to change the length of this interval according to circumstances,—after which the administration is resumed, if necessary, in a different form.

It may also be well to get the drug from a different source, bearing in mind the conditions detailed above as influencing the efficacy of the drug.

* Compare Penzoldt, "Münchener medicinische Wochenschrift," 1886, No. 42.

If the remedy again proves ineffective, a third attempt should certainly be made. No general rules can be given as to how long an interval should be allowed to elapse between attempts nor about the size of the dose; in fact, it cannot be repeated too often that the successful use of digitalis cannot be learned from books.

If the remedy fails after the third attempt, the outlook, in the great majority of cases, is unfavorable. As we have seen, the virtue of digitalis resides in a peculiar effect it has on the heart muscle, the nature of which is still unknown. If the drug fails to produce this effect, the muscle has probably ceased to be susceptible of improvement because of some irreparable structural disturbance. This is the universal tacit opinion, and is probably correct. But we have no conception whatever what the nature of a structural change in the muscle can be that renders it susceptible or non-susceptible to digitalis.

Two things must be remembered: the first is never to discontinue the administration of digitalis until the appropriate doses really have been utilized. It not infrequently happens that the suitable or necessary dose is found to be larger than the generally accepted dose of the drug.

Traube * noticed this and I myself have had the same experience. In chronic cases in which the remedy has been given repeatedly there is no doubt that enormous doses are sometimes of use. I prefer not to give any special directions; we must feel our way cautiously, keeping a close watch on the pulse.

Not infrequently good results are obtained by giving the remedy in small doses for a considerable length of time. This method is particularly useful in patients with weakness of the cardiac muscle, such as results from myocarditis, for example.†

Although the prospect of influencing the heart muscle by medicinal agents is, generally speaking, unfavorable if the administration of digitalis is without effect, the other heart stimulants presently to be mentioned may, nevertheless, be tried. There are said to be isolated cases in which these drugs have been known to produce a result after digitalis had failed, but it would be a mistake, in my opinion, to give them before trying digitalis, for in the great majority of cases the latter drug is our most effective weapon.

In *administering digitalis* we usually employ the crude preparation and have to accept its undeniable disadvantages because these disadvantages, in my opinion, at least for the present, represent the lesser evil as compared with the employment of the active agents. The leaves of digitalis are given in the form of an infusion (1 : 150 without dilution). A tablespoonful of this is given every two hours until one gram (15 gr.) has been used during the day. From two to three grams are given in this way. It is now often preferred to give the leaves themselves in the form of powder, 0.1 gm. (1½ gr.) or 20 pills to the gram (¾ gr. to each pill), two powders or pills making a dose. Generally speaking, adults should receive 1 decigram ([0.1 gm.—Ed.] 1½ gr.) of the powdered leaves three or four times a day.

The infusion is weaker than the pure drug; on that point opinions are unanimous. Accordingly, it is also less toxic; it is better borne

* Traube, "Beiträge," vol. iii, p. 220.

† Compare Naunyn, "Therapie der Gegenwart," May, 1899. Groedel, "Congress für innere Medicin," 1899, p. 283. Kussmaul, "Therapie der Gegenwart," January, February, 1900.

by many patients than the drug itself. The infusion is rather apt to undergo decomposition.

Older children may be cautiously given the same doses as adults; for younger children (between six and ten years of age) the dose should be one-half, and in still smaller children one-quarter. The effect of the drug must be carefully noted, and we must always feel our way; for it is impossible to determine the size of the necessary dose beforehand in every individual case.

There are, however, certain guides that will assist one in administering the remedy. The individual physician's habit and personal preference, of course, play an important rôle. The man who knows how to handle digitalis—which is the essential thing—will get results no matter what method he uses.

Personally, I use the infusion when I wish to produce a slow and feeble effect, and give pills or powder when a definite, powerful effect is to be produced rapidly.

When the drug is to be given for some time in small doses, the above-mentioned pills or the tincture of digitalis may be employed; of the latter, 25 [U. S. P., 33 minims—Ed.] drops correspond to 0.2 gm. (3 gr.) of digitalis. I have had no personal experience with the preparation [which best represents the crude drug, since some of the active principles, like digitoxin, are insoluble in water—Ed.], but I know eminent physicians who like to prescribe it with iron, for example. Many patients do not tolerate the administration of digitalis by the mouth; it frequently produces gastric symptoms, which are probably due in part to the local action of the drug. In such cases digitalis sometimes has to be administered per rectum, somewhat after the following formula:

℞. Infus. digitalis.....2–150 c.c. (30 gr.–5 oz.)
Muc. acaciæ..... 50 c.c. (2 oz.).
M. To be divided into four doses, of which two or three are administered in a day.

Böhringer's preparation of digitalinum verum may be used. Romberg recommends hypodermic injections of 1 to 3 mg. ($\frac{1}{60}$ to $\frac{1}{20}$ gr.) in a 10 per cent. alcoholic solution, two or three times a day. The injections are painful. The needle must be introduced deep into the corium or into the muscle.

Digitoxin in the form of Merck's tablets, 0.25 mg. ($\frac{1}{240}$ gr.), is also recommended. The dose is one tablet two to four times a day, and from eight to twelve tablets in all. I have also seen good results from these tablets.

Numerous drugs have been recommended as *substitutes for digitalis*, and it cannot be denied that in a purely pharmacologic sense, a number of substances closely resemble in their action the constituents of digitalis. I consider it my duty, however, to insist once more that not one of these remedies is in any sense capable of taking the place of digitalis.

Among these substances **strophanthin** is undoubtedly the one that is most commonly used. It is employed almost exclusively in the form of tincture of strophanthus, an alcoholic extract of the seeds of *Strophanthus hispidus* [an inferior variety, not admitted in U. S. P.—Ed.] and *Strophanthus Kombé* [1 to 10—Ed.].

The dose of the tincture is five to ten [minims, U. S. P.—Ed.] drops three times a day, and two to five drops for children. The remedy should be regarded rather as complementary to digitalis than as a substitute for it. I would use it instead of digitalis only in case the latter were not well borne on account of severe gastric symptoms. But when

it is desired to keep the heart slightly under the influence of digitalis for some time, tincture of strophanthus is a very excellent preparation. The remedy is also eminently suitable in the numerous cases in which, while there is no strict indication for the administration of digitalis, that is, no cardiac weakness in the sense used above, the physician, nevertheless, wishes to improve the strength of the heart either for a certain definite purpose or to enable it to bear up against certain strains. In these cases excellent results are sometimes seen to follow the administration of strophanthin. Pharmacologically the effect of the drug on the heart is exactly the same as that of digitalis, that is, it increases the function of the ventricular muscle.*

Occasionally, although it is true very rarely, tincture of strophanthus causes nausea, vomiting, and diarrhea. When these symptoms appear, the drug must be withdrawn.

[A. Fraenkel's claim that ¾ to 1 mg. of Boehringer's strophanthin will bring on digitalis effects in a few minutes deserves further investigation.† Strophanthin, like digalen, is well adapted to intravenous injections. Both preparations are put up in sterile tubes suitable for a single dose each. In severe cases of heart weakness they deserve thorough clinical study.‡—Ed.]

The remaining "substitutes" for digitalis, for example, *adonis vernalis* [not official, U. S. P.—Ed.], *convallaria*, *spartein*, *scilla*, can practically never be expected to produce a better result than digitalis. Personally, I have had experience only with *infusum adonidis vernalis*, 1 : 15. This has *sometimes* worked as well as digitalis, but never better. [Strychnin is used to a very wide extent in all forms of heart disease, but without sufficient pharmacologic warrant. Whether the drug acts as a general or vasomotor stimulant or, as Cabot intimates, chiefly by suggestion, should be determined by careful investigation.—Ed.]

Diminution of the velocity of the blood and simple cardiac weakness are not to be regarded offhand as identical. That the vessels are largely concerned in the production of circulatory disturbances is in itself probable, and had been surmised even before Romberg's investigations established the proposition on a firm basis. Previously it unconsciously influenced methods of treatment, and many conditions of enfeebled circulation were treated with measures which, as later investigations show, not only stimulated the heart, but, indeed, predominantly affected the blood-vessels. Most of these measures appear to influence heart and vessels at the same time, and in this respect resemble nerve stimuli,§ which formerly were believed to act exclusively on the blood-vessels. As yet we have no accurate knowledge that would enable us to determine how much of the alteration in the circulation ought to be attributed to the heart and how much to the blood-vessels.

It appears, therefore, that the conditions we have to deal with in using the substances presently to be mentioned are quite different from the conditions present when drugs of the digitalis group are employed. Contraction of the vessels forms at most an insignificant part of the action of the latter, and for that reason the increase effected in the work of the

* Popper, "Zeitschrift für klinische Medicin," vol. xvi, p. 97 (von Basch's laboratory).

† "Congress für innere Medicin," 1906.

‡ See Hatcher, R. A., "The Pharmacology of Digitalis," "Journal of the American Medical Association," December 22, 1906.

§ Grossmann, "Zeitschrift für klinische Medicin," vol. xxxii, pp. 219 and 501.

heart throws much less strain on the organ than if the peripheral resistance were increased at the same time. But if the diminution of the arterial tension is chiefly due to paralysis of the vessels, remedies which combat vascular paralysis are evidently the most suitable, and the incidental stimulation of the heart is merely an additional advantage. On the other hand, if the diminished quantity of blood in the arteries is due chiefly to insufficient contraction of the heart, a drug which should chiefly stimulate the vessels would make it more difficult for the heart to empty itself and would, therefore, be not only unsuitable, but even directly harmful.

Cardiac weakness associated with paralysis of the vessels is the chief indication for the use of cutaneous irritation, which is discussed on p. 589, as this procedure chiefly affects the vasomotor tone. The best medicinal agents are the following:

Camphor is administered subcutaneously or by intramuscular injection whenever its immediate effect is necessary; in all other cases it may be given by the mouth. Hypodermic injections are painful and, therefore, unpleasant to the patient; nevertheless, the method is not to be condemned, and in many cases it is possible that the pain itself has some desirable effect on the circulation.

For administration by the mouth we use camphoræ tritæ [camphor pulverized by rubbing with a little alcohol—Ed.] in doses of 0.2 gm. (3 gr.) with sugar, three or four times a day; or for small children, in an emulsion with mucilage and syrup, as in the following formula:

R. Camphoræ tritæ ..... 1 gm. (15 gr.)
Muc. acaciæ .....
Syr. simpl .....āā 20 c.c. (5 dr.)
Aquæ destill. ..... 150 c.c. (5 oz.).
M. S.—One tablespoonful every two hours.

For hypodermic injection we use the 10 per cent. officinal [German—Ed.] camphorated oil or a 10 to 20 per cent. solution with oil and ether in the proportion of 6 : 4. From 0.1 to 0.2 gm. (1½ to 3 gr.) may be given every quarter or half-hour, and numerous doses a day. If the injections are given too frequently, unpleasant fat emboli in the lungs occasionally result.

Camphor is the type of substances that improve the action of the heart and stimulate the vasomotor centers; hence if the drug is to benefit the heart, the organ must still possess a certain degree of functional capacity. This remedy not infrequently saves life, especially in cases in which the circulatory disturbance is of brief duration.

The relation existing between the action of the drug on the heart and that on the vessels we are as yet unable to decide, nor is this the proper place to discuss the possible theories. As a matter of fact, camphor does not act exclusively on the vasomotor nerves, so that the drug is not injurious to a weak heart; at least I have never seen it do any harm, nor have I ever seen any statements to that effect.

Musk (moschus) appears to resemble camphor in its action. Personally, I have not had much experience with the drug, but it is recommended by excellent physicians, even for cases in which all other remedies have failed. Musk is administered internally in powders of 0.05 to 0.2 gm. (1 to 3 gr.) every half-hour, or in the form of tincture of musk, 30 to 40 drops, frequently repeated. [Dose of U. S. P. tincture, ½ to 2 drams.—Ed.]

Ether administered subcutaneously or by intramuscular injection in the dose of 1 gm. (15 gr.) (see Camphor) is regarded by most physicians as a heart stimulant. Its action is rapid and transitory, and ether injections are, therefore, used chiefly when haste is necessary, the dose being repeated at short intervals.

These injections are painful and I cannot help thinking that in the use of this drug also pain has something to do with the effect produced; theoretically, that would explain why ether raises the blood-pressure, an effect which is otherwise difficult to understand.

Wine is quite generally employed in the acute disturbances of the circulation which are often present in collapse. The strong wines of the south, champagne, and old Rhine wine, as well as cognac, enjoy a special reputation for this purpose. In common with most physicians I cannot help believing that the ingestion of large quantities of one of the above-mentioned wines increases the amount of blood in the arteries and raises the blood-pressure, especially in acute disturbances.

The mode of action, however, is equally obscure. What is the active substance—is it the alcohol or is it the other ingredients contained in the wine? As yet it has been impossible to demonstrate that alcohol has any definite action on the heart. It is true that the "exact" observations, so far as I can see, have again been made exclusively on individuals with sound hearts, and, as I have frequently remarked, such observations do not tell us anything positive about the behavior of diseased hearts. In Wendelstadt's recent paper * from Binz's laboratory, which deals with other aspects of the action of wine, it is shown that the effect of the drug on healthy tissue is quite different from its effect on diseased tissue.

Large doses of alcohol cause dilatation of the vessels, paralysis of various portions of the central nervous system, and a reduction of many of the functions of the body. Hence even a patient should not be given too much wine, say several bottles of strong wine a day; although it is true that many patients can take much more wine without any bad results than persons in good health.

All these things are in urgent need of further investigation. For the present I think we physicians will have to adhere to the view that the above-mentioned varieties of wine improve the action of a diseased heart and strengthen the circulation.

Caffein also stimulates the heart as well as the blood-vessels. Theoretically, it may, therefore, be included among the substances of this class and is also most frequently used to satisfy the indications, which have been sufficiently explained. Coffee is undoubtedly the best form in which to administer caffein. A cup of coffee prepared from 20 gm. (5 dr.) of good coffee-beans corresponds to a dose of 0.1 to 0.2 gm. (1½ to 3 gr.) of caffein. Certain double salts of the alkaloid are now chiefly used, especially caffein and sodium salicylate or sodium benzoate, containing respectively 50 per cent. and 45 per cent. of caffein, in doses of 0.25 to 0.6 gm. (4 to 10 gr.), three or four times a day, either by the mouth or by hypodermic injection.

I would advise that freshly prepared hot coffee be given the preference over ready-made preparations, and recommend its use especially in the treatment of acute cases of failing circulation.

* Wendelstadt, "Pflüger's Archiv," lxxvi, p. 223.

Coffee has also been recommended for cases of simple cardiac insufficiency,* but evidently has not proved particularly useful; at least it has been given up by most physicians, which is not surprising in view of its marked action on the vasomotor nerves.

None but the most important and well-tried remedies have been mentioned in the preceding pages. This has been done purposely, because the best and most eminent physicians find that they answer every purpose, and there is no doubt that constant experimentation with new drugs, unless they are unquestionably good, does the patient no good and distinctly injures the physician. Outside of Germany, particularly, numerous other remedies are in vogue; but I purposely refrain from mentioning them.

Narcotic Remedies in the Treatment of Heart Disease.—Quite frequently narcotics are simply indispensable in the treatment of heart patients because they alleviate the horrible conditions of heart pain, heart fear, and dyspnea, without in the least injuring the patient.

The object in giving such drugs, therefore, is to mitigate the patient's sufferings, and it is quite immaterial whether, by doing so, the so-called causal indication can be satisfied at the same time or not. A certain length of time often has to elapse before the causal indication can be fulfilled—quite often we are altogether powerless to satisfy it—in short, there are many conditions in which it would be simply inhuman to abandon the patient to his suffering.

I always advise that *morphin* be tried first. It is the most reliable remedy for any patient who is not used to it and is willing not to resist its action. It is also relatively free from danger in conditions of cardiac weakness, because it does not influence the circulation in the least for a long time; at least, the dilatation of the cutaneous vessels which takes place in normal individuals does not, so far as we now know, materially affect the circulation as a whole.

Morphin is most effective when given by hypodermic injection. The beginning dose should never be more than 1 cg. [0.01 gm.—Ed.] ($\frac{1}{6}$ gr.), and the remedy should be given only when immediate relief is imperatively demanded. Morphin injections may also be given unhesitatingly in any temporary disturbance that is not repeated at frequent intervals; but as regards chronic conditions, it must be remembered that the sword is two-edged. There are persons who cannot resist the fascination of morphin injections, either because they suffer terribly without them or because they have no will power. In such cases the physician often cannot be exonerated from the charge of having caused morphinism. I do not say that injections of morphin must be avoided altogether in chronic, frequently recurring conditions; for there are cases of angina pectoris and cardiac asthma, for example, in which the drug is indispensable. *But whenever it is at all possible, an attempt should first be made to give morphin internally* in the form of a powder or of a solution with aqua amygdalarum, beginning with a dose of 1 cg. ($\frac{1}{6}$ gr.). The various preparations of opium are also given for this purpose.

Codein (phosphate), 0.02 to 0.03 gm. ($\frac{1}{3}$ to $\frac{1}{2}$ gr.), may be given instead of morphin. I have not found that it possesses any special advantages for our purpose. It is especially adapted to cases with marked irritative cough, provided an immediate subcutaneous injection of morphin is not required.

*Riegel, "Berliner klinische Wochenschrift," 1894, No. 19.

If, in patients who suffer at night, insomnia is a more prominent symptom than dyspnea and anxiety, a dose of from 1 to 1.5 gm. (15 to 25 gr.) of trional or sulphonal in the evening may be recommended and sometimes proves extremely useful in combination with morphin. With regard to the other substances of the alcoholic group,—paraldehyd, amyl hydrate, urethane, chloral hydrate, and chloralamid,—a certain caution is undoubtedly necessary when they are given in large doses.

APPLICATIONS TO THE PRECORDIA. CUTANEOUS STIMULATION. DERIVATIVE MEASURES.

Whenever the heart action is excited and accelerated, cold compresses or the application of an ice-bag to the precordia should be tried. To avoid pressure on the precordia it is well to have the ice-bag suspended from barrel-hoops the ends of which are placed in the bed. The chest-wall should always be covered with a thin piece of linen to prevent immediate contact of the skin with the ice-bag. Small pieces of ice should be used, and the bag should never be filled quite full; also the ice must be frequently renewed. [If the bag is not heavy, suspension is not necessary. In the case of restless patients the ice-bag, in an outer bag, can be pinned to the shirt.—Ed.] Leiter's coil, made of rubber or metal and of a suitable form, may also be used to advantage.

The effect of the application is almost always to render the heart action slower and more uniform and to diminish or relieve altogether any distress that the patient may have suffered on account of the abnormal force of the heart or on account of hyperesthesia.

Two things must be considered in explaining the action of the procedure. The first of these is the cooling of the heart's substance. We know that the number of the muscular contractions diminishes as the temperature falls. It appears doubtful, however, whether the cooling of the skin extends as far as the organ itself. I have been unable to find any reliable observations bearing on this point. From the analogy of the gastro-intestinal canal, it might be assumed to be the case; but reasoning by analogy in the case of organs so differently situated does not advance the subject. The second thing to be thought of is the cutaneous reflex. As the blood-vessels are speedily paralyzed by cold, it does not appear altogether impossible that this local hyperemia may have something to do with the action of the ice-bag. On this theory it would represent a true derivative measure, if we define the latter as the production of a local hyperemia for the purpose of relieving congestion in some other organ. The physiologist will probably laugh at this notion, and from his standpoint he is quite right; for, if the physiologic laws of blood distribution apply strictly to the conditions found at the bedside, it would be senseless to expect to relieve the congestion in a distant organ supplied by an entirely different set of vessels by means of local hyperemia or by the abstraction of fluid. But since it may be assumed that cooling or warming the skin diminishes the amount of blood in the diseased organ, and as the explanation of this fact on the assumption of a reflex is most unsatisfactory or at least altogether unproved, the thought naturally suggests itself that tissues affected with certain forms of disease give up part of their blood more readily than all other (healthy) tissues, and that such tissues, therefore, permit the accession of fluid to the part of a body which has been artificially rendered hyperemic. Observations might decide this point.

There are a good many people who cannot bear the application of cold to the precordia. In such cases warm applications often act very favorably—either a simple Priessnitz compress or Leiter's coils filled with warm water.

Whether cold or heat will be more suitable cannot be determined beforehand any more than in persons suffering from inflammatory con-

ditions of the abdomen. It is always best to experiment, and to consult the patient's wishes as fully as possible.

Derivative measures are well adapted to combat pain, particularly about the heart, and the same effect is produced by local bleeding. Mustard poultices, mustard plasters, leeches, or Heurteloup's artificial leech may be used, applied either to the precordia itself or to the left axilla.

Whether or not *large quantities of blood shall be abstracted* must be decided on similar principles. We all know that at different periods in the history of medicine venesection played a very important rôle in the treatment of pathologic conditions of the heart, as in that of most diseases. Now the method has been almost entirely given up, and there is no doubt that the aversion we entertain toward venesection at the present time is exaggerated. For my part I cannot believe that a method which survived so long in the hands of able physicians is altogether worthless.

If there is any sense in venesection, we must assume some change in the physiologic laws of blood distribution and regulation of the circulation. This, indeed, seems probable; at least the supposition finds support in a number of observations. It really seems that in cardiac insufficiency the heart in many cases is very sensitive to any increase in the amount of inflowing blood and responds very readily if the quantity is diminished. On this theory one would be justified in expecting good results from venesection, and it also enables us to understand the value of other well-tried measures which render individual portions of the body hyperemic. Traube long ago showed the advantage of rendering the intestine hyperemic in such patients by means of an active purge, and similarly hot foot- and leg-baths at 40° to 45° C. (104° to 113° F.) often bring magic relief in morbid conditions in which it must be assumed that the heart is finding it difficult to handle a large quantity of blood.

Nevertheless, the conditions are somewhat different in the case of *cutaneous hyperemia* and *inflammation produced by irritating substances*, such as mustard poultices, mustard plasters, embrocations of various kinds, painting with tincture of iodin, blisters, cauterizations, and issues. These measures, it is true, produce a local hyperemia of the skin, limited to a very small and circumscribed spot. There is no doubt that cutaneous reflexes and the absorption of irritating substances have some share in the effect produced. An explanation is as yet out of the question, but clinical experience so far justifies the conclusion that the above-mentioned procedures are undoubtedly useful, not only in conditions of sudden failure of the circulation, but also to combat oppression and simple cardiac weakness. I strongly recommend that they be tried in every case of this kind, particularly well-prepared mustard plasters to the feet, mustard-baths, and brushing the soles of the feet with a hard clothes' brush. How far these methods act on the vessels by direct pressure and how far they improve the activity of the skin has, so far as I know, never been investigated. As clinical experience recommends them chiefly for acute cases of failing circulation in the production of which paralysis of the vessels plays a large part, it may be assumed that they act chiefly on the vasomotor apparatus.

BATHS AND OTHER HYDROTHERAPEUTIC PROCEDURES EMPLOYED TO STRENGTHEN THE HEART.

How and in what form baths may be used for purposes of cleanliness and the care of the skin in heart patients has been explained. In this section we have to deal with the use of hydrotherapeutic measures

for stimulating and strengthening the action of the heart. These procedures may act as cutaneous stimulants; but, so far as I can see, we have not as yet any accurate knowledge of how the stimulation of cutaneous nerves influences the action of the heart and vessels. The effect on the action of the heart would depend on the contraction of some vessels, the dilatation of others, and possibly on an increase in the total cross-section of the blood-channels. The behavior of the coronary vessels would have to receive special attention, and after that the heart itself. Sensory stimuli, as an interesting investigation carried on by Grossmann * in v. Basch's laboratory has shown, are capable of increasing the action of the heart even aside from their influence on the vasomotor tone, namely, by directly acting upon the heart. Marked constriction of the blood-vessels in certain regions of the body—in man the vessels supplied by the splanchnic nerve are probably the most important in this respect—exercises the left ventricle and may do a great deal of good if the heart is still capable of responding to an increased demand. Whether it may be employed therapeutically in a given case depends on the same considerations that determine the advisability of muscular movements. If the action of the heart can be directly increased through reflex nerve action, another valuable means of treatment is thereby afforded; only the conditions necessary to develop the effect on the heart are very complicated and are still in need of careful investigation. We have no detailed knowledge whatever, and there is a wide field for accurate observation. But such observations must be made with a full knowledge of our present opinions in regard to the physiologic conditions of the circulation.

The practical application of hydrotherapeutic measures must be guided solely by the experience gained at the bedside. To a large extent actual treatment is independent of our theoretic views in regard to the action of these measures. Although this theoretic knowledge is still relatively slight, the experience that has been accumulated in regard to the kind of hydrotherapeutic measures and their mode of application in diseases of the heart is already quite respectable; it points chiefly to the *use of baths with water containing carbon dioxid and salts.*

The stimulation exerted on the skin by carbon dioxid causes a rise of the arterial pressure when the heart is weak. It is probable that the effect is produced chiefly through vasomotor stimulation. In other words, an extra demand is thrown on the heart, and the procedure, therefore, constitutes a heart exercise. If the heart is to be benefited, it must still have at its disposal a certain degree of reserve force. A heart with passive dilatation (due to congestion) frequently becomes smaller under the influence of the baths. This fact and the general impression gained from the effect of carbon dioxid on the heart no doubt strongly suggest that the heart muscle is directly strengthened; in other words, that the effect of carbon dioxid to a certain extent corresponds to that of digitalis and of sensory stimuli. But here, again, there is need of more observations to clear up the conditions.

In practice these baths are employed either to improve mild grades of cardiac insufficiency or to insure, as it were, the permanence of the effect produced by digitalis—indeed, in a few cases the use of carbon dioxid baths has been followed by success after digitalis had failed.

But carbon dioxid baths can in no sense be said to rival digitalis in their action on the heart. Although there are certain points of contact between the two remedies, digitalis, nevertheless, is and remains

* Grossmann, "Zeitschrift für klinische Medicin," vol. xxxii, pp. 219 and 501.

the only proper remedy in cases of marked cardiac insufficiency with abnormal blood distribution. It seems to me improper to begin by giving baths in such cases; by doing so discredit is cast on the procedure and the patient is injured. But they are unquestionably of great value when it is desired to exert a sustained effect, not to mention the milder forms of cardiac weakness. In addition they often have a very favorable effect on many sensory disturbances of various kinds, including angina pectoris in nervous and arteriosclerotic individuals.

The manner in which the heart reacts to the procedure must be tested in every individual case in order to determine whether it will be tolerated. This cannot be known beforehand, and caution is, therefore, necessary under all circumstances. The treatment should always be begun with weak baths. The general condition must receive due attention; the patient should feel perfectly comfortable and well during the bath, not anxious or oppressed. The dyspnea, if there is any, should not be increased by the baths; the tension of the pulse should be improved; quite often a retardation of the latter is effected. The baths must never be allowed to affect the action of the heart unfavorably; if such an effect is produced, the bath must be modified or, if that fails, omitted altogether. The effect of the bath depends on a great many different conditions, such as temperature, percentage of carbon dioxid and salt, and the duration of the bath. All these factors can and must be carefully adapted to the individual case. Proficiency in the selection of appropriate baths to suit an individual case is only gained by practice and experience. It is practically impossible to lay down any more rational principles; the method is purely empirical, and its proper use, therefore, imperatively demands experience on the part of the physician.

There are a number of *natural carbonated springs* which contain variable quantities of carbon dioxid gas, besides many kinds of salts. Just how these salts modify the effect of the bath we do not as yet know. As salt solutions in themselves exert a stimulating influence on the cutaneous nerves in a variety of ways, it is quite natural that they should increase the effect of the carbon dioxid. Not infrequently simple brine baths (Soolbäder) are used to obtain the same effect.

Among the carbon dioxid baths those which are most frequently visited by heart patients are Nauheim, Oeynhausen, where there are thermal waters, and Cudowa, a cold spring. There is no doubt that the physicians who practise in these places, especially in Nauheim, have had a very large experience in the treatment of heart patients, and this I regard, *a priori*, as a strong argument in favor of sending patients to these places. It is needless to say that the accommodations generally are especially good in all the more popular baths.

Hamm and Werne in Westphalia, Kissingen, Marienbad, Homburg, Tarasp, Langenschwalbach, Driburg in the Teutoburg Forest, Franzensbad, St. Moritz, Pyrmont, Reinerz, Rippoldsau, and other springs also contain carbon dioxid in combination with a variety of salts. Some of these are already being used for the treatment of heart patients, and there can be no doubt that equally good results could be obtained in diseases of the heart with these waters as at the above-mentioned places, and for many reasons—such as the distance from a patient's home and others—some of these places are unquestionably better adapted for some patients than Nauheim and Oeynhausen.

Whenever possible, the patient should be sent to one of the above-

mentioned springs; for a variety of reasons a course of treatment at one of these regular spas is much superior to treatment with artificial carbon dioxid baths, provided, of course, a critical examination is made of the method to be employed for the individual patient. For only certain very definite conditions are amenable to treatment with carbon dioxid baths, and we must absolutely avoid exposing unsuitable patients to the discomforts and expense of the journey.

In many cases, however, it is not possible to use the natural spring water because the patient cannot be sent to the health resort. In such cases *artificial carbon dioxid baths* sometimes render excellent service. It is important to have the carbon dioxid as finely divided as possible, so that the patient's skin will be acted upon by small bubbles of gas. This is best accomplished by introducing liquid carbon dioxid into the bath-water. In the case of bathing institutions the arrangements necessary to effect this should be urgently recommended, but in private practice they cannot be used on account of the comparatively high price.

To prepare a bath in a private house from 0.1 to 1 kg. ($\frac{1}{3}$ to 2 pounds) of sodium bicarbonate, with an equal quantity by weight or 0.15 to 1.5 kg. ($\frac{1}{3}$ to 3 pounds) of crude hydrochloric acid, are added to a bath of about 250 liters (60 gallons) at a temperature of 35° to 28° C. (95° to 82.4° F.). The bicarbonate of soda is thrown into the water. The hydrochloric acid is poured into bottles with narrow necks which are introduced under the water, and the acid is allowed to run out slowly, in about five minutes, on the bottom of the tub. By this means small bubbles of carbon dioxid are slowly evolved.

The firm of Sandow, in Hamburg, manufacture tablets which are very convenient for the preparation of carbon dioxid baths. With this method the gas is generated by the action of potassium sulphate on sodium bicarbonate. These baths are expensive, however,—1 Mark (25 cents) as against 40 to 69 Pfennige (10 to 17 cents), the cost of a bath prepared with hydrochloric acid,—and, besides, it is my impression that when the carbon dioxid is generated by this method the bubbles are considerably less minute.

As a rule, the temperature of the water rises as the mineral acids combine with the sodium. If this reaction takes place, the temperature must be taken again after the acid has been put in and, if necessary, the temperature reduced by cautiously adding cold water. The patient then enters the tub and keeps as quiet as possible. Owing to the stimulating effect of the carbon dioxid, cold baths are quite well borne—in fact, often better than baths at higher temperatures. The suitable temperature must be determined for each individual patient.

Salt (Stassfurt or "artificial Nauheim salt") can also be added to the baths to make a concentration of about 3 per cent.

Gradually the percentage of hydrochloric acid and carbon dioxid is increased, carefully noting the effect, and the temperature of the water lowered. At first the duration of the bath should be from five to ten minutes; later this may be extended to twenty-five minutes. At the beginning of a cure the baths should be given only every other day.

Numerous other kinds of medicated baths * have been recommended for heart patients, and it is quite possible that the effect of these baths on the circulation is similar to that of the carbon dioxid baths. That this is the case with saline and brine baths has already been mentioned,

* Matthes, "Lehrbuch der klinischen Hydrotherapie," Jena, 1900, p. 3.

and Matthes has found that oil of turpentine and ammonia act in a similar manner. It is, therefore, perfectly safe to try substances of this kind. Any substance, provided it is fresh and has a pleasant odor, is particularly useful when a suggestive effect is desired. It should be remembered, however, that, so far as we know, carbon dioxid baths are by far the most certain in their action and are better known than any others.

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DISEASES OF THE HEART OCCURRING AFTER BODILY EXERTION.

The innervation of the skeletal muscles has a very great influence on the circulation; for functionating organs always attract large quantities of blood, which are then carried to the heart. At the same time the heart also fills and empties itself more completely than under normal conditions, and the number of contractions is considerably increased.

Hence the velocity of the blood is greatly increased in other vascular areas than is the case when the body is at rest. While an inquiry into the causes of the individual factors which produce this change in the circulation is most alluring, such an inquiry would carry me too far afield. For the present purpose it is enough to know that during exercise the power of accommodation of the heart is called into action to enable the organ to take up and propel large quantities of blood, and that the number of cardiac contractions is at the same time increased. Even if the pressure in the aorta and pulmonary artery should fall,—which is probably not the case (see p. 487),—the fall would certainly not be enough to prevent or neutralize an increase in the work of the heart. We must, therefore, reckon with the fact that *any extensive innervation of the skeletal muscles increases the work of the heart.*

These matters were mentioned in discussing the factors which are, generally speaking, capable of exerting a harmful influence on the activity of the organ. Now, however, our point of view is different: what we

wish to know is what kinds of cardiac disturbances are to be regarded as directly and solely due to severe muscular exertion. These disturbances are:

Laceration of a valve, more frequently at the arterial, rarely at the venous, orifices. Our discussion does not include these conditions, as they have already been considered in this work by our colleague, v. Jürgensen.

Conditions Associated with Diminished Functional Capacity of the Heart.—These possibly include hypertrophy, which will be referred to at the end of the article.

Causes.—According to the definition, the direct cause of these disturbances must be sought in the increased demand made upon the heart on account of the muscular exertion. The quantity of blood in the ventricles is increased, and the peripheral resistance, far from being diminished, is heightened. To what extent violent movements of the body are accompanied by an unusual preponderance of these two factors is not infrequently determined by a certain accidental combination of circumstances which especially favors the occurrence of overexertion. As I have mentioned elsewhere, emotions such as anger or fear furnish illustrations, inasmuch as they interfere with the proper observance of precautionary measures. Another illustration is the irresistible desire to accomplish something out of the common, to achieve some unusual feat of strength, because in doing so the individual usually holds his breath in forced expiration, which causes an excessive rise of the arterial pressure. Examples of either of these conditions are to be found in medical practice. The literature contains numerous cases in which the overexertion is mentioned as having taken place during a moment of emotion. Personally, I have also known numerous examples of overexertion under such circumstances, as, for example, at students' duels.

Overexertion is also more apt to take place when violent muscular movements are made while the abdomen is constricted and the breast confined by the clothing, as, for example, in the case of workmen who wear a belt, soldiers with a heavy pack, and tightly laced women while dancing. The harmful factors in these cases are the overfilling of the thorax and the increase in the intrathoracic pressure, as well as the interference with the excursion of the lungs, possibly also, as specially pointed out by Sommerbrodt, inadequate vasodilator reflexes.

In these cases the strain on the heart is sometimes so enormous that one hesitates to deny the possibility of a healthy heart failing to respond to the sudden demand made upon it; the more so as the beginning of insufficiency can be artificially induced in any heart, providing the individual exerts himself sufficiently, and as in animals, which may be presumed to have sound hearts with especially good functional capacity, the same thing has been observed. Senac * found the heart completely flaccid in hunted stags. It is, therefore, in my opinion, undoubtedly possible that a previously healthy heart may give out under the strain of excessive exertion, even though of brief duration. Later I shall try to show how far clinical observations justify this theory. The objection might be urged that the animals had been ill, although there is not the slightest ground for making such an assumption.

It is much more difficult to judge whether and to what extent *moderate*

*"Traite de la Structure du Coeur, etc.," translated, Leipzig, 1781, p. 170.

demands on the heart, such as the majority of individuals under similar circumstances are able to meet without any difficulty, are capable of producing the symptoms of overexertion in a few individuals with hearts that pass for normal. Cases are, in fact, not infrequently encountered in which the heart is injured by muscular movements attended by an amount of work which, although evidently injurious to the individual, by no means appears to be excessive. Such cases must be explained by assuming some individual factor or the coincidence of several injurious influences; but there still remains the question whether many of these hearts, although up to that time they had been apparently healthy, did not exhibit some anomalies of texture even before the discovery of their insufficiency. The present state of our knowledge of the pathology of the heart does not give any positive information on this point, which is not surprising, as opportunities for making autopsies in such cases are obviously rare.

Formerly, when I was in the habit of seeing clinical patients chiefly, that is, for the most part, grave cases of heart disease, I was very much inclined to attribute the occurrence directly to well-marked disease of the myocardium when the function of the heart was found to be seriously impaired by muscular movements which were not out of the ordinary. But in dispensary work and in private practice one learns that this assumption is not justifiable or well founded in all cases. It is true that in these milder forms of overexertion of the heart the entire organism is very frequently under the influence of general injuries which diminish its power of resistance and its functional capacity. This, as Romberg points out, has a most unfavorable effect on the heart. In some cases in which muscular exertion is followed by heart symptoms there is a history of an antecedent infectious disease. Observations made on soldiers in war and peace have taught us that convalescents, various kinds of patients, and persons in a depraved state of health are exceedingly sensitive to even slight bodily movements. Malnutrition, gastrointestinal diseases, nervousness, and alcoholism injure the powers of resistance both of the individual and of the heart; great heat, bad air, and especially damp air have the same effect, nor must we underestimate the influence of care and worry.

Just how these things act on the heart is, unfortunately, not clearly known as yet, but there is no doubt that these factors diminish the resisting power of the heart, especially when several such injurious factors are operative at the same time.

The condition of the heart itself is another factor in the problem. If the organ is diseased, the slightest demands of any kind may be injurious. But this point requires no further elaboration; the reader is referred to the section on General Etiology.

We may, therefore, say that the phenomena of overexertion of the heart occur as soon as a disproportion exists between the demands made upon the heart and its functional capacity.

Symptoms.—We speak of acute and of chronic overexertion according to the length of time during which the muscular movements exert their injurious influence on the heart. In a few cases a single overexertion of the heart suffices within a very short time to bring on the symptoms of functional incapacity. But, as a rule, the process is slower and the strength of the organ does not begin to suffer until the same injury has been repeated a number of times.

We shall first discuss *the clinical symptoms in those cases in which a single violent muscular movement produces circulatory disturbances.*

During or after a single muscular exertion symptoms of dilatation and insufficiency make their appearance, consisting of dyspnea, a feeling of pressure on the chest, and frequently more or less severe pains, which are seated in the precordia and often radiate to the upper epigastrium or to the left arm. The pain not infrequently begins during the exertion, and convincingly shows the patient that something abnormal has happened. It is attended by anxiety, and the prostration is usually so great that the patients are no longer able to stand, let alone walk, by themselves, and either have to be carried or in milder cases at least supported. If the individual is alone when the accident occurs, he is forced to lie down wherever he is, or, at the best, is only able to crawl along the ground slowly on hands and knees.

Examination of the circulatory organs at once shows a great fall in the arterial pressure; the pulse is always soft and small, often irregular and unequal, usually accelerated, rarely retarded. The apex-beat is displaced outward and sometimes downward as well, but it may also be found displaced upward, and, as Martius has pointed out, it is not necessarily soft in spite of the cardiac weakness (see p. 432).

The result obtained by percussion is not always abnormal; but, as a rule, and always in severe cases, percussion reveals enlargement of the heart to the left or right or in both directions. I have been unable to discover what factors in a given case determine whether the dilatation will affect the left or the right ventricle.

Auscultation either reveals nothing abnormal or a systolic murmur; the mitral shows insufficiency of its valves. Of course, signs of mitral or aortic insufficiency may sometimes be due to a laceration of the valve.

The lungs and the abdominal organs, especially the liver and kidneys, at first present nothing abnormal; edema also is absent at first, but we shall presently see that a change soon takes place.

As a rule, the patients from the beginning look as if they were in a serious condition. I have elsewhere spoken of the anxious, sunken features seen in patients with angina pectoris. A similar expression, although less marked, is encountered in these conditions also; besides, the patients are usually cyanotic and complain of extreme lassitude. If the overexertion occurs among soldiers on the march in very hot weather, the face may be red and covered with perspiration; but more frequently it is cool and moist.

Overexertion of the heart may be immediately followed by death. For example, I recall the impressive case reported by Düms. A soldier, fearing he would not reach the barracks in time one evening, ran a long distance, part of the way over a freshly plowed field, and fell down dead in the barracks yard. At the autopsy the heart was found to be dilated and the muscle degenerated.

Fortunately, this is a rare accident, and almost equally rare are the cases of permanent insufficiency of the heart developing after a single, or a very brief, bodily exertion. Such a condition may last for a long time—weeks, months, or even years, or may bring on death at any time. The characteristic feature is that the functional capacity of the organ has suffered an injury which shows itself after any brief, violent bodily movement; death takes place with the well-known symptoms of insufficiency of both ventricles.

Cases of this kind have frequently been described,* and are particularly difficult to understand. As a rule, in these histories the individual had been for a long time, often for years, in the habit of performing, without the least injury to his health, the identical muscular movement which suddenly ends his life. In such a case one cannot help wondering whether some intercurrent condition may not have occurred to impair the functional capacity of the muscle in some chemic way, or to cause an unfavorable change in its structure. I refer to my explanation at the beginning of this section. It is a question in these very cases whether, granting that the severe muscular exertion was the final exciting cause of the fatal symptoms, the heart and the body as a whole were *absolutely healthy* before the blow fell. One cannot help having one's doubts on this point in these rare cases. In many, perhaps in most, alcohol had already developed its devastating influence. How far infectious diseases are responsible in these cases cannot as yet be determined, nor are we as yet in a position to exclude severe structural lesions in the weakened heart; for the cases so far reported are not, in my opinion, sufficiently numerous to justify a decisive conclusion because the entire heart ought to be subjected to microscopic examination before giving such an opinion, and so far we have no observations of that kind.

In a third series of cases, which are by far the most frequent and are, in fact, not rarely encountered, the cardiac insufficiency persists only a short time after the bodily exertion has ceased. As soon as the patient rests, the symptoms disappear, or at least improve, very much, and the abnormal rhythm and the dilatation of the heart also disappear in the course of hours, days, or weeks. Some of these patients are as well as ever; that is, they bear bodily exertion just as well as before. But although in some cases the course is thus favorable, it certainly is not so as a rule; much more frequently disturbances persist, at least for some time, and not rarely become permanent.

We shall discuss this point in detail as soon as the symptoms of more gradual overexertion of the heart have been mentioned.

The symptoms of "heart-strain," as has been stated, *not infrequently make their appearance gradually;* so slowly, in fact, that the patient is hardly able to tell when they began. This happens after very hard work. The condition is also observed, however, after very slight movements, such as unquestionably have no effect whatever on the majority of persons; its occurrence in a given case again depends on the disproportion between the strength of the heart and the demand made upon it. These mild, slowly developing cases of heart-strain are very frequently seen at the present time. For a number of reasons a great many people are now in the habit of taxing their muscular strength; among these reasons are the impetus recently given to industrial enterprise, the large number of working people and the resulting competition, the wide-spread popularity of athletic pursuits, which are frequently indulged in to a ridiculous excess, and, on the other hand, the practice of employing very young workmen or undeveloped girls in a great many pursuits, and the prevalence of debilitating factors, such as alcohol, sexual excesses, insufficient sleep, and the like—these things all have an effect on the prevalence of heart disease. They explain why this particular form of cardiac insufficiency is chiefly found among weakly individuals—young apprentices, domestic servants, students who are

* For example, by Fräntzel, "Herzkrankheiten," vol. i, p. 112. Zunker.

beginning to fence [or take part in other sports without proper training or fitness—Ed.]. On the other hand, old gentlemen and fat, anemic women who insist on trying to ride a bicycle furnish a large contingent. It is, of course, difficult to distinguish this form from those varieties of cardiac insufficiency which occur under ordinary circumstances under the influence of factors, such as infectious diseases and especially alcoholism, for example, which primarily reduce the resisting power of the myocardium.

The course of this form of cardiac insufficiency also may be pernicious and progressive.

A classic example is furnished by the case of the Cornish miners reported by Peacock. After a hard day's work, during which they were obliged to wield heavy hammers in a crouching position, the men formerly had to put in another hour climbing a long series of ladders in order to get out of the depths of the mine. In many, not to say most, of these miners the well-known signs of cardiac insufficiency appeared during the fifth decade and proved the cause of their death. Similar descriptions are also found in the German literature, as, for example, in the classic works of Seitz, Münzinger, Jürgensen, and numerous others.

Here, again, the conditions are extremely complicated. There is no doubt that individuals who for years had been in the habit of doing hard work, often combined with privations, have been known to die of progressive cardiac weakness. But this I think is as much as can be said with absolute certainty. I think it would be a veiling of the truth to regard it as positively settled that in these cases the muscular exertion was the only cause, and that the fibers of the heart muscle were structurally intact. Since the publication of these famous clinical histories and postmortem reports our knowledge of diseases of the heart has been considerably enlarged, and much greater accuracy and minuter technic are expected in examination. I cannot as yet bring myself to consider these observations as decisive in the above-mentioned sense, and venture to recall, for example, the interesting findings made by Nauwerck. In this case a patient showed the signs of cardiac weakness after continuous hard work. The case had been regarded as one of heart-strain, while the autopsy showed a wide-spread mural endocarditis.

From an etiologic standpoint, also, the conditions in these cases are very complicated. Although these patients were exposed to severe muscular exertion, it does not follow that this muscular exertion alone or preëminently produced the weakness of the heart and placed the organ, so to speak, in a permanent condition of fatigue. Many other factors come into play. The beer and wine habit must also be reckoned with. Not a few of these weak hearts were reported to be hypertrophic, and we know that beer-drinking, according to our present views, very frequently leads to cardiac hypertrophy. Again, many of the patients who were included in this group had never done any unusually hard muscular work. Hence in these grave cases it is quite as difficult to find the line of separation between cardiac insufficiency due to exertion and cardiac insufficiency due to *disease* of the musculature as in the milder cases now to be discussed.

Much more frequently chronic overexertion runs a mild course, and from a practical standpoint cases of this kind are, therefore, much more important.

In these milder cases the patients complain that they have noticed

a gradual loss of power in performing muscular movements. On close interrogation the chief symptoms are found to be heart symptoms—palpitation, dyspnea, a feeling of pressure on the chest. As a rule, the symptoms are complained of only during exercise, but not infrequently during rest also. Dilatation of the heart may or may not be present; disturbances of the heart action, such as have been described in the acute cases, are, as a rule, observed. Altered blood distribution, that is to say, the signs of cardiac insufficiency during rest, are not present.

Quite often the symptoms disappear very quickly if the patient stops work and gets absolute rest and good nursing. In some cases the patient recovers entirely and is later able to resume his former work without any difficulty whatever. This is certainly not the rule even in these cases. Much more frequently a certain degree of functional disturbance remains, and from this point on what I shall have to say will apply also to the course of acute strain. It is true that these disturbances vary a good deal in severity and, fortunately, they are in most cases quite slight. During rest the symptoms disappear altogether, and it may be possible for the patient to undergo muscular exertion, although the latter is apt to be attended by unpleasant sensations. Sometimes the physician is able to demonstrate dilatation of the heart by its objective signs, and the patient is made aware of it by the sense of pressure and oppression and the dyspnea that accompanies every movement of the body, so that he instinctively avoids any severe exertion. His comfort in life will depend on his ability to avoid exertion. Individuals of this kind sometimes change their entire mode of life, and that alone is often exceedingly characteristic.

In not a few cases hearts which have once suffered from an attack of overexertion, instead of reacting to subsequent demands on their functional capacity by dilatation, exhibit abnormalities of action—usually acceleration, often irregularity and inequality of the heart-beat. Sometimes the apex impulse is strong and heaving and the second sounds at the base remarkably loud. As a rule, this is accompanied by unpleasant sensations, like those referred to above, namely, pain and pressure on the chest, dyspnea, and especially palpitation and vertigo. Da Costa * has described these things very fully and has given to the condition the very appropriate name of "irritable heart" ["soldier's heart"—Ed.]. The phenomena of irritability occur in some individuals only after some special demand has been made on the functional capacity of the heart (excitement, muscular exertion). But in other cases they persist even when the patient does not expose himself to any special insult. Palpitation and acceleration of the pulse are observed in such cases and may be constant or paroxysmal; quite often there are marked and unexpected changes in the frequency of the pulse. The patients get in the habit of observing their hearts—they become *nervous*. As a result, they become more sensitive to any demand on the heart, and thus the trouble, as it were, reacts on itself and grows constantly worse.

It is not infrequently observed that in the same individual bodily overexertion is sometimes followed by the phenomena of irritability and at others by those of simple weakness and dilatation of the heart.

The *diagnosis* of insufficiency of the heart after muscular exertion is comparatively simple. Impairment of the functional capacity having

*Da Costa, "On Irritable Heart, etc.," "American Journal of the Medical Sciences," 1871, n. s., vol. lxi, p. 17.—Ed.

been determined, the next thing to do is to look for the cause, and in these cases the patient's own statements are practically the only data obtainable. But this has no special disadvantage; for in cases of acute overexertion the patient, as a rule, gives an accurate account of what has happened. His observation of the injury to the organism, and sometimes even to the heart, is quite accurate.

In chronic heart-strain the case is much less clear, because it has to be decided whether or not exercise, which for a variable time had been well borne, has become the cause of disturbances. I shall presently return to this question, as soon as I have disposed of one more point.

For it is as impossible as it is difficult to decide whether the overexertion that has taken place is due to the fact that the heart is in itself diseased or has been primarily damaged by some special injury, or whether one has to deal with a case of so-called simple strain. This question can, of course, be determined only by accurate examination of the heart, supplemented by a most careful interrogation of the patient; and sometimes it will be impossible to decide the question until after the acute sequels of the overexertion have disappeared. I do not here refer to cases in which there is no valvular lesion; for that can, as a rule, be determined without any difficulty. But the existence of coronary sclerosis, myocarditis, and especially of "beer heart" may be very difficult to recognize, especially in an individual who really has cardiac insufficiency brought on by bodily exertion. In deciding this point the student must bear in mind all that has been said in the respective sections about the recognition of these conditions. By doing so he will, in many cases, at least, be able to make the differential diagnosis from coronary sclerosis and myocarditis; but it will often be difficult to decide how much of the blame for a morbid condition is to be attributed to alcoholism and how much to muscular exertion. It is evident that the question is not infrequently incorrectly put; for it must be remembered *that the heart frequently falls a victim to the coöperation of several factors.* Alcoholism and severe muscular exertion are so often found in the same individual that it is simply a matter of taste whether a clinical history of this kind is classified under one head or under the other. In such a case differential diagnosis is, of course, out of the question.

In general it may be positively stated, in the majority of cases of so-called chronic overexertion, especially cases in which some form of exercise which the patient has been in the habit of performing for a long time—perhaps years, or even decades, without any difficulty and without suffering any damage—is regarded as the cause of the injury, that the heart gives out because it is diseased. The more closely these things are studied, the more one is inclined to adopt this view.

The matter assumes a different aspect in those cases in which some unaccustomed occupation, accompanied by considerable exertion, is regarded as the cause of the cardiac symptoms. In such a case one's judgment will be formed after duly weighing the patient's constitution, the amount of work that he has performed in the past, and the conditions under which the work has been performed.

The principal conditions to be considered in the differential diagnosis of irritable heart are nervous diseases of the heart. For this purpose, again, the history is important. To justify the diagnosis of overexertion it must be possible to demonstrate that unusual exertion actually

has preceded the attack. It has already been pointed out that this conception is, after all, only relative.

Enlargement of the heart is rare in cases of simple nervousness, but frequent in cases of overexertion. The effect of muscular movement must also be taken into account; for it often produces no disturbances in nervous individuals, while in the cases now under consideration it brings on the symptoms quicker than anything else. In addition it is, of course, important to determine whether there is any general nervousness in the case which could be regarded as the cause of the cardiac disturbances. But this point must be determined with great care because, in the first place, as has been said, patients with an irritable heart are very apt to become nervous, and another special difficulty is that it is perhaps impossible to draw a sharp line of separation between an irritable heart and cardiac disturbances produced by local changes in the nervous structures of the heart independently of any condition in the central nervous system (see the section on Nervous Diseases of the Heart).

Who shall venture to say that nervous changes in the organism may not greatly favor the development of conditions of so-called irritable heart? The fact that they occur so frequently in war is certainly no argument against this hypothesis.

In the case of young people the differential diagnosis also includes the changes in the heart which occur during the period of growth and which will be explained later. But here, again, the problem is not quite accurately stated; for these conditions are not infrequently due to a dilatation of the heart which must be ascribed to some muscular exertion too severe for the individual concerned.

Prognosis.—The first examination of the patient will suffice to show whether one has to deal with one of the severest type of cases, in which the prognosis is always grave; for even after the immediate danger to life has passed, the prospect of complete recovery after such a severe attack is extremely doubtful.

And even in the mildest class of cases of acute overexertion, which at the time do not threaten life, an opinion in regard to the future must be given with great reserve; for, as has been mentioned, the condition often leaves behind a certain diminution of bodily, and often also of mental, vigor. The severity of the accident, previous diseases, the mode of life, demands on the heart to which the patient is exposed either in the course of his work or otherwise, these are the determining factors for the prognosis, which must be finally determined on general principles. In severe cases of so-called chronic overexertion the prognosis will depend on the impression gained at the time in regard to the condition of the heart muscle in the individual case; for this condition is probably associated with disease of the myocardium. In the milder cases the condition of the heart muscle is also important, and the prognosis also depends in part on whether the patient can be placed under favorable surroundings or not.

Treatment.—Immediately after the occurrence of acute overexertion absolute bodily and mental rest is the first requisite. The patient should be put to bed and, if possible, undressed to his shirt. An ice-bag or cold compresses to the heart are always to be recommended when the action is greatly accelerated.

If the dyspnea, anxiety, and oppression persist after the patient has been put to bed, it is well to give him strong coffee and some kind of

heavy wine. Some form of cutaneous stimulation, mustard poultices, a hot foot-bath possibly containing mustard, or brushing the soles of the feet with a stiff clothes-brush may also be recommended.

Quite often, especially in all the milder cases which make up the majority, these measures will be found to suffice. But if the symptoms do not improve, the administration of wine and coffee must be continued, and, if necessary, camphor may be tried in addition.

Theoretically, digitalis is indicated; I do not know whether any reports have been made about its use in these cases. In an urgent case digitalin might perhaps be injected hypodermically.

The main thing is to keep the patient absolutely at rest for a sufficient length of time after the first symptoms have subsided. This is often very difficult to do; for while at rest the patients frequently feel so well that they think they can do anything, although, as a matter of fact, the heart symptoms are apt to return on the slightest exertion.

Gradually the patient should be instructed to try muscular movements, and this may then be kept up in the form of gymnastics or accurately regulated walks in such a way as to obtain an accurate account of the amount of work performed. This is often accomplished more easily by sending the patient to Baden-Baden, Wiesbaden, or Meran, and the carbon dioxid baths administered under medical supervision, which can be obtained in one of these resorts, will greatly assist, as experience has shown, in training the heart muscle to perform a larger amount of work. Artificial carbon dioxid baths are also very useful in cases of this kind.

But these measures do not always suffice. If the tendency to weakness of the heart muscle recurs again and again, patients whose work calls for muscular exertion must be instructed to change their mode of life altogether. But this is a grave step, fraught with serious consequences, and no physician will give such advice without very carefully considering the circumstances of the case. Sometimes, however, there is no choice. It is almost needless to say that bad habits and unwise indulgence in athletic exercises usually have to be restricted. On that point the physician must be firm and must not allow himself to be swayed by his sympathy for the patient. Strict orders must be given, especially in the matter of smoking, drinking, and sexual intercourse.

FORMS OF CARDIAC HYPERTROPHY ATTRIBUTED TO OVEREXERTION.

It appears to be a generally accepted fact in medical literature that violent muscular exercise may lead to cardiac hypertrophy. In healthy men who work hard, the weight of the heart, so far as we know, increases in proportion to the increase in the weight of the body, or, strictly speaking, that of the musculature; but the organ does not hypertrophy in the true sense of the word. This point has been fully explained on p. 484.

The possibility that in some individuals the heart muscle may undergo much greater development as a result of hard muscular work alone than is usually the case in the majority of persons under similar circumstances might be explained by one of several suppositions, one of which is that the bodily exertion in these cases is absolutely enormous. This seems to be *a priori* improbable, because numerous other persons, who perform exactly the same amount of work, do not develop hypertrophy. It is

true that one individual has to exert himself much more than another to perform the same amount of work; but this difference is most marked in unusual and unaccustomed kinds of work, and as in the conditions under consideration the individual is almost always used to the work and is, therefore, trained to perform it, the element of individual variation is practically negligible.

We have seen that the organism has at its command a mechanism whereby it can diminish the work of the heart during muscular exercise; this is effected almost exclusively by diminishing the arterial pressure. I do not believe that the effect of the enormous increase in the systolic output on the work of the heart can be equalized by this diminution of the arterial pressure, and am, therefore, no longer inclined to see in the failure of this mechanism of equalization the cause for the production of cardiac hypertrophy.

Another supposition is that the above-mentioned relation between increase in the amount of bodily work and increase in the muscle of the heart may become modified in favor of the latter. This, in a certain sense, would imply that the muscle-cells are exceedingly susceptible to the stimuli which induce contraction. But this theory also is open to many objections, because no one will be inclined to admit a change in the fundamental properties of the tissues unless it is demanded by very urgent reasons.

Finally, it is possible that the period of recovery, the diminution of the work of the heart during the pause between contractions, is insufficient. Nothing positive can be said on this point.

It is evident, therefore, that the nature of these forms of cardiac hypertrophy after muscular exertion is shrouded in obscurity. But it would be altogether wrong to close our eyes to facts because we do not understand them.

But are they facts? No one who has made any extensive and careful study of the literature dealing with these questions can doubt it, but the proofs are either exceedingly slender or altogether lacking.

In many works it is stated that hypertrophy of one or both ventricles may be caused by muscular exertion, but we unconsciously look on this statement with a certain misgiving when we remember how rare it is for an experienced physician who sees a large number of patients every year to have under his care cases in which hypertrophy of the heart can be positively diagnosticated either during life or at the autopsy, and in which muscular exertion is the only cause that can be assigned for the condition. There is no doubt that this is exceedingly rare, especially when compared with the many cases of cardiac dilatation produced by the same cause, which all of us have opportunities of observing. For instance, I do not remember a single case of positively demonstrated cardiac hypertrophy in which muscular exertion could be regarded as the only, or even the most probable, cause.

One who has had this experience unconsciously assumes a more skeptical attitude in reading the literature, and it is remarkable how rarely the anatomic diagnosis of hypertrophy is based on careful weighing, and particularly how very rarely muscular exertion is the only one of the possible causes of hypertrophy. Persons with a cardiac hypertrophy explained in this way are very frequently arteriosclerotic, and give a history of overindulgence in beer or wine, nor can nephritis by any means be excluded from all the cases. In other words, it is another case of a multiplicity of

causes, and among them we find beer-alcoholism and nephritis, the etiologic significance of which in the production of hypertrophy is beyond all doubt.

I do not wish to be misunderstood. I do not venture to say that there is no such thing as a cardiac hypertrophy due to muscular exertion alone. Only, it is a very rare condition and has so far never been positively proved. For that reason further observations seem exceedingly necessary, and every clinical history of such a case that is above criticism has distinct value.

The only observation which, in my opinion, can be used as an argument for the production of cardiac hypertrophy by muscular exertion is that of Henschen, which was mentioned on p. 486. The Upsala clinician observed enlargement of the heart with a high, heaving apex-beat and a hard pulse in certain Laplanders who were in the habit of making long and exhausting runs on skees. Repeated observation of the same patient to determine the presence of the phenomenon would, of course, be most desirable; but I freely admit that even in the absence of such observations these symptoms distinctly speak for the presence of cardiac hypertrophy. Whether the condition was due to other causes besides muscular exertion is more than I can say, as I am not familiar with the habits of Laplanders. In any case Henschen's observations are extremely important.

A Few Clinical Histories.—N. N., twenty years of age, law student; father nervous; mother said to have heart disease. At the age of six, whooping-cough; no other infectious diseases. From December, 1897, until February, 1898, articular rheumatism. At that time it was said there was no heart disease. The summer semester of 1898 was spent in Switzerland; while there the patient drank much wine and beer, although only at times. Mountain-climbing was well tolerated.

Since the winter semester of 1898–99 the patient has been a member of a society in Marburg, during which time he has fenced a good deal but has not drunk much. Since then he has had attacks of palpitation which occur mostly at night. The patient goes to sleep in the evening without any trouble, but wakes up during the night. He is then anxious; the pulse is accelerated and irregular. An attack of this kind lasts about two hours; then the patient goes to sleep again, but early the next morning he is still conscious of palpitation. Palpitation and dyspnea are brought on by every form of exercise and psychic excitement. All the phenomena are worse during the semesters than during vacation. The patient, for example, cannot fence continuously for fifteen minutes, being obliged to stop on account of dyspnea and palpitation. The pulse on these occasions is 170 and often intermittent.

Status præsens January, 1900: Emaciated; fairly well muscled; not nervous; of a quiet temperament.

Heart: Apex-beat in fifth intercostal space, extending a little beyond the nipple-line; broad, soft, not forcible, 68, regular, uniform. Boundaries of absolute dulness: fourth rib, left sternal border, nipple-line. Boundaries of relative dulness: upper border of third rib, two finger-breadths to the right of the right sternal border, apex-beat. Sounds pure; second pulmonic and second aortic sound very loud, equal; pulse shows nothing peculiar.

Other organs negative.

N. N., student of philology, twenty years old. Father nervous, said to complain of palpitation; mother said to be dyspneic. One brother healthy; one sister and one brother delicate.

As a child had measles and scarlet fever, otherwise was always healthy. Has been a student for four semesters; for the past three semesters has been a member of a students' society. Illness began during the present semester. Dyspnea on movement; sometimes flashes of heat; always very tired. Cannot fence a bout to a finish on account of dyspnea. Has dyspnea on going uphill. He denies ever having drunk to excess—never more than five to eight glasses [pints?—Ed.] a day and never as much as that several days in succession. Patient always feels perfectly well while resting.

Status præsens July, 1899: Strongly built, looks healthy. Apex-beat and size of heart quite normal. First sound at the apex is not quite pure; second pulmonic

sound somewhat accentuated. Pulse 90, regular and uniform, of good volume and moderate tension. Arterial walls somewhat hard.

Other organs normal. Urine normal.

B. A., twenty-four years old, student of natural history. Belongs to a healthy family and in early childhood had measles, diphtheria, and middle-ear inflammation. As a boy he was always healthy until the fall of 1888. In that year, being a Tertian,* he took part in a very fatiguing excursion and carried large stones up a hill. Palpitation, dyspnea, and a feeling of profound fatigue at once made their appearance. The next morning a physician who knew the boy found that the heart was greatly enlarged, a condition which he is sure was not present before.

Since that time the patient has had dyspnea and palpitation after exertion. The degree of exertion that he can bear varies greatly; sometimes B. is almost well and can accomplish a good deal; at other times these symptoms make their appearance very early.

No subjective symptoms, such as pain or fear during the night; never while he is at rest. Drinking of coffee is immediately followed by palpitation.

Status præsens January, 1899: Quiet, sensible fellow. Absolutely not nervous. The heart at certain times—I have often examined him—is somewhat enlarged to the left and to the right; at other times the size is entirely normal; the apex impulse presents nothing peculiar. The sounds are always pure. The pulse is always somewhat accelerated, 88 when he is completely at rest, always irregular and unequal, usually of good volume and tension.

The lungs and abdominal organs are normal.

K. H., nineteen years old; man-servant. Father died of some pulmonary disease; mother and two other members of the family are well. Denies alcoholism and venereal diseases. Had diphtheria at the age of eight; after that always healthy. Three years ago the patient one day "for fun" wrestled several hours in succession with three vigorous friends of his and immediately afterward developed pain in the chest and back.

During the succeeding week palpitation and dyspnea made their appearance. Since that time the patient has been unable to do hard work, often has pain in the epigastric region, and the bowels are irregular.

Status præsens July, 1900: Emaciated, somewhat cyanotic. Lungs healthy.

Heart: Apex-beat in fifth intercostal space in nipple-line; broad, strong, and distinctly heaving. Boundaries of absolute dulness: fourth rib, left border of sternum, left nipple-line. Boundaries of relative dulness: third rib, one finger-breadth to the right of the right sternal border, the left as far as the apex impulse. Systolic murmur at the apex and at the second intercostal space on the left side. The second pulmonic sound is accentuated; the second aortic is also distinctly loud. Brachial arteries tortuous. Pulse irregular and unequal, soft, of moderate volume; the rate varies between 56 and 110. The pulse is extremely labile and much affected by bodily movements and mental excitement.

The liver is negative. The urine on the first day showed traces of albumin without any formed elements; after the first day it was normal. No edema.

The patient was first treated with rest, then with carbon dioxid baths. The size of the heart soon become entirely normal. The systolic murmurs and accentuation of the second pulmonic sound disappeared, but the first sound at the apex remained impure. The heart action is always unequal and irregular. The pulse-rate always fluctuates.

Later the patient's nervous symptoms became very prominent. The man was depressed and constantly complained of pressure and discomfort in the epigastric region. He never had any more cardiac symptoms. Gastric digestion was quite normal. Treatment of the gastric symptoms was without result. Unfortunately, the patient was not intelligent enough to give an accurate account of his nervous disturbances, especially of his mental disposition.

The case shows very clearly that disturbances of the nervous system may be superadded to a cardiac disturbance caused by excessive muscular exertion. Is there any myocarditis in this case?

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*A member of "Tertia" or third class from graduation, the graduating class being the first or "Prima." The average age in this class (Tertia) is sixteen.

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DISEASES OF THE HEART CAUSED BY THE EXCESSIVE INDULGENCE IN ALCOHOLIC BEVERAGES.

After the excessive drinking of alcoholic beverages the symptoms of cardiac insufficiency develop in many individuals under circumstances which make it absolutely certain that the circulatory disturbances are caused by the nutritional anomaly.

Beer-drinkers furnish the largest contingent of patients with this disease. Bavaria, especially Munich, is its home par excellence. In Bavaria excessive beer-drinking is very wide-spread in every walk of life, and the people in that country in every class of society fall victims to this form of heart disease. Nevertheless, certain classes show a peculiar predisposition, namely, those who are engaged in the manufacture and sale of beer. These people drink all day long—they absolutely live on beer, and, besides, another important factor enters their lives, namely, that of heavy manual labor.

But the so-called "beer heart" is not altogether rare in countries outside of Bavaria. The incidence appears to bear a direct relation to the geographic distribution of good beer. Again, of course, the disease is most frequent among persons whose occupation or the bad habits peculiar to their class afford them an opportunity, or in a certain sense force them, regularly to drink large quantities of beer. I have seen this form of heart disease in Saxony, Thuringia, and Hessia, especially among brewers, beer-wagon drivers, waiters, innkeepers, merchants, butchers, and students. In the case of many of these people severe muscular exertion perhaps also enters into the question. Among students, at least, I was struck by the fact that the disease is found almost exclusively among members of the dueling corps. Not that other students are not frequently quite as heavy drinkers, but the former have, in addition, the exertion of fencing and dueling and frequently suffer also from want of sleep. I think these things are all important, and they will be referred to again in connection with the pathogenesis. There is certainly no doubt, however, that excessive beer-drinking alone may produce this form of heart disease; at least, cases have been reported from Munich which seem to prove this to be possible.

Among *wine-drinkers* these forms of heart disease appear to be distinctly less frequent, at least such is my impression from a study of the literature and my personal experience. It is not absolutely certain, however, for it may be merely an accident that most of the reports come from beer regions, and it is a curious fact that in some of the earliest cases of this form of heart disease, I refer to those reported by Traube and Fraentzel, the patients were wine-drinkers. If heart disease develops at all in the latter class, there are usually certain other bad habits besides the excessive drinking of wine. Most of these people eat too much, and especially eat large quantities of proteids; many also drink spirits and smoke large quantities of imported cigars; and not a few of them are addicted to sexual excesses. The nutritional anomalies referred to suggested to Fraentzel the term "Luxusconsumption," which he used; but in my opinion this term would better be discarded, because it is misleading and some people might imagine that the word helps to explain the pathogenesis.

Dilatation and cardiac insufficiency due to excessive eating and drinking occur chiefly among the wealthier classes of the population, especially among people who attach an exaggerated value to food and drink among

the things of this world. Stokes mentions the frequency of the condition in gouty subjects.

Whether the *drinking of spirits* (Schnaps) alone produces an analogous form of heart disease seems doubtful to me. At least such a thing is very rare, and I, at least, know of no such case. It should be mentioned, however, that in the opinion of other physicians spirits (Schnaps) have some influence on the production of heart disease.*

The disease is characterized by a gradually developing insufficiency of the heart. The cardiac insufficiency may be primary and may represent the only effect of the above-mentioned factors; or a hypertrophy may be produced in addition to and before the impairment of cardiac function. In studying the pathogenesis we must, therefore, explain how the strength of the heart is diminished by the excessive drinking of beer and wine and how, in many cases, its labor is increased.

That *hypertrophy of the musculature really can be produced by the excessive drinking of beer* may be regarded as positively proved by the extensive observations of Bollinger.

In the Munich cases, chiefly described by Bollinger, the weight of the heart is exceedingly large as compared with that of the body. It is true that the pericardial fat was not removed from the heart before the organ was weighed, although some of the individuals must have been obese. But this omission cannot possibly be said to vitiate the value of his figures, not only because Bollinger distinctly excluded hearts with an unusual amount of fat from the investigation, but also because the relation is so very different from the normal. The mere presence of the pericardial fat could not cause an error of any consequence.

In these cases of hypertrophy the weight of the heart often attains an extraordinary degree, and, judging from the reported observations, the walls of both ventricles are thickened.

Hypertrophy the result of copious beer-drinking is observed mostly in persons between twenty and forty years of age; it is needless to say, almost exclusively among men, and it has been found most frequently in men whose occupation continually demands or compels them to drink large quantities of beer.

Some of the individuals, besides consuming large quantities of beer, did very heavy work; many of them smoked to excess, some of them were addicted to sexual excesses, and most of them were very well nourished. The beer itself contains large quantities of carbohydrates, which are thus introduced into the body. There is no doubt, therefore, that a combination of causes is at work in these cases also; but the ingestion of alcoholic beverages is, nevertheless, so much the more prominent, that it must be held chiefly responsible for the effect produced. It is the factor that is repeated again and again and, besides, it is sometimes, according to reliable authorities, the only factor present.

So far as I can see, the statements of the Munich investigators for the present justify the assertion that the immoderate drinking of beer is capable of producing hypertrophy of the heart.

But I am less confident in the assertion that *hypertrophy of the heart develops from excessive eating and the drinking of wine.* Hypertrophy doubtless occurs also in wine-drinkers and gluttons; but so far as I see, in at least a great many cases arteriosclerosis and diseases of the kidney may be held responsible for its development. A discussion of a cardiac hypertrophy due to excessive indulgence in alcoholic beverages should

* Compare Aufrecht, "Archiv für klinische Medicin," vol. liv, p. 615. Tresillian, "Edinburgh Journal," June, 1898.

include only those cases in which there is no known cause for the occurrence of hypertrophy other than the ingestion of these beverages.

According to the Munich investigators, hypertrophy is an absolutely frequent result in beer-drinkers. Bauer, Bollinger, and Rieder, whose description I chiefly follow in this work because they had the best opportunity to make a large number of observations, assert that the hypertrophy is from the beginning associated with dilatation—that is, with an increased quantity of blood in the ventricles. The explanation of its production rests on the fact that each individual portion of the heart, owing to the increase in the systolic output, performs more work, and this increase in the work is quite marked. Hypertrophy is the result.

Unfortunately, we still lack the data necessary for a detailed discussion of the pathogenesis. The drinking of moderate quantities of beer and wine causes a rise in the blood-pressure lasting a few hours, this rise being quite variable in different individuals.* As it is not to be supposed that the filling of the various portions of the heart is correspondingly diminished, —since the opposite is much more probable,—one might assume an increase in the labor of the heart for this reason alone. But we do not know whether this increase also occurs when the individual has become accustomed to this definite quantity of wine or beer, which is the all-important point. It does not seem probable that the increase in the labor of the heart is due simply to the propulsion of a larger quantity of fluid. Such a supposition is not even justified on theoretic grounds, and, besides, it is well known that the drinking of excessive quantities of water produces only a very slight effect. We, therefore, have to consider either a direct increase in the work of the heart, or an indirect increase brought about by modification of the blood or changes in the blood-vessels and associated with an increase of the volume of the heart-beat.

Here we begin to tread on uncertain ground; we do not know what else is responsible for the increase in the labor of the heart. The theory that alcohol is the sole active agent may be discarded at once. Against this theory we have, in my opinion, the very great rarity, not to say complete absence, of cardiac hypertrophy among exclusive whiskey (Schnaps) drinkers. As has been pointed out, beer-drinking is the most important cause; but beer contains, besides alcohol, a number of other active agents, as well as large quantities of readily assimilable carbohydrates, and, therefore, has a high nutritive value.

Bollinger lays great stress on the fact that there is plethora. It is probable that plethora increases the labor of the heart, nor can the possibility of its existence by any means be denied. But neither has it been proved; and if we admit the existence of plethora, we open the door to various other questions that must be answered. Does plethora develop in all beer-drinkers, and if so, why does the heart hypertrophy only in some and not in others? Again, is plethora always accompanied by hypertrophy of the heart, and why does plethora develop only in some drinkers and not in others? We know nothing at all about the effects of the friction of the blood, nor about the condition of the vessels in beer-drinkers.

In regard to the production of cardiac hypertrophy in gluttons, certain speculative theories have been advanced which I need not enter into. Plethora may be thought of in these cases also, but, of course, any explanation of the *modus operandi* meets with the same difficulties. Although all that has been said about the habitual overfilling of the abdominal vessels by excellent investigators assuredly does not belong to the realm of fable, any attempt to argue on the basis of these statements that there is an increase in the work of the heart would, in the present state of our knowledge, be absolutely futile.

Any theory that may be advanced in the future will have to reckon with a number of circumstances. Cardiac hypertrophy after the excessive

* Rieder, "Archiv für klinische Medicin," vol. lv, p. 8.

drinking of beer only occurs in a certain proportion of those who are exposed to the same injurious influence in the same way. It follows that the conditions necessary for increasing the work of the heart arise only under certain circumstances. Hence the mechanism is not the same as in cases of valvular lesions, for example. An aortic insufficiency, if severe enough, under all circumstances causes dilatation and hypertrophy of the left ventricle; but the excessive drinking of beer does not necessarily produce any effect at all. Perhaps, after all, the fault lies in the coöperation of several factors, as I have frequently pointed out. One cannot help thinking that muscular exercise exerts a peculiar influence on the organism in alcohol drinkers. As I tried to show in the preceding section, active innervation of the skeletal muscles causes a marked increase in the work of the heart and the demands made upon the heart. It is quite conceivable that this effect is specially marked in drinkers.

In any case there are two more reasons that incline us, for the present, to abandon any attempt to explain this cardiac hypertrophy both in beer-drinkers and in wine-drinkers. The first of these reasons is found in the *relation of hypertrophy to arteriosclerosis*. I need not here discuss the connection between the cardiac and the arterial disease in detail, because it will be explained in another place. It is a fact, however, that sclerosis of the vessel is present in not a few of the patients belonging to this class, especially in those who have been addicted to excessive eating and wine-drinking, and in such a case the sclerosis must, of course, be taken into account in explaining the cardiac hypertrophy. The second reason is found in the *behavior of the kidneys*. In beer-drinkers these organs have been very carefully examined.* They often present atrophic processes which manifestly bear the closest resemblance to granular atrophy, and the latter undoubtedly has a very considerable, although still unexplained, influence on the work of the heart. The frequency and intensity of the renal changes ought, therefore, to be made the subject of careful observation in the future.

While hypertrophy of the heart occurs only in a certain proportion of cardiac disturbances following the excessive drinking of beer and wine, insufficiency of the heart is regularly found. I have never seen hypertrophy without insufficiency, although I have seen numerous cases of cardiac weakness presenting nothing whatever to justify the assumption of hypertrophy. Nor have I ever found in the literature a description of cardiac hypertrophy due to the abuse of beer without insufficiency of the organ.

In many respects, however, we are still far from a clear understanding of the pathogenesis of cardiac insufficiency. In a number of hypertrophic hearts which had become weak I was myself able to demonstrate the presence of inflammatory processes in the musculature as the cause of the insufficiency.† These processes consisted of round-cell infiltration beneath the endocardium or pericardium or in the muscle alone, foci of connective tissue in various stages of development, and inflammatory processes in the vessels. Degeneration of the nuclei and the protoplasm of the muscle-fibers was also not infrequently found. Curschmann, in his well-known

* Puricelli, in Bollinger's "Arbeiten aus dem pathologischen Institut zu München," Stuttgart, 1886, p. 262. Horn and Schmaus, "Die cyanotische Induration der Nieren," Wiesbaden, 1893.

† Krehl, "Archiv für klinische Medicin," vol. xlviii, p. 414.

case,* saw a wide-spread fatty degeneration of the muscle-fibers, and the same alteration is mentioned by other authors.

The inflammatory processes in the cases which I examined were infectious or toxic in character; at least they exhibited the greatest similarity to processes observed in infectious diseases and, as a matter of fact, the history of these patients often contained mention of infectious diseases, especially syphilis, so that one might be inclined to regard these processes as the remains of such diseases. It is also possible, however, that persons with a cardiac hypertrophy of this kind are more susceptible to a fresh infection,—for instance, the well-known fact that a diseased heart is greatly susceptible to injury during infectious diseases,—or that the alcohol or some other ingredient of the beverages taken in excess is not altogether free from the blame of having produced such inflammatory degenerative processes. I may also point out the complication with inflammation of the coronary vessels, which we all know greatly injures the function of the heart.

Hence in these cases the causes of the cardiac insufficiency are, at least in part, understood, and one might be inclined to generalize from these cases, were it not for certain definite assertions, especially those of Bollinger, to the effect that in the Munich beer-drinkers inflammatory degenerative processes are usually not found. It is true that in his investigations the hearts were not examined systematically, which undoubtedly ought to be done. In short, it will not be possible to give a final judgment until the question has been further investigated.

As we have already seen, the process known as fatty degeneration has frequently been found in hearts of this kind—for example, by Curschmann. Our present views in regard to its relation to the functional capacity of the heart are discussed on p. 500. But fatty degeneration is also frequently absent in the conditions under consideration (see Bollinger's observations), or at least it is so slight in extent that it cannot be regarded as of vital importance.

Finally, it must be remembered that alcohol, through the poisons it contains, perhaps produces a certain weakness of the heart muscle, so that it perhaps more readily succumbs not only to the ordinary demands of life, but still more to any unusual exertion. Again I wish to point out the injurious influence of the combined action of severe muscular exertion and the excessive drinking of beer and wine. Among students, for example, one often does not know which of the two factors to consider the chief cause of an existing cardiac insufficiency.

Symptoms.—The first to appear are usually *symptoms of diminished functional capacity* of the heart in every conceivable gradation. Often the symptoms are at first only subjective; the patients say they are unable to perform certain muscular exertions as well as formerly. At the same time slight sensory disturbances are often present—pressure and a certain sense of pain in the chest and sometimes a feeling of anxiety.

The objective examination at this time need not necessarily show anything abnormal, or slight modifications of the heart action, consisting in acceleration and irregularity, and especially a certain labile quality, are found, so that even slight muscular movements or mild emotion have an altogether disproportionate effect on the frequency and rhythm of the heart.

The next sign that is noticed is a slight dilatation of both ventricles,

* Curschmann, "Archiv für klinische Medicin," vol. xii, p. 193.

and now the first sound at the apex frequently becomes impure. Not rarely the second sounds at the base are accentuated, split, or unequal. The pulse is often extremely soft. Even now one practically never finds the signs of altered blood distribution nor congestion in the liver, skin, or kidneys. I do not remember ever to have seen albuminuria under these conditions.

These symptoms are, therefore, signs of diminished cardiac function. I first made their acquaintance among students, and the symptoms observed in those who eat and drink to excess are quite similar. Why the excessive drinking of beer in young men, like students, at once leads to the appearance of signs of cardiac insufficiency is not altogether clear; possibly the reason is to be found in the above-mentioned coöperation of beer-drinking with factors which in themselves are apt to injure the functional capacity of the organ. In the case of excessive eaters I always suspect that coronary sclerosis has a good deal to do with the etiology; at all events, the prognosis in such cases is much less favorable than among young men.

As has rarely been stated, *hypertrophy with dilatation* now develops in addition to the above-described dilatation. This condition is found chiefly in persons who, for years, have drunk large quantities of beer in the course of their work. This point seems to be positively established by a large number of autopsies made in Munich. During life we find the signs of enlargement to the left and to the right, a more forcible apex impulse, accentuated second sounds at the base, and frequently also a systolic murmur at the apex, which may be due to muscular insufficiency of the mitral valve. Minute examination of the peripheral vessels frequently shows tortuosity and sclerosis of the arteries. The pulse is often accelerated and often, at the best of times, slightly irregular and unequal, rarely of abnormally high tension.

The symptoms which induce patients with this form of hypertrophy and dilatation to consult the physician are either signs of cardiac insufficiency or *sensory disturbances*, that is, a sense of pressure and discomfort in the chest, often associated with a certain anxiety. These symptoms develop either after muscular exercise and emotion or without any direct cause; they usually appear first at night. The patient goes to bed feeling perfectly comfortable, falls asleep, and awakes after from one to three hours with dyspnea and a sense of fear and pressure on the chest. These symptoms vary greatly in intensity; they often disappear after a quarter to a half-hour, not to return for days or weeks, and then either with the same or with increased severity. Later, severe attacks of dyspnea or cardiac fear develop, but these are rarely the first symptoms.

In discussing the diagnosis it will be mentioned that the latter is often rendered very difficult by the fact that these nocturnal or diurnal attacks may be extremely mild for months. This is eminently a case in which nature refuses to be bound by rules, and it must be remembered that stenocardia, like asthma, presents many more rudimentary and transitional forms than is generally thought.

The *phenomena of cardiac insufficiency* next make their appearance, or, as frequently happens, they may form the first link in the chain of disturbances. The mildest degrees of cardiac insufficiency have already been described, and these may be present in exactly the same way in the cases here under consideration. As a rule, however, the morbid process does not stop there, and all the signs of altered blood distribution develop; edema

makes its appearance and the liver enlarges. The albuminuria, which until now had been only temporary and mild, now becomes quite marked. White and red blood-cells, as well as hyaline and not infrequently granular and epithelial casts, make their appearance. I have often seen patients of this kind come to the Policlinic with dilatation and hypertrophy of the heart; the urine contained large quantities of albumin and the above-mentioned morphologic elements. The diagnosis was in doubt between "beer heart" with congestion of the kidneys and hypertrophy with dilatation of the heart accompanying a chronic nephritis. After a few days' rest at home or in the wards and the administration of digitalis both albumin and formed elements disappeared from the urine. The diagnosis of "nephritis" might seem erroneous; but it is a question whether it is not often correct.

The enfeebled heart is enlarged in both directions. The apex impulse is displaced to the left and sometimes downward; it is either relatively or absolutely weak, or, in cases of hypertrophy, not infrequently more resistant and sometimes, on account of the dilatation, abnormally high (heaving). The heart action is, as a rule, irregular and unequal, mostly accelerated, although I have known "beer hearts" in which the rate became abnormally slow during the last weeks—50 beats and even less. Not infrequently a systolic murmur is heard at the apex. The radial pulse is always small and soft.

In the lungs and abdominal organs the same signs of congestion are found as in all other forms of congestion; embolism, of course, may also occur; the patients develop jaundice—in short, one sees the well-known picture of profound cardiac weakness.

In addition to the findings in the heart, not a few of these patients present the ordinary appearance of chronic alcohol intoxication, which again may be of importance in forming an opinion of the changes in the heart. The liver is not infrequently large and hard, and one may at first be in doubt whether the case is not one of simple hepatic congestion. But the organ retains its form and consistency even when all other signs of congestion subside; ascites remains, and cirrhotic changes due to alcohol are at least probable (compare p. 523).* It is also possible that the kidneys themselves are not infrequently directly injured by the alcohol.

The *clinical course* in this group of cases is extremely variable. There are some patients,† albeit very few, who, after enjoying practically perfect health, more or less suddenly develop cardiac insufficiency either as a result of some external cause, such as an infectious disease, severe bodily exertion, and especially alcoholic excesses, or from some unknown cause; and either die in a short time or, if suitably treated, may recover and continue relatively well. But a more protracted course is much more common,‡ and personally, although I have had a large experience, I have never seen this rapid, malignant type, in which the patient dies at once in the first attack of cardiac insufficiency.

As a rule, after the patient had complained for months or even years, true cardiac weakness finally developed, subsided again on suitable treatment, and was followed by a variable period of relative health. Ultimately, however, the condition unavoidably leads to death, unless the bad habits are given up altogether. If the patient has sufficient energy to change his

* Compare Aufrecht's communications.

† Compare the statements of Bauer in the "Festschrift" to Pettenkofer.

‡ Compare Rieder, Aufrecht, and many experiences of my own.

mode of life, the condition is often completely arrested and the patient lives on in tolerable comfort and may possibly be able to do his work. In milder cases recovery is not at all rare—even perfect recovery as regards bodily and mental capacity. I have seen such results among students, for example.

So it is among beer-drinkers and so it may be among those who eat and drink to excess; but I have an impression that in the latter nocturnal attacks of stenocardia, asthma, or mixed attacks early become a prominent feature and may for some time constitute the only symptom, while the patient's ability to stand physical exertion is still practically unimpaired. In beer-drinkers this is also sometimes the case, but I believe much more rarely.

Toward the end cardiac dyspnea plays an important rôle in all these cases; the patients often suffer horribly day and night.

No one who has had any experience in diseases of the heart will fail to see the great resemblance of many conditions belonging to this class to the conditions occurring in coronary sclerosis; this resemblance is, indeed, quite striking. It has both theoretic and practical importance. As a matter of fact, distinct changes in the coronary vessels are not infrequently found at the autopsy, especially in those who eat and drink to excess—in fact, I believe in the majority of these cases. It once more shows how very complicated the etiologic relations are—so-called uncomplicated cases are extremely rare.

Diagnosis.—In spite of the diminished functional capacity of the heart, objective examination does not necessarily yield any positive results; or dilatation of the ventricles may be found, possibly associated with hypertrophy. Whether, and to what extent, these conditions are present must be determined according to the rules laid down on p. 492. Anomalies of the heart action and sensory disturbances complete the clinical picture.

The latter is not very characteristic. At first the appearance may be exactly the same as in myocarditis, the form of heart disease produced by severe muscular exertion, or the forms occurring in the obese, in nephritis, and after coronary sclerosis. The history here asserts its rights. It shows, at any rate, to what extent muscular exertion is the *causa morbi* and whether there is alcoholism in the case.

With regard to the statements obtained in regard to alcoholism, two things must be remembered: In the first place, the patient is very likely to misrepresent the facts; and, in the second place, one man may be able to dispose of a quantity of wine and beer without any trouble which would be distinctly harmful to another.

The existence of any of the ordinary forms of nephritis can be determined by examining the urine (compare p. 526), and the condition of the eye-ground must also be taken into account. It is true that the relations between the conditions here under consideration and diseases of the kidneys are still obscure, and I have often had occasion to point out that certain peculiar forms of nephritis perhaps play an important rôle in the pathogenesis, at least in those cases in which there is hypertrophy of the heart muscle.*

There only remains to discuss the differential diagnosis between the form of heart disease that occurs in the obese, on the one hand, and coronary sclerosis and myocarditis, on the other. This distinction may be

* Compare the interesting observations by Hirsch, "Archiv für klinische Medicin," vol. lxviii, p. 55.

exceedingly difficult and often quite impossible because the conditions merge into one another. For example, who would venture to exclude sclerosis of the coronary arteries when stenocardiac conditions are present? How many of those who are addicted to excessive eating and drinking are not also obese? How often is not obesity due to excessive eating and alcoholism? Even myocarditis not infrequently occurs under the same circumstances. Hence a very guarded opinion must be given in such a case, especially if one's acquaintance with the patient is only slight. If the physician already knows whether treatment has been given and whether it has been effective or not, he is in a much better position to give an opinion.

Treatment and Prognosis.—The first and most important part in the treatment consists in the complete withdrawal of alcoholic beverages. If the heart retains any functional power at all, this should, in my opinion, be strictly and inexorably insisted upon because, in the first place, it is best for the heart, and, in the second place, it is impossible to keep patients of this class within bounds if they are permitted to take alcohol even in small quantities. As a rule, they are not amenable to reason.

In a few cases, when the patient is on the other side of fifty and already exhibits a considerable degree of cardiac weakness, the sudden withdrawal of alcohol may seem a daring procedure. In such cases beer, at least, must be forbidden altogether and the patient must be advised to take small quantities of some rather light Rhine or Palatinate wine.

Next the diet must be carefully regulated. In the first place, the excessive quantities of meat that many of these patients are in the habit of eating are certainly not beneficial, and, in the second place, the non-nitrogenous substances must also be restricted, for there is obviously a certain degree of obesity in many of these cases. Many of the principles that obtain in the treatment of heart disease due to obesity must be followed in these cases alone.

If the patients are still free from any obvious signs of circulatory disturbance, such as edema and congestion of the liver, a carefully supervised course of exercises (Bewegungscur) may be ordered. Exercises often do a great deal of good in these very cases by strengthening the heart muscle. It is of the utmost importance to read the indications rightly, that is to say, to determine whether the heart still responds to the demands incident to the muscular movements, which can, of course, only be determined by general principles. Whenever excessive eating and drinking are at the bottom of the trouble; when the patient, either from the necessities of his calling or from choice, had led a sedentary life; when abdominal plethora—a condition that is perfectly familiar to the physician, although theoretically but imperfectly understood—is present, a so-called reduction cure may be tried, that is, the patient may be ordered to drink some alkaline saline mineral water or may be sent to Carlsbad, Marienbad, Kissingen, Neuenahr, or Tarasp.

But the treatment should never be confined to one of these "cures." The main thing is that the patient's entire mode of life in regard to eating, drinking, and exercise must be established on a rational basis, not only for a time, but for the rest of his life. If this is done, really good results and even recovery may be effected in early cases of this disease; the dilatation may subside and the hypertrophy perhaps also; the greatly damaged functional capacity of the organ may become moderately good or even be completely restored.

Even if severe circulatory disturbances have already developed, very satisfactory results can be achieved by a combination of dietetic and mechanic (mechanotherapeutic) measures, as Oertel's well-known observations have shown. But too much should not be expected from this method of treatment. After ascertaining in a general way the functional capacity of the heart muscle in a given case, one should carefully determine how much in the way of exercise the heart is still able to bear. As soon as edema, congestion of the liver, and marked albuminuria make their appearance, I urgently advise rest, restriction of the diet, and digitalis; and not until after these remedies have had their effect—or even if they fail, but then with only the greatest caution—an attempt may be made to strengthen the heart by means of exercises and carbon dioxid baths. In general, it seems to me much more advisable to adopt as vigorous measures as possible, exactly as in other "disturbances of compensation." The outlook is often less favorable in these cases than in lesions of the mitral valve; although similar results are, nevertheless, not infrequently obtained; partly as the result of rest and changing the mode of life and partly under the action of digitalis the congestion phenomena subside, the dilatation of the heart diminishes, and its contractions become more vigorous. It is true that complete recovery is no longer to be thought of; physician and patient must be content if the condition becomes tolerable and the organ regains a certain degree of functional capacity. This is by no means always the case. Many beer-drinkers die when congestive phenomena first appear, as observations in Munich especially show, and there is no doubt that after recovery from the first attack every renewed impairment of the strength of the heart renders the prospect less favorable. Finally, there comes a stage in which digitalis fails. This is the beginning of the end, although it is sometimes weeks before the poor sufferer is relieved from his torment and terrible dyspnea.

Clinical Histories.—H. A., twenty years old, student in philology in Jena. Mother very nervous; father and one sister healthy. Had measles, scarlet fever, and frequently "diphtheria." Was delicate, and until the twelfth year wore a brace. Never suffered from heart disease. Since the fall of 1897, when he became a student, has been drinking a good deal of beer.

On the evening of the fifteenth of May, 1898, although nothing special had happened during the day, he was suddenly seized while in bed with oppression, dyspnea, and a sense of fear. The condition persisted during the entire night, and the patient did not get to sleep until toward morning. He slept late and then felt well. In the afternoon the assistant at the Policlinic discovered a slight enlargement of the heart to the right, but nothing else. The patient gave up drinking beer, stopped smoking, and felt quite well.

On the twenty-fifth of May he drank four cups of strong coffee in a village, 4 km. (2½ miles) from Jena. On the way back to town he became ill. He felt oppressed and anxious, and it took him three hours to cover the distance of 4 km. It was only with the greatest difficulty and by crawling along the ground part of the way that he reached home. During the night enlargement of the heart to the right was again found; the pulse was of normal frequency, very soft and small, and the slightest movement of the body caused acceleration and irregularity. The patient was in bed four weeks. The second pulmonic sound was sometimes reduplicated, but except for that there were no findings other than those mentioned. Rest and digitalis caused a disappearance of all the symptoms.

He made a good recovery during the vacation, but whenever he took any violent exercise he became distressed. This tendency persisted after the winter semester of 1899. Once he had to run up three flights of stairs, which again brought on a feeling of fear and oppression. After an hour he felt well; no objective findings.

In December, 1898, the patient had become exceedingly pale, and excited and anxious "on account of the disease." Constipation, excitement, or emotion of any kind caused palpitation. He says the heart at such times beats forcibly and rapidly.

As a rule, he also has a peculiar feeling of weight in the left arm. Has already accustomed himself to going upstairs slowly. Pulse, 108, very soft; otherwise the heart is negative.

M. N., thirty-one years of age, waiter. Father was an inn-keeper, drank a great deal, and died of disease of the heart and kidneys. One brother, who does not drink, has heart disease; the mother is well. The patient has drunk a great deal all his life, "as is the custom in a tavern." "He learned it from the students." Later the patient became a waiter in hotels, wine-rooms, and beer-saloons; he never drank much wine and whiskey, which he does not like, but always drank a good deal of beer. Owing to the large quantity of beer that he drank his appetite was always small and he ate very little.

Has to exert himself a good deal and has much to annoy him. About five cigarettes a day. Very little sexual indulgence.

During childhood he had scrofulosis; at the age of fourteen, scarlet fever. Once he broke his arm. At one time he had a chancre; the disease was said not to be syphilis; at all events he never had an eruption, inflammation of the throat, or inunction cure.

Was taken sick about the end of 1897, with palpitation, pressure on the chest, dyspnea, and swelling of the feet.

Status præsens April 5, 1898: Cyanotic and jaundiced, very dyspneic. Apex-beat in the fifth intercostal space, one finger-breadth outside of the nipple-line, weak and soft. Absolute dulness enlarged to the left, relative cardiac dulness enlarged in both directions. Systolic murmur at the apex; nothing special at the base; pulse 114, small, soft, regular, and uniform. Bronchitis. Large, hard, sensitive liver. The urine contains much albumin, white and red blood-corpuscles, but no casts.

The administration of 3 gm. (45 grains) of digitalis is followed by a perfect flood of urine—from 5 to 6 liters (quarts) in twenty-four hours. Albuminuria, bronchitis, and edema disappear. The congestion in the liver diminishes considerably. Heart impulse in the fifth intercostal space in the nipple-line, of average force and resistance; absolute dulness only a little enlarged to the left; relative dulness normal. The first sound at the apex not quite pure. Otherwise auscultation reveals nothing special. Pulse negative. No symptoms except a slight pressure on the chest. Urine free from albumin.

N. was able to resume his work as a waiter. Did a good deal of hard work and resumed his drinking.

March, 1899: During the past weeks dyspnea has returned.

Status præsens March 3d. The patient is deeply cyanotic, icteric, has severe dyspnea and extensive edema. Apex-beat in the fifth intercostal space, two finger-breadths outside of the nipple line, exaggerated, soft, quite irregular, and unequal; 110. Absolute heart dulness extends to the left nipple-line; relative dulness two finger-breadths beyond the right sternal margin. Systolic murmur at the apex. Second pulmonic sound not accentuated. Pulse very soft and small. Bronchitis, congestion of the liver. The urine contains much albumin, no casts.

The administration of 3 gm. (45 grains) of digitalis is followed by a copious excretion of urine, the edema disappears, the dyspnea improves. The patient frequently sweats at night.

The heart does not regain its former dimensions. Toward the end of March the precordia bulges. Apex-beat in the fifth intercostal space one finger-breadth outside the nipple-line, of medium height and very resistant. Limits of absolute dulness: fourth rib, left sternal margin, one finger-breadth within the nipple-line. Limits of relative dulness: third rib, two finger-breadths to the right of the right sternal border, apex-beat. All the sounds are perfectly pure; the second sound at the base is everywhere uniform and of moderate strength. The pulse is 72, regular, uniform, slow (*tardus*) and of moderate volume. The brachial arteries are tortuous. The lung and urine are now negative. The liver is still somewhat larger and harder than normal.

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The literature on diseases of the heart produced by muscle movements contains numerous important contributions to the knowledge of the disturbances here discussed.

THE DISTURBANCES OF CARDIAC FUNCTION FOLLOWING INTOXICATION WITH TOBACCO, COFFEE, AND TEA.

The influence of tobacco, coffee, and tea on the function of the heart has been discussed in a former section in connection with general remarks on treatment. As these products are not without influence on the heart's substance, they must be exhibited cautiously to heart patients. For the same reason they become the causes of cardiac disturbances and play a not unimportant part in the etiology, particularly in these days when their unlimited use is on the increase in many classes of the population.

I do not refer to acute intoxication with these substances. In these the cardiac symptoms are no more prominent than in a number of other intoxications, and we have already stated that we shall not include in this treatise every condition in which anomalies of the cardiac function occur.

The long-continued use of tobacco, coffee, or tea not rarely produces cardiac symptoms which are so prominent that the condition may easily be mistaken for heart disease.

Tobacco, as a rule, causes heart symptoms only when used to excess and for a long period of time. I only once saw heart symptoms caused by chewing tobacco, although such conditions are unquestionably much more frequent in places where the custom of chewing is more wide-spread.

In Germany heart symptoms due to tobacco-poisoning occur practically only among cigar-smokers. Many authorities believe that the disease is much more rarely produced by smoking cigarettes and long pipes, while, on the other hand, it is said to be quite frequent among those who use the short French pipes. There is a wide-spread impression, borne out by Fraentzel's statements, that heart disease is most frequent in those who smoke so-called imported cigars. This seems remarkable; for this variety is said to contain less nicotin than cigars of domestic manufacture, and most authorities believe that nicotin is the active substance (see p. 478); at least we know of no other of the same importance, and all careful investigations point to nicotin as the most significant constituent of tobacco. But I purposely refrain from a minute discussion of these questions because there is still too much uncertainty, both as regards toxicology and purely clinical observation. So far as I can see, the percentage of nicotin contained in cigars is extremely variable. I do not know whether I am justified in making a general statement that imported cigars contain less nicotin than those of domestic manufacture. It is true that we cannot as yet venture to say that it has been definitely proved that heart symptoms really are more frequent among smokers of imported cigars, and it would be much better if physicians who base their opinions almost exclusively on the examination of the well-to-do people would report a few

accurate observations instead of summary statements. It is not to be denied, however, that ordinary, every-day experience is strongly in favor of the theory. How many individuals daily smoke a great number of domestic cigars without becoming ill, and then develop cardiac disturbances after smoking one or two imported cigars? It is much more frequently the case in men who are not used to cigars! Much depends also on the number of cigars a man smokes and how he smokes; so-called swallowing and inhaling of smoke seems to be particularly harmful.

The first symptom that the patient becomes aware of in chronic intoxication is palpitation. Some patients have it permanently; more frequently the palpitation is paroxysmal. The attacks occur after smoking, but quite frequently also spontaneously; for example, they often occur at night. The heart becomes more irritable in general: muscular movements, eating, digestion, psychic emotion, easily bring on a feeling of palpitation.

The action of the heart may remain altogether unaffected, although frequently disturbances are observed. The characteristic change is acceleration to about 100, more rarely retardation to 50 pulsations. Irregularity and inequality are not at all rare; the two phenomena are observed in association with the feeling of palpitation. Apex-beat and pulse may be weak; but occasionally a high, heaving apex-beat is observed in these conditions.

The size of the heart in the great majority of cases is not altered. Farwarger saw a dilatation which, so far as can be judged from the clinical history, he quite rightly attributed to tobacco-poisoning. We must, however, be very cautious in forming such an opinion, because tobacco-poisoning comparatively rarely occurs unaccompanied by any other injury that influences the heart. It is very frequently associated with alcoholism.

But in Farwarger's case there was no other cause, and I myself once saw enlargement of the heart which I had to attribute to excessive indulgence in imported cigars, and which disappeared as soon as smoking was given up.

In some cases the subjective symptoms are more severe; there is a feeling of oppression, fear, and pain, and occasionally attacks of stenocardia occur (see p. 541).

Animal experiments have shown that nicotin has a peculiar action on the heart and blood-vessels, but the knowledge so obtained is not as yet sufficiently complete to explain the phenomena observed in man in chronic tobacco-poisoning. It has not, in my opinion, been determined as yet how much the injury attacks the nervous system of the heart and how much the muscle or the vessels; and I do not think that we are as yet justified in attributing tobacco angina to a spastic condition of the coronary arteries.

In addition to the tobacco symptoms in the heart we, of course, not infrequently observe other manifestations of tobacco-poisoning in other organs, especially the eye and central nervous system. These symptoms may have diagnostic significance. Tobacco-poisoning is diagnosed chiefly by the presence of a sufficient cause, which can usually be demonstrated without difficulty. In heavy smokers it may be difficult to distinguish between symptoms due to tobacco-poisoning and symptoms of a nervous or arteriosclerotic nature. But whenever the patient has been under observation for some time, it is always possible to determine how many

of the symptoms are due to intoxication; for all observers are agreed that every symptom due to intoxication disappears as soon as smoking is given up—in fact, often as soon as the quantity of tobacco is restricted. But until the physician has had an opportunity of thus observing the patient he should be very cautious in giving an opinion, especially in regard to excluding coronary sclerosis, for the latter condition is also favored by excessive smoking.

The excessive use of *coffee* or *tea* (see pp. 478 and 479) may give rise to palpitation, which is frequently associated with irregularity and inequality of the pulse and occasionally with oppression and anxiety. The heart action sometimes appears to be increased in force. In Germany and France these symptoms are most frequently found as a result of coffee-poisoning; in England they are more often due to the excessive use of tea,* a difference which is unquestionably due to the distribution and manner of preparation of these two beverages in the different countries.

The diagnosis is based on exactly the same considerations as those mentioned above.

The **prognosis** of all these conditions is favorable whenever the state of intoxication is recognized and interrupted by eliminating the harmful agent. It is best to forbid the use of coffee and tea altogether for a time, and smoking also has to be stopped altogether in some cases. But it is often better, especially in old people, to allow a small number of mild cigars (two or three a day), at least during the transitional period.

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HEART SYMPTOMS AND HEART DISEASES OCCURRING IN OBESITY—SO-CALLED FATTY HEART.

Although the physician is probably not prepared as yet to surrender the idea of "fatty heart," it must be remembered that it is anything but accurate, and is made up of a number of different factors, that is to say, it includes purely clinical, pathologic, as well as etiologic elements. By far the best definition, in my opinion, is that given by Leyden. According to him we mean by the term fatty heart "the *heart symptoms occurring in obese individuals* if the cardiac symptoms have developed in association with, and at least chiefly as a result of, obesity." I have been unable to find in the literature, nor have I been able to devise, a better definition for this term fatty heart, which is so difficult to explain.

The fact is that a clinical picture has been erected on the foundation of certain symptoms and etiologic factors. As soon as we attempt to provide it with an anatomic basis we enter the region of error. For the symptom-complex characterized by the term fatty heart does not correspond to

* Potain, "Clinique médicale," p. 150.

any well-defined, uniform morphologic changes. There is no doubt whatever that the heart muscle in obese persons who present cardiac symptoms frequently contains and is surrounded by fat. But the presence of this fat cannot be determined clinically from definite symptoms; we only know, from a wide experience, that individuals who present a certain appearance and have certain definite symptoms, when examined post-mortem are, as a rule, found to have a fatty heart. Moreover, as will be shown presently, the functional significance of these pathologic conditions and their effect on clinical symptoms are anything but clear. And, finally, the pathologist and, unfortunately, the physician, also applies the term "fatty heart" to another, altogether different condition from the one now under consideration, and not only different, but what is more important, independent of it, namely, the accumulation of more or less minute fat-droplets within the muscle-fibers. This condition is explained in connection with the other degenerative processes in the myocardium, and, like the latter, is found in a great variety of diseases. It is no longer regarded as a nosologic entity, but rather as a pathologic phenomenon which may be due to a great variety of causes.

In almost every human subject, and in most animals, we see adipose tissue under the visceral layer of the pericardium, chiefly at the base of the aorta and pulmonary artery and along the vessels, with which it penetrates the heart in varying degrees of abundance. The quantity is subject to great variation even in healthy individuals. The effect of differences in mode of life and nutrition is best shown by observations on fattened animals and those that are allowed to run loose. The hearts of fattened sheep or hogs exhibit enormous layers of fat. In man also the causes of this obesity of the heart (adipositas cordis) in the main coincide with those of general obesity,* and it is not within our province to enumerate these causes here.† Just why in this disease the fatty deposits occur in definite regions of the body is absolutely unknown. The important point in this connection is that there is no strict parallelism between the general obesity and the deposit of fat around the heart; indeed, what is no less curious than the choice of these sites of predilection for the accumulation of fat is that, in general obesity, the heart in some cases is profoundly involved and very much less in others.

Before attempting to explain the *effect of this adipositas cordis* on the functional capacity of the heart one must first inquire how it affects the bulk of the muscle. As a matter of fact, the latter, especially in the right ventricle, is frequently found to be attenuated, even thinner than in a normal heart,‡ and we are involuntarily reminded of the conditions that obtain in so-called dystrophy of the striated muscles of the body, namely, that there is a connection between atrophy of muscle and hyperplasia of fat. Two possibilities may be considered: the muscle may be destroyed and replaced by fatty tissue; or the proliferating, fat-containing connective tissue may encroach upon and destroy the muscle. What the actual conditions are is, for the present, as impossible to decide as it is to determine the *extent* of the muscular atrophy; although by using the methods sug-

* W. Müller, "Die Massenverhältnisse des menschlichen Herzens," Leipzig, 1883, p. 59.

† See von Noorden, "Nothnagel's spec. Path. und Therapie," vol. vii. Also Krehl, "Pathologische Physiologie," Leipzig, 1898, p. 337.

‡ Compare Birch-Hirschfeld, "Pathologische Anatomie," fourth edition, vol. ii, p. 140. Stokes ("Heart Diseases," p. 249) is of the same opinion.

gested by W. Müller* any one having a large amount of autopsy material at his disposal could answer at least the second question. Until we have some numeric data in regard to the extent of the muscular atrophy, we cannot estimate its clinical significance, because the impressions on which our judgment must for the present be based are too uncertain.

As a matter of fact, the heart muscle in obese persons, as Hirsch† directly demonstrated, is extremely light in comparison with the body-weight. This is not difficult to understand, because the increased demand which the fat-cells make on metabolism and thereby on the circulation is unquestionably much less in proportion than the increase in body-weight which they produce. Indirectly, by adding to the body-weight, the number of fat-cells undoubtedly increases the demands on the heart considerably, which is certainly unfavorable for obese persons. But whether the infiltration of the myocardium with fat has any special connection with the low weight of the heart, and whether the right ventricle under such circumstances is especially attenuated, remains to be decided by future investigations.

Moreover, the quality of the muscle-fibers in a "fatty heart" of this kind has never been sufficiently cleared up. Fatty degenerations occur; but recent observers, like v. Leyden, for example, after careful investigations do not regard them as frequent. This view must be upheld in opposition to a wide-spread impression; indeed, it should be specially emphasized, because this general impression is very largely responsible for the confusion existing between the two "forms" of fatty heart. One might be tempted to quote no less an authority than Stokes as the champion of the theory that the two processes are frequently combined;‡ but a more minute examination of his clinical histories seems to me to show that the term "fatty heart" in his patients embraced a variety of other things which we now usually distinguish from that condition, for example, fatty degeneration of the heart muscle secondary to sclerosis of the coronary arteries. I have been unable to find in the recent literature any reports of thorough microscopic examination of the myocardium from cases of fatty heart as above defined. It is true that a few cases have been reported of obese persons dying with heart symptoms, in which microscopic examination (although only of a few pieces of tissue) failed to give any clue to the pathogenesis of the heart failure; but, on the other hand, reports of naked-eye changes in the myocardium are much more frequent. Very often the muscle is exceedingly flaccid; in Leyden's cases, it will be remembered, the myocardium is frequently described as friable and "squashy" ("matsch"). It is perfectly true that all kinds of chemic changes in the myocardium might develop after obesity, and might greatly interfere with the function of the heart. Indeed, we have reason to suppose that obesity is not so very rarely due primarily to far-reaching changes in metabolism. Again, a great variety of more minute changes in the muscle-fibers may be present. The first thing to be done is to collect enough material for the discussion by careful and extensive observation. Not until this material has been supplied shall we be able to decide, for example, whether inflammatory processes are not frequently present in obesity.

v. Noorden points out that the large deposits of fat between the muscle-

* W. Müller, "Die Massenverhältnisse des menschlichen Herzens," Leipzig, 1884.
† Hirsch, "Archiv für klinische Medicin," vol. lxiv, p. 608.
‡ Stokes, "Diseases of the Heart," p. 250.

fibers perhaps interfere mechanically with the systole of the heart and with its dilating capacity during diastole. This is quite conceivable. To prove the point it would be necessary to determine the distensibility of fatty hearts in animals at body-temperature.

In addition to the interference with the circulation caused by the change in the heart itself, numerous other disturbances necessarily accompany obesity, the severity of which is proportionate to the degree of obesity present. Such conditions are the increased weight of the body, which cannot help affecting both the skeletal muscles and the heart during exercise, the accumulation of fat in the peritoneal and in the pleural cavities, and the interference with respiration which it produces, and a number of others. These things are fully explained in the description of the symptoms of obesity.

It is evident, even now, from a careful consideration of the subject, that the above-mentioned factors, the deposit of fat among the muscle-fibers and friability of the muscle, very rarely represent the only changes in so-called fatty heart, at least, in those cases in which the functional disturbances cannot be explained by simple disproportion between the mass of the heart muscle and that of the body. As a general rule, other phenomena are also present, especially sclerosis of the coronary arteries and the processes to which it gives rise. The not infrequent association of obesity with coronary sclerosis no doubt depends chiefly on the fact that both conditions are often due to the same cause, especially alcoholism and luxurious living. And, in addition, heavy eaters frequently smoke to excess, which also injures the heart.

But abnormal obesity is by no means regularly attended by cardiac symptoms. There are many excessively obese persons who are perfectly strong, who feel well, and must be considered well. They look healthy and flourishing, have strong muscles, and are sound in every respect. On the other hand, some obese persons look anemic; their whole appearance and complexion give one the impression of illness, and these patients are very apt to complain of dyspnea and other cardiac symptoms that other anemic individuals suffer from. One cannot help thinking that anemia has much to do with these various manifestations of obesity, but they may be due to a variety of metabolic processes, and investigation along that line would be both interesting and valuable.

Symptoms.—As a rule, the physician is first consulted because the patient notices signs of diminished functional capacity of the heart, particularly a difficulty in breathing and palpitation during exercise. Often these are the only early symptoms, but sometimes vertigo is also present. These are the cases that present the best prognosis, because the chief factor in the etiology is the disproportion between the size of the body and the size and efficiency of the heart muscle, so that even a slight reduction of the strength of the heart, such, for example, as follows lack of exercise, suffices to produce this disproportion. Obese persons are frequently indolent, and hence their muscles, including the heart, atrophy. It is, therefore, not astonishing that, as the bulk of the body increases and the weight of the heart diminishes, or, at least, does not grow, a stage is finally reached in which even ordinary movements can be performed only with difficulty and with great exertion. The heart need not be diseased, and in many cases certainly is not. Fortunately, the symptoms can often be made to disappear altogether, and in some cases permanently, by means of suitable treatment and by sufficiently reducing

the weight of the body. In such cases the physical examination of the heart either shows nothing abnormal or only a slight enlargement to the right. Owing to the obesity of the patient, no definite conclusions can be drawn from this physical sign, partly because percussion is often very difficult in these subjects and because it is also possible that the increase in the size of the dulness may be due to accumulations of fat on the heart. It is possible, however, that in some cases a slight degree of dilatation of the cavities is present, and in view of the above-mentioned disproportion between the force of the heart and the demands made upon it, it is not surprising that it should be so. The pulse in these cases is often diminished in volume and soft, and, as a rule, also more rapid.

Whether the above-mentioned disproportion by itself is capable of producing circulatory disturbances during rest—in other words, a permanently abnormal blood distribution—I would not venture to decide. Personally, I think it is more probable that, when cardiac insufficiency is present during rest, a graver form of disease must be assumed to exist. The question can, of course, be decided only by a large number of anatomic investigations.

But the above-mentioned symptoms are by no means always the only ones present during the incipient stage, and still less during the subsequent course of a case of heart weakness due to obesity; the greatest variety of unpleasant subjective symptoms also develop later on—cardiac fear, stenocardiac conditions, and profound disturbances of the rhythm. The functional capacity of the heart diminishes more and more. When once these symptoms are present, there is no doubt that the process is already quite advanced, and, in our opinion, the case by that time has, as a rule, gone beyond the condition of simple "fatty heart" and is complicated by morbid processes in the coronary vessels or by changes in the muscle. As we know but little about changes in the muscle, it is wiser, for the present, to abide by the theory of coronary sclerosis.

The symptoms present in this stage cannot, in my opinion, be separated from those observed in coronary sclerosis, a statement that is fully borne out by the descriptions found in the literature, especially those of the famous Stokes. I, therefore, do not think that it is necessary to give a detailed description, for I practically identify these conditions with those seen in coronary sclerosis. From a clinical and symptomatic standpoint, at least, the two conditions are absolutely identical, and the present state of our knowledge does not permit us to attribute these conditions to fatty degeneration of the fibers. Such a theory is absolutely contradicted by the fact that in the gravest cases of myocardial degeneration, as, for example, pernicious anemia and phosphorus poisoning, these symptoms are always absent.'

Granted that we are justified in attributing these cases of so-called "fatty heart" to coronary sclerosis, then we may also ascribe the symptoms of the condition made famous by the observations of the great Irish physicians, the attacks of syncope with paralysis, the Cheyne-Stokes breathing, and the extreme slowing of the pulse, essentially to diseases in the arteries. This, in my opinion, is entirely in accord with the experience gained at the bedside. All these phenomena are most frequently observed in arteriosclerosis, and I think I may say they are rarely found without arteriosclerosis. I have never seen them after degeneration of the heart muscle not due to sclerosis of the coronary arteries.

Physical examination, even in these severe cases, reveals but little

that is characteristic, aside from the general obesity; but in many of the cases the patient conveys a general impression of profound illness. The accumulation of fat in the subcutaneous tissue renders examination of the heart very difficult in every way. Palpation of the apex-beat, as well as percussion and auscultation, is more uncertain and less accurate than in lean persons. The difficulties are especially great in the case of women. In fact, successful percussion in such cases is often quite impossible.

As a rule, the heart impulse is displaced outward and feeble, although, for obvious reasons, these findings have but little value. The heart dulness, so far as it can be determined at all by percussion, is usually enlarged transversely; but this finding also is of little importance, for it may be due to one of several ambiguous causes—accumulation of fat under the sternum, about the heart, and dilatation of the organ. The heart-sounds are mostly very feeble; in ordinary cases they are pure, but systolic murmurs may be present at the apex or the sounds at the base may be modified. The latter is apt to be the case especially when there is sclerosis of the ascending aorta.

In the main, we have to depend on our observation of the arteries and especially of the radial pulse in determining the activity and functional capacity of the heart. The pulse is often small and soft, although this finding may be modified by the presence of arteriosclerosis or an accompanying chronic nephritis.

Quite frequently the heart action is irregular and unequal, often accelerated, and not so very rarely retarded. For these matters also the reader is referred to the descriptions given in the section on Arteriosclerosis.

The ill effects of abnormal blood distribution later make their appearance in a variety of combinations and in a variable order. Both may occur at any time. Sometimes the patients die a sudden and unexpected death.

With the reservations mentioned above and observed in this discussion the relation between obesity and disturbances of the heart appears tolerably clear. But is it not possible that in order to present the facts in an intelligible way too narrow a view may have been taken of the processes of nature? I should like at least to mention that this cannot be altogether denied. In the old literature the very definite statement is met again and again that fatty degeneration of the muscle-fibers occurs as an independent disease, with or without general obesity, and produces symptoms which, as has been described, are regarded chiefly as the expression of a coronary sclerosis. Unquestionably, the signs of arteriosclerosis are also present in these cases, and with equal certainty processes that are followed by severe fatty degeneration of the muscle-fibers, such as anemia and many forms of intoxication, etc., do not produce these symptoms. But there are yet other possibilities. The accumulation of fatty granules in the muscle-fibers may occur as the expression of unknown processes in the heart, and these processes may be associated with the same symptoms as coronary sclerosis in other cases; for the latter also ultimately acts through certain definite effects which it produces on the muscle. I merely throw out this supposition because the assertion that fatty degeneration is an independent disease is so confidently made by excellent authorities in the old literature, especially the English literature. Since we know that the causes and conditions of disease are not quite the same in England, owing to differences in climatic and artificial conditions, we have no right, in my opinion, to render an absolute judgment on these statements.

The **diagnosis** practically follows from the definition. The main difficulty consists in distinguishing coronary sclerosis from the cardiac condition produced by overeating; all that is to be said on this point will be found in the corresponding sections. I shall merely remark here that

there can be no hard-and-fast lines of separation, particularly as it is my opinion that many heart troubles which even here we are in the habit of ascribing to obesity are caused by coronary sclerosis, and alcoholism unquestionably also plays an important part in the production of obesity.

Treatment.—As during the beginning of the trouble at least a considerable share of the symptoms in these patients are slightly due to the large amount of fat in the body, and possibly also about the heart, a method of treatment directed toward the removal of fat is always indicated. It has already been stated that such a method of treatment is occasionally followed by the disappearance of all symptoms. The treatment consists in judiciously combining dietetic measures with a system of exercises. Both the dietetic measures and the exercises must be begun with great caution and carefully adapted to the strength of the heart, according to the general principles laid down both in v. Noorden's essay and in the present article. The reduction cure must be begun slowly, and the physician must constantly keep in mind the necessity of strengthening the heart muscle at the same time; for, owing to the fact that the patients, before treatment is begun, are usually fat and heavy and, therefore, lazy, and owing also to the fact that the symptoms develop rapidly, the heart is usually in relatively bad training. Hence a reduction cure must be carried out with the greatest caution in these cases, and it is obviously preferable, in many instances, to send the patient to a place where this is easily accomplished both on account of the local conditions and the familiarity of the resident physicians with the treatment of heart patients. A great many different health resorts may be considered, and, as I have already stated, I am guided in my choice, aside from social and pecuniary considerations, chiefly by the skill of the attending physicians. It is now possible to reduce fat without materially disturbing the proteid balance. This is, of course, most easily possible when the physician has a wide knowledge of all the factors that enter into the problem,—and they are numerous,—and has at his command the best conveniences that can be obtained. As compared with this, the question how to reduce the fat is really quite secondary. In this matter it is, indeed, true that, under the guidance of the experienced physician, many roads lead to Rome. But whatever therapeutic measures are ordered, the condition of the heart must always be taken into account. Thus, for example, the ingestion of large quantities of fluid may have to be avoided under certain circumstances if it appears probable that the fluid will be absorbed. It is also necessary to determine accurately the amount and degree of exercise to prescribe. In these respects the treatment of heart patients differs from that of ordinary cases of obesity. But in all other respects the same principles obtain, and I would urgently advise that the treatment be as simple as possible and that every kind of charlatanism, such as has been and still is so much practised in the treatment of this disease, be rigorously excluded.

Whenever the excessive ingestion of nutritive material must be regarded as the cause of the obesity, the food must be restricted, at the same time preserving the proteid balance of the organism as much as possible. The food must not only be accurately adapted in quantity and quality to the functional capacity of the heart, but must also be regulated with the above object in mind.

Reduction of the body-fat and simultaneous strengthening of the heart are the first indications in the treatment of all those cases in which the

above-mentioned disproportion between the mass of the body and the strength of the heart must be regarded as the sole, or at least the chief, cause of the existing symptoms. If good results are to be obtained, the heart muscle must still possess sufficient power to enable it to perform the extra amount of work ordered for the purpose of strengthening it, and must not be still further damaged by the exercises. This point must be carefully determined in every individual case by means of the history and the objective examination. The plan of treatment depends altogether on the decision of this point.

It would follow, therefore, that young people, in whom integrity of the heart muscle may be assumed as probable, furnish the most suitable cases for this kind of treatment. In these cases the use of a mild laxative mineral water, especially at some of the appropriate health resorts (see p. 562), is a useful adjunct to the treatment. As I have already mentioned, the aversion to this method of treatment was greatly exaggerated at the time when dry diet was in vogue.

It seems very important in these cases to exercise a careful supervision over the amount of beer and wine that is taken. As individuals of this class are usually vigorous, it is best to forbid alcoholic beverages altogether; the patients then know where they stand, and are much less exposed to temptation.

The conditions are, of course, entirely different when heart symptoms are present in obese persons in whom the obesity cannot be attributed to the excessive ingestion of food or to deficient exercise. Unfortunately, cases of this kind have never been carefully investigated, and, I am unable to say, for example, how frequently marked cardiac symptoms are observed in such individuals. It is evident that the accumulation of fat in these cases is also dependent on some anomaly of metabolism. Reduction cures are hardly indicated; on the contrary, strengthening of the heart and reduction of the fat must be affected by carefully supervised exercises.

In the presence of pronounced cardiac insufficiency with edema, albuminuria, etc., it is absolutely necessary, at first, to influence the heart by means of rest and digitalis, and, if there is reason to suspect coronary sclerosis, great caution must be observed in ordering exercises. Such patients, as everybody knows, are very intolerant to any kind of violent procedures, among which we must include strict reduction cures.

If there is any reason to suspect grave changes in the muscle, and particularly coronary sclerosis, all other considerations must give way to the indications derived from these conditions. Reduction of the fat in itself appears to be an altogether secondary consideration under such circumstances, and the physician's entire aim must be to improve the muscular strength of the heart. Indirectly the strength of the heart is, of course, also benefited by reducing the body-weight, and for that reason the latter object should not be altogether lost sight of in these cases. The best plan is to try, by a suitable diet, to prevent the further increase of the body-fat,—prohibiting beer alone often accomplishes this object,—and gradually one often succeeds by means of more accurate regulation of the diet, especially by *changing* it, indirectly reducing the amount of body-fat. I repeat that any violent treatment is to be deprecated; laxative mineral waters are usually too severe.

It is needless to say that the use of alcoholic beverages must be greatly restricted, but I do not go so far as to counsel total abstinence, especially

in older persons. The beer may have to be changed to wine, and many of the patients will have to be allowed to take their wine as usual.

Carbon dioxid baths are often excellent for increasing the strength of the heart, and are usually well borne by obese subjects.

The most difficult question to decide is whether to try to train the heart by means of exercises and how far to carry the experiment. In my opinion no general rules can be given, as it all depends on whether, in a given case, the condition of the heart is such as to make it seem probable that it is capable of increasing its strength without being injured by the exercises necessary for that purpose. The first attempts will have to be made with extreme caution, carefully noting how the heart reacts. I refer to what has been said on p. 563, and shall merely emphasize once more that the question of exercises is much less important than the manner in which they are carried out; everything depends on the physician in charge.

The *prognosis* depends on the nature of the processes which are responsible for the symptoms in a given case. As a rule, it is very favorable when there is merely a disproportion between the mass of the body and the strength of the heart. If marked circulatory disturbances with abnormal blood distribution have developed, the prospect is usually very doubtful; while signs of coronary sclerosis, of course, always render the prognosis extremely grave.

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INFLAMMATION OF THE HEART (CARDITIS).

The term myocarditis is applied to those processes in which the phenomena of true inflammation of the heart muscle are clinically the most prominent features.

There always has been and still is a great divergence of opinion in regard to the definition and scope of the process designated as inflammation. In this article the term will include those processes in which the

tissue reacts to certain injuries by well-known changes in the blood-vessels, blood-cells, and connective-tissue cells. Thus defined, myocarditis is more restricted in its scope than other forms of inflammation are held to be; for it does not include the purely parenchymatous degenerations, for example, the processes belonging to granular and fatty degenerations.

Certain phenomena that follow occlusion of the coronary arteries—Ziegler's myomalacia—must, so far as the anatomic changes are concerned, be included among the inflammations; for in this condition true inflammatory processes follow the necrosis of certain portions of the muscle due to the anemia.* From a clinical point of view, however, it seems to me wiser to separate these processes from those of primary inflammation. For in myomalacia the most prominent feature is the sclerosis of the coronary arteries, with a number of phenomena peculiar to and directly dependent on the arterial disease. As these arteriosclerotic diseases of the heart are probably more frequent than diseases which are primarily inflammatory, the latter are in danger of being ignored by the physician—as, indeed, happened for a number of years—unless a special effort is made to distinguish them from the conditions due to anomaly of the coronary vessels. In this article the term myocarditis will include primarily inflammatory processes in the musculature of the heart, and we shall distinguish between acute and chronic forms of myocarditis. The view upheld by the old clinicians, that inflammation of the heart muscle, in the great majority of cases, is associated with pericarditis and endocarditis, is unquestionably correct from an anatomic standpoint. Inflammation of the heart in the great majority of cases involves the entire organ, a maxim which was steadily maintained by the older investigators, like Corvisart,† Bouillaud, and Rokitansky.

That *carditis* is not a delusion would appear from the following description. But there are inflammatory conditions involving the entire heart in which the whole pathologic process and the symptomatology are entirely dominated by the disease in the muscle itself, and with these we have to deal in this article. Endocarditis also is, as a rule, accompanied by inflammation of the entire heart, that is, of its three layers; and in speaking of endocarditis, that is, disease of one of the layers only, we merely follow this same custom. We do so because clinically, or rather so far as the results obtained with the various methods of physical examination are concerned, the phenomena of valvular endocarditis and the resulting valvular defects are by far the most conspicuous features in the case. The failure to recognize for so many decades‡ that the muscle is also involved in the inflammatory process in cases of valvular disease has, in my opinion, had much to do with preventing the physiology of these processes from being properly understood. One who overlooks the fact that the heart muscle shares in the inflammatory process in cases of valvular disease will be unable to understand, or at least to gain a perfectly clear insight into, the symptoms and the course of the disease in cases of this kind.

* Ziegler, "Archiv für klinische Medicin," vol. xxv, p. 588. *Ibid.*, "Lehrbuch der speciellen pathologischen Anatomie," ninth edition, p. 23. Weigert, "Virchow's Archiv.," vol. lxxix, p. 106. Huber, *ibid.*, vol. lxxxix, p. 236. Sternberg, "Ueber Erkrankungen des Herzmuskels in Anschluss an Störungen des Coronararterienkreislaufes," Dissertation, Marburg, 1887.

† Corvisart, "Herzkrankheiten," German translation by Rintel, 1814, p. 239.

‡ Compare Krehl, "Archiv für klinische Medicin," vol. xlvi, p. 454. Bard, "Lyon médic.," 1893, No. 9.

Practically the same thing is true of pericarditis. In the majority of cases the muscle is more or less profoundly damaged, and there is no doubt that the injury to the muscle is, in a large degree, responsible for the functional disturbances which are the most prominent features in the clinical picture. It is my duty to mention these matters here, although the discussion of the endocarditis and pericarditis form no part of the task allotted to me in this work.*

As frequently happens, the great progress in diagnosis had the effect of rendering our views one-sided in regard to inflammations of the heart. The physical signs of endocarditis and pericarditis being so much more striking than the signs of muscular disease, which are more modest and unobtrusive as symptoms, but unquestionably of much more importance functionally, the latter were for a long time completely overshadowed in the minds of physicians by the more conspicuous signs of endocardial and pericardial disease. Another unfavorable influence on the development of our knowledge of myocarditis is found in an old superstition that the heart muscle is incapable of becoming inflamed. So far as I can see, there has always been a great diversity of opinion among various physicians in regard to the occurrence and frequency of carditis. While Craigie,† for example, as late as 1847, considered it worth while to collect all the cases known up to this time,—sixteen in all,—Kreysig‡ states that the disease is "very frequent, but unfortunately mostly unrecognized up to that time." Even since that period opinions have fluctuated, and it is only now that something like unanimity is being achieved. We are able not only to describe the clinical picture, but also to define its differences from other conditions, and the disease is by no means rare. In describing the symptomatology we have to depend chiefly on the more recent observations, because in most of the older cases no attempt was made to distinguish between endocarditis and pericarditis. Laennec never saw an absolutely clear case of myocarditis, and even Latham considered such cases extremely rare and impossible to recognize.

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‡ Kreysig, preface to Davis' "Observations," see Literature, below.

THE HEART IN ACUTE INFECTIOUS DISEASES; ACUTE INFECTIOUS MYOCARDITIS.

A modern description of the processes that take place in the circulatory organs in the infectious diseases must include a consideration of all the various mechanisms that serve to maintain the circulation; for the toxic agents that develop during infections may attack the circulatory organs in a variety of places; heart, blood-vessels, as well as the nervous apparatus which are intrusted with the task of regulating the action of the heart and the caliber of the vessels, may be diseased either singly or combined.

We may see the most variegated clinical picture, the disentangling and interpreting of which often present great difficulties. For the physician at the bedside sees only the results of these complicated processes, that is, their ultimate effect on the circulation. The case is exactly parallel with disturbances of the heart rhythm (see p. 458). For what the physician observes in these conditions due to infection are the effects of the most complicated influences, and he at first has no means of knowing by which one of a number of possible methods they have been brought about. As was explained in connection with disturbances of the cardiac rhythm, the processes are so complicated that it does not seem possible that they can ever be cleared up by observation at the bedside alone; the question must be still further investigated, not only by systematic observation of patients by trained physiologists, but also by imitating the phenomena observed in man by means of animal experiments. These two methods have, in fact, enabled us to obtain a certain insight into this question.

Infection and fever always have an effect on the rhythm of the heart. Not merely that a rise in the body temperature is accompanied by an increase in the frequency of the heart-beat, and the latter is either increased or diminished when the force of the heart is impaired; but, in addition, the various diseases exercise different effects on the heart rhythm, evidently dependent on the nature of the intoxications with which they are associated. I recall merely the relative slowing of the pulse in typhoid fever and its acceleration in scarlet fever and most streptococcus-infections. These things are discussed in connection with infectious diseases, and need not, therefore, detain us here.

The function of the tissues is very much under the influence of the pressure and velocity of the blood. Both these factors *may* remain entirely unaffected in infectious diseases, as the investigations made by Mosen* demonstrate with absolute certainty. But this is by no means always the case. On the contrary, we cannot help being reminded of the abundant opportunities in infectious diseases for causing disease of the heart, blood-vessels, and vasomotor centers.

Although it has never been absolutely proved it may be assumed with confidence that the poisons generated by the bacteria may produce degeneration of the muscle-fibers and true inflammation. The blood-vessel walls also, both in the heart and in other organs, as the kidneys, for example, sometimes become inflamed. These changes, as we have seen elsewhere, diminish the functional capacity of the heart, and it should be part of the description of the individual disease to set forth to what extent this impairment takes place; by what symptoms it is accompanied, and at what period of the disease it occurs.

* Mosen, "Archiv für klinische Medicin," vol. lii, p. 601.

We also know that a great many different parts of the nervous system sometimes manifest functional disturbances during infectious diseases. Here we are interested only in the central endings of the nerves going to the heart, and in those of the vasomotor nerves. The rhythm, as well as the strength, of the cardiac contractions can be influenced by stimuli from the medulla oblongata, and it is easily possible that such an influence actually is exerted in infectious diseases.

There is unquestionably a disturbance of the vasomotor apparatus for the cutaneous vessels in all infections associated with fever; in fact, there is the closest possible connection between this disturbance and the occurrence of the fever. Irritative and paralytic phenomena are closely interwoven, but a closer understanding of these important and interesting matters still remains to be gained. Changes in the condition of the cutaneous vessels do not directly affect the general circulation, if we may be allowed to judge by the results obtained in animal experiments made for the purpose of determining the influence of the individual vascular regions of blood-pressure; there is no doubt that in the case of man also the splanchnic vessels exert a marked influence on the blood-pressure. One is forced to conclude, therefore, that if the central endings of the nerves of one vascular region become labile,—and we know that this is actually the case during fever, so far as the cutaneous vessels are concerned,—it is probable that the endings of nerves belonging to any other vascular area may be equally labile.

The clinical picture of vasomotor paralysis, or, more correctly, paralysis of the splanchnic vessels, has been rendered familiar by animal experiments: overfilling of the very wide abdominal vessels, resulting in anemia of the rest of the body; incomplete filling of the heart, and accelerated heart action with low blood-pressure. There is no marked dyspnea, and stasis is absent; the color of the skin is extremely pale, not blue. The clinical picture, therefore, presents a number of points of similarity to that of ordinary heart failure, although there are also a number of points of dissimilarity, and the mere appearance of some patients suffering from circulatory disturbances during an infectious disease sometimes suggests that vasomotor palsy is the most important part of the morbid process. Now it has been shown, by an excellent experimental investigation carried out by Romberg and his collaborators,* that the circulatory disturbances which not infrequently make their appearance in profoundly infected animals are caused almost exclusively by central paralysis of the vasomotor apparatus. The heart plays only a secondary part in the production of these circulatory disturbances.

These facts are exceedingly important and valuable; for they may be utilized to draw conclusions applicable to man. In the infectious diseases of man, also, vasomotor palsy quite possibly plays an important part in the explanation of circulatory disturbances. It remains to be shown to what extent this is really the case, in the several diseases, and to what extent the heart itself becomes diseased in these conditions.

It is always found that the heart and vasomotor apparatus are only injured in a certain proportion of the cases. What are the factors that favor disease of the heart and vasomotor apparatus? Is anything known about this point? In the case of palsies in the nervous system the great question, which has not even been touched upon as yet, whether what we call a weak—hereditarily weak—nervous system is more apt to fall a victim to the poisons of infectious diseases than a sound one, remains to be decided. Many observations might be interpreted as supporting this view, and the

* Romberg, Pässler, Bruhns, and Müller, "Archiv für klinische Medicin," vol. lxiv, p. 652.

question is in urgent need of careful investigation. But it is obvious that this question can be answered only in the case of patients with whose family history and conditions the physician is familiar, and, therefore, as in so many other matters, the family physician alone can help us.

It would be most desirable to know, in the case of the heart, whether factors which affect its functional capacity unfavorably, such, for example, as anomalies in the nutrition or improper habits of life, also render the organ more sensitive to the poison of the infectious diseases; nor has the significance of antecedent diseases of the heart ever been determined with sufficient accuracy.

The pathologic changes observed in the heart muscle in the various infectious diseases are essentially the same; they are briefly the degenerations and inflammations that develop under the influence of microbiotic poisons. The skeletal muscles not infrequently become diseased as well as the myocardium; in fact, the morphologic processes were first studied by Virchow and Zenker on skeletal muscles,* and Hayem, in an excellent article,† showed his acumen and breadth of view when he drew a parallel between the processes that occur in the two kind of tissues. The first observations were made on a typhoid heart, but the most minute observations were obtained in diphtheria. We shall begin with diphtheria, because we know most about its relations to the changes in the circulatory organs from both an anatomic and a physiologic standpoint

DIPHTHERIA.

The frequency of circulatory disturbances is quite characteristic of the general picture of diphtheria. It is true that some uncomplicated cases of the disease run their course without any marked symptoms referable to the heart and blood-vessels; but, on the other hand, the poison of diphtheria is evidently specially apt to injure the circulatory organs, and the frequent complication of the diphtheria infection with other bacterial invasions and local diseases affords special opportunities for the development of the disease of the circulatory system.

It has been learned, by a long series of very careful investigations, that diphtheria is very often followed by profound changes in the parenchyma fibers of the heart—vacuolated, granular, fatty, and waxy degeneration as well as degenerations of the nuclei, besides true interstitial inflammation of the musculature.‡ These alterations are almost always accompanied by exudations on the pericardium and frequently also on the endocardium. There is still a good deal of discrepancy between the statements and views of different authors in regard to numerous details, the frequency of the kind of change, and especially the relation between parenchymatous and interstitial processes. This discrepancy is probably due in part to the methods of examination employed; but it seems to me that the disease itself produces a great variety of phenomena. There is no doubt that degeneration of the parenchyma fibers takes place gradually and that, until the middle of the second week, the most minute changes in the muscle are

* Virchow, in the celebrated article, "Ueber parenchymatöse Entzündung," "Virchow's Archiv," vol. iv, p. 261. Zenker, "Ueber die Veränderungen der willkürlichen Muskeln bei Typhus abdominalis," Leipzig, 1864.

† Hayem, "Archives de physiol. norm. et pathol.," 1869, vol. ii, p. 699, and 1870, vol. iii, various papers, especially p. 284.

‡ For a detailed description of these changes see Romberg, Rosenbach, Hallwachs, Ribbert.

chiefly parenchymatous. About this time the interstitial exudation begins, and then dominates the process in the circulatory apparatus. The exudate may be unattended by any visible parenchymatous changes, or it may be combined with such changes in a variety of ways. As has been stated, there is still much confusion on this point. It does not seem to me that general pathologic observations or observations made on other organs, as, for example, the kidneys, justify the assumption that there is a fundamental distinction between the two kinds of processes; but it is nevertheless true, as Romberg points out, that the observations reported are against the obvious assumption that the accumulation of leukocytes between the fibers is always secondary to known degenerative changes. More investigations are required to determine what effect the causes of inflammation under all circumstances have on the structure of the parenchyma cells.

There is no doubt that the *circulatory disturbances during convalescence*, presently to be described, must be attributed to the existence of an acute interstitial myocarditis;* the two processes are absolutely parallel,† and it will not be difficult to show, by the description of the symptoms, that the clinical phenomena rest on insufficiency of the inflamed heart muscle as a foundation.

The conditions are much more obscure during the *early period of the disease*. At this time there is no interstitial myocarditis; the degenerations in the muscle-fibers have already begun, and may have attained a variable degree and extent in different cases on the same day of the disease. The intensity of these degenerations is unquestionably great enough in many cases to afford a perfect explanation of the diminution in the strength of the heart; if for no other reason, because a large quantity of functionating plasma has been destroyed.‡ In other cases the changes in the fibers are insignificant or altogether wanting at this period. In such cases toxic disturbance of the heart is by no means impossible in my opinion; § for all that we know about the subject indicates that these parenchymatous degenerations develop under the influence of poisons. Besides, Rolly's‖ investigations demonstrate with absolute certainty that the heart itself is damaged by the diphtheria poison.

But, as Romberg has very correctly pointed out, there is a third factor: the disturbance of the circulation due to vasomotor palsies. The above-mentioned investigations have shown that in acute fatal diphtheria-infection in the rabbit vasomotor palsy is an important factor. This observation may be unhesitatingly applied to human diphtheria, for the clinical picture of the profound circulatory disturbance during the early period of the disease presents many features which point to a vasomotor palsy. Of course, in thus using the results of animal experiments to explain the processes occurring in man, it must be borne in mind, as in all cases of this kind, that the morbid phenomena, owing to the fact that they are often forcibly produced and that the clinical course is abnormally con-

* The historic aspect of the question is treated by Romberg.

† The proofs are given by Romberg, Hallwachs.

‡ Compare Jäger, "Münchener medicinische Abhandlungen." Romberg, "Herzkrankheiten."

§ Hesse, "Jahrbuch für Kinderheilkunde," vol. xxxvi, p. 19 (from Heubner's clinic).

‖ Rolly, "Archiv für experimentelle Pathologie," vol. xlii, p. 283 (from Gottlieb's institute). Compare Beck and Slapa, "Wiener klinische Wochenschrift," 1895, No. 18. Enriquez and Hallion, "Archives de physiologie," 1898, p. 393.

centrated, sometimes exhibit a certain onesidedness—an exaggeration in some one direction. I think it will require further observation on man to determine how much each system of the body contributes to these disturbances, and I believe it will be found that the heart takes some part in these disturbances also.

I will not venture to express an opinion about the changes found in the nervous apparatus, for example, the disease of the abdominal nerves described by Veronese. Equally premature, in my opinion, would be the attempt to explain the clinical picture by some disease of the heart nerves.* For the present all we can say is that *the diphtheria poison injures the heart muscle and the vasomotor apparatus.*

Symptoms.—During the first week of the disease circulatory disturbances are probably less frequent than later on. But they do occur at this time, especially when the infection is very severe from the beginning and there is a pulmonary complication. The pulse becomes small and soft. The rate may be unchanged, but more frequently the heart action is greatly accelerated, sometimes also distinctly infrequent, not rarely irregular and unequal. The usual physical examination of the heart does not necessarily reveal anything abnormal. Dilatation and systolic murmurs, such as we shall presently discuss more fully, are sometimes present even at this early stage of the disease.

The patients are often exceedingly pale and usually a little cyanotic. Sometimes they complain of a sense of pressure and pain on the chest or in the abdomen; the latter is most frequently due to swelling of the liver. The albuminuria, which, as a rule, is already present, may increase or, if it was not present before, it now makes its appearance. Occasionally vomiting is observed. Without the occurrence of severe stasis, the patients sometimes become apathetic or somnolent, but occasionally retain consciousness to the end, and die, unfortunately very often, within a few hours, or, in other cases, days. At this early stage of the disease any severe symptom is always of grave prognostic omen. Mild symptoms may subside even at this stage.

These are the cases in which vascular palsies unquestionably play an important part, as no one will doubt who attentively studies his patients with the eyes of the physiologist and is familiar with Romberg's animal experiments. But I believe in the case of man the heart also shares in the process. I need only call attention to the slowing, irregularity, and inequality of the heart-beat and to the dilatation which is not infrequently encountered even at this period of the disease in combination with the above-mentioned phenomena.

During the latter stages of the disease, abnormality of the heart action becomes more and more prominent as the cause of the circulatory disturbances. Symptoms of disturbed heart action often make their appearance even at this stage; while the diphtheritic infection in addition manifests itself by a number of other phenomena referable to a great many different organs.

But the most curious thing is that, for a long time, weeks after the disease itself has run its course, and when the patient is convalescent, there is still danger of the occurrence of severe circulatory disturbances; in other words, acute myocarditis—for that is always the condition in these cases—may occur even at this late date. Possibly this fact justifies

* Vincent, "Archives de médecine expérimentale," vol. vi, 1894, p. 513, contains bibliography. Compare Schamschin, "Ziegler's Beiträge," vol. xviii, p. 64.

Romberg's view in regard to the production of myocarditis. He attributes it not to the continued action of the diphtheria poison, but, so to speak, to the compensation of injuries which the poison has caused. To this might be added the curious changes which the specific poisons of infectious diseases appear to undergo in the body, or possibly secondary infections. This will be referred to again. Patients who develop this form of myocarditis—they are usually children—often present no general signs whatever. In other cases, however, they become restless, excited, peevish, or apathetic, and even somnolent as the cardiac symptoms begin. Marked pallor, coming on with variable rapidity, is observed in a number of cases, and violent vomiting not infrequently makes its appearance. Both these symptoms are always extremely grave. Fever is usually absent in post-diphtheritic myocarditis; very rarely slight elevations of temperature are observed in the beginning.

It is a fact to be remembered in connection with all the symptoms that occur in association with this form of myocarditis that they are only in part dependent on the myocarditis, and that the primary trouble is also partly responsible for them. The physician must determine by careful observation the condition of each individual case in this respect: paralysis, severe bronchitis, bronchopneumonia, and inspiration pneumonia, of course, complicate the clinical course very much, but have nothing to do with myocarditis as such. Among the cardiac symptoms the most important are disturbances of the heart action, especially an increase of the frequency. This may develop gradually and may persist; sometimes, however, it occurs suddenly, and in such a case there may be at least a certain outward resemblance to the symptoms of paroxysmal tachycardia.*

Slowing of the action is not infrequently encountered, and is said, as a rule, to be a bad prognostic omen, because it indicates a severe degree of cardiac weakness; rapid sinking of the pulse-rate is particularly ominous. In order to determine these symptoms it is almost needless to say that the patient's age, the temperature, and other existing complications must be carefully taken into account. Other changes are irregularity and inequality of one kind or another, enlargement of the heart to the left, to the right, or in both directions; reduplication of one or both sounds; very often impurity of the first sound or a systolic murmur, which must be attributed to muscular insufficiency of the mitral valve. The accentuation of the second pulmonic sound is another evidence of mitral insufficiency. After severe cardiac weakness gallop rhythm or embryocardia is sometimes observed.

The arterial pressure falls,† the pulse becomes small and soft, passive congestion not infrequently develops in various regions of the body; the liver becomes enlarged, edema makes its appearance, the quantity of urine diminishes. The face, and especially the regions surrounding the eyelids, are usually puffy; although this symptom may be in part due to the local affection and possibly to disease of the kidney.

Albuminuria is very frequent. It recurs in those cases in which it had already disappeared. If it is still present as the result of the infection itself, the excretion of albumin usually increases. With the microscope, a few white blood-cells and hyaline casts are found.

The entire illness may run its course without any subjective symptoms

* Compare the cases cited by Proebsting on p. 360 of his essay in "Archiv für klinische Medicin," vol. xxxi.

† Compare Friedmann, "Jahrbuch für Kinderheilkunde," vol. xxxvi, p. 50.

referred to the heart, but this again is in part explained by the fact that most of the patients are children. Quite frequently, however, they complain of pressure on the chest and heart-fear, pain, and palpitation. Conditions very closely resembling angina pectoris may even occur, and all these symptoms, or some of them, by themselves cause many patients a good deal of distress. The children sometimes suffer discomfort from congestion of the liver. There is no doubt that the subjective symptoms are much greater in the second period of the disease than in the first, probably in part due to the fact that the heart is more profoundly involved during this period of the disease, and partly to the fact that the children are perfectly conscious because the general infection by that time has run its course.

It is said, however, that all these cardiac symptoms may be absent and that the pulse even may retain its normal character until shortly before sudden and unexpected death. It would obviously be improper for a writer to call positive statements in question. It must be remembered, however, that a perfect familiarity with the morbid symptoms is necessary before they can be discovered, and that our accurate knowledge of diphtheritic myocarditis is very recent; so it will surprise no one that, as this knowledge spreads, these symptoms of diphtheritic myocarditis will be recognized correspondingly earlier in the disease. Romberg and Veronese express themselves to the same effect: cases of *unexpected* death will diminish in proportion as the heart is more regularly subjected to minute examination in diphtheria.

The above-mentioned circulatory disturbances are encountered in from 10 to 20 per cent. of all the cases of diphtheria. On this point the statements of Romberg and Schmaltz agree. So far as can be judged at the present time, serum treatment has no appreciable influence on their frequency. While the prognosis is very unfavorable when the phenomena occur during the early stages of the infection, probably depending on the profound intoxication of the central nervous system, recovery takes place in more than half of the cases which begin later.

It has been stated that this myocarditis of convalescents may appear after the intoxication proper has run its course, and is often protracted for weeks or even months. Either the symptoms of cardiac insufficiency gradually increase and then subside again, or they persist with variable intensity for weeks; the patient, so to speak, hovers between life and death. These cases afford a striking illustration of the curative power of uniform, absolute rest and of the great injury caused by even the slightest bodily exertion or psychic excitement. Other cases of this kind progress irresistibly to complete cardiac insufficiency and death, which may occur in a few days or may be deferred for two or three weeks. Toward the end the child often becomes somnolent; although, on the other hand, death may be accompanied by the most frightful conditions of intense excitement and terror.

Death may come on gradually as the result of a progressive increase of all the symptoms; or it may, as has been mentioned, occur suddenly. At all events, these patients have to be watched with the very greatest care and attention by the parents as well as by the physician, often for many weeks; and so long as the objective findings render such supervision necessary, a guarded prognosis will have to be given.

If the child escapes,—the proportion of recoveries is about two-thirds,—all the symptoms may disappear completely. Acute myocarditis,

as we know, is a curable disease, even when it occurs under other circumstances, and the heart of a child possesses extraordinary powers of recuperation. It may be a long time, however, before the signs of disease disappear from the heart; for instance, the muscular mitral insufficiency may persist for weeks and months and may disappear at the end of that time.

Complete recovery does not occur in all cases. It cannot be denied, either theoretically or on the basis of clinical experience, that the inflammation of the muscle may progress and that the acute myocarditis may develop into a chronic form.

French investigators particularly lay great stress on the previous occurrence of diphtheria as a factor in the etiology of chronic myocarditis. Whether or not valvular lesions develop after diphtheria I will not venture to decide. While the endocardium is frequently inflamed in diphtheria, it is certainly rare for the valves themselves to be affected. One thing is certain, however; a considerable proportion of the children for years after the acute disease has run its course still have dilatation of the heart, a systolic murmur, and accentuation of the second pulmonic sound; in other words, all the signs of mitral insufficiency. It is quite possible that this mitral insufficiency may become permanent. The question is whether we are dealing with phenomena of a purely myocarditic nature or whether the inflammation which takes place in other portions of the endocardium finally also attacks the valves.

Schmaltz* has made a careful study of the course and termination of the diphtheritic circulatory disturbances and has reported his results in a number of fine treatises. He shows how very obstinate these processes are. Even when the heart presents nothing morbid during the acute disease or the weeks immediately following, the symptoms of carditis may, nevertheless, develop and become the starting-point of a chronic inflammation. We, therefore, observe a close similarity to the processes that occur in rheumatic polyarthritis.

And, finally, there is another very important point. As has been stated, the *restitutio in integrum* in many cases of diphtheritic heart disease that end in recovery is so complete that an ordinary physical examination reveals nothing whatever.

But it is a question, nevertheless, whether the heart is really absolutely sound, especially whether it has the same functional capacity as before, or, in other words, whether its capacity is adequate to the demands incident to the individual's mode of life. The more we study heart diseases, the more we are impressed with the extreme complexity of the etiology; an infectious disease that has run its course many years before a definite disease of the heart develops, appears, nevertheless, to be capable of influencing the development of that disease, although the interval between the two events may have been marked by perfect health. Possibly the first disease prepared the soil for the second.

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RHEUMATIC POLYARTHRITIS.

Among infectious diseases polyarthritis stands out as the classic cause of heart disturbances and is, accordingly, discussed at length in the section on Valvular Lesions. But it cannot be neglected altogether in this connection because, like diphtheria, articular rheumatism also leads to acute myocarditis. That this is relatively so little known is due to the prevailing and in many respects incorrect views in regard to the pathology of the conditions designated valvular lesions.

Many of the processes that take place in the heart in these diseases are attributed to endocarditis without sufficient grounds for doing so. To give a special instance, we now know that it is altogether wrong to make a diagnosis of endocarditis as soon as a systolic mitral murmur is discovered, even if the murmur is accompanied by accentuation of the second pulmonic sound. The great significance of the muscular function in the closing of the valves is now well known, and in diseases of the heart characterized preëminently by anomalies of the muscular action, muscular insufficiency of the mitral valve is, in fact, extraordinarily frequent.

Anatomic studies have shown that the entire heart becomes diseased in consequence of rheumatic polyarthritis, exactly in the same way as in the other infectious processes. The signs of inflammation with the exudation of numerous round-cells make their appearance in the myocardium, the pericardium, and the endocardium, in the latter chiefly at the valves. Extensive parenchymatous degenerations are also said to occur.*

[For a more detailed account of the myocardial changes after rheumatism see Romberg, "Lehrbuch der Krankheiten des Herzens und der Blutgefässe," 1906; Krehl, "Pathologische Physiologie," fourth edition, 1906; Aschoff and Tawara, "Die heutige Lehre von den path.-anat. Grundlagen der Herzschwäche," 1906; Aschoff, "British Medical Journal," 1906, vol. ii, p. 1103. Aschoff and Tawara describe not only simple necrotic areas and scars that might follow such areas, due to emboli derived

* West, "Lancet," January 30, 1886. Cited from "Virchow-Hirsch," 1886, ii, p. 64.

from the endocardium or to stenoses of the vessels, but also submiliary nodules in the perivascular connective tissue and under the endocardium. The nodules are made up of cells of various kinds, including large ones with giant nuclei. They were found in rheumatic cases, but not in all of the cases examined. Geipel has confirmed the findings in a few cases. Aschoff is properly reserved in the interpretation of the nodules, admitting that they may cause cardiac weakness, in some cases, from interference with the conducting fibers. His general conclusion regarding heart weakness in such cases is that it is due to causes outside the heart. The possible relation of even slight changes in the myocardium to the conducting fibers, however, must be worked out by extensive and painstaking examinations on the lines laid down by Aschoff.—Ed.]

The extent to which the individual parts of the organ are involved in the morbid process varies a good deal in different cases; one part may apparently be so much more affected than the others as to give the physician the impression that it is the only part affected by the disease.

Rheumatic pericarditis and endocarditis are discussed at length in other sections of this volume. In these processes, especially in endocarditis, the muscle, as a rule, is also diseased. In order to understand the nature of endocarditis we must have recourse to the old conception of carditis, and as disease of the muscle is unquestionably the determining factor in the explanation of functional disturbances, it would be quite possible to include these matters in the present discussion. But custom forbids. To avoid misunderstanding, however, it must be emphatically stated that the distinction is purely artificial and based only on intrinsic reasons; endocarditis and myocarditis, more particularly, in the broadest sense of the term, represent parts of the same whole.

It is characteristic of both conditions that they frequently begin after articular rheumatism and run their course in one of three ways. In very rare cases the course of the disease is very severe and ends in death; or complete recovery may take place; but this is not the usual course either. In the majority of cases the acute inflammation becomes chronic and persists for years and decades. The reason why the process becomes permanent is not definitely known; it may be, as Romberg suggests, that it is due to the frequent relapses of the acute arthritis. I am inclined to think there are, in addition, certain other factors; for chronic endomyocarditis is observed even without renewed articular symptoms, and such cases must be explained in the same way as chronic inflammation in general.

These chronic processes, which rarely attack the muscle alone, more frequently involve the endocardium as well, and are, therefore, as has been mentioned, treated chiefly in connection with valvular lesions of the heart; as a rule, they lead ultimately to fatal cardiac insufficiency. In many cases severe symptoms of asystole may have been present at different periods of the patient's previous life, and the disease frequently lasts many years. These temporary disturbances of the circulation, as well as the terminal attack, are brought on partly by the interference with the circulation caused by the valvular defect and partly by the impairment of the function of the muscle. And it will depend chiefly on the nature of the injury to the muscle whether the condition can be influenced by treatment or is irremediable.

Myocarditis accompanying articular rheumatism, at all events, begins frequently as an acute disease. During the course of a polyarthritis,

symptoms of a disturbed heart action make their appearance, and the extent and intensity of the articular rheumatism do not have any direct influence on their production. The heart may become diseased in the mildest cases, as is frequently observed among children,* and in such cases an observant examiner not infrequently fails to discover the origin of the circulatory disturbances, especially in those rheumatic conditions in which there are no joint symptoms whatever.†

The patient may complain of a sense of pressure, dyspnea, oppression, and painful sensations in the precordia; but not infrequently such phenomena are altogether absent, and only a conscientious objective examination reveals the cardiac affection. Dilatation of the cavities of the heart, disturbances of the rhythm, and insufficiency of the mitral valve are the first signs; any one of these signs may be present by itself or they may be combined. Slight deviations from the normal are more frequent than marked, not to say severe, symptoms; in fact, myocarditis in this infection, more than in any other, generally develops gradually and runs a slow course.

The **diagnosis** is difficult in a good many respects, particularly at the very beginning. It is regarded as particularly difficult because it is said that in polyarthritis disturbances of the myocardium occur that have nothing to do with structural disease, especially inflammation of the muscle, being merely "functional" in character; that is to say, attributable to poisons or some other unknown cause. These conditions, it is asserted, may give rise to alterations of the heart action, systolic murmur, or even dilatation of the ventricles. If that were true, the diagnosis of myocarditis would, indeed, be not only difficult, but altogether impossible, at least at the beginning of the trouble. Nor can it be denied, if it be permitted to draw analogous conclusions from other infectious diseases, that there is no reason why one should not assume toxic conditions of the heart capable of producing functional anomalies. Hence it is, no doubt, extremely difficult at first to make the distinction. But since, as we saw in diphtheria, the intoxications are intimately concerned with the development of myocarditis, I do not believe that their sphere should be too much restricted. At most the question might be asked whether it is possible for toxic conditions of the heart muscle to occur in polyarthritis rheumatica and run their course without causing any signs of reaction in the musculature.

In the second place, it is quite difficult to determine whether, in a given case, endocarditis or myocarditis is chiefly responsible for the clinical phenomena. I do not think the question ought to be put whether endocarditis or myocarditis is present; for both conditions are usually associated in the form of a true carditis. In forming an opinion of the development of a valvular lesion it is important to determine to what extent the valves are implicated in the process. Disease of the aortic valves is not likely to present any difficulties, because aortic insufficiency without changes in the semilunar valves is extremely rare. Again, a diastolic murmur at the apex renders the diagnosis of mitral stenosis practically certain; but, as a matter of fact, mitral stenosis is only exceptionally found at the beginning of myocarditis. The chief difficulty is encountered with endocarditic mitral insufficiency. All that can be said on this subject for the present is that endocarditis may be present without, and myocarditis with, a systolic murmur in the second left intercostal space or at the apex. Of course, one

* Hirschsprung, "Jahrbuch für Kinderheilkunde," n. s., vol. xvi, p. 324.
† Bruce, "British Med. Journal," 1890, vol. i, p. 937.

will always be inclined, in the absence of murmurs, to attribute disturbances of the heart action and dilatation of the heart to myocarditis; and if there is a murmur, its significance will be determined chiefly by its constancy or variability. In any case, however, great caution is to be advised.

The **course** of rheumatic myocarditis is relatively little known. It very rarely leads to fatal cardiac insufficiency.* How often inflammation of the myocardium, chiefly confined to the muscle, becomes chronic, we are as yet unable to determine. As a rule, at least, myocarditis is combined with endocarditis, and in that case the muscular disease, in my opinion at least, has a very great influence on the course of the valvular lesions.

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TYPHOID FEVER; TYPHUS FEVER; SCARLET FEVER; MEASLES.

The changes in the circulatory organs that occur in typhoid fever and typhus fever have been known longer than any others;† they furnished the material for the first observations, and on them the first general views were based. The reason that we now know more about diphtheritic myocarditis than about the typhoid form is partly because cardiac symptoms are more frequent in diphtheria and partly because of the possibility of attacking the question by means of animal experiments. In the older writings the distinction between typhoid and typhus fever is not sufficiently clear. In typhus fever we have no extensive clinical and anatomic observations from the modern point of view. In the case of typhoid fever and scarlet fever more abundant anatomic investigations of the circulatory disturbances occurring in the various stages of the disease would be, to say the least, desirable. In measles no anatomic observations whatever have been made, so far as I know.

Circulatory weakness may occur in these infections at the height of the disease. It is more apt to occur and occurs more frequently in some of these infections than in others, being unquestionably most rare in measles and probably most frequent in typhoid fever. In the latter disease, especially, as it chiefly attacks adults, the effect on the circulation varies a great deal, depending on the individual's constitution, on previous exposure of the heart to injurious influences, and, in fact, on the structural and functional conditions of the heart and oblongata in general.

These questions cannot be discussed in this connection—practically, the same conditions are encountered in all infectious diseases.

During the febrile period of these diseases, when the function of the various portions of the brain suffers in so many different ways, central vascular palsies manifestly play the most important rôle. The whole

* Compare the cases of Romberg and Weill and Barjon.
† See historic review by Romberg.

appearance of typhoid-fever patients who develop "heart weakness" at the height of the disease clearly indicates paralysis of the splanchnic nerves. This, taken in connection with the facts experimentally demonstrated by Romberg and his collaborators, in my opinion shows very positively that the circulation has been damaged by vasomotor paralysis. But, as was explained in connection with diphtheria, it cannot be said that the heart has no share in producing the circulatory disturbance. For no one who will carefully observe the heart during the febrile period of any of these infectious diseases will fail to find phenomena which can only be explained on the ground of functional anomaly. These phenomena are irregularity and inequality of the heart action; dilatation of the left, more rarely of the right, ventricle; displacement of the apex-beat to the left; weakening, and occasionally accentuation of the apex-beat; systolic murmurs, sometimes accompanied by accentuation of the second pulmonic sound; weakening of the second sounds; and accentuation of the second pulmonic sound without mitral insufficiency (probably due to weakness of the left ventricle, while the right ventricle does better work than usual). Besides, there are a few isolated anatomic reports about the changes of the heart muscle during this period of the disease. It is true they are still much too scanty to afford an adequate explanation, and further investigation would be very welcome.

Another difficulty arises from the impossibility, in these conditions, of denying the occurrence of functional disturbances due to toxic effects on the heart muscle. It is almost needless to say that one is not justified in assuming a functional disturbance due to intoxication until the heart has been properly examined for the purpose of excluding structural changes. It is true that structural changes also would be produced by poisons, and in that sense there is no contradiction, nor even a fundamental difference, between intoxication and myocarditis. But, fortunately, we still have sufficient anatomic training to desire a morphologic basis on which to base our etiologic craving.

In my opinion there is nothing gained by mentioning all these things specifically in connection with each individual infection, since all we want here is a general idea of the extent to which the heart is involved in these morbid conditions. It has already been mentioned that some of the causal micro-organisms and the substances which they produce affect the circulatory organs more easily than others. But these are things that can only be discussed in connection with a full description of infectious diseases.

In typhoid fever structural changes in the musculature of the heart are known to begin at the end of the second week. This does not imply that such changes may not be found earlier under certain circumstances. But after the second week the physician must always be prepared for more or less profound changes in the circulation, and especially in the heart action.

In the same way as after diphtheria, but much more rarely than in the latter disease, the febrile period in the infections now under consideration is followed by the appearance of symptoms which must be regarded as the expression of an infectious myocarditis, exactly as in diphtheria. This interpretation is partly based on direct observation and partly on analogy.

In *typhoid fever* we owe our knowledge of these matters to Romberg.* One or several weeks after defervescence the pulse becomes accelerated, not only as the result of muscular movement or excitement, but also during

* Romberg, "Archiv für klinische Medicin," vol. xlix, p. 434.

rest. Later it also becomes irregular and unequal, and the patients at the same time have a sense of palpitation. This is followed by dilatation, sometimes by functional mitral insufficiency, as in the other forms of myocarditis. As a rule, recovery takes place after a few weeks; but as in diphtheria, sudden death occasionally occurs either quite spontaneously or after bodily exertion.

I have often seen this form of myocarditis. The great increase in the frequency of the pulse, which in one of my cases was accompanied by a marked rise of the blood-pressure, and the variability of the auscultatory phenomena, seem to me exceedingly characteristic. Sometimes the findings varied from time to time. At one time a loud systolic murmur with considerable accentuation of the second pulmonic sound was heard; at another time only the accentuation of the second pulmonic was present; or the sounds were perfectly pure and uniform. The dilatation also, especially toward the right, is subject to great fluctuation. Some patients are quite unaware of these disturbances; others complain of palpitation and anxiety and a certain degree of pressure on the chest. Temperature and urine present nothing unusual. After several weeks the symptoms all disappear. I was unable to learn anything about the subsequent condition of the heart.

Scarlet fever often begins with the most violent symptoms. As we know, the progress of events is very rapid in such cases. Thus Romberg observed dilatation of the heart during the very first days of the illness. In the second week he saw,* in children, while the quantity of urine was diminished and the temperature was high, retardation or acceleration, irregularity, and inequality of the heart action. At the same time the first sound was impure, and there was slight dilatation of the ventricles. Occasionally there were subjective symptoms, pain in the precordia, and oppression. In one case the symptoms gradually disappeared in the fourth week. Nothing more definite is known about the duration of the disease. Sommer† saw a case of fatal recent myocarditis, with marked dilatation of the heart, following an attack of scarlet fever. Purulent myocarditis has also been observed.‡ I myself once saw, seven weeks after the end of a slight attack of scarlet fever, an increase in the frequency of the pulse, which continued for several weeks, in the neighborhood of 120. The heart was enlarged; at the apex a systolic murmur was heard, and the second pulmonic sound was accentuated. Four weeks later the sounds had become pure again, but the increase in the pulse-rate persisted. The kidneys at no time presented anything abnormal.

At the height of the disease in *measles* circulatory disturbances occur, but probably only in connection with severe capillary bronchitis and bronchopneumonia, and even then the complication is not frequent.

After the febrile period, also, disease of the heart appears to be very rare. At least, as good an observer as Jürgensen§ remarks: "there is no such thing as injury to the heart due directly to the poison of measles." Nevertheless, the heart does occasionally suffer, although this statement is based chiefly on clinical observation. Wunderlich‖ long ago mentioned measles as the cause of chronic heart disease. Working under Gerhardt,

* Romberg, "Archiv für klinische Medicin," vol. xlix, p. 43.
† Sommer, "Charité-Annalen," 13. Jahrgang, 1888, p. 647 (Henoch).
‡ Goodhart, "British Medical Journal," 1879, p. 818.
§ Jürgensen, "Measles," in "Nothnagel's Practice," English edition.
‖ Wunderlich, "Archiv für physiologische Heilkunde," vol. i, 1849, p. 451.

Breyer* in Würzburg observed heart symptoms accompanied by a slight elevation of temperature in a number of patients during convalescence from measles. The heart action was slow and irregular; there was dilatation and a systolic murmur, with accentuation of the second pulmonic sound.

According to the views of his time, Breyer regarded this phenomenon as due to endocarditis, and as we lack sufficient anatomic material, nothing positive can even now be urged against his assertion. But, reasoning by analogy, from the observations made in typhoid fever, diphtheria, and scarlet fever, it seems to me much more probable that the functional disturbances were due chiefly to disease of the muscle. In one case Breyer saw a pericardial exudate.

I myself have seen three large epidemics of measles, two in Jena and one in Marburg, and in two of the Jena cases I observed exactly the same phenomena as those described above; although in both cases I am certain that the circulatory apparatus had previously been entirely normal, especially during the febrile period. There were no subjective symptoms whatever in my cases. Within a few days mitral insufficiency, with dilatation and acceleration, irregularity, and inequality of the heart action developed. The symptoms disappeared completely in the course of two or three months.

I wish to mention specially that neither in the Marburg nor in the Jena cases was there any complicating diphtheria to which the cardiac symptoms might have been attributed.

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FIBRINOUS PNEUMONIA.

The acute infiltration of large portions of the lungs under all circumstances throws an increased demand on the right heart, and not infrequently accentuation of the second pulmonic sound occurs to show that the demand is being met. But sometimes the heart is unable to respond, and its failure to do so in these cases manifests itself clearly by a dilatation of the right ventricle. It is well known that the condition of the heart and vascular apparatus exerts a very great influence on the course of pneumonia. Whether the injury to the heart is more important than the injury to the blood-vessels, how much significance is to be attached to the increase in the resistance due to the infiltrative process, and what is the effect of the pneumonic poison on the heart and the central terminations

* Breyer, "Mittheilungen über die Masernepidemie der Stadt Würzburg," Dissertation, Würzburg, 1883.

of the vasomotor nerves, are questions that cannot as yet be definitely settled. One gets the impression, however, that the resistance due to the infiltration in the lungs which must be overcome, is in proportion to the severity of the infection, the quantity and virulence of the circulating poison, and to the powers of resistance and functional capacity of the heart itself. Hence three factors influence the conditions of the circulation in pneumonia patients, and the course of the disease ultimately depends on the combination of these three factors.

The effect of pulmonary infiltration on the action of the heart need not be investigated in this section, as the essential points are given in the chapter on Modifications of the Heart Action in Diseases of the Lungs.

Nor have we anything to do with the question whether the condition of the heart in the beginning is more or less adapted to meet the demands thrown upon it by an attack of pneumonia; for that question must be decided chiefly by the results of clinical experience with pulmonary infections, which can, of course, only be done in the treatise on pneumonia (see vol. on Diseases of the Bronchi, Pleura, and Lungs).

The only questions we have to deal with here are: What effect is produced on the heart and vessels by the infection, and what morbid changes, if any, are produced in the heart? At the height of the disease, if there is any circulatory disturbance at all, the pulse is accelerated, small, soft, and occasionally irregular; dilatation of the heart is not infrequently present, the first sound is impure, and a systolic murmur may even be heard. In addition there may be venous congestion, and death often occurs within twelve to thirty-six hours with the picture of profound collapse. To what extent these disturbances are caused by toxic or inflammatory conditions of the heart and to what extent by intoxication of the nervous system, it is as yet impossible to say. So far as I know, no accurate investigations have been made, and the presence of pulmonary infiltration as a complication acts as a deterrent to *a priori* surmises. Lépine* reports that recent alteration of the myocardium is rarely found in pneumonia, and Homburger† also failed to find such a change in a number of patients in Kussmaul's clinic.

During or immediately after the crisis the circulation is endangered for the second time, even in the case of children, in whom the conditions are otherwise favorable for the most perfect functional capacity. As the temperature falls, the pulse-rate rises enormously, and the pulse becomes small and weak, and the patients go into collapse. They almost always recover, however, in spite of the grave symptoms. But we still lack any positive data to explain these phenomena. The most obvious explanation is that marked changes take place in the condition of the vessels, such as unquestionably accompany such a tremendous revolution in the organism.

During convalescence circulatory disturbances are encountered for the third time. This I have had occasion to observe in Jena in children of different ages. After the fever and the crisis had run their course without producing any anomaly in the circulation; after the child had fully entered upon convalescence and the condition in the lungs had subsided entirely or showed only slight remains of the process, a relapse occurred with general malaise. The temperature either remained normal or showed a

*Lépine, "Die acute lobäre Pneumonie," translated from "Nouveau dictionnaire de médecine," Vienna, 1883, p. 133.

†Homburger, "Untersuchungen über croupöse Pneumonie," Dissertation, Strassburg, 1879.

moderate rise to 38° C. (100.4° F). There was nothing to call attention to disease of the heart except the objective examination, which revealed a slight enlargement in both directions, an impure first sound at the apex, irregularity, inequality, and slight acceleration of the heart action, moderate swelling of the liver, and sometimes small quantities of albumin in the urine. The child would be kept in bed on indifferent treatment, and in one or two weeks all the symptoms disappeared. I never saw any dangers occur, nor was the attack followed by sequelæ.

No anatomic investigations were made, and the number of clinical observations is very small. But if we compare the symptoms with those observed after other infectious diseases, the supposition that they are due to myocarditis seems to be justifiable; at least, I can think of no other way out of the difficulty.

ACUTE MYOCARDITIS DURING AND AFTER OTHER INFECTIOUS DISEASES. PRIMARY ACUTE MYOCARDITIS.

An attempt was made in the foregoing to give a description of the symptoms which occur when the heart is specially involved in known infectious processes. The result of this description is that, just as the kidneys suffer both in structure and in function in many general infections, just as they may be involved in any of these infections although, as a matter of fact, they are not attacked in every instance, so the heart is always exposed to danger whenever micro-organisms or their products enter the circulation. Whether disease of the heart actually develops or not will depend on a variety of factors, which we as yet understand but very imperfectly. There is no doubt that very much depends on the nature of the disease-poison, which again shows the similarity to the behavior of the kidneys. Unfortunately, we know very little about the nature of the bacterial poisons, and for the present it looks as if it would be some time before we know anything about the common properties of those poisons which chiefly attack the heart. Nevertheless, there are a few data which justify the assumption that there are certain properties common to many different bacterial poisons, which in turn explains how it is that an affection of the heart may develop in practically every one of the infectious diseases.

On the other hand, it is possible that there may be properties inherent in the heart itself which either predispose it to, or protect it against, participation in the morbid processes. This point has already been mentioned. The natural disposition, as well as the constitution of the organ, no doubt has something to do with the question. Unfortunately, I have no facts to report.

I repeat, then, that in the infectious diseases so far enumerated the heart is comparatively often involved. It may also be attacked in any of the other infectious diseases, and the physician must always be prepared for it. Thus in the Leipzig clinic I once saw a typical myocarditis develop immediately after an attack of non-diphtheritic **gangrenous tonsillitis.** All the well-known signs (dilatation, arhythmia) were present. The disease ran a short course and ended favorably.

In a number of other infectious diseases isolated facts are known in regard to the occurrence of cardiac disturbances, for example, in *variola*, *malaria*, and *gonorrhea*. My reason for not discussing these diseases at greater length is that I cannot venture to give a positive

opinion about the behavior of the heart muscle in these conditions. That *endocarditis* occurs in gonorrhea has been definitely established, and it seems probable that it occurs also in malaria and smallpox. But whether the clinical symptoms are due to the endocarditis or to *changes in the heart muscle* it is, in my opinion, impossible to say, and the symptomatology of most of these diseases is even more unsatisfactory. The gonorrheal symptoms have been considered in connection with endocarditis, and as regards the other diseases, we shall have to wait for additional reports from observers familiar with the present views.

From these forms it is but a step to those cases in which **myocarditis,** so far as our present knowledge goes, is **primary** or idiopathic, or whatever other term one may wish to employ. We may be quite sure, reasoning by analogy, that these cases also are due to an infection of some kind; but how the patient acquires the infection is not known. The heart in these cases is the organ that is preëminently or even solely affected, and hence the myocarditis, the exact pathology of which is probably in every respect similar to the forms of acute infectious inflammation which have been carefully studied, must be designated as a primary myocarditis of unknown origin. This assumption is, at least, based on a foundation of well-known analogous cases; as regards the infection, I shall merely refer to the cryptogenetic diseases due to the streptococcus, and, as regards the organic lesion, to acute nephritis. Many of the cases which are still included in this group possibly belong to septicemia, for example, those described by Romberg ("Lehrbuch," p. 960), and others possibly owe their origin to a rheumatic infection.

In the midst of perfect health people suddenly fall ill with the symptoms of an acute infection. In one of my cases the attack was preceded by a complete drenching of the body; in two others, by an outbreak of violent temper. The latter observation is perhaps not without significance in view of the time-honored popular belief that mental excitement plays a large part in the production of heart disease.

Sometimes the cardiac disturbance is apparently at first entirely overshadowed by the symptoms of the general infection, although to the eye of an expert it is, nevertheless, quite plain. The patient complains of oppression, anxiety, and a certain painful sensation in the chest. The left arm may be the seat of drawing pains; it may feel heavy and cramped, and as if it had gone to sleep. This gives rise to a certain similarity to the symptoms of angina pectoris. The frequency with which anginoid symptoms develop in a great variety of conditions has already been explained and probably furnishes an explanation of the statement that true angina pectoris also occurs in association with infectious myocarditis. The heart action is, as a rule, disturbed, almost always irregular and unequal; sometimes it is accelerated, but quite frequently retarded to 52, 50, or 48. The pulse is always soft and usually small as well. Enlargement of the heart and modification of the heart-sounds may be present or not, or may develop at any time. The condition of the lungs, liver, kidneys, and subcutaneous tissues depends on the strength of the heart, particularly that of its individual portions. In this respect the cases manifest wide differences; as a matter of fact, the conditions in general are the same as have been discussed in the foregoing paragraphs.

Romberg had two cases which ended fatally after an illness of several weeks, and in which the autopsy showed a diffuse interstitial myocarditis.

The patients whom I personally observed were also ill for several weeks. At times there was high fever, with marked nervous phenomena, such as headache, restlessness, hallucinations, and delirium. At other times there was great prostration, and, toward the end of the illness especially, lassitude became extreme. The intensity and prominence of cerebral symptoms may give rise to great diagnostic difficulties, and as the pulse also is frequently slowed, one is very apt to assume the existence of some grave process in the brain, such as meningitis or abscess.

The fever was irregular in my cases, although it often persisted for days between 39° and 40° C. (102.2°–104° F.). The symptoms referable to the heart and other organs have already been mentioned. In my cases also there was a marked reduction of the strength of the heart for several days, and it is both practically important and theoretically interesting to know that this reduction in the strength of the heart is accompanied by slowing of the rhythm. As a matter of fact, Romberg's observations show that the patient's life is always in danger.

During convalescence, after the temperature has fallen and the heart has regained its normal dimensions, the action for some time continues labile and is easily influenced. The same therapeutic precautionary measures are necessary as in other forms of acute myocarditis. Ultimately, however, complete recovery may take place. I have even known patients who completely recovered their original strength and efficiency. But in others a certain permanent weakness of the organ remained. During rest the heart responds perfectly to all demands made upon it; but as soon as these demands are increased, either by psychic or physical influences of any kind, the patient experiences discomfort, although there is no actual cardiac failure.

The literature contains reports of a series of cases which, if they do not belong to this category, at least bear a close resemblance to the cases here described; but as in all these cases we know nothing about the cause, or rather, one may say, the identity of the causal micro-organism, it is very difficult to say positively whether these older cases in the literature belong in the same category or not.

In many respects there are points of contact with well-known conditions, such, for example, as the cardiac affections in polyarthritis. As has been mentioned, the irregular symptoms in these conditions are not necessarily pronounced, and in many instances one may remain in doubt whether one has to deal with genuine rheumatism or merely with pains in the limbs, such as occur in so many infectious diseases. Especially when the muscles alone are painful, this distinction may be very difficult to make.

There is also some similarity to the affections of the heart muscle which occur in the course of infections with pus micro-organisms. The invasion of the organism by streptococci or staphylococci may be local, or may take place from the local focus in the course of a general intoxication, or may be accompanied by a diffusion of the bacteria into the blood. The clinical picture of the disease varies accordingly, especially in respect to its intensity. These conditions are still designated by such terms as pyemia, septicemia, erysipelas, phlegmon, etc.; but it would be much better either to substitute for these terms definite etiologic concepts or to add the latter to the old names. The heart is quite generally involved in these conditions, but the details of the process await further investigation. Possibly some of the cases are due to in-

toxication, but there is no doubt that acute insufficiency and especially diffuse purulent myocarditis also occur. The latter is the form of inflammation of the heart muscle that has always been most widely recognized. Many of the older descriptions of "inflammation of the heart" refer to "purulent" myocarditis. It is frequently associated with diseases of the valves, that is to say, with endocarditis.

In severe cases of general streptococcus or staphylococcus infection the cardiac symptoms are completely merged in the general clinical picture; dilatation, murmurs, and especially great acceleration and weakening of the heart-beat are found. It is difficult to distinguish between a condition due to poisoning of the heart and interstitial and purulent inflammation of the muscle with or without involvement of the endocardium. Perhaps it will never be possible to do this; at all events, it is impossible now and will be until the pathologic process has been more minutely investigated and described.

Here we approach the transition to idiopathic acute myocarditis—at least, perhaps. In streptococcus and staphylococcus diseases the source of the infection may remain hidden and the process may be, at least chiefly, if not altogether, confined to one organ. If this organ is the heart, it is obvious, even on theoretic grounds, that the invasion of such microbes may easily produce the picture of acute primary myocarditis. From the symptomatic and anatomic standpoint there is nothing to be said against this view; on the contrary, the clinical phenomena tally perfectly, and it is only a question how far the theory is justified by etiologic investigations.

Even after streptococcus and staphylococcus infections at first local, cardiac symptoms are observed which possibly belong to this same category. Myocarditis following necrotic angina has already been mentioned. In erysipelas H. Lenhartz * observed dilatation and systolic murmurs during the febrile period; Traube reports profound circulatory disturbances during convalescence.

Romberg † observed dilatation of the heart and symptoms of insufficiency after a great many different forms of infection with pyogenic micro-organisms, both in local processes (phlegmon, abscesses) and in cases of general infection. We do not as yet know exactly to what such things are due. The effect of the nutritional disturbance and exhaustion following a severe illness no doubt has something to do with it. And it is equally certain that the other factors that have been mentioned so often, functional disturbance due to intoxication and inflammation, are also significant; personally I consider them very important.

Here the influence of inflammatory disease of the genitalia ought to be mentioned. Processes which are probably produced by the gonococcus have already been mentioned. To these I do not here refer, but rather to the inflammations in the female abdominal organs which are probably caused chiefly by streptococci and staphylococci. I have occasionally seen in women affections of the heart which were unquestionably myocarditis, and could have no other origin than an antecedent puerperal infection. Romberg reports similar observations. These will have to be mentioned again in connection with chronic myocarditis; for it appears to be a fact that the germ of a chronic inflammation is

* H. Lenhartz, "Nothnagel's System," vol. iii, p. 49. Traube, "Abhandlungen," vol. iii, p. 579.

† Romberg, "Herzkrankheiten," p. 961.

not infrequently derived from acute streptococcus and staphylococcus infections.

Perhaps the causal connections are even more far-reaching. That is the great question, whether the infections of the heart which have been described as occurring in the various infectious diseases must be attributed directly to the action of the original disease-poison, or whether they may possibly be connected with secondary infections. There is no doubt that the latter, even in ordinary, well-known diseases, play a more important part than is generally supposed. Experiments have proved that the diphtheria poison by itself is capable of producing diseases of the heart, especially parenchymatous degenerations. Whether inflammatory processes in the myocardium may also be produced by the diphtheria poison has never, so far as I know, been made the subject of animal experimentation, and nothing more accurate is known of other disease-poisons in this respect, because we are too little acquainted with them. Streptococci and staphylococci unquestionably possess the power of producing inflammatory processes in the valves as well as in the muscle, and it is also known that they readily become superadded to injuries caused by other microbes. In diphtheria, for example, mixed infection plays a very important part. In the case of other diseases the question of mixed infection has not been minutely studied. But it is probable that the process is significant in typhoid fever and in small-pox also. It would be possible, therefore, to arrive at a uniform conception of these acute diseases of the heart; but I cannot venture to suggest more than that this possibility exists.

In regard to the purulent myocarditis mentioned in the foregoing pages, it is to be understood that the diffuse variety was meant. This form cannot be distinguished clinically from interstitial, non-purulent inflammation, and even anatomically the two conditions present many points of resemblance. But purulent myocarditis also occurs in the form of a more or less circumscribed abscess in the muscle. These abscesses may be combined with diffuse, purulent inflammation of the rest of the muscle tissue; or they may be isolated, in which case the clinical picture is very confused, as has been explained in connection with focal diseases of the heart. In many cases, especially when other metastases are present, the symptoms of general infection dominate the picture; in other cases the symptoms produced by the primary localization of the disease occupy the foreground. There is weakness of the heart, usually accompanied by fever, and if the abscess ruptures ("acute heart ulcer"), a good many different complications may develop, such as communication between the two ventricles or the two auricles; multiple embolism, or rupture of the heart followed by fatal hemorrhage into the pericardial cavity.

Clinical History of a Case of Acute Primary Myocarditis.—A. B., thirty-nine years old, physician. Formerly had typhoid fever and migraine. On the sixth of August, 1897, in the evening, while on his rounds, he was drenched and afterward sat three hours in a closed room, and although he was not actually chilly, he felt uncomfortable. During the days preceding the disease the patient had been very greatly vexed. On the seventh of August, in the forenoon, the patient had a slight chill and complained of lassitude, with dull headache. The headache increased in severity, so that on the tenth of August the patient was hardly able to move his head. Until the eighth of August he continued to attend to his practice. Then he had to stay at home and his temperature was taken. Until the twelfth of August it was never less than 40.1° C. (104.1° F.), and went as high as 41.1° C. (105.9° F.).

On the eleventh of August I saw the patient. His mind was not perfectly clear, and he was exceedingly excited; complained of violent headache and pressure on the chest. He thought he had acute tuberculosis.

Nothing to be found in the nervous system. The lungs, liver, and urine were negative. The heart was somewhat enlarged both to the right and to the left. The first sound at the apex was not quite clear. The pulse was below 60, of moderate volume, irregular, and unequal.

Diagnosis: Acute infectious myocarditis. He was given 1.5 gm. (22½ gr.) of sodium salicylate a day. On the fifteenth of August he was free from fever. The violent headache continued until the seventeenth of August.

The heart action continued slow for some time and was exceedingly labile. The slightest movement caused great acceleration.

August 20th: Patient without fever, perfectly clear in his mind, and quite comfortable while in bed. While he is at rest, the action of the heart is quiet, but not quite regular; still very labile.

He made a slow recovery. By the beginning of September he was able to walk, and on the thirteenth of September he performed a major operation.

He has never lost a certain unpleasant feeling of "tension," "pressure," or "dragging" in the precordial region, which occurs quite frequently and is apt to be brought on by psychic excitement. During muscular exercise the heart is perfectly equal to its work. I have not had another opportunity to examine it, but it is said to be perfectly sound.

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DISEASES OF THE HEART OCCURRING AFTER INFLUENZA.

The disturbances of the heart which occur during *influenza* are the most obscure, and, at least for the present, more difficult to understand than any others, and this is particularly troublesome to the physician because they, unfortunately, appear to be quite frequent.

In the years when the great influenza epidemics took place there was much discussion about the diseases of the heart that occur in connection with influenza. If all that has been laid to the door of this infectious disease were to be accepted, it would be impossible to speak of a definite form of heart disease, or even of several different forms; for every conceivable symptom that has ever been known to occur in the heart has at one time or another been described as a direct result of influenza.

The epidemic of the year 1890 found most physicians completely unprepared. Many knew the "grip" only by name; but few had any conception of the protean nature of its manifestations. Besides, the enormous and unexpected distribution of the disease made such a profound impression on the profession that it was made to bear the blame for many things with which it had nothing to do. That alone makes it very difficult to judge of the organic changes.

Persons who were ill and weak were especially apt to fall a victim to the disease, and many of these died. In such cases it was difficult to distinguish between the symptoms and the anatomic findings due to the original disease and those produced by influenza. Besides, the disease very frequently leads to mixed infection, and in such cases the effects of the mixed infection greatly complicate the clinical picture.

Finally, it is often exceedingly difficult to determine whether influenza exists or has previously existed; indeed, the diagnosis in this respect must often remain uncertain. There is a widespread tendency to attribute any condition that is difficult to define to influenza, and thus many things are described as influenza which certainly have nothing whatever to do with the disease.

To add to all these difficulties, the etiologic and anatomic foundation for an orderly classification of circulatory disturbances following influenza are extremely scanty.

As in all other infections, so in influenza, many persons pass through the entire disease without presenting any special symptoms referable to the circulation. Not only healthy persons, but heart patients also, may get through the disease without suffering in this respect. This I have often observed, and Leichtenstern makes the same statement.*

On the other hand, the circulation, and particularly the heart action, is sometimes affected at the very beginning of the illness. That is not what I refer to here. Leichtenstern has given a detailed description of these conditions in his section on Influenza.† *Endocarditis* and *pericarditis* have also been observed. The distinction between the latter and diseases of the heart muscle presents the difficulties that have been repeatedly mentioned. That the myocardium is structurally damaged by the infectious process is an established fact; several descriptions of parenchymatous changes in man ‡ and in animals § are found in

* "Nothnagel's Practice," American edition. Volume on Influenza.

† *Loc. cit.*, p. 148.

‡ Marchand, "Berliner klinische Wochenschrift," 1890, No. 23. Kuskow, "Virchow's Archiv," vol. cxxxix, p. 414.

§ Bollinger, "Münchener medicinische Wochenschrift," 1890, p. 2.

the literature. But, unfortunately, we lack such systematic investigations as have been made in diphtheria. Whether, and if so with what frequency, genuine myocarditis occurs at the height of the disease has never, so far as I can find out, been accurately investigated.

With the *clinical symptoms* of cardiac disturbances accompanying influenza I have had but little personal experience. It is certain that at the height of the disease one does not often see cardiac symptoms that could be regarded as an expression of acute myocarditis, reasoning from analogy with the symptoms observed in other infectious diseases. Sometimes irregularity and inequality of the heart action are observed, and very occasionally dilatation; but the latter is comparatively rare.

I have seen, in a number of patients, cardiac symptoms which were said to be due to a former influenza. Like Romberg, I also have an idea that the conditions in these cases are exceedingly complicated. Nobody will deny the possibility of chronic myocarditis (possibly also endocarditis) developing from an acute infectious disease of the heart occurring during influenza, especially since, as has been mentioned above, the heart *may* become involved during the height of the disease. But it is probable that in most cases the cardiac disturbances are chiefly to be explained by an increase of an already existing cardiac affection, and by the influence of the general damage to the nervous system and the general health, which is such a conspicuous result of influenza.

It is evident that Romberg has a much larger experience with these things than mine has been. That is probably more than an accident. His practice was composed chiefly of persons belonging to the so-called better classes in a large city. Among such individuals the nervous system and the nutrition are relatively very labile, at least, much more so than among my Thuringian peasants and workmen; among such people as Romberg's patients "influenza" is a much more formidable disease. Romberg has given an excellent description of the symptoms; those referable to the heart often represent a great variety of sensory symptoms. The symptoms following influenza quite often resemble those of nervous diseases of the heart in every respect. Disturbances of rhythm are also very frequent. The functional power of the heart is diminished; even slight exertion suffices to cause fatigue and dyspnea; palpitation and shortness of breath follow the least movement. Many patients have dilatation of the heart and systolic murmurs. As I have said, this may be the result of a disease of the heart muscle caused by the infectious process. But in these cases also the symptoms are often due to an increase of an already existing morbid condition, or changes in the strength, size, or rhythm of the heart, such as occur during the ordinary pursuits of life when there is a reduction of the nutrition. The symptoms, therefore, would fall under various heads, and are accordingly described in several chapters. Hence the treatment need not be specially discussed. The prognosis in diseases following influenza is not very favorable; as a rule, these conditions prove exceedingly obstinate.

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THE TREATMENT OF ACUTE INFECTIOUS MYOCARDITIS.

With the treatment of the *circulatory disturbances occurring at the height of the disease* we are not here concerned. Nor can they be discussed separately from the methods of treating the remaining symptoms. It is needless to say that *active measures to combat the cause of the disease,* such as are now available at least in diphtheria and articular rheumatism, also benefit the circulation. For the rest, remedies are selected that are capable of influencing the heart and the vessels. These remedies are enumerated on p. 585, and the physical methods of treatment that are employed for the same purpose are discussed on another page (see p. 588).

As yet it is beyond our power to prevent the occurrence of cardiac disturbances late in the attack or *after the actual infection has run its course.* But there is much that the physician can do to prevent their injurious effects. The heart of any patient convalescing from an acute infectious disease must be subjected to the most minute examination at short intervals. Some diseases, such as diphtheria, require special care in this respect. If there is any suspicion of the slightest anomaly of the heart, strict rest in bed must be ordered, and if there are marked symptoms of cardiac disturbance, the patient must even remain perfectly quiet while in bed. The chances that an acute disease of the heart will run its course without any injurious consequences are greatly increased by diminishing the work of the heart, and it must be remembered that any muscular movement makes demands on the powers of the organ. In severe cases, especially in the myocarditis following diphtheria, this precaution must be carried to the extreme of forbidding every kind of movement; the patient remains perfectly quiet in bed and even has to be fed. The diet must be regulated according to the nature and duration of the disease. The patient must be kept at absolute rest, both mentally and bodily, so long as there are any signs that the heart is not perfectly competent to meet any increase in the resistance or the filling of its chambers. In some cases the patient may have to be kept at rest for weeks, and in this respect, again, diphtheria is the disease which most severely taxes the physician's and the patient's perseverance. As soon as there is reason to believe that the functional capacity of the heart is good, muscular movements may be permitted, even if the heart action has not become entirely normal. To hit upon the exact moment is a matter of medical skill; the considerations which determine the question have been repeatedly explained in the foregoing pages.

The transition from rest in bed to the activities of ordinary life must be very gradual. During this period it has been found, by experience, that cutaneous stimulation in the form of carbon dioxid baths is very useful in improving the strength of the heart. Romberg orders these baths as soon as the patient has become accustomed to being up a few hours a day. The time when the patient may be allowed to return to his work depends altogether on the nature of his occupation, the amount of bodily exertion or mental excitement which it involves, as well as on the convalescent's ability to save himself, a question into which the personal equation, of course, enters to a large extent (compare p. 556).

All morbid manifestations in other organs require careful treatment, especially anything that is capable of disturbing or interfering with the circulation; for example, constipation must be avoided under all circumstances.

Cold, cool, or even warm compresses should be applied to the precordial region. I think one should always try cold compresses first; but if they are not comfortable to the patient, warm compresses should be substituted.

Except for these measures, an expectant policy may be pursued so long as no signs of altered blood distribution make their appearance. But if the latter occur, I should advise that digitalis or strophanthus be tried. In most cases the remedies, unfortunately, are of no great benefit. Why, it is impossible to say. Possibly the action of these substances depends on certain definite changes of the heart muscle; or it may be that in these conditions there is not enough susceptible substance to enable the drugs to develop their influence.

In cases of profound cardiac weakness coffee, wine, camphor, and the other remedies mentioned above must be used. They are indicated because they are also capable of influencing the *heart*. By far the most important part of the treatment in all cases consists in regulating the hygienic conditions and muscular exercise. This part of the treatment should never be neglected for the administration of medicines in acute myocarditis.

CHRONIC MYOCARDITIS.

The inflammatory changes described on p. 628 develop insidiously and slowly in the heart muscle, and again, from the purely anatomic standpoint, are almost always associated with pericarditis and endocarditis. The *symptoms* of the two latter processes, however, are in many cases entirely overshadowed by the symptoms of myocarditis; the signs of effusion into the pericardium and of valvular defects being absent.

The inflammatory processes are always diffusely scattered over the myocardium. As a rule, large foci occur only when a number of smaller patches coalesce. The left ventricle is usually more severely involved than the right, and in it the upper part of the posterior wall and the lower part of the anterior wall especially (Köster*). The papillary muscles and the subpericardial layers first become the seat of inflammatory foci, and are more thickly set with these foci than any other portion of the heart. For this reason Köster is perfectly justified in saying that when there exists a suspicion of chronic myocarditis the pathologist should examine thin sections parallel to the surface, in addition to those usually examined.

Owing to the long duration of the process and the tendency to local regeneration even in the worst forms of the disease, the different stages are nearly always found combined in the same heart. Thus, for example, quite recent, round-cell infiltration is found between muscle-fibers that are but little damaged and side by side with well-developed scars containing few cells. As inflammation also attacks the walls of the large vessels; as, in addition, the blood-vessels may become involved secondarily in the older foci and may ultimately become occluded; as, finally,

* Köster, quoted by Rühle, "Archiv für klinische Medicin," vol. xxii, p. 85.

the parenchyma, both primarily during the myocarditis and secondarily from disease of the blood-vessels, may undergo granular and fatty degeneration in a great variety of ways, there results a combination of arterial, interstitial, and parenchymatous processes, and it may be exceedingly difficult in an individual case to determine positively what was the primary lesion. Hence even anatomically there is often much difficulty in distinguishing the changes from those due to coronary sclerosis, especially since, as we have already seen, that condition also is sometimes associated with inflammatory symptoms. Perhaps a strict distinction between the two processes is absolutely impossible. Among the causes of coronary sclerosis infectious conditions unquestionably must be included, and it is quite conceivable that these inflammatory processes might, in some cases, first attack the coronary vessels and in others exert their action directly on the heart muscle. It may be said, therefore, that chronic myocarditis in many cases develops as the direct result of the action of some injury on the muscle-fibers, while in other cases it is secondary to disease of the blood-vessels; in the etiology of chronic myocarditis coronary sclerosis is an important factor. The two things, however, are not identical. Sclerosis of the coronary vessels does not necessarily produce chronic myocarditis, and, in addition, produces certain other symptoms besides those of myocarditis.

The inflammation may be chronic from the beginning, or it may begin acutely, in which case chronic myocarditis represents a sequel of one of the processes discussed under acute myocarditis. Instead of recovery or acute and rapidly fatal cardiac insufficiency, a chronic insufficiency of the heart develops. This is seen most frequently after rheumatic polyarthritis; more rarely, after typhoid fever, scarlet fever, diphtheria, and pus-infections.

Chronic myocarditis may be insidious from the beginning, the transition both anatomically and clinically between health and disease being imperceptible. In these cases the causes, so far as I can see, are still very obscure. One is naturally inclined to think of infections and intoxications. In the case of intoxication, especially that due to alcohol, the theory finds support in the fact that in the diseases of the heart which develop in many forms of chronic alcoholism inflammation of the muscle has been found to be the cause of the cardiac insufficiency in not a few instances (see p. 610). In many of these cases with insidious onset one of the above-mentioned infections must be regarded as the direct cause of the myocarditis. Sometimes a considerable interval elapses between the acute disease and the appearance of the cardiac symptoms.

The disease of the heart-wall leads to defective contraction of the organ and diminution of the muscle tonicity, and, finally, if the process is protracted and the work of the organ is permanently diminished, true atrophy of the heart develops. Parts of the wall in which a great deal of the muscle substance has been destroyed may bulge or even tear through, but these two processes are probably more frequent in coronary sclerosis.

According to our theoretic and purely mechanic views, *hypertrophy of the muscle* ought not to occur in pure myocarditis. Nevertheless, one sometimes gains the impression that the muscle has become thickened. I will not venture to say more than this because in these slight degrees of hypertrophy it is impossible to give a definite opinion without an

accurate examination after Müller's method, and observations of that kind have never been made.

When there is reason to believe that hypertrophy is present, one generally looks for some other cause, either of endocardial or ectocardial origin. But it may fairly be doubted whether this method of reasoning is justifiable in all cases. It depends on whether the idea of hypertrophy is absolutely inseparable in our minds from the idea of overcoming increased resistance, and of evacuating larger quantities of blood. But who, in the present state of our knowledge, can successfully deny that inflammatory irritation of the muscle substance might be capable of increasing the extent and rapidity of the contractions and thereby the amount of work performed, or that the muscle-fibers may undergo hyperplasia under the direct influence of the inflammatory stimulation? (See p. 491.)

The disease, so far as I can see, attacks men and women in about equal proportions. It shows a distinct predilection for youth and middle age, but is also met in older persons. In children I have never seen it except in the form of the processes which follow the acute exanthemata and diphtheria.

I do not think it is possible, as yet, to give an opinion in regard to the frequency of myocarditis. For a time the disease was regarded as extremely rare, while some insisted that it was quite widespread—for example, Köster,* Rühle†, Romberg,‡ and myself.§ If I am now rather inclined to hesitate on this point, it is because, during the last seven years, during which I have seen a large number of patients, and among them many suffering from heart disease of various kinds, I have seen comparatively few autopsies; and during life it is often quite impossible to determine whether, in the presence of the chronic cardiac disturbance described in the foregoing sections, chronic myocarditis exists and whether it or coronary sclerosis is responsible for the clinical symptoms. For the present, however, I am still inclined to think that the incidence of the disease is by no means so very limited, and I am convinced that this view would be confirmed if sufficient autopsies could be made, with extensive and systematic microscopic examinations.

Symptoms.—Excellent descriptions of the symptomatology of chronic myocarditis were given by many authors as early as the first half of the nineteenth century—for example, by Sobernheim (see also Stein's analysis of the older views on this disease‖), and in the French and German text-books on diseases of the heart. A comparison of these views with those held during the following decades, for example, during the sixties and seventies, clearly shows how much the doctrine of myocarditis has suffered from the undue prominence that was, perhaps naturally, given to percussion and auscultation. It is true that some of the older reports cannot be utilized for our present purposes without a certain degree of reserve, because the term myocarditis at that time embraced a great many different conditions which we are now able to separate and, therefore, must separate.

*Köster, in Rühle's article, "Archiv für klinische Medicin," vol. xxii, p. 82, and in the Bonn "Program," 1888, "Ueber Myocarditis."

† Rühle, "Archiv für klinische Medicin," vol. xxii, p. 82.

‡ Romberg, "Herzkrankheiten," p. 755.

§ Kelle, "Archiv für klinische Medicin," vol. xlix, p. 444.

‖ Stein, "Myocarditis," p. 117.

Köster and Rühle, in their contributions, were probably the first to call attention to chronic myocarditis and to emphasize its importance.

For us myocarditis is characterized by the symptoms of an increasing, although very slowly progressing, insufficiency of the heart muscle. As has been mentioned, this insufficiency often follows some acute disease affecting the entire body and the heart, and in that case the first symptoms of the disease are identical with those described in the section on Acute Myocarditis.

In other cases the patient notices a gradual loss of power. This general deterioration shows itself chiefly during muscular exertion, although the patient also feels the effect of increased demands on his psychic powers more than before. Slight provocations suffice to cause excitement, dyspnea, and fatigue. Dyspnea especially is apt to develop early and to make itself greatly felt during muscular exertion, and may occur in paroxysms. Digestive disturbances may be present from the beginning, and must always be attributed to passive congestion in the lungs and in the abdominal organs.

Physical examination often reveals marked cyanosis quite early in the disease. As a rule, the heart is dilated, generally in both directions. The apex-beat is weak and soft, or may have disappeared altogether. One point upon which the authors of the first half of the century laid great stress is that the apex-beat, at times at least, is increased in force; but it would be wrong to conclude at once that the heart is hypertrophied, for the phenomenon can readily be explained by inflammatory irritation and exaggeration of the heart action, especially if the increase in the force of the apex impulse is only occasional. The action of the heart is very frequently irregular and lacking in uniformity, rarely retarded, almost always accelerated; the corresponding properties are observed in the pulse, which is generally soft as well. The irregularity and inequality of the heart-beat rarely follow any special order of periodicity, a complete absence of anything like regularity being the rule. In fact, total irregularity of the heart action is absolutely typical of myocarditis; for coronary sclerosis, so far as the production of this kind of symptoms is concerned, is on an absolute par with genuine inflammation of the muscle, the important factor being the disease of the musculature. But it would be a very great mistake to exclude myocarditis because of the absence of disturbances of the heart action.* And now that we know the significance of the auricular muscle [and the auriculoventricular bundle of His—Ed.] in determining the rhythm of the heart, this no longer excites our surprise. The physician must, at least, remember that anomalies of the rhythm or size of the heart may be altogether absent; for it is probable that all these symptoms depend on the nature and localization of the anatomic processes, which present the greatest possible variations.

But if irregularity of the heart action is present, as is the case in most patients, it usually becomes permanent. This point is of some diagnostic value.

The results of *auscultation* may be entirely normal, but sometimes the first sound at the apex is weak or impure. It is very unfortunate for our understanding of these matters that we know so little about the influence of abnormal contractions of the muscle on the first heart-sound.

* Compare Ebstein, "Zeitschrift für klinische Medicin," vol. vi, p. 97. *Ibid.*, "Archiv für klinische Medicin," vol. lxv, p. 81.

Systolic murmurs at the apex indicate a mitral insufficiency. This may, of course, be due to endocarditis; but these cases of associated endocarditis and myocarditis, as has been stated, are not included in the present discussion. But in cases of pure myocarditis also a murmur is not infrequently heard at the apex, either accompanying or replacing the first sound. Such a murmur must, as a rule, be regarded as due to muscular insufficiency of the mitral valve. That this insufficiency is particularly common in chronic myocarditis needs no special explanation. In most cases the deficiency of the muscular contractions which causes the valvular defect is inconstant, and the signs of mitral insufficiency are, accordingly, heard at times and absent at others. This is the rule, but there are exceptions. In one case under my observation the muscular insufficiency of the valve was constantly present during many months.

Anomalies of the second sound at the base must be attributed to changes in the aorta or to valvular lesions, although in a few instances I have found diastolic murmurs at the aorta due to muscular insufficiency of the semilunar valves. It may be remembered that these valves are able to sustain the great difference in pressure between the aorta and the left ventricle only when they are assisted by the contraction of fasciculi of muscle-tissue situated at the base of the valves, in the septum. If these fasciculi fail to contract sufficiently, all the signs of aortic insufficiency may be produced even in the absence of any actual change in the semilunar valves. This is particularly apt to occur after chronic myocarditis. In a few cases a diastolic murmur of precisely the same character as that heard in mitral stenosis was present at the apex, although the autopsy failed to reveal any changes in the valves or any narrowing of the orifice. In the two cases which I observed there was much dilatation of the left ventricle, although the mitral orifice was not at all enlarged. Our ideas in regard to the mode of production of this diastolic murmur are not very clear. According to the prevailing views, the current of blood, as it enters the dilated ventricle through the mitral orifice, sets up eddies which cause vibration of the valves and heart-wall.

The *character of the arterial pulse* depends solely on the heart action, more particularly on the completeness with which the heart supplies the vessels with blood. The arteries themselves have no special effect on the pulse—at least nothing is known about any such effect.

Sensory disturbances are scarcely ever absent. Pressure on the chest, fear, and actual pain are the most frequent. The question whether there is a pain characteristic * of myocarditis has been much discussed in the past. It is denied by most authorities, and personally I must agree with them. There is no doubt, however, that sensory disturbances form an important part of the patient's discomfort. Even angina pectoris is said to occur, the attacks varying from the mildest to the most severe; but it is undoubtedly much more rare than, for example, in coronary sclerosis.

Shortness of breath, a feeling of pressure, and fear not infrequently lead to great restlessness. The patients are continually moving from one place to another and changing their position, unable to find any comfort; they toss about and so increase their sufferings.

The anomalies of the heart action may persist for a long time and the patients may, to a certain extent at least, retain their ability to work.

* See, *e. g.*, Bamberger, "Herzkrankheiten." Stein, "Myocarditis."

In the end, however, the signs of severe cardiac insufficiency develop. Even when the patient is at rest, abnormalities in the distribution of the blood and symptoms of congestion are observed; sometimes, chiefly in the pulmonary circulation (when they are accompanied by violent dyspnea) or only in the systemic veins, or, as most frequently happens, in both the pulmonary system and the systemic veins, although not in the same degree in both. The type of cardiac weakness depends altogether on the distribution and localization of the inflammatory processes in the muscle, that is to say, on which parts of the heart are most severely damaged.

In other cases the symptoms of passive congestion make their appearance early, being often confined to one or two organs. One patient may have an obstinate bronchitis; another, an enlarged liver; a third, edema of the ankles. The process may extend to the kidneys either in the form of congestion or true Bright's disease. But in the latter case one probably has to deal with an accidental combination or with the association of two diseases with a common etiology, such as syphilis, for example.

In the end, and after an interval which varies in different individuals, the well-known picture of profound cardiac insufficiency always develops. Some patients live in tolerable comfort for years before they reach this stage. This final stage of cardiac insufficiency may last for some time, its duration being probably greatest in chronic myocarditis and some forms of mitral valvular lesions. In the latter, also, it must be attributed to the presence of myocarditis.

The patient's sufferings during this stage of the disease are too well known to need description. It sometimes lasts a long time, and many myocarditis patients suffer a great deal, especially when the left ventricle is weaker than the right and the dyspnea caused by the slowing of the blood-current is exaggerated by the overfilling of the lungs.

The symptoms referable to other organs, unless they are the result of the disturbance of the heart's function in the manner explained, are in every instance completely overshadowed by the cardiac symptoms. In a few cases I saw hectic fever with an evening rise to 40° C. (104° F.), and occasional chills. The autopsy clearly showed that there could be no other cause for this fever but the inflammatory process in the heart muscle. At that time we did not examine the heart for the presence of micro-organisms; nevertheless, these cases of myocarditis may quite properly be compared to the observations of subacute or chronic endocarditis with fever which were first described by Leyden * and carefully investigated by Harbitz.† Streptococci and pneumococci play an important part in the etiology of these conditions. Owing to the close connection between myocarditis and endocarditis, one is naturally inclined to attribute these cases to the same or similar causes.

In some cases I observed severe grades of anemia along with the myocarditis. I cannot explain its occurrence, and it is of some interest, from a diagnostic standpoint, because in such cases it may be difficult to interpret the cardiac symptoms.

The **diagnosis** of chronic myocarditis rests on the demonstration of cardiac insufficiency in its various degrees.

At the very beginning, if the functional power of the heart appears

* Leyden, "Zeitschrift für klinische Medicin," vol. iv, p. 321.

† Harbitz, "Deutsche medicinische Wochenschrift," 1899, No. 8.

to be well preserved and the disturbances of its action are the most prominent features in the case, a doubt may exist as to whether the heart symptoms may not be purely nervous. This question must be decided by the identical considerations that are given at length in the section on Arteriosclerosis; only in regard to myocarditis I wish to emphasize that it never occurs *without* some diminution of the functional power of the heart, even if at first this diminution manifests itself only when certain definite demands are made upon the organ. Again, the rhythmic disturbance in nervous individuals much more commonly occurs only at times and as the result of certain definite influences, while in myocarditis the irregularity is generally permanent and follows no rules whatever.

But even in these incipient cases the necessity of differentiating the condition from the effects of coronary sclerosis must be borne in mind, a necessity which arises again and again at every stage of the disease. And in every stage the results of such efforts at diagnosis are equally small. It is true that a few diagnostic points of importance can be mentioned, such as the presence or absence of disease in the peripheral arteries, the greater prominence of sensory disturbances in coronary sclerosis, especially angina pectoris,—which is rare in myocarditis,—in the fact that many cases of arteriosclerosis are associated with increased arterial tension and hypertrophy of the heart, both of which conditions are always absent in pure myocarditis, and, finally, if one has an opportunity of observing the course of the disease, it should be remembered that an atypical, bizarre course is characteristic of coronary sclerosis, while in myocarditis the disease progresses slowly and steadily, but irresistibly, toward an unfavorable termination. In the individual case, however, and especially at the first examination, the distinction between the two conditions is always exceedingly difficult and often quite impossible.

Chronic myocarditis must be differentiated from the cardiac affections occurring in the obese, in alcoholics, and in persons who do hard manual work. This distinction must be based on the development and course of the symptoms and on consideration of the etiologic conditions. The presence of cardiac hypertrophy in many cases, the nature of the clinical course, and the effect of treatment must also be taken into account. The information gained by examining the heart has but a limited value in this respect and affords no positive data on which to base a differential diagnosis. This will astonish no one who is familiar with the conditions; for the anatomic, physiologic, and pathogenetic aspects of all these morbid states are far from being clearly understood. It is possible that not a few of these cases, after all, belong to the category of myocarditis. The older I grow, the more I see, and the more familiar I become with these things, the more guarded I am in giving an opinion. The distinction is particularly difficult when cardiac insufficiency exists. But when the functional power of the heart is tolerably good, observation of the rhythm has a certain value in so far as persistent, marked irregularity with relatively good heart action is chiefly characteristic of myocarditis and coronary sclerosis, particularly the former,* and is distinctly more rare in the other conditions that enter into the differential diagnosis.

It is quite impossible to distinguish between the forms of myocarditis here under consideration and inflammation due to syphilis. Nothing but the knowledge that syphilis is present and the possibility or prob-

* Compare also Riegel.

ability of its being responsible for the cardiac trouble will guide the physician on the right track.

Finally when a systolic murmur is present at the apex in myocarditis, the disease must be differentiated from ordinary mitral insufficiency, and, if the heart-sounds are pure, from mitral stenosis without murmurs. But so long as a fair degree of compensation is maintained, the marked involvement of the pulmonary circulation and the hypertrophy of the right ventricle (indicated by dyspnea and accentuation of the second pulmonic sound) usually make it possible to distinguish between the two conditions. In the presence of marked cardiac insufficiency, however, a careful physician will refuse to give a specific diagnosis, at least in the beginning.

The distinction between muscular and endocarditic mitral insufficiency is not possible when a systolic murmur is always present and the second pulmonic sound is accentuated. I am well acquainted with cases of this kind, in which the above-mentioned signs are observed constantly until death, and all we can hope to do in these cases is to give a diagnosis of mitral insufficiency with myocarditis. But in uncomplicated myocarditis the murmur is much more frequently inconstant,—present at one examination and absent at the next,—and the murmur disappears as the heart action improves. I will not say that this is absolutely characteristic. In endocarditis also the mitral insufficiency is undoubtedly due in a great measure to functional deficiency of the muscles at the base of the heart.* But in muscular insufficiency inconstancy of the murmur is a more frequent and more conspicuous feature. If, in addition, the disturbances of the heart action are taken into consideration, a positive diagnosis of myocarditis will be possible, as a rule, and it will often be possible to say whether there is a probability that the mitral insufficiency is due to endocarditis as well as to deficient contraction of the muscle. Quite frequently, however, the careless examiner will make a mistake, and even the man with experience will not venture to give a positive opinion. For it must be remembered that in endocarditis the disturbance of the heart action is, in a large measure, due to the accompanying disease of the myocardium.

The presence of a *diastolic* murmur renders the diagnosis of myocarditis exceedingly difficult. Since it is absolutely impossible to distinguish the murmur from that of aortic insufficiency or mitral stenosis, a careful diagnostician will not venture beyond a tentative diagnosis. However, owing to the great rarity of these murmurs in the absence of endocarditis, this point is of little practical importance.

Treatment.—The causal indication in the forms of myocarditis here described cannot be satisfied. Syphilis is treated in a separate section. We know of no remedy to combat the final effects of rheumatic processes. If, however, alcohol is the cause of the myocarditis, the root of the evil may be attacked by forbidding its use. But alcoholic beverages must be restricted to a minimum in every case; for our principal duty is to avoid everything in the diet and in the mode of life capable of making a greater demand on the heart or of directly injuring the organ. The necessary explanations on this point will all be found in the section on General Principles, and nothing would be gained by adding any special remarks here. The question whether the patient shall stay in bed or may be allowed to sit up or go about must be determined on

* Compare Romberg, "Archiv für klinische Medicin," vol. liii, p. 141.

general principles, while, at the same time, carefully taking into consideration the individual patient's peculiar circumstances. Accordingly, the physician's instructions must be absolutely specific and include every detail of the patient's mode of life.

Although no definite measures can be adopted to combat the causes of the disease or to prevent its progress, a good deal can be done, nevertheless, to keep up the strength of the heart by preserving the power of the muscle tissue or even increasing its functional capacity. For this purpose the patient should be instructed to take exercise as long as possible, not, of course, without observing due caution and carefully controlling the individual reaction, for there is always great danger of overexerting the heart. We all know how much damage Oertel's treatment can do in this class of cases, as well as in cases of coronary sclerosis. In any case I advise the greatest caution.

Carbon dioxid baths are distinctly less harmful, and there is no doubt that good results have been obtained by their use, especially the baths given in Nauheim; some surprising results have come within my own observation. But even in administering carbon dioxid baths the effect of each bath on the organism must be carefully noted.

If actual stasis develops, digitalis or its substitutes, as well as diuretic remedies, must be administered in the usual way. I know of no special advice to give in this connection. In some cases I find it useful to give small doses of digitalis (0.1 gm.—1½ gr.—infusion or pill, twice a day) for weeks at a time, and have in this way at least succeeded in keeping the functional power of the heart from diminishing. Under these circumstances, also, especially if the cardiac insufficiency is of a minor degree, carbon dioxid baths are useful.

CARDIAC STENOSIS.

In exceedingly rare cases, when inflammation of the heart muscle is associated with endocarditis, scars may be formed, and in contracting may produce considerable narrowing of certain portions of the heart, thus simulating stenosis of one of the orifices. This occurs relatively most frequently at the **conus arteriosus,** producing a clinical picture which resembles that of pulmonary stenosis. In the first case of this kind, described by Dittrich,* it was thought that there might have been a myocarditis chiefly affecting the right ventricle and in some way connected with a blow received on the chest. The inflammation had developed at the **conus arteriosus** below the pulmonary valves, and the cicatricial contraction had produced a considerable narrowing of the cone. As a result, the muscular tissue of the right ventricle, which propels the blood into the pulmonary artery, had undergone great hypertrophy. Hammernjk † believes that the cause of the myocarditis in this case was a former attack of intermittent fever. Dittrich suggested for the conditions found in this case the rather pertinent term, "true heart stenosis." This term has since been adopted into the literature, and, owing to our weakness for curious and rare or interesting cases, a number of observations of a similar kind have since been described.‡

* Dittrich "Vierteljahrsschrift für die praktische Heilkunde," 6. Jahrgang, 1849, vol. i, p. 157. † Hammernjk, *ibid.*, p. 180.

‡ For the literature up to 1881 see Rollett, "Medicinische Jahrbücher der k. k. Gesellschaft der Aerzte in Wien," 1881, p. 161.

It has already been mentioned that symptoms similar to those observed in stenosis of the pulmonary arteries may be produced by narrowing of the *conus arteriosus*. If the approach to the aortic orifice becomes narrow as the result of cicatricial processes,—and it is possible that this disease also begins during fetal life,—the patient will, of course, exhibit essentially the same symptoms as in stenosis of the aorta. As a matter of fact, the diagnosis of aortic stenosis is usually made during life in these cases, although it has been possible in some to recognize stenosis below the opening of the aorta even before death.*

Such a diagnosis might be arrived at by carefully considering those circumstances which, although, on the whole, the clinical picture is one of aortic stenosis, militate against the presence of that lesion; but in all such cases the possibility of other rare diseases of the heart, such as tumors and echinococci, must also be kept in mind. I am not inclined to lay much value on the success of this kind of diagnosis in judging an individual case. It is more a clinical feat than a piece of scientific work. At all events, most diagnoses of this kind are wrong; only now and again one of them proves correct.

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DISEASES OF THE HEART MUSCLE IN TUBERCULOSIS.

Tuberculous infection of the entire body may involve the heart in a variety of ways. With tuberculous pericarditis and the form of endocarditis which not infrequently follows pulmonary tuberculosis we have nothing to do; we shall follow our program by confining ourselves to

* Rollett, *loc. cit.*

the diseases of the heart muscle. First, we must mention hypertrophy of the right ventricle, which is more particularly apt to follow the more chronic forms of pulmonary tuberculosis and the forms accompanied by considerable interference with the pulmonary circulation. This will be referred to later.

Genuine tuberculosis of the heart may cause the formation of numerous miliary nodules in the myocardium. This condition is found not infrequently as part of a general miliary tuberculosis, but does not give rise to any special clinical signs. Labbé found a diffuse tuberculous infiltration of the heart muscle in a case of profound cardiac insufficiency. If one may be permitted to reason by analogy from other inflammatory processes, the anatomic alteration in this case may be regarded as the primary cause of the functional disturbance.

Solitary tubercles are very rarely found in the heart. They run their course without producing any clinical symptoms, or, if they attain large dimensions, cause the symptoms of a tumor. This question will be discussed in the following section, where it will be mentioned how scanty the diagnostic data in these cases are. Anything like certainty in differentiating solitary tubercles from other tumors is altogether out of the question.

I purposely refrain from going into these things at greater length because it is impossible to give a satisfactory presentation of heart tuberculosis unless the relations of the endocardium, the myocardium, and the pericardium are considered together. The pathologic processes in these three structures are often very intimately dependent upon one another in tuberculosis. The process frequently begins as a tuberculosis of the pericardium, and, on the other hand, an inflammation of the myocardium is always a great menace to the endocardium and to the blood. But these matters do not fall within the province of diseases of the heart muscle.

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TUMORS AND PARASITES OF THE HEART MUSCLE.

The observations of pathologic anatomists have taught us that echinococci, cysticerci, and a great variety of tumors may occur in the heart muscle. Tumors may be primary and may begin in the muscle, in the connective tissue, or in the endocardium. Primary growths are

very rare, however. Metastatic tumors derived from other portions of the body are much more frequent. A number of observations of this kind have been collected, and any one who wishes to make an exhaustive review of the literature will find no lack of material.

Nevertheless, Oppolzer's remark that these things have no clinical interest still holds good, "because it is almost never possible to make the diagnosis." With the purely oncologic aspect of heart tumors and their general pathologic importance we, of course, have nothing to do here. What we have to know is to what extent the physician must take account of their occurrence and whether they can be recognized.

It must be stated, to begin with, that a large number of cases of heart tumors, as well as tumors reported in the literature, represent accidental findings. The subjects either had not presented any symptoms at all, or the symptoms were those of a primary tumor in any part of the body without any disturbances referable to the heart. Some of the patients died a sudden and unexpected death. In such cases, assuming that the existence of a primary focus is known, it may occasionally be possible to form a guess as to the causes which ultimately bring on death; but a diagnosis is out of the question. In some cases in which the previous clinical picture had afforded no explanation of the morbid process whatever, particles of tumor or portions of a ruptured echinococcus-cyst may be carried away and lead to the formation of multiple emboli, but even in these cases one would not venture to do more than suggest certain possibilities.

But in some cases parasites or tumors may produce a clinical picture of heart disease. Even in such cases, however, the disease during the patient's life will often be confounded with more common and frequent morbid conditions. And quite rightly, too; for, unless we wish to indulge in the merest guesswork, we must hold fast to the principle that in an individual case the probabilities are more in favor of ordinary clinical conditions than of the rarer anomalies.

Occasionally, however, it is possible to surmise the presence of one of these rare conditions; especially when the symptoms of cardiac insufficiency are present and the phenomena taken as a whole do not fit any of the usual clinical pictures. For example, a tumor or a parasitic cyst may obstruct one of the orifices of the heart only at certain times or only in certain positions of the body. Or the enlargement of the heart obviously extends only in one direction in a way that does not tally with any of the usual morbid states. Or marked congestion may take place in the distribution of one vein, while other portions, which ought also to be affected in an ordinary case of cardiac insufficiency, escape altogether. These are a few individual possibilities, and there are numerous others. Nothing is gained by going into detail, for every case is different; and to be comprehensive one would have to give a complete review of the literature. To be familiar with the literature is, no doubt, an advantage; but what is more valuable to the physician in my opinion, is diagnostic instinct, which is simply the fixed habit of carefully and minutely taking into consideration all the signs and symptoms referable to the heart, the circulation, and all the other organs.

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SYPHILIS OF THE HEART.

Syphilis may attack any portion of the heart. In the pericardium the disease produces fibrous inflammation and gummata; the blood-vessels may be attacked by syphilitic endarteritis, and all the effects of coronary sclerosis may make their appearance. The lesions of the muscle are: first, the effects of the vascular disease; second, diffuse interstitial inflammation or true myocarditis; and, third, gummata, which vary greatly in number, size, and location. Add to these changes cicatricial, verrucous, and gummatous processes in the endocardium, and it is evident that a great variety of disturbances are possible.

Disease of the blood-vessels and of the muscle is decidedly more important and more frequent than any other. Any one of the changes may occur by itself. In the case of arteritis this is readily understood, since death may result before the effect of the disease has had time to show itself in the muscle.* Formations of gummata and interstitial myocarditis may also occur independently; but, of course, the *combina-*

* See Kockel's case.

tion of all these processes is what we observe most frequently, especially the association of arteritis and myocarditis. Diseases of the endocardium and pericardium are generally secondary and dependent on the muscular disease; they practically never occur alone.

In chronic fibrous myocarditis, which may occur alone or not infrequently accompanies gummata in the muscular wall, large portions of the ventricular walls may undergo connective-tissue degeneration. This may cause bulging of the wall, so-called aneurysm of the heart, exactly as in simple myocarditis or as the result of coronary sclerosis. Such an aneurysm or diseased spot in the auricular wall is liable to rupture and cause fatal hemorrhage.

Syphilis also frequently attacks the blood-vessels of the body, particularly the arteries. Syphilitic disease of the beginning of the aorta should be specially mentioned in this connection; for, owing to the constriction or occlusion of the orifices of the coronary arteries, the condition manifests itself chiefly by heart symptoms. Like ordinary sclerosis, syphilitic aortitis may produce all the effects of occlusion of the coronary arteries, especially severe attacks of angina pectoris and sudden death.

Syphilis of the heart is certainly not frequent. But whether it is as rare a disease as would appear from some statistics on the subject seems to us more than doubtful. The diagnostic difficulties are exceedingly great; for, so far as I can see, the only condition that is absolutely characteristic of syphilis in a cardiac process is the formation of gummata. As for the remaining forms of endocarditis, myocarditis, and pericarditis, as well as arteritis, which unquestionably occur in syphilis, their origin cannot be seen. Hence it is largely a matter of choice on the part of the observer whether and to what extent processes which occur in notorious syphilitics and are not characteristic shall be regarded as specific or not, and statistics in regard to the frequency of syphilitic processes in the heart are, therefore, bound to vary. For the same reason the diagnosis is equally difficult at the bedside, as Curschmann has so clearly shown.* If even the pathologist is not always able to tell us positively whether inflammatory processes in the heart are directly due to syphilis or not, how are we clinicians to know?

It is true that the effect produced by certain drugs affords some information. I am perfectly ready to admit that mercury and iodin in themselves may act on the circulatory organs. Mercury often causes a marked increase in the excretion of urine, and in that way alone its services to the circulation are most valuable. The value of iodin in arteriosclerosis, and especially coronary sclerosis, is constantly gaining more and more recognition. Nevertheless, the fact that in the cases reported by Curschmann the administration of these two drugs was promptly and unquestionably followed by the disappearance of severe cardiac symptoms leaves no doubt of the specific nature of the fundamental disturbances. Hence the effects of treatment retrospectively afford very valuable data, which, if properly interpreted, enable us to determine whether cardiac symptoms are syphilitic or not. But in proportion as the remedy is efficacious the scope of pathologic anatomic investigation is necessarily restricted, and thus the extension of our knowledge in this field meets with certain not inconsiderable obstacles. The same is true of the nervous system. Owing to the lack of post-mortem observations, especially in the milder, curable diseases and in

*Curschmann, "Arbeiten aus der medicinischen Klinik zu Leipzig," 1893.

recent cases, our knowledge of the anatomic changes underlying a number of clinical conditions is in many respects imperfect.

Syphilitic disease of the heart usually occurs late, say from six to ten years after infection—at least in most cases of the disease. Exceptions have, of course, been observed, just as we know that, for reasons which for the present we do not quite comprehend, tertiary phenomena sometimes occur very early.

How often it happens during the so-called *secondary* period of the disease that the heart becomes involved in the syphilitic process sufficiently to give one the right to speak of a cardiac disease, cannot as yet be decided. After Fournier had called attention to these matters, Grassmann examined a number of syphilitic individuals during the secondary period, and Mracek (see p. 389 of his work) also discussed the symptoms referable to the circulation which occur during the secondary period. For obvious reasons there is a total lack of anatomic data, while from a clinical standpoint the symptoms are very much like those which occur in other infectious diseases. In accordance with the scope of this work I have dealt with these symptoms only in so far as they are due to pronounced changes in the heart, and for that reason shall not discuss the condition of the circulatory organ during the secondary period of syphilis.

Specific disease of the heart occurs also in *congenital syphilis*. Virchow, Kantzow, and Mracek describe such cases, but they may be omitted from a clinical description because nothing is known as yet in regard to the symptoms.

In studying the *clinical phenomena of syphilis of the heart* it will have to be shown what symptoms are produced by gummatous processes and how inflammation of the heart muscle and coronary arteries caused by the poison of syphilis can be distinguished from inflammations due to other causes.

Gummata may run their course without producing any symptoms, and ultimately bring on sudden and unexpected death. In syphilis, as in the case of other tumors of the heart, it is not a very rare occurrence for persons who have never complained of any cardiac symptoms to die a sudden death; in such cases one or several gummata are found in the heart at the autopsy. It is astonishing how many cases of sudden and unexpected death are reported in the literature of heart syphilis.

But the growth of gummatous neoplasms in the heart may cause distinct symptoms even during life, and these symptoms are essentially the same as those which are produced by diffuse syphilitic processes in muscle. *A priori* the latter do not differ essentially from the symptoms of other chronic diseases of the muscle or those produced by coronary sclerosis.

Irregularity and inequality of the heart action, acceleration or slowing of the pulse, dilatation of one or both ventricles, with systolic murmurs at the apex or at the base—all these symptoms are possible. The functional capacity of the ventricles may be impaired in any conceivable degree, from the faintest diminution of force to the well-known picture of ordinary, well-marked cardiac insufficiency.

The objective signs are very frequently accompanied by subjective symptoms, such as palpitation, pressure on the chest, oppression, a sense of anxiety, and pain, going on to a fully developed attack of true

angina pectoris. Paroxysms of cardiac asthma may also occur early in the disease and may, indeed, be among the first symptoms.*

Diagnosis.—It appears, therefore, that heart syphilis presents the well-known and, unfortunately, ambiguous phenomena of chronic disturbance of the muscular function of the heart. How and under what circumstances is it possible in a given case to recognize the syphilitic nature of such phenomena? The anatomic difficulties have already been pointed out, and the clinician is baffled by the fact that the presence of gummata, which alone renders the diagnosis positive, cannot be recognized during the patient's life.

The most significant feature is the rather sudden development of cardiac disturbances in persons known to be syphilitic, either unaccompanied by any characteristic phenomena or in association with distinct syphilitic symptoms, such as changes in the skin, the mucous membranes, the bones, or the liver. In such cases there is at least a *possibility* that the heart has been damaged by the same influences, and this possibility becomes a probability when all other causes for the occurrence of a cardiac affection are wanting. But the greatest caution is necessary in deciding this question "of the absence of other causes"; for of the two conditions which are unquestionably the most important in the differential diagnosis,—coronary sclerosis and simple chronic myocarditis, which has been described,—the former is very frequent and the latter is, to say the least, not a rare disease; and, as has been shown in the respective sections, it is always difficult in such cases to find an adequate cause. Nevertheless, we do know something about the etiology, especially the conditions that are apt to lead to coronary sclerosis. If a symptom which points to disease of the coronary vessels with some degree of probability, as, for example, a typical attack of stenocardia, occurs in an individual in whom the existence of ordinary coronary sclerosis would at least excite surprise,—as, for instance, a young man, and especially a young woman,—such an occurrence might suggest syphilis of the heart or blood-vessels. I am well aware of the fact that diseases of the arteries are encountered more and more frequently among young individuals, probably because the conditions of life are becoming more and more complicated. Nevertheless, such a discovery cannot help causing surprise and gives a definite direction to the physician's thoughts if there is any possibility of syphilis in the case or the signs of the disease are found. The most painstaking inquiry into the history, a certain guessing, and, above all, a minute examination of the entire body are, of course, the most essential requisites in every case. Again, certain reservations must be borne in mind. Thus every heart symptom that occurs in a syphilitic patient need not be of luetic origin; nor, on the other hand, is it always necessary to find similar changes in other organs in every case of heart syphilis. Nevertheless, the probability or certainty of the existence of syphilis has considerable weight in the diagnosis. One should be very careful before assuming that syphilis is "impossible."

In cases of pronounced syphilis one's suspicions should be aroused by apparently quite insignificant symptoms, such as palpitation and change of rhythm, for a study of the literature shows distinctly that persons who die a more or less sudden death and whose hearts at autopsy

*Compare Rosenfeld, "Medicinisches Centralblatt des Württembergischen ärztlichen Vereines," 1882, vol. lii, No. 38.

present syphilitic and especially gummatous changes, had previously suffered from slight symptoms of the same kind. When the patient's general condition is not suspicious, the present tendency is to regard these symptoms as nervous or at most to consider the existence of a coronary sclerosis, but I cannot help thinking that a timely diagnosis in these cases might do a great deal of good.

But little has so far been said about the symptoms of the heart disease itself. But I cannot discover that these symptoms are in any sense characteristic. The circumstances accompanying their appearance may possibly point to a special origin, and in this connection I must again refer to the factors which indicate disease of the coronary vessels.

Any deviation in the clinical picture from the usual type must receive special attention. Above all, I would urge a careful examination of the abdomen, especially the liver and spleen. Syphilis may attack the liver at the same time as the heart; it is not only possible, but indeed actually occurs, that abdominal symptoms, such as ascites and enlargement of the spleen, are more prominent than any others in the clinical picture, and such a state of affairs may be explicable only by assuming some special disease of the liver. The latter would support the assumption of syphilis, especially when palpation of the liver yields characteristic signs.

Although it was said above that chronic myocarditis and coronary sclerosis are the conditions most easily mistaken for syphilis of the heart, the differential diagnosis, nevertheless, must include all other disturbances of the heart muscle, such as, for example, dilatation produced by muscular exertion. It is evident that the decision may be exceedingly difficult, not to say impossible, in an individual who is known to have syphilis, if he develops cardiac disturbances as a result of severe bodily exertion.* It is to be remembered in this connection that infectious diseases in themselves not infrequently impair the resisting power of the heart, and for that reason cardiac disturbances of various kinds are especially apt to occur in syphilitic individuals.

Treatment.—Owing to the difficulty and uncertainty of diagnosis it is not easy to make up one's mind whether, and if so, when, to begin active antisyphilitic treatment. In view of Curschmann's observations I would strongly advise that if there are any positive indications to suggest the probable existence of heart syphilis,—an absolutely positive diagnosis is practically always out of the question,—not to wait too long before putting the patient on a mixed treatment of mercury and potassium iodid. Whether digitalis shall be given, either at the same time or before antisyphilitic treatment is begun, must, of course, be decided on the merits of the individual case; it depends solely on the functional power of the heart. Whenever it is possible and the delay is justifiable, it is better, in my opinion, not to give digitalis, because by withholding the drug there is a better chance of clearing up the diagnosis, which is such a great advantage later on. It is needless to say, however, that one should not run any risk on this account.

There need be no hesitation about administering mercury and iodin even when the patient's condition is grave; for we are now able, under all circumstances, to give both these drugs without doing harm, and, besides, it has already been pointed out that both these substances have

*Rosenthal, "Deutsche medicinische Wochenschrift," 1897 (No. 9), Vereinsbeilage, p. 57.

a favorable influence on the circulatory apparatus. For administering mercury I advise inunctions of gray ointment, 4 to 5 gm. (1 to 1¼ dr.). Potassium iodid may be given about two or three times a day in the dose of 1 gm. (15 gr.). In urgent or obstinate cases larger doses of both remedies may be required.

Prognosis.—If antisyphilitic treatment is instituted early, there is some prospect that the prognosis of heart syphilis may improve in the future. In fact, this is already borne out by some clinical observations. The reason why the prognosis has usually, in the past, been regarded as hopeless probably is that syphilis of the heart was practically known only through autopsy reports. As we know from our experience with the other organs of the body, antisyphilitic treatment is of no avail when begun very late because the effect of the drug is exerted against large masses of parenchyma which have been destroyed and are no longer capable of regeneration.

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DISEASES OF THE HEART OCCURRING AS THE RESULT OF ARTERIOSCLEROSIS.

Owing to its great frequency, arteriosclerosis has an extraordinary claim on the physician's attention and is, of course, discussed at length in a separate section in [the original of—Ed.] this system. Nevertheless, we cannot help devoting a section to it here, for no other pathologic process is so frequently the direct or indirect cause of functional disturbances of the heart muscle.

But our treatment of the subject must necessarily be more or less incomplete. Chronic arteritis usually involves the entire body; the morbid symptoms to which it gives rise have their origin in all the various organs and exhibit the greatest imaginable variety. As we have been intrusted only with the discussion of matters relating to the heart muscle, we can only present certain portions of the entire picture of arteriosclerosis. Here, again, we meet with special difficulties, chiefly didactic. Arteriosclerosis may affect the heart in one of three ways: First, through the structural alterations in the entire valvular tract and the changes in the blood-current; second, through disease of the coronary and renal vessels; third, through sclerotic valve lesions, especially of the semilunar valves of the aorta, and, much more rarely, of the mitral leaflets. The third factor is beyond our province, because the discussion of valve lesions has been undertaken by our colleague, v. Jürgensen, and because it is impossible, for many reasons, to separate sclerotic from the endocarditic valve lesions. Similar difficulties are encountered in sclerosis of the renal vessels.

There remain for discussion, therefore, the influence of general endarteritis on the heart and disease of the coronary arteries. Each of these conditions produces a great variety of symptoms, almost any one of which may occur by itself. On the other hand, however, any one symptom may be combined with any other; and the number of possible combinations, as well as the number of symptom-complexes that actually occur, is astonishing. Almost every patient presents some special feature.

In order to find one's way through such a maze of morbid phenomena the essentials of the disease must be firmly kept in mind. On this point some preliminary remarks appear to be called for.

Observations on the Pathogenesis and Pathologic Anatomy of Diseases of the Heart Due to a General Arteriosclerosis.—Although we cannot give a full description of the *pathology* and *etiology* of arteriosclerosis, we shall have to touch upon one or two questions belonging to each of these subjects. Since we are exclusively interested in the heart, the first question that suggests itself is: *What is the effect of general arteriosclerosis on the heart?* Many views are possible. A sclerotic artery is, in general, less elastic [*i. e.*, less capable of stretching, more rigid—Ed.] than an artery with a healthy wall;* hence the blood, as it flows into the vessel, meets with increased resistance. Whether an increase in the labor of the heart can be attributed to the greater rigidity of the vessel-walls will depend on the extent and distribution of the rigidity in the individual case. If the hardening process has attacked so many arteries that the increased resistance due to the greater rigidity can no

*Compare Polotebnow, "Berliner klinische Wochenschrift," 1868, No. 35. Israel, "Virchow's Archiv," vol. ciii, p. 461.

longer be equalized by dilatation of other arterial regions; or, if arteriosclerosis attacks arteries which, owing to the extent of the capillary region supplied by them, have a controlling influence on the pressure and quantity of blood in the vascular system, the effect of the arteriosclerosis simply is to throw a greater strain on the left ventricle, on account of the altered condition of the vessel-walls and, if this extra demand is met, to produce hypertrophy—in other cases, dilatation.

Under Romberg's direction Hasenfeld settled these matters by means of a careful investigation.* For reasons that everybody knows cardiac hypertrophy is found chiefly after disease of the intestinal arteries. Sclerosis of the first portion of the aorta also influences the work of the heart. The vessels of the brain and extremities are without any influence. Only the left heart becomes hypertrophic as a result of arteriosclerosis.

In view of the great frequency of arteriosclerosis it is to be hoped that these observations will be still further extended. In fact, such further observations are rendered absolutely necessary by the extraordinary variety of the pathologic processes and organic diseases produced by arteriosclerosis.

In the foregoing it has been assumed that a diseased artery is more rigid than a sound artery. This is unquestionably true in certain definite anatomic processes, but the available observations are extremely scanty, and so many different lesions occur in the vessel-wall (new formation, disappearance of connective tissue and elastic fibers, fatty changes, calcareous deposits, ulcers) that general statements must be made with great caution, for it is possible that the effect on the degree of elasticity of the wall may be quite different in different cases. This question cannot be settled by surmises, but requires careful observations; and these we must have.

It is important to distinguish between the *strength* of the elasticity and the *completeness* of elasticity (perfect elasticity) in speaking of diseased arterial walls; in sclerotic vessels the elasticity is in many cases at least diminished. This should cause dilatation of the diseased arteries, and the effect on the blood-current should, therefore, be quite different from the effect produced by hardening of the wall. But, as the very important investigations of Thoma have taught us, vessel-walls in yielding to the pressure of the blood suffer an alteration in their structure. Hence in attempting to answer the question how the blood-current behaves in sclerotic arteries, the conditions encountered are found to be most complicated; and, first of all, minute investigations of all the *physical* properties of diseased vessels, such as have already been begun in the case of normal tissues, are urgently needed.†

Among the vessels which influence the action of the heart when their walls become altered the *renal arteries* occupy a peculiar position. It is positively known that many forms of chronic inflammation of the kidneys cause extreme hypertrophy not only of the left ventricle but of the right ventricle, and both auricles as well. In addition, sclerosis of the renal arteries often leads to disease of the kidney itself, which in many respects resembles nephritis, and the thought, therefore, naturally suggests itself that changes in the renal arteries on that account also have a special influence on the action of the heart. But before this point can be settled, further clinical and pathologic observations are needed.

In renal diseases the problem is extraordinarily complicated because in many forms of nephritis, especially in a number of cases of contracted kidney, it is as yet impossible to decide what relation exists between disease of the parenchyma and disease of the renal vessels. Similar conditions, as we know, have an important bearing on the occurrence of cardiac hyper-

* Hasenfeld, "Archiv für klinische Medicin," vol. lix, p. 193.

† Compare Triepel, "Merkel-Bonnet's anatomische Hefte," vol. x, i, p. 1; vol. xiv, p. 315.

trophy in many different kinds of Bright's disease and have repeatedly been utilized for explaining this hypertrophy. According to competent observers, the central fact is a general change in the arteries which is probably identical with sclerosis, or at least resembles it very closely, and either precedes* the renal affection or is dependent upon it.† We cannot, of course, enter minutely into these matters; it must be mentioned, however, that many cases of cardiac hypertrophy associated with arteriosclerosis are in part at least, due to concomitant disease of the kidney. In such a case the relations between the various morbid processes may be conceived in a variety of ways. Thus, the nephritis may have been produced by disease of the vessels, but, nevertheless, itself exerts a peculiar influence on the circulation (arteriosclerotic contracted kidney?). Or the nephritis may be primary and the change in the arteries secondary (genuine contracted kidney?).

First, I wish to point out a special reason that renders the discussion of these things extremely difficult; it is the complicated character of the etiologic conditions. Many of the factors which we are in the habit of including among the possible causes of arteriosclerosis are also capable of injuring the kidneys, and, above all, as appears from the preceding sections, they have a direct influence on the heart. The excessive indulgence in alcoholic beverages, particularly when combined with the ingestion of large quantities of food, tobacco, coffee, and with bodily exertion, according to our present views, influence the kidneys as well as the arteries, and also affect the work of the heart. That is why these factors are so frequently encountered in the previous history of patients suffering from vascular, cardiac, or renal changes, and why it is so difficult to decide what etiologic relationship exists between the various organic diseases. Again and again I have emphasized the great importance of a combination of several factors in the causation of morbid conditions in individual patients, and it will now be understood why it is so comparatively rare to find an uncomplicated clinical picture and simple pathologic conditions; why heart, blood-vessels, and kidneys so frequently suffer together; and why arteriosclerotic diseases of the heart and diseases of the kidney with their sequelæ cannot always be strictly separated in practice from the conditions described in the foregoing sections. We shall advert to this important point again in the following pages.

Aside from the fact that under special circumstances the disease of the arterial wall as such is capable of increasing the work of the heart either directly or indirectly, there is another possible view in regard to the relations existing between general arteriosclerosis and the action of the heart; and this view is closely connected with the causes and origin of the vascular disease. It is more than doubtful whether this can be referred to a single cause; there is hardly a factor that has not been regarded as a cause of arteriosclerosis. In fact, we know nothing positive except that the action of toxic substances and, as the very important observations of Thoma have shown, distention of the arterial wall, must be regarded as important etiologic factors. Of course, these two things may also be closely related to one another.

The essential thing for us to determine is: if the development of general arteriosclerosis is in fact preceded by distention of the vessel-walls, to what must this distention of the vessel-walls be attributed? In that case the first thing in the whole clinical picture must be an obstruction to the blood-current in the smallest arteries or capillaries of the body, that is, a general increase of the arterial pressure.

*Gull and Sutton, "Medico-chirurg. Transactions," vol. lx. Lancereaux, "Dictionnaire encyclop. des sciences médic.," Art.: "Rein," ser. 3, vol. iii, p. 198. Cited after Edgren; compare Leyden, "Zeitschrift für klinische Medicin," vol. ii, p. 148.

†Senhouse Kirkes, "Medical Times and Gazette," 1855, vol. ii, p. 516.

Such an increase of the arterial pressure undoubtedly occurs as the result of many renal diseases, but the latter can by no means be regarded throughout as the ultimate cause of arteriosclerosis. For, in the first place, it has been mentioned that in nephritis and in cases of simple arteriosclerosis different parts of the heart become hypertrophied, and, in the second place, a great many arteriosclerotic patients do not suffer from nephritis.

We are, therefore, forced to attribute this increase in the pressure to circulatory processes which, so far, have entirely escaped our apprehension.

This hypothesis, which is defended by Traube and Huchard, is in many respects very attractive because it views diseases of the blood-vessels and of the heart, as well as the renal changes with which we are dealing (arteriosclerotic contracted kidney), from a common standpoint,—that of obstruction to the circulation in the small arteries,—and assumes that the former are coördinate phenomena produced by the latter.

But on examining the question more closely we encounter a great many difficulties. In the first place, if an increase in the pressure really precedes every form of arterial disease, to what are we to attribute this increase in pressure? Spasm of the smaller arteries, either of all the smaller arteries or at least those of the splanchnic region, is quite conceivable. Such a spasm need not be severe, since the cross-section of the vessels is in the fourth power in the formula for the work of the heart. But the spasm would have to be continuous, and we shall hardly venture to assume a permanent tonic contraction of the smaller vessels! So far as I know, such a phenomenon would be without example, and, therefore, the promoters of the theory would find themselves forced not only to maintain, but to prove, that such is the case. This question leads to discussions quite similar to those which have taken place on the explanation of renal hypertrophy of the heart.

Our judgment of this matter would largely depend on the behavior of the heart in detail. After wide-spread arteriosclerosis the heart may be free from any sign of hypertrophy. Can this mean anything else except that arteriosclerosis may develop without any increase in the arterial tension?

On the other hand, there are cases of chronic hypertrophy which, according to our present views, are directly dependent on arteriosclerosis. In the cases of this kind hitherto examined, in which the technic was unexceptional, only the left ventricle was found to be thickened, as has been mentioned. This positively indicates that there is obstruction in the greater circulation. In nephritis, and especially in contracted kidney, a condition which undoubtedly is closely related to the conditions with which we are now dealing, every portion of the heart, including the auricles,* undergoes hypertrophy. This can only mean that there is something which increases the function or—unless we persist in limiting the discussion to mechanic theories—the nutrition of *all portions of the heart and of the arteries.*

Whether the same thing is not true in many cases of arteriosclerosis has not, in my opinion, been definitely settled. A good many more observations conducted according to Müller's method are, as we have already seen, most urgently needed. Clinically, the thought of some connection between arterial, cardiac, and renal changes in the sense of general disease of the vascular system would appear to be a happy one. To explain the abnormal resistance in the circulation the same causes which were elsewhere mentioned for diseases of the kidney† might be considered, as, for example, the increased internal friction in the blood.

But this whole question requires investigation. Above all, it needs to be decided whether arteriosclerosis, on the one hand, and diseases of the kidney, on the other, always cause hypertrophy of different portions of the heart.

One thing is certain, sclerosis of certain vascular regions increases the demand on the left ventricle and thereby leads to hypertrophy or dilatation of the ventricle, depending on its power of accommodation. From the following it will be seen why functional weakness of the heart is particularly apt to develop in association with arteriosclerosis.

Remarks on the Pathologic Anatomy of Sclerosis of the Coronary

* Romberg and Hasenfeld, "Archiv für klinische Medicin," vol. lix, p. 193.
† Krehl, "Pathologische Physiologie," p. 32.

Arteries.—Sclerosis develops in the arteries of the heart for the same general reasons that cause the vascular alteration in other organs. Just why the coronary vessels are attacked must, in my opinion, for the present remain a complete mystery.

One thing may perhaps be mentioned: every form of psychic emotion accompanied by a marked influence on the action of the heart, especially care and worry, seems to favor disease of the coronary vessels to a marked degree.

The sclerotic process impairs the blood-supply of the heart, and this, in turn, is followed by a great many different sequelæ. Wherever the arteries are completely occluded, the muscle-fibers whose nutrition and gaseous interchange has ceased die (Ziegler's myomalacia cordis*)—in this sense the coronary arteries may be regarded as end-arteries—their substance is gradually removed and replaced by connective tissue which, at first, is quite cellular, but later loses many of its cellular elements. In this way some of the well-known fibroid patches are produced. They vary in size, which is directly dependent on the order of the occluded artery.

Old scars are apt to become the seat of extensive calcareous deposits. The entire heart-wall may become as hard as stone, and it is difficult to understand how the muscle can contract at all. This condition has been described in literature under the term "osseous heart," and I have personally seen quite a collection of such specimens in the possession of my honored teacher, Curschmann.

It is extremely difficult, if not impossible, to estimate the functional value of the fully formed connective tissue. It unquestionably depends on the size, number, and location of the foci; for the conversion of a large portion of the heart-wall into connective tissue cannot help influencing unfavorably both the contractility of the heart and its power to expand. And who will venture to deny, in the present state of our knowledge of the structure of the heart, that a focus of degeneration may not be much more harmful in one place than in another? Nevertheless, one often sees hearts with a large number of *old* scars in which the functional capacity is still relatively quite good.

When extensive scars occupy a circumscribed portion of the heart-wall, especially the apex,—more rarely other portions of the organ,—the condition may lead to thinning and bulging, or so-called aneurysm of the heart.†

In itself this condition has no more pathologic significance than large fibroid patches in the heart, and cannot, of course, be diagnosticated. In rare cases, when the thinning of the wall attains a very high degree, rupture takes place with all the symptoms of rapid accumulation of fluid in the pericardial space.

A deficiency in the blood-supply of living muscle-fibers has, in my opinion, much greater functional significance. This condition occurs from narrowing of the coronary arteries or their branches, and during the production of these myomalacic areas. This view is borne out by numerous experiments which show that in animals also a disturbance of the heart action is most easily caused by changes in the condition of the cells.

When the blood-supply of individual portions of the heart is impaired without being completely abolished, a variety of different changes appears in the muscle-

* For the literature on this condition and its causes see the sections on Carditis and Myocarditis.

† This term, as at present used, is not to be confounded with Corvisart's acceptation. The great French physician used the term *aneurysma cordis* to describe the condition which we now call dilatation.

fibers of the diseased area. The chief of these are the so-called granular and fatty degenerations, which, according to the theories prevailing at the present time, gradually merge into complete necrosis. The process may be rapid or slow, depending, of course, on the changes present in the arteries; and, accordingly, the degenerations vary in distribution and may assume many different forms in individual portions of the heart at the same time.

There is no doubt whatever that those biologic processes which are associated with the deficient blood-supply of the entire heart muscle or some of its portions are chiefly responsible for the production of the symptoms observed in coronary sclerosis. This applies to the sensory as well as to the motor symptoms. Of the fact itself it seems to me there can be no doubt; but with regard to the interpretation or explanation of the connection between the clinical and the anatomic processes, it is, in my opinion, difficult to formulate hypotheses.

The sensory symptoms (angina pectoris) have already been described on p. 531. As regards the disturbances of the functional power and of the action of the heart, all we can say is: we know that both are caused by changes in the condition of the muscle-fibers, such as accompany inflammations, degenerations, and a sudden impairment of the blood-supply.

The well-known experiments of ligating the coronary arteries, which will be mentioned later, have shed some light on this question, although not as clear a light as these experiments have often been credited with. Experimental occlusion of coronary vessels frequently injures the action and capacity of the heart, but not always; and, above all, does not regularly produce death provided the occluded vessel is not one of the first order and the distortion of the muscle-fibers, which is so apt to accompany the experiment, is carefully avoided.

These results of animal experimentation agree very closely with clinical experience. Not infrequently anemic infarcts of considerable size are found which must have been formed more or less suddenly weeks or months previously, and are due to occlusion of large vessels. Sometimes the history reveals anginoid symptoms, which may be assumed to have coincided in time with the accident; but sometimes these symptoms are wanting, and, at all events, it is found that the accident happened long before the patient's death.

The most important factor in the functional impairment of the myocardium when the blood-supply is deficient is the disproportion between the degree of metabolic activity required by the muscle *in full action* and the degree of which it is capable in disease of the coronary vessels. On this theory it is easy to understand why the insufficiency increases in proportion as the demands on the heart are increased. This point, so far as it applies to sensory phenomena, was taken up in the discussion of stenocardia.

It follows, from what has been said, that any one who attempts to epitomize the changes in form and size that may take place in the heart in the course of arteriosclerosis must be prepared to find the greatest imaginable variations. Hypertrophy of the left ventricle undoubtedly occurs, although it does not usually attain a very high degree.

In addition we have functional weakness and passive dilatation of various portions of the heart. Both conditions, which, as a rule, are due chiefly to coronary sclerosis, occur in a great variety of combinations with hypertrophy. On the other hand, dilatation occurs not only without thickening, but even with considerable thinning, of the wall. The latter occurs when the arteriosclerosis has chiefly or exclusively attacked the coronary vessels.

If the coronary disease is followed by a progressive destruction of muscle-fibers; if nothing develops to stimulate the myocardium to increased activity, the heart may attain a considerable degree of atrophy in

coronary sclerosis. Many a functionally "weakened" heart, besides being structurally diseased, is absolutely small and puny.

Symptoms.—The difficulty of giving an isolated presentation of a portion of the arteriosclerotic symptoms, which has been hinted at above, at once makes itself felt in describing the symptomatology. Our province is the heart. Heart symptoms caused by arteriosclerosis may, of course, at any time be combined with symptoms referable to other organs; and, as the arterial disease may attack practically all the other organs of the body, it is found that the symptoms of the cardiac disease are frequently associated with a great variety of other processes. To get over this difficulty we shall here describe only the symptoms referable directly to the circulation, although, for diagnostic reasons, we shall be obliged to mention some facts relating to the kidneys. Everything else the reader is requested to supply himself.

There is, however, another and a much greater difficulty, namely, to give such a description of the manifold and variously combined symptoms caused by general and by coronary sclerosis as will absolve us from the criticism either of adhering to a preconceived scheme in defiance of nature or of presenting the symptomatology in an utterly irregular and unintelligible way.

Huchard, whose book abounds in excellent clinical descriptions, adopts the usual expedient of his countrymen, that of erecting types. Although, for many reasons, I am unable to follow him in all the details of his classification, I must confess that I have no better rule of action. I shall first try to describe a few clinical pictures, such as the physician often sees, and then proceed to explain how they may be combined.

Before beginning the description proper of the clinical phenomena, attention must be called to the fact that not a few individuals with sclerosis of the peripheral arteries (as we know from clinical observations), as well as persons with sclerosis of internal vessels (as we know from our anatomic experience), present no cardiac symptoms whatever. I find no difficulty in understanding this; for it follows from what has been said above (p. 674) that arteriosclerosis exerts its influence on the heart only by virtue of a general distribution or, if the process is local, through the agency of certain vascular regions, particularly the thoracic aorta and the splanchnic and coronary vessels. It seems fair to assume, therefore, that in cases of arteriosclerosis without cardiac symptoms the above vascular regions have escaped the disease. We mention the occurrence of vascular disease without any symptoms referable to the heart partly because the fact has a diagnostic significance, but partly also because of its great theoretic interest. For how can we reconcile this fact with the theory that arteriosclerosis is always produced by a general increase in the arterial pressure?

In a number of arteriosclerotic individuals we also find the clinical symptoms of hypertrophy of the left ventricle, associated, as a rule, with a slight degree of dilatation. These patients are usually men belonging to the wealthier classes, about forty years of age, who have lived well—as the saying is—in every respect and have not stinted themselves in wine, beer, plenty of animal food, and other articles of diet that are usually eaten at the same time. They present a healthy appearance, have florid complexions, tinged, it is true, with a suspicion of cyanosis. Not infrequently, indeed usually, the adipose tissue is excessively developed. Sometimes these patients have no subjective symptoms and the physical signs are discovered, so to speak, by accident. But quite often their efficiency is impaired in every way, and that is what takes them to the physician.

There may also be dyspnea, at first only after muscular exertion. This may be all; but on careful interrogation other symptoms are elicited: tendency to bronchial catarrh, peculiar dyspneic conditions, anxiety, pain in the chest, and palpitation, which occur either without any provocation at night or as the result of muscular movements which had been previously performed without any discomfort whatever, and not rarely also after a copious meal. Severe attacks of cardiac asthma or angina pectoris are not infrequently present. Quite often combinations of cardiac asthma with angina pectoris are observed. These things have all been described in detail in the section devoted to the Sensory Heart Symptoms (see p. 531).

The heart in these patients shows all the signs of hypertrophy and slight dilatation of the left ventricle; the second aortic, and sometimes the second pulmonic as well, is accentuated and usually ringing on account of sclerosis of the aorta. The walls of the arteries in the head, neck, and arms are generally hard and tortuous. The pulse is often large and always hard. The dicrotic wave is frequently situated high up on the descending limb of the sphygmographic curve. Irregularity of the heart action is usually due to sclerosis of the coronary arteries; but it would be altogether wrong to exclude arteriosclerosis because of the absence of arhythmia.

This form or stage in the cardiac changes brought about by arteriosclerosis is unquestionably much more common in large cities, where life is luxurious, than in the country and in places where there is less so-called "culture." It is certainly more than an accident that Traube, Fraentzel, and Huchard from their consultation practices in Berlin and Paris, have given us so many descriptions of this condition, while in the Policlinic and in the public hospitals it is observed comparatively seldom; but I believe it is too early as yet to express an opinion in regard to its frequency in general. For although in these cases, owing to extension of the boundaries of the lungs, one cannot depend on the heaving character of the apex-beat to establish the diagnosis of hypertrophy of the left ventricle, the diagnosis was, nevertheless, and still is, based on "the heightened tension of the arteries" (to use Traube's expression). But it is necessary to make a technical distinction between increased tension of the walls of the vessels and heightening of the blood-pressure. The latter must be permanent, to be used as an indirect argument of the presence of cardiac hypertrophy; for increased tension of the vessel-walls is by no means always concerned in its production. It is always difficult and often impossible, to distinguish between increased tension of the vessel-wall and heightening of the blood-pressure, and there is no doubt that the two conditions are frequently not differentiated unless a special effort to do so is made. Nevertheless, I do not regard it as proved that these two conditions were not formerly confounded even by excellent observers. In the future the difficulty will be, at least in part, eliminated by the use of instruments for determining blood-pressure, provided the estimation is made with the proper precautions. What needs to be done, first of all, is to investigate, by the aid of this method, the frequency of this form of arteriosclerotic heart disease among as large a number of patients as possible, from clinical material that shall be both abundant and of heterogeneous composition.

In general medical practice, however, there is no doubt that the symptoms of simple dilatation of the left ventricle without hypertrophy are much more frequent. This is a typical and, according to my experience at least, an extremely frequent clinical picture. The patients are mostly

between thirty and thirty-five years of age and of the male sex, although cases are not very rare among women and among young persons who are still in their early twenties. The disease attacks individuals of every social rank and every variety of nutrition, and the traditional views about the cause of arteriosclerosis often leave one in the lurch in an individual case.

As a rule, the patient's first complaint is, that he is less able than formerly to bear the strain of his regular work and of life in general. During bodily and mental rest he feels perfectly well, at least in the beginning of the trouble; but as soon as he exerts himself, and sometimes after psychic emotion, he quickly becomes tired and short of breath and is apt to suffer from a certain sense of pressure on the chest. After a renewed period of rest all the symptoms disappear and the patient feels perfectly well, although sometimes various sensory disturbances occur spontaneously, and at night the patient's rest may be disturbed by sensations suggesting cardiac asthma or even by genuine attacks of the disease.

The physical signs are equally typical: displacement to the left and sometimes also downward of the apex-beat, the force and resistance of which are only moderate or even diminished. Unless, as frequently happens, the boundaries of the lungs are enlarged and obscure the results of percussion, the absolute and the relative heart dulness are both enlarged to the left. The sounds are either altogether unchanged, or the first sound at the apex is impure—that is, accompanied by an adventitious sound which must be attributed to muscular insufficiency of the mitral valve. The second aortic sound often appears to be somewhat louder than normal, higher in pitch, and quite often ringing; but it must always be remembered that this ringing quality of the second sound has hardly anything to do with increased pressure in the aorta, being probably caused by local changes in the vessel-wall. The peripheral arteries referred to above are tortuous, and the walls in places are sclerosed. In not a few cases the patients complain of the same subjective symptoms, although examination of the heart shows not the slightest abnormality; the apex-beat is normal, the size of the heart and the sounds are not altered. Nothing but careful palpation of the vessels reveals the origin of the trouble. This is not difficult to understand, for there is no reason whatever why hypertrophy should occur. Dilatation does not develop, because no great demands are made on the heart, and even the sounds at the base are found unchanged because the wall of the ascending aorta in these cases is evidently not diseased. The subjective symptoms are explained by the reduction in the strength of the heart which renders it incapable of meeting the demands made upon it.

Renal symptoms are not infrequently present in patients of both these groups. I do not here refer to passive renal congestion, which frequently develops when the right ventricle is weak; I refer to the association with well-known forms of renal inflammation. Thus, we not infrequently observe in association with arteriosclerosis the typical, genuine, contracted kidney, and the combination is particularly frequent when gout is the prominent feature in the clinical picture.

In arteriosclerotics with increased arterial tension the quantity of urine is not infrequently increased, the specific gravity is low, and the urine contains no albumin. If the same conditions are found again and again, it is practically impossible to draw any positive conclusion; in such a case the increase in the excretion of urine may be due to an increase in the velocity of the blood. But great caution is necessary in giving an opinion, because

it is possible that the kidney may be the seat of morbid processes belonging to the group of genuine contracted kidney. This point will be discussed presently. Or the urine may be free from albumin and the quantity about equal to the average, but the specific gravity is considerably above the normal. In such a case one is justified in saying that the quantity of urine is relatively diminished—sometimes it really is remarkably low. But in both groups of cases some of the urinary findings not rarely indicate a definite change; occasionally, especially after marked exercise, traces of albumin make their appearance. Formed constituents are usually not found, but a patient and attentive search not infrequently reveals blood-corpuscles and very few hyaline or granular casts, or casts with only one or two epithelial cells. These are very important matters and emphatically indicate the existence of a morbid process in the kidneys. Indeed, the renal disease not infrequently manifests itself positively by some uremic symptom.

Thus uremia may occur at any time in the course of arteriosclerosis and often causes the physician an unpleasant surprise if he has overlooked the possibility of a nephritis because of the absence of albumin.

What is the condition in the kidneys? The question will have to be answered by future investigations. The process is probably either closely related to, or identical with, so-called arteriosclerotic contracted kidney, possibly sometimes with the above-mentioned "congested and contracted kidney." More I cannot say for the present. But it cannot be denied that there is a close connection between arteriosclerosis and many forms of exceedingly chronic diseases of the kidneys. In spite of distinct inflammatory and contracting processes in the kidneys, no albumin may be found in the urine for months in a specimen representing the average of the twenty-four-hours urination. The quantity of the urine is normal, but the specific gravity is considerably increased. Whether albuminuria is present even after active exercise, has not, I believe, been sufficiently investigated.

The above described clinical symptoms of those forms of arteriosclerosis which influence the action of the heart are accompanied by irritative and paralytic phenomena. The latter are by far the most important; for even in cases of increased arterial tension and hypertrophy of the heart muscle the patient's complaints and many of the objective signs directly show that the functional power of the organ has suffered. In the second group of symptoms this diminution is quite marked.

How is it brought about? We may answer, confidently, by disease of the coronary arteries and processes in the heart muscle resulting therefrom. At least when arteriosclerotic subjects die with symptoms of cardiac insufficiency, the explanation is invariably found in these factors, and the same reasoning may, therefore, be applied to the earlier stages of the disease.

This diminution in the power of the heart as a result of coronary sclerosis calls for a few remarks. It must be remembered, in the first place, that it occurs in every imaginable degree. As has been mentioned, the mildest cases do not necessarily present any physical signs in the heart, and the diagnosis is based solely on the history and on the patients' statement that they get out of breath on exertion. On the other hand, we have the most extreme degrees of cardiac insufficiency, the highest grades of dilatation, with all the symptoms of abnormal blood distribution. Between these two extremes there are all kinds of curious variations—sometimes enlarge-

ment of the left ventricle, sometimes enlargement of the right, and again of both; in every case unquestionably dependent on the seat of the arterial disease and the kind of demand made upon the organ. It has been stated that the left coronary artery is more frequently attacked than the right. If that is the case, it will help us to understand why the left ventricle is so frequently dilated in coronary sclerosis. The increased resistance in the arteries no doubt contributes, at least in a good many cases, to the dilatation of the left ventricle; but it seems to me that the changes in the heart muscle are much more important, because the pressure in the arteries, as has been stated, is often not increased.

The greater and more frequent involvement of the left ventricle also agrees with the occurrence of cardiac asthma. This curious condition has been described both in its clinical and in its pathogenetic relations on p. 509; but we have to call attention to it again in this connection because, after contracted kidney, arteriosclerosis is distinctly its most frequent cause. Here, again, coronary sclerosis is probably the all-important alteration. Either it produces absolute weakness of the left ventricle, in which case dyspnea is present with a low pulse tension; or it produces a relative insufficiency of this portion of the heart, and in that case the dyspnea is associated with a high blood-pressure and a high pulse, as in diseases of the kidney; indeed, it may be said that the dyspnea is due to these factors.

It has already been stated that cardiac asthma not infrequently gives rise to local or general pulmonary edema, and this again happens chiefly in patients with arteriosclerosis. All that is found described on p. 510, etc., is present in this condition; in fact, the descriptions referred to were taken from the clinical pictures of this condition. The reason they are given at that point rather than here is that they occasionally, although much more rarely, occur in other diseases of the heart muscle as well.

It can hardly be assumed, from our present views, that coronary sclerosis as such produces hypertrophy of the ventricles; although some of the older investigators, as Buhl, for example, were inclined to attribute the thickening of the heart muscle to some inflammatory process, and sometimes one is almost inclined to adopt this view at autopsies. But at present hypertrophy occurring during disease of the coronary arteries is attributed to sclerosis in other vascular regions. More detailed observations might possibly help to clarify this question also.

In addition to these quantitative changes in the action of the heart certain qualitative alterations may also occur in coronary sclerosis. Quite frequently the rhythm of the heart is disturbed. Single pulse-beats may be delayed, or omitted, or may occur prematurely; the pauses between the systoles become unequal even to the sense of touch, and the systoles themselves vary in force. These modifications sometimes occur only at long intervals—say every twenty to thirty beats, or even more, or they may be constantly present. The greatest variations are found in this respect. Embryocardia and especially gallop-rhythm also occur; their significance has already been discussed. But it must always be remembered that coronary sclerosis without any disturbance of the rhythm whatever is not so very rare.

Changes in the frequency of the heart-beat are also quite frequently observed. There may be a persistent acceleration, so that the pulse, at least during the daytime, never falls below 80 to 100; or the pulse is normal while the patient is at rest, but increases after the slightest muscular

exertion, to a rate out of all proportion to the cause. Persistent acceleration is quite the rule in the later stages of coronary sclerosis.

An even greater increase in the frequency of the heart-beat not rarely takes place, but such extreme rapidity is always paroxysmal. It is possible that a good many cases of so-called paroxysmal tachycardia are really cases of coronary sclerosis. In the midst of absolute or, at least, relative health, often after some moderately severe exercise or psychic emotion, the pulse frequently goes up to 180, 200, or 250, and all, or at least part, of the remaining symptoms described at length in the section on Paroxysmal Tachycardia make their appearance.

Retardation of the heart-beat is also observed after coronary sclerosis. It has already been mentioned on p. 460 that it is difficult to recognize the milder grades of these conditions on account of the individual variations under normal conditions. But a reduction of the pulse-beats to 56 should always arouse attention; and if they are reduced to 48 or less, the diagnosis of pathologic bradycardia is assured. There is no other disease of the heart in which bradycardia is as frequent as it is in sclerosis of the coronary arteries. The most extreme degrees of retardation are reached. Thus, eight contractions in a minute have been observed, and I have personally found the pulse between 50 and 30 on various occasions. In spite of this extreme bradycardia the patients may continue in relatively good health for years, but, as a rule, their efficiency is greatly reduced. The pulse, besides being slow in these cases, is always slightly irregular as well. The failure of atropin to influence the frequency of the heart is a positive sign that the bradycardia is due to some cause residing in the heart muscle,* probably in the auricles.

In these patients with bradycardia due to coronary sclerosis curious nervous phenomena occur which are known in the literature under the name of *Adams-Stokes' disease.*† Consciousness is lost either suddenly and without previous warning or after an aura of some kind—a peculiar sensation of smell, taste, or hearing—or a tactile sensation or something similar. The warning given by this aura enables many patients to reach a couch before the attack comes on, others fall to the ground at once. Consciousness is always completely abolished; the breathing is often retarded and stertorous, and sometimes exhibits the well-known phenomenon of Cheyne-Stokes' respiration, while in other cases it ceases altogether. Palsies are absent both during the attack and afterward. The pulse during the attack is even less frequent than ordinarily. The rhythm of the heart, as well as the rhythm of the auricles, may be altered in a variety of ways,‡ both independently and in their relations to one another.

The attack usually lasts a few seconds or minutes, rarely hours. The patient then awakes without any paralysis, and in many cases is at once restored to his former condition. But most of these patients complain of lassitude after the attack, and do not fully recover for some time. Quite often the attacks are repeated. I know a patient who has had several hundred of them in the past ten years.

The *causes and pathogenesis of the attacks* are quite unknown. Many patients cannot give any reason for them whatever; others mention

* Dehio, "Petersburger medicinische Wochenschrift," 1892, No. 1.

† Stokes, "Diseases of the Heart," Ch. v. Huchard, "Maladies du cœur," p. 255. His, "Archiv für klinische Medicin," vol. lxiv, p. 316. Contains the literature. Krause, "Ueber einen Fall von Bradycardie," Dissertation, Göttingen, 1895.

‡ See the interesting case reported by His, *loc. cit.*

exercise and emotional excitement. One of my patients attributes his attacks to the eating of various kinds of food, such as dark meat, for example; but I have never been able to satisfy myself that suggestive and unknown causes may not have been much more important.

In studying the pathogenesis the first question that presents itself is: how is the heart attacked by the morbid process? It hardly seems possible as yet to give an answer to this question, and one of the principal reasons is the want of uniformity in the etiology and nature of these conditions.

A few cases must be regarded as due to uremia. In others one is inclined to think of vascular disease of the cerebrum and of the medulla oblongata. Such cases, as Huchard very correctly points out, naturally suggest a comparison with the symptoms of intermittent claudication. These questions ought to form the subject of investigation in any future anatomic studies of the condition of the brain in these cases.

It cannot be doubted that the abnormality of the heart action in some way contributes to the causation of the attacks, and the cerebral anemia due to bradycardia undoubtedly has something to do with the pathogenesis. But to ascribe everything to cerebral anemia in the simple way that is often done is, in my opinion, altogether out of the question.

[Our knowledge of Stokes-Adams' disease entered a new phase in 1905, when Joseph Erlanger demonstrated its relation to *heart-block*. The elucidation began with Stannius, Gaskell, and Engelmann, but could not be completed until the discovery of His' bundle (1893) and the production of heart-block, *i. e.*, the impairment (partial heart-block) or the destruction (total block) of the stimulus conduction from the auricle to the ventricle, by His, Jr. (1895). Erlanger, Humblet, and Hering independently demonstrated the mechanism of heart-block, and Erlanger especially, by a brilliant series of experiments on dogs, worked out the physiologic processes involved and was able, in that way, to complete the analysis of two cases of Stokes-Adams' disease in the wards of Professor Osler.

Experimentally, Erlanger showed that, by compression of His' bundle, all stages of heart-block may be produced, *i. e.*: "(*a*) An increase of the intersystolic pause; (*b*) an occasional ventricular silence; (*c*) regularly recurring ventricular silences—*e. g.*, one in 10, 9, 8, 7, 6, 5, 4, 3, and 2 auricular beats; (*d*) a 2 : 1 rhythm; (*e*) a 3:1 rhythm; (*f*) complete heart-block," in which the ventricle beats independently of the auricle, as the result of the automaticity of heart muscle.

"As a rule, the ventricles take on a constant slow rate at the moment complete heart-block is established.

"When the block is complete, stimulation of the vagus has no, or but minimal, effect upon the rate and force of the ventricular beats, whereas, the auricles still react normally.

"When the block is complete, stimulation of the accelerator nerve increases the rate both of the auricles and the ventricles.

"When the block is complete, the rate of ventricular beats may not be materially affected by variations in the general blood-pressure, nor by asphyxia, nor by interference with the coronary circulation" (Erlanger, 1896).

The relation between experimental heart-block and heart-block in man is very close, and the slight differences, such as the impossibility of keeping up a partial block in the dog for a long time, may be due to the differences between "the crudeness of experimental methods as compared with the subtle inroads of disease" (Erlanger).

The pulse and cardiac phenomena of Stokes-Adams' disease are exactly those of heart-block. The condition is not a simple bradycardia, as the shrewd Stokes perceived, but a loss of coördination of the heart in which the auricles may beat with normal or increased rate, but the ventricles always at an infrequent rate. The syncopal attacks can be explained by slowing of the ventricles (Erlanger). Webster showed that the epileptiform attacks may be due to cerebral anemia; the apoplectiform attacks can be produced in the same way or by venous congestion. It is not always clear what produces the attacks, but it is reasonable to suppose that either a change in the ventricular rate or force is the most common cause. The former has been seen by His and others.

Clinical and pathologic-anatomic proofs of the relation of Stokes-Adams' disease to lesions of the auriculoventricular bundle have accumulated rapidly since Erlanger's experiments were published. The lesions have included such processes as sclerotic change in the endocardium, involving the bundle, gummata in or on the bundle, cartilaginous tumors, fatty infiltration with atrophy of the bundle. Gummata are relatively frequent. In some cases no gross lesions have been found. In one of mine there was a small fibroid area in one leg of the bundle; in another, only minute fibroid areas between the fibers of the bundle, with moderate endarteritis in the artery in the bundle. Aschoff reports a similar case. Whether disease of other parts of the heart or of the nerves can produce the symptoms is not yet known. Though the subject is still incomplete, it can be said that Stokes-Adams' disease is a disease of the heart and not of the central nervous system.

The diagnosis of the disease is based upon the clinical symptoms and the demonstration of heart-block. The latter can often be made by observing a characteristic difference of rate between the cervical venous pulse and that of an artery. This was especially striking in one of Stokes' cases (Case XXXIII), but in some cases the jugular pulse is not distinct enough to be of assistance, or it may be impossible to determine its rate by the eye. Synchronous tracings of the jugular (external, if possible), and the radial, apex, or carotid, are the most certain guides, using the polygraph of Mackenzie, or any other efficient instrument. By the use of the fluoroscope it is possible to see the dissociation of the auricular and ventricular contraction. Alternating pulse and some forms of extrasystole can probably always be distinguished from the heart-block by a study of tracings. Heart-block is probable when a pulse is near 30. In some cases of Stokes-Adams' disease the radial pulse may disappear for many seconds, minutes, or even days. The faint contraction of the auricles may still be audible at such times. Experiments show that the ventricles remain free from contraction during the long pauses, becoming greatly dilated.

The diagnosis of heart-block does not prove a case to be one of Stokes-Adams' disease. The former occurs temporarily as the result of infections or intoxications (digitalis!). When such causes can be excluded, and when a patient has arteriosclerosis, especially with evidences of coronary disease, the Stokes-Adams' syndrome should be suspected.

Besides the cardinal symptoms, all the subjective and objective symptoms of angina pectoris may be present, with Cheyne-Stokes breathing at times. Syncopal attacks or convulsions may not occur until long after the heart-block is discovered. Or the attacks may cease and the slow pulse continue. So in one of my cases there were very many syn-

copal and epileptiform attacks with extreme general and cardiac weakness for about six months, and then the patient was free from subjective symptoms, was able to work in his store, but with a pulse never above 30, for more than twelve months until he dropped dead while preparing for supper.

The prognosis depends upon the underlying condition and is, therefore, as uncertain as angina pectoris. Patients may die in the first attack, or may live for years with variable symptoms and sometimes with considerable preservation of the circulation. In cases with syphilitic histories prompt and active treatment may bring about recovery.

In other cases the treatment is symptomatic. In two of my patients the persistent use of atropin and strychnin, singly or together, seemed to improve the general condition and force of the circulation, though in neither case did the pulse exceed 32 in the minute. Nitroglycerin does not seem so useful as in many cases of arteriosclerosis without the peculiar symptoms. Alcohol seems to cut short the paroxysm in some cases; in others, changes of posture, such as getting on the hands and knees. Digitalis is usually not indicated and may do harm.

It is not possible to cite many, even of the most important, of the numerous recent articles. See Erlanger, J., "On the Physiology of Heart-block in Mammals, with Especial Reference to the Causation of Stokes-Adams' Disease," "Journal of Experimental Medicine," vol. vii, 1905, p. 676, and vol. viii, 1906, p. 8. Discussion at the Toronto meeting of the British Medical Association, "British Medical Journal," vol. ii, 1906, p. 1103 *et seq.* Ashton, Norris, and Lavenson, "Adams-Stokes' Disease (Heart-block) Due to a Gumma in the Interventricular Septum," "American Journal of the Medical Sciences," January, 1907.—ED.]

As has been mentioned, sclerosis of the coronary arteries produces sensory heart symptoms. All the symptoms described in the general portion (see p. 531), as a matter of fact occur in coronary sclerosis; for disease of the coronary vessels is a classic, and by far the most common cause of heart pain and heart fear. These symptoms will not be described again in detail because everything is mentioned on the page referred to.

The faintest, apparently most insignificant degrees of palpitation and pain in the chest and oppression are observed in coronary sclerosis—so slight that only the expert considers them worthy of any attention. On the other hand, the most frightful angina pectoris is also seen. A great variety of transitional and, so to speak, extremely mild forms of these conditions occur; but even apparently heterogeneous conditions—although in reality very closely related—occur side by side.

These sensory disturbances may accompany any degree of cardiac weakness and are also observed independently, at least without any demonstrable abnormality in the strength of the heart's contraction or in the rhythm.

In addition to the symptoms referable to the circulatory organs, there are all sorts of symptoms referable to other organs and occurring in every conceivable combination and chronologic order. Arteriosclerosis causes structural disturbances in numerous tissues, and it is very curious that the vascular alteration may be especially well developed in one organ, or may even be confined to that organ, while all the others—perhaps including the heart—are still quite normal. These phenomena interest us chiefly because they often accompany cardiac disturbances and may thus make it possible to recognize the arteriosclerotic nature of these disturbances. Occasionally they also afford very definite indications for treatment.

The important pathogenetic factor in the development of the organic diseases is the insufficient blood-supply of tissue-cells and a congestion caused by the first degrees of cardiac insufficiency. We can do no more than summarize these matters. The well-known cerebral symptoms; arteriosclerotic changes in the eye-ground; hepatic, gastric, and intestinal symptoms; the effects of an insufficient blood-supply to nerves, muscles, or an entire extremity, which were recently described so well by Erb,* are the most important.

The Combination of Symptoms and the Clinical Course.—Almost all these phenomena, numerous as they are, may occur separately, or, on the other hand, may be combined or follow one another in many different ways. Irregularity and everything that is peculiar and bizarre is characteristic of this disease; the rule is that there is no rule.

In general the development is progressive; mild and insignificant phenomena are gradually succeeded by graver symptoms, and in the end we have the well-known picture of bilateral cardiac insufficiency with cyanosis, low arterial pressure, soft and rapid pulse, bronchitis, dyspnea, hepatic congestion, edema, and albuminuria. The irregularity in the order of these symptoms, which has already been pointed out in the general description of cardiac insufficiency, applies with special force to the arteriosclerotic form. Many patients develop enlargement, induration, and tenderness of the liver early in the disease; others may have slight edema about the ankles or an obstinate bronchitis along with the earliest symptoms in other parts of the body. Any one of these symptoms may have an important bearing on the diagnosis, as well as on the whole general conception of the condition; hence a careful examination of the entire body is always absolutely indispensable.

Thus, cases which, so to speak, gradually pass through all the various stages of the disease from the mildest to the most severe may be protracted for years or even decads, or may end fatally in a few months or weeks. In this respect also the rule is more elastic than in other forms of heart disease.

When the disease is protracted, the patients often suffer horribly. We have already spoken of cardiac dyspnea: it is more agonizing in coronary sclerosis than in almost any other condition. The poor sufferers are often forced to spend weeks or months sitting up in bed or even in an arm-chair day and night. Anginoid conditions and the many other distressing consequences—edema, bed-sores, and the like—which so often follow permanent cardiac weakness fill the measure of their misfortune.

Again deviations from this gradually progressive course (from good to bad) are very frequently encountered. Sometimes the disease becomes stationary at various stages, and relative recovery may take place. There are patients who get along for years with a certain degree of cardiac insufficiency; the already existing structural changes and the morbid symptoms depending on them persist, but no new ones develop. The morbid process may progress in one organ and become stationary in others; in the kidneys this may account for the above-described moderate or, one might say, indistinct symptoms.

Finally, relative recovery from individual symptoms may occur, because those portions of the heart muscle in which the fibers have been injured by insufficient blood-supply undergo complete atrophy. This does not necessarily result in the production of any morbid symptoms,

* Erb, "Deutsche Zeitschrift für Nervenheilkunde," vol. xiii, p. 1.

either because the functional loss of certain portions of the muscle is borne without any disadvantages or because it is compensated for by the activity of other cells. In this connection I call attention to Curschmann's interesting cases* in which angina pectoris of a severe type disappeared after the complete occlusion of coronary vessels which had previously been stenotic.

On the other hand, a patient with arteriosclerosis may at any time die a sudden death, which may be due to a rapidly fatal cardiac insufficiency, an attack of angina, pulmonary edema, or pneumonia, not to mention possible intercurrent diseases. The latter are particularly significant because the heart in patients of this class is so very apt to get out of order. The slightest provocation of whatsoever kind is capable of bringing on the most severe and not infrequently fatal conditions of cardiac insufficiency.

Indeed, sometimes the duration of the entire disease is limited to seconds, minutes, or hours. This is the case in those individuals who retain their full powers until the very end and succumb to the first or second attack of angina pectoris, or, without any such attack, to coronary embolism, which is, of course, very apt to occur in these conditions. Sudden heart failure from coronary sclerosis is unquestionably the most frequent form of sudden and unexpected death.

It is thus seen that one must always be prepared for surprises in the course of arteriosclerosis. The most unexpected may occur; any phenomenon, of whatever nature it may be, may develop with extraordinary rapidity and disappear again quite as rapidly and unexpectedly. In giving an opinion of the duration of the disease the physician must, therefore, be extremely careful and reserved. Persons who look as if they were certainly doomed to die within a short time may recover apparent health and be able to return to their work.

Diagnosis.—It is impossible to discuss the diagnosis in the lump; all we can do is to discuss the individual symptoms, their mode of combination, and the clinical course.

Obviously the first question is: *how is arteriosclerosis in general recognized?* By examining the arteries. Of course. But since we have seen that excellent clinicians believe that the actual alteration in the vessel-walls is preceded by a stage of general increase of arterial tension, it is proper to inquire whether, under certain circumstances, it may not be possible to make the diagnosis by the change in the tension of the pulse before distinct changes have developed in the vessels.

According to v. Basch,† the determination of "angiorhigosis," or increased blood-pressure due to rigidity of the vessel-wall, is of great value, but requires more accurate demonstration than is possible with the palpating finger. I believe it is true that one obtains a more correct notion of the tension of the pulse by means of the apparatus designed by v. Basch and Riva-Rocci, although the skill in palpation displayed by experienced and clever physicians is quite wonderful. For the inexperienced the use of instruments for determining the blood-pressure is undoubtedly to be recommended, and even the expert may use them without disadvantage. [See p. 470, *et seq.*—Ed.]

If nephritis can be excluded, increased arterial pressure is a fairly good indication of beginning vascular disease and is, therefore, extremely useful. It is to be remembered, however, that arteriosclerosis is by no means always associated with rise in the blood-pressure.

* Curschmann, "Congress für innere Medicin," 1891, p. 275.
† v. Basch, "Wiener medicinische Presse," 1893 and 1896.

The observer bases his judgment on the condition of the vessels accessible to sight and touch, and in doing so it is absolutely necessary to make a careful and systematic examination of all the accessible arteries, that is, the temporals, carotids, subclavians, radials, ulnars, and femorals. The large vessels of the neck, and especially the subclavians, are not infrequently dilated and pulsate unequally on the two sides. Sometimes the dilated innominate artery or even the aorta can be felt in the suprasternal notch. It is advisable to subject all the upper regions of the chest to careful percussion and palpation, as by this means dilatation of the ascending aorta is oftener demonstrated than is generally believed.

It must be emphasized again and again that the inference of arteriosclerosis of the important vessels of the internal organs from the existence of sclerosis of the external arteries is altogether indirect, and should be made only with a certain reservation. The age also is but an untrustworthy criterion of the presence or absence of arteriosclerosis. On the other hand, sclerosis of the ascending aorta, if present, is of distinct and direct value; for the only condition for which it could be mistaken is aneurysm, and for our purposes such a mistake would not be serious, since aneurysm also, in the majority of cases, is primarily due to arteriosclerosis. Some information may also be obtained from an x-ray examination.

Insufficiency of the aortic valves, when it occurs in the second half of life and there has not been an antecedent polyarthritis, is a most valuable sign of arteriosclerosis.

It has been stated repeatedly that arteriosclerotic symptoms in many different organs may be associated with sclerosis of the coronary vessels. They need not be enumerated separately, because that would involve a complete discussion of the entire clinical aspect of arteriosclerosis. Their diagnostic significance should, however, be mentioned. When the patient exhibits, in addition to heart symptoms that cannot be readily explained, phenomena referable to any other portions of the body which are undoubtedly caused by vascular changes, this, of course, has an important bearing on the diagnosis. If necessary, a search for such symptoms must be made, for instance, in the eye, and this is another example of the value of the ophthalmoscope as an aid to the recognition of internal disease.*

Before an existing hypertrophy of the heart can be attributed to arteriosclerosis, it is first necessary to prove the existence, or, at least, the possibility of the existence, of arteriosclerosis, and, on the other hand, to exclude all other factors capable of increasing the heart action. It is most difficult in these cases to determine the exact condition of the kidneys, and after what has been said, this will readily be understood. In not a few cases the renal disease appears to form part of the general arteriosclerotic process; but it is so mild that it is apt to be overlooked, and yet the patient may at any time be most disagreeably surprised by uremic symptoms. The condition of the urine has already been described. If it presents anything abnormal and the hypertrophy of the heart is pronounced, one should always think of the possibility of kidney disease.

The same considerations must decide the question whether a dilatation of the heart is due to arteriosclerosis. Here, again, the etiology supplies the most important data. The differential diagnosis from alcoholic heart disease and the form due to obesity in many cases can only be made with a certain degree of probability, even when all the attending circumstances

*Compare Raehlmann, "Zeitschrift für klinische Medicin," vol. xvi, p. 606.

are taken into consideration; for the difficulties encountered in the differential diagnosis are very great. Many alcoholics also have arteriosclerosis; many arteriosclerotics drink to excess, and many of them, finally, suffer from obesity.

The differential diagnosis from chronic myocarditis, which is enormously difficult and often practically impossible, has been in part discussed in another section (p. 662). Here it may be well to remind the reader once more that chronic myocarditis and the effects of coronary sclerosis resemble one another very closely, and hence there must necessarily be a close resemblance between the functional disturbances in the two conditions.

If there is marked cardiac insufficiency with abnormal blood distribution, lesions of the mitral valves must be considered in addition to all the other conditions that have been mentioned; for systolic murmurs may be present in arteriosclerosis owing to deficient contraction of the muscles at the base of the heart or to sclerotic changes in the valves; while, on the other hand, diastolic murmurs may be absent in endocarditic processes. The history, the age, and the general impression derived from the clinical picture must be taken into account in such cases.

Under these circumstances, also, the conditions from which it is most difficult to make the differential diagnosis are chronic myocarditis and cardiac insufficiency occurring in the course of kidney diseases. The diagnostic points have been explained partly in this section and partly in the sections devoted to these conditions.

Finally, we have the cases in which the subjective symptoms—pressure on the chest, pain, anginoid conditions, palpitation, and disturbances of the heart action—are the most prominent factors—either the two groups of phenomena combined or each one separately. In these cases a differential diagnosis must be made chiefly between disease of the heart muscle and the nervous affections. The general impression conveyed by the patient is of the greatest value for this purpose. Is the patient nervous? Does he present in addition to the cardiac symptoms other symptoms of neurasthenia? Here we at once meet with difficulties. For many heart patients become nervous, especially when grief and worry are superadded to their disease, and who can then decide what is primary and what is secondary?

Then, as to the findings in the heart itself; the absence of any objective changes in the heart or vessels would fit equally well with the diagnosis of a nervous heart affection and that of disease of the muscle or of the coronary arteries. Nevertheless, such a condition is absolutely frequent in nervous affections, while it is distinctly the exception in myocarditis and coronary disease. As a rule, some abnormality is present in coronary sclerosis, even if it is no more than a somewhat ringing second aortic sound or some other symptom mentioned in the symptomatology. The most careful examination is urgently necessary and exceedingly important.

Dilatation in itself is more in favor of some organic disease, but does not absolutely exclude the existence of a nervous affection.

The *character of the arhythmia* and inequality of the heart action, in my opinion, have only a limited value for the differential diagnosis. Nervous patients undoubtedly exhibit fluctuations in the pulse-rate extending over a considerable number of cardiac contractions. But, on the other hand, these patients also exhibit exactly the same disturbances as those described on p. 684 as occurring in coronary sclerosis as well as in the other diseases

of the heart muscle, especially myocarditis. Others may base their opinion on the character of the arhythmia, if they like. After a long experience, during which I have paid special attention to this question, I have ceased to place any dependence on this symptom alone or even to attach very much value to it. At most I am willing to admit that persistent and absolutely atypical irregularity may have some value as a symptom, for it is not observed in nervous patients to the same degree as in diseases of the heart muscle (coronary sclerosis, myocarditis).

The symmetry of the pulse must be carefully noted. Inequalities in the volume and time of occurrence of the brachial and radial pulses are quite frequent, and often occur early in sclerosis of the ascending aorta. This point has considerable diagnostic value whenever careful examination of the arteries shows that the phenomenon is not due to local changes in the vessels.

In the majority of cases the functional power of the heart during exercise is unquestionably of great diagnostic value. No certain means have as yet been devised to test the function of the heart objectively, and one's opinion has to be based chiefly on the patient's statements that he suffers from oppression, palpitation, and shortness of breath. In nervous patients these symptoms are, as a rule, not induced by exercise, although there are, of course, exceptions (see the section on Nervous Diseases). On the other hand, dyspnea is frequently, not to say regularly, found after coronary sclerosis and myocarditis. On this point all are agreed. Thus Huchard attaches a very great deal of importance to dyspnea on exertion (*dyspnée d'effort*). Nevertheless, even this symptom may be altogether absent in individual cases of this disease, perhaps more frequently than is generally supposed.

This seems to me to be borne out by a good many cases. Thus I know a man, forty-two years of age, who has had a number of what I consider genuine attacks of angina and presents a slight dilatation. Nevertheless, this man can ride a bicycle for long distances without the slightest discomfort. I might quote other cases of this kind if I had anatomic data at my disposal. A few similar cases are found in the literature. Thus, an elderly gentleman after a typical attack of genuine angina pectoris made difficult mountain excursions without developing any symptoms whatever.* This is readily conceivable on anatomic grounds. The heart in these cases is probably not the seat of a diffuse disease, and the symptoms are probably produced by a local affection, such as sclerosis of a branch of the coronary artery. If occlusion of the vessel takes place, possibly accompanied by the symptoms of violent stenocardia, the patient is permanently relieved† and may quite possibly recover all his former strength.

The case is quite clear whenever there are *nocturnal attacks of shortness of breath or pulmonary edema*. If these conditions are present, the patient, in my opinion, is certainly suffering from a grave disease of the heart muscle, even if no physical signs are present in the organ. For the occasional attacks of nervous dyspnea can be at once distinguished from cardiac asthma by the peculiarity of their behavior; and genuine pulmonary edema, finally, admits of no misinterpretation whatever. If the patient gives a statement of nocturnal attacks of dyspnea and nothing whatever is found on physical examination, one may possibly hesitate between pulmonary and cardiac asthma.

* Meyer, "Archiv für klinische Medicin," vol. xliii, p. 379.

† Compare Curschmann, "Congress für innere Medicin," 1891, p. 275.

The *character of the sensation of pain and fear* is not without distinct value in distinguishing between nervous heart symptoms and coronary sclerosis. In fact, in uncomplicated and well-developed cases it may decide the diagnosis, especially if all the cardiac symptoms are either absent or strongly in favor of nervousness; or, on the other hand, all the positive signs of arteriosclerosis are present.

But it is only in very rare cases that the diagnosis is as clear as this. Well-developed attacks of true angina are often preceded by rudimentary attacks, bearing a strong resemblance to the nervous form. If the patient has other signs of arteriosclerosis, these rudimentary attacks will not obscure the diagnosis; but in many cases the physical signs are altogether negative, or the examination yields only very uncertain results, particularly in the case of men between thirty-five and fifty. In the heart nothing at all is found; the only symptoms complained of are rudimentary attacks of stenocardia after violent exertion or emotional excitement; there is some nervousness. Is it a case of genuine or false angina? I have already given as full a discussion as possible of this subject on p. 543. In every case the greatest caution must be enjoined.

Treatment and Prognosis.—The first object of treatment, whatever line may be adopted, must be the removal of those factors which we believe to cause arteriosclerosis or to be capable of favoring its extension. First of all, the patient's mode of life must be accurately regulated. Here we can do no more than give general directions, for to go into detail would occupy too much space, and as this work is intended for physicians, details are unnecessary, and at best would only be suitable for a few cases.

The food should be just sufficient in quantity; as a rule, it is not desirable that the patient should put on fat. The reasons for both these things have already been mentioned.

Generally speaking, a mixed diet with a preference for fruit and green vegetables and with a minimum of condiments should be advised. Most physicians are agreed that the excessive ingestion of proteid food, especially meat, which is so popular in many quarters, is not suitable for patients of this class. The reasons for this are yet to be discovered, and there is still some doubt about the truth of the statement.

Eminent clinicians, as, for example, Huchard, say they have seen excellent results from an absolute milk diet. If possible, I should advise that a diet consisting chiefly of milk be tried, at least for a time. The attempt is most likely to prove successful in the case of patients who have no work to do, because they do not need a large quantity of nutritive material. But if the patient is exercising to any extent, it is very difficult to get him to take enough milk to insure the necessary quantity of nutritive material. Bread, at least, must be added to his diet-list; in fact, I would do that under any circumstances. In my opinion it does no harm whatever, the main thing being to avoid meat and condiments; and, finally, it is to be remembered that when a patient is living on an absolute milk diet he ingests in the course of a day much more fluid than is desirable.

In many cases, especially when there is angina pectoris and the attacks are brought on by dyspeptic disturbances, a strict supervision of the gastro-intestinal tract becomes necessary. The special directions to be given will depend on the condition of the alimentary canal. It is often very important to order light meals at frequent intervals because a full stomach is apt to bring on an attack. The directions to be given in regard to wine, beer, coffee, and tobacco have been discussed on p. 560. This question must

also be largely determined by the presence or absence of stenocardia. I would advise special caution with regard to tobacco, and Huchard is quite right in warning his patients against sitting in a room full of smoke.

The question whether *absolute rest*, possibly even *in bed*, is to be ordered, or the patient be permitted the usual amount of bodily activity can only be decided on general principles (see p. 554). At all events violent exercise, particularly when associated with emotional excitement, as, for instance, mountain-climbing and athletic pursuits in general, which are nowadays carried to such an absolutely morbid extent, must be forbidden altogether. Although prolonged muscular rest unquestionably does the patient no good in the early stages, the physician must, on the other hand, be extremely cautious in ordering very active exercise in arteriosclerosis, and must take into account the special features of each case. I particularly caution against any attempt to improve the strength of a weak heart by means of forced movements according to Oertel's method. I do not say that exercises for training the heart must be avoided under all circumstances, but I do recommend the greatest caution, as dilatation is very apt to develop or to increase if it is already present. When attacks of stenocardia have already occurred, the patient's activity must be restrained, and all those things which are known to produce the attacks, especially walking after meals, must be strictly forbidden.

Sexual intercourse must be restricted in this disease more than in any other; old men with coronary sclerosis not so very rarely die during coitus.

Most of these things, which I merely mention in a general way, have to be explained to the patient in detail in every individual case. If this precaution is neglected, the physician cannot count on a proper observance of his instructions.

There is no doubt whatever that *emotional excitement*, even excessive joy, but especially worry, grief, and care, are dangerous for arteriosclerotic patients. A good physician will guard against these things also as much as he can; but he must necessarily depend mainly on the patient's own efforts—the patient must learn self-restraint.

Those clinicians who incline to the view that increase of arterial pressure is a factor in the pathogenesis give their greatest attention during the early stage of the disease to the question of diminishing the blood-pressure. The desirability of being able to accomplish this will, indeed, appeal to every physician, even if he does not consider arteriosclerosis solely the effect of an increase of pressure in the arteries. For in any case an abnormally high blood-pressure is unnecessary for the demands of daily life, and is in many respects dangerous; for example, it predisposes to rupture of blood-vessels and throws more work on the left ventricle. I need only refer to cardiac asthma.

One of the first remedies to be mentioned is venesection, and the removal of from 150 to 300 c.c. of blood. The experience of the older physicians chiefly, but also of some of the moderns,* proves that the procedure may be directly useful. The profession has become somewhat "blood-shy." The reaction against venesection was quite natural, but it is exaggerated, and we ought to get over it more and more. In so-called "full-blooded" arteriosclerotics, that is, those who are not anemic, with high pressure, venesection may be confidently resorted to and even repeated if it proves useful.

If the increase in blood-pressure is the direct cause of certain symptoms

* Fraentzel, "Herzkrankheiten," vol. i, p. 100.

and discomforts, the indication for venesection is especially clear. It acts like magic in cardiac asthma with hard pulse, and should be employed at once in such cases.

Personally I cannot indorse the use of *diaphoresis* and diuretic remedies for the sole purpose of reducing the arterial pressure. It does not appear to me that the value of such treatment has ever been positively proved in practice.

On the other hand, measures calculated to produce arterial hyperemia of the intestine are distinctly to be recommended. That this is followed by a fall in the blood-pressure (splanchnic vessels!) appears *a priori* probable, and it is a fact that many arteriosclerotics feel worse *whenever the bowels do not move regularly*. This must, therefore, receive attention, and, if necessary, rhubarb, cascara sagrada, or compound licorice powder must be prescribed. In many cases it is wise to give these remedies for a long time, so as to produce two daily semifluid evacuations.

Mineral waters must be given with the greatest caution. Of this, there can be no doubt; much harm has been done by indiscriminately ordering a drinking-cure in some health resort with sodium chlorid or sodium sulphate waters. The combined effects of the drinking-cure and plenty of exercise seem to be too much for these patients and are to be deprecated. On the other hand, however, I have seen good results follow a carefully regulated drinking-cure with the above named waters in arteriosclerotic patients; for instance, when there was much obesity or constipation and abdominal pain was a prominent feature.

A drinking-cure must never be ordered unless the patient is absolutely free from cardiac insufficiency with altered blood distribution. If mineral waters are ordered at all, they must be given in small quantities—only enough to produce one or two bowel movements—and the exercise must be accurately adapted to the functional capacity of the stomach.

A regular bathing-cure at a suitable resort may also be recommended under these circumstances, provided the patient can be placed in the care of a physician who is familiar with the treatment of heart diseases.

French physicians recommend the *iodin preparations* and assert that they have a direct action on arteriosclerosis. Huchard is quite enthusiastic about them. He asserts that he has seen a diminution of the arterial pressure after only a few days' use. Vierordt* has also seen good results from the use of these drugs, and I myself have often seen similar good results, which I believe were positively due to potassium iodid.

From 1.5 to 2 gm. (24 to 30 gr.) of potassium iodid or sodium iodid are given in the course of the day, divided into two or three doses after meals in a large tumblerful of water or milk.

It appears to be immaterial whether potassium or sodium iodid is given for this purpose; at all events, there is no need of avoiding potassium iodid under the impression that it injures the circulation, as that is an illusion. On the contrary, I believe it is a mistake to substitute the sodium for the potassium salt, as seems to be the custom in many clinics nowadays, if good therapeutic results have been obtained with potassium iodid. The effect of the salt when given in the form of the potassium salt is altogether different, and anyhow we do not know how these substances ultimately produce their beneficial effect.

At any rate, the iodin preparations must be continued for a long time —more than a year. Huchard's plan of stopping the drug for a week out of each month may be adopted, or the remedy may be given steadily for

* Vierordt, "Congress für innere medicin," 1897, 277.

several weeks and then withdrawn for a considerable period. Even after it has been given for from one to two years, its administration should be continued still longer, with suitable interruptions.

In the next place nitroglycerin is recommended by a good many physicians, among whom Huchard may again be mentioned. This clinician gives a 1 per cent. alcoholic solution of nitroglycerin for from ten to fifteen days in each month, two drops twice a day, and if this is well borne, increases the dose up to two drops six times a day. [Larger daily doses may be taken, and continued longer.—Ed.] Sodium nitrite and amyl nitrite are less efficient for this purpose. The action of the latter is too evanescent, and the former is believed to be not quite free from danger. In these cases it is necessary to give the drug for a long period. [Erythrol tetranitrate is said to act slowly, but to persist longer than the above; its explosiveness is a decided objection.—Ed.] I am unable to give a personal opinion as to the value of these remedies in arteriosclerosis in general.

The various degrees of cardiac weakness must be treated according to the principles that have been laid down in the section on the general principles of the treatment of heart disease. A number of special points have to be considered in arteriosclerosis; but these need not be mentioned again, because all that is necessary has already been said elsewhere.

The management of an individual case of angina pectoris is discussed on p. 544.

The treatment of the individual organs and the disturbances to which they give rise must, of course, be attended to with all due care. If these disturbances are caused by anomalies of the circulation, they will be best corrected by suitably influencing the heart, and this must be done even when the remedies that are called for seem to be contraindicated by the conditions of certain organs. The various dangers and disadvantages must be carefully weighed against each other, and the evil must, if possible, be attacked at its root.

The *prognosis* of arteriosclerotic disease of the heart is exceedingly difficult to determine even in the individual case, and no rules of general application can be laid down, because the prognosis depends in the highest degree on the condition of the blood-vessels in all the other organs of the body; and these, as every one knows, are quite different in different individuals.

A "sensible" mode of life, rest, a careful diet, and potassium iodid are unquestionably capable of delaying the progress of the arterial process. This seems to be important for patients who are just beginning to have cardiac symptoms. But how rarely it is possible to carry out adequate directions long enough! If the patient is compelled, in spite of all his symptoms, to continue a life of activity, care, and excitement, the prognosis is certainly less favorable. There is often a tendency for the recurrence of the same symptoms, such, for example, as angina pectoris; and as every attack directly endangers life, the occurrence of genuine stenocardia always renders the prognosis extremely grave. The same thing applies to cardiac asthma. On the other hand, I have already mentioned that severe attacks of this kind may occur a few times and then disappear permanently.

Whether the prognosis is better in arteriosclerosis with high than with low blood-pressure I will not venture to say. Distinct kidney symptoms are certainly unfavorable. When actual cardiac insufficiency with altered blood distribution and passive congestion has developed, it is very

much more difficult to remove than any other disease of the muscle, especially the cardiac insufficiency due to a valvular lesion at the mitral orifice. Even if it yields to digitalis it is apt to return in a short time.

Arteriosclerosis, Arteriosclerotic Contracted Kidney.—B. L., spinster, sixty-six years of age. Previous health good, on the whole, until the last few years, during which she had several attacks of influenza. The heart is said to have been somewhat out of order for about a year. At all events, she found that walking was difficult and rapid movement brought on dyspnea.

October 14, 1897: Violent emotional excitement was followed by an attack of pulmonary edema; the patient was pale and cyanotic, extremely anxious, and covered with cold perspiration; the pulse was 132, very small and soft. The attack lasted two hours. From that time on and until her death in April, 1899, the patient's health was broken; she never recovered.

Patient is dyspneic, anxious, excited, and restless.

The heart is moderately enlarged to the right and to the left. The apex-beat is in the fifth intercostal space, one finger-breadth outside the nipple-line, of moderate height, and more resistant than normal. The first sound at the apex is impure. The second aortic and pulmonary sounds are exceedingly loud; the aortic sound is somewhat ringing. Marked sclerosis of the peripheral arteries. Pulse, 88 to 92, not quite regular, of average volume, always hard. During the attacks the pulse is always 132 to 136, and then is irregular and always small and very soft. There is never any fever, but there is bronchitis, which varies very much in intensity. During many months the right pleural cavity contained a moderately large serous exudate. Swelling of the liver; almost constant edema of the feet. Quantity of the urine, 500 to 1000 c.c.; specific gravity, 1012 to 1017. Very rarely a trace of albumin; at all events, the urine was generally free from albumin for many weeks in succession, as determined by a daily examination. A few white blood-cells.

The patient's condition varied a great deal from time to time. Most of the time she was confined to bed. There were many attacks of dyspnea accompanied by anxiety, during which the pulse would be 112 to 116, small and soft. Altogether she had ten attacks of genuine pulmonary edema lasting from three-quarters of an hour to two hours.

July, 1898: Profound uremia lasting four days; headache, vomiting, stupor, slow pulse, convulsions. For twenty-four hours she was almost pulseless. The quantity of urine was very small, and the secretion sometimes contained traces of albumin; at others it was entirely free. The recovery from the attack was astonishing. The quantity of urine immediately increased very much. Edema disappeared altogether. The patient felt comparatively well, could go out and busy herself about the house until December, 1898. After December 17th attacks of anxiety and dyspnea recurred, with soft and rapid pulse (116), but since the attack of uremia there has never been true pulmonary edema. The urine continued to be free from albumin. The condition of the heart was always as given above.

In March, 1899, uremia again gradually developed. The disease ran an insidious course and terminated fatally on the first of April, 1899. During the last week the patient was constantly stuporous, and tonic and clonic convulsions occurred almost continuously. The pulse until death was almost regular, of good volume and tension, and average size. The urine never contained albumin, not even on the day of death.

Autopsy: Generalized, marked arteriosclerosis. Slight hypertrophy of both ventricles. Coronary sclerosis, fibroid heart. Bronchitis. Congested liver. Slight passive congestion of the kidneys, which were normal in size; here and there retracted areas on the surface of the organs. Corresponding to the areas of retraction, and in other scattered portions of the cortex the glomeruli were atrophied, and these portions were the seat of round-cell infiltration or of more or less cellular connective tissue.

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OCCLUSION OF THE CORONARY ARTERIES.

Complete occlusion of the coronary arteries is usually due to thrombosis, more rarely to embolism. It is only in the worst cases that a previously healthy vessel becomes occluded. Almost always the walls of the occluded artery are already the seat either of sclerosis or of arteritis. If the accident is due to embolism, complete occlusion of the lumen, of course, occurs suddenly. Thrombosis, in the majority of cases, develops gradually, but in thrombosis also the process may be comparatively rapid. The transition from stenosis to occlusion frequently develops so rapidly that the resulting clinical phenomena are comparable to those which are produced by embolism.

The anatomic and clinical effects of gradual occlusion, especially of the smaller branches, have already been discussed in connection with coronary sclerosis and myocarditis; for these two processes always form the pathogenetic groundwork, and their symptoms are inseparably bound up with those of gradual obliteration of the arteries.

The question to be decided is: what symptoms are produced by the sudden obliteration of small or large coronary arteries? The emboli, if any there be, are derived from the lungs, the cavities of the heart, or the arteriosclerotic wall of the coronary arteries themselves. If thrombosis runs a rapid course, it is almost always due to disease of the vessel-wall.

In the first place, it is positively known that in man more or less complete occlusion of at least a portion of a branch of the first order, if not of an entire coronary artery,—for example, the ramus descendens anterior of the left coronary vessel,—is perfectly compatible with continuance of life. This we know from the fact that large infarcts of the heart, often of old standing, in a state of calcification and corresponding to the distribution of one of the above-mentioned vessels, are not infrequently seen at the autopsy table.*

It is possible that the accident sometimes happens without any symptoms that the patient is aware of; he lives on and never suspects what an abyss he has escaped.

But in other cases it is possible, at least, to surmise, with a fair degree

*See, *e. g.*, Birch-Hirschfeld, "Lehrbuch der pathologischen Anatomie," fourth edition, vol. ii, p. 110. Also Sternberg, "Ueber Erkrankungen des Herzmuskels im Anschluss an Störungen des Coronararterien-Kreislaufes," Dissertation, Marburg, 1887.

of probability, that an attack of severe stenocardia or a temporary attack of cardiac failure has had something to do with the occlusion of the vessel. The heart failure in these cases is, as a rule, associated with marked subjective symptoms, especially pressure on the chest, pain, and a sensation of fear—in short, all the symptoms described under stenocardia. The heart action is weak and almost always retarded. In other words, there is genuine weakness of the organ. Sometimes the condition is temporary and its connection with obstruction of a coronary artery is not recognized until later, when the patient dies and a large infarct of some standing is found. The attack may, however, end fatally after a variable length of time, or even at once, or after a few hours.

Sometimes the final catastrophe is preceded for a period of minutes or even hours by symptoms which, at first, do not specially attract the attention of the patient or those around him, but which, nevertheless, in retrospection reveal themselves as the beginning of the end,—since death is not infrequently due to rapidly progressing thrombosis of the coronary arteries,—a certain feeling of anxiety, pain in the chest, lassitude, and vague discomfort.

In another class of patients the softening of the portion of the muscle from which the blood-supply has been cut off leads to laceration of the heart-wall and the whole symptom-complex of rupture of the heart.

Finally, some patients fall dead at the instant when occlusion of the vessel takes place, either without presenting any special symptoms or immediately after the beginning of an attack of angina pectoris. Stenocardia and coronary embolism are the most frequent causes of sudden death in persons in the second half of life.

Why the clinical symptoms of occlusion of the coronary vessel are so different in different cases is not, in my opinion, as yet susceptible of explanation. I do not believe that the contractions of the heart cease because part of the organ no longer contracts. My reason for not believing this is that large portions of the heart-wall sometimes suffer structural as well as functional death without the activity of the organ being completely abolished, and, on the other hand, sudden death may occur solely as the result of stenosis at the orifice of a coronary vessel, without complete occlusion.

The heart is extremely sensitive to many different kinds of influences. Any one who has performed experiments on the circulation of animals, especially dogs, must be fully aware of this fact. Fatal fluttering of the heart, or *delirium cordis*, often follows the slightest manipulation, such as merely grasping the heart or exerting very moderate traction on the viscus, manipulations which, at other times, are not followed by untoward results. Physiologists are still unable to say what is the ultimate reason for this curious phenomenon; it can be produced by ligation of the coronary arteries.* On the other hand, the ligation is often tolerated, provided the ligated vessel is not a main artery,† as is complete anemia of the entire heart lasting many minutes and more,‡ and fluttering is much more often observed when nothing whatever has been done to the coronary vessels. I myself have seen this again and again after many different interventions, and very often when I

* v. Bezold, "Untersuchungen aus dem physiologischen Laboratorium zu Würzburg," vol. ii, p. 256. Cohnheim and v. Schulthess-Rechberg, "Virchow's Arch.," vol. lxxxv, p. 503. Samuelson, "Zeitschrift für klinische Medicin," vol. ii, p. 12. Michaelis, "Zeitschrift für klinische Medicin," vol. xxiv, p. 270.

† Porter, "Pflüger's Archiv," vol. lv, p. 366, and "Journal of Physiology," vol. xv, p. 122.

‡ Panum, "Virchow's Archiv," vol. xxv, p. 312. Tigerstedt, "Skandinavisches Archiv für Physiologie," vol. v, p. 71. Compare *ibid.*, "Lehrbuch der Physiologie des Kreislaufes," p. 190.

was not conscious of having done anything special to the heart Hence in my opinion there is some intermediate link of which we are still ignorant* between occlusion of a coronary artery and the occurrence of heart failure. This unknown connecting event sometimes follows occlusion of the coronary arteries, and at others does not. It occurs more readily in some species than in others, and also in some individuals of the same species than in others; for example, the dog's heart is distinctly more sensitive than that of the rabbit. The reason for this is not known.

Nevertheless, anemia of large portions of the heart muscle is anything but an indifferent condition. Although the human subject sometimes tolerates occlusion of large branches of the coronary arteries, the reason probably is that the occlusion always takes place in arteries that are already diseased, that is, in organs that are already accustomed to certain insults and have in part lost their sensibility. Intact coronary vessels and hearts appear to be distinctly more sensitive, and usually lose their functional power at once after occlusion of one of the larger branches.† This is quite in accord with the observations discussed in connection with fatal stenocardia. In such cases it is quite common to find stenosis at the orifice of the coronary arteries, which are more or less unchanged. I have frequently seen this myself, and sometimes the condition can even be diagnosticated during life.

In these cases of sudden death occurring after occlusion and narrowing of the coronary arteries (in angina pectoris) some special change unquestionably occurs which ought to interest the physiologist quite as much as the clinician. When the heart action ceases from some other cause, the circulation is suddenly extinguished and the well-known symptoms of acute asphyxia are observed. The frightful convulsions of respiratory and skeletal muscles can never be forgotten by one who has once seen them. In occlusion of a coronary artery the interruption of the circulation is also quite abrupt—more sudden, in fact, than under any other circumstances, and yet there is no trace of these terrific symptoms. The patients die without changing countenance, so to speak, especially those who die in an attack of angina; they maintain after death the very same attitude—often a very curious one—that they had last assumed during life. It appears, therefore, that the heart must exert some very special and, from a physiologic standpoint, mysterious influence on the nervous system. This again, in my opinion, points to some peculiar process in the heart in this condition.

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A FEW REMARKS ON THE DISEASES OF THE HEART OCCURRING AFTER RENAL AFFECTIONS.

Even the older physicians believed that a close connection probably existed between the function of the heart and that of the kidneys, but it was not until the nineteenth century that the many pathologic relations which actually exist between these two organs began to be more accurately known. In fact, every relation that is conceivable on theoretic grounds

* Compare v. Frey, "Zeitschrift für klinische Medicin," vol. xxv, p. 158.
† Oesterreich, "Deutsche medicinische Wochenschrift," 1898, No. 10, and Barth, *ibid.*, No. 17, p. 269.

actually occurs. The changes in the renal function caused by diseases of the heart are discussed on p. 524; a simultaneous disturbance of both organs is found chiefly in arteriosclerosis (see p. 675). The latter disease is, in fact, capable of injuring the heart as well as the kidneys in a great variety of ways.

We here have to deal with a large group of abnormal cardiac conditions which are dependent on changes in the kidneys. We cannot devote more than a few words to the subject, because most of it cannot be discussed without going into the details of renal pathology, and with the latter we have to do only in so far as it has any bearing on actual diseases of the heart.

In a great many renal diseases the labor of the heart is increased, chiefly that of the left ventricle, but also that of the right. In many forms of nephritis the pulse becomes hard and the second sound at the base accentuated. If the disease is protracted, *hypertrophy of the heart muscle* develops. It is true that the various forms of nephritis by no means produce increase of the work of the heart and hypertrophy to the same degree, and this may possibly have an important bearing on the theoretic explanation of the symptoms. We cannot go into this question at this point, nor shall we discuss the reasons for the occurrence of increased arterial tension and hypertrophy of the heart.*

So long as the hypertrophied heart performs its work, the condition rarely presents any diagnostic difficulties. For, owing to the renal disease, there is, in the majority of cases, some albuminuria, which, however, is absent when there is disease of the heart with insufficiency of the muscle. All the other signs of nephritis, such as edema, anemia, albuminuric retinitis, are also present. It may be difficult to distinguish between cardiac hypertrophy accompanying genuine contracted kidney and the cardiac hypertrophy produced by arteriosclerosis. The physician should be extremely cautious in giving an opinion after the first examination in cases of this kind. For albumin may be absent at times in granular atrophy, and, on the other hand, it may be present in arteriosclerosis during periods when the heart is able to do its work—because the changes in the blood-vessels lead to peculiar morbid processes in the kidney (arteriosclerotic contracted kidney, see p. 675).

Careful observation, however, will in many cases, at least, permit a differential diagnosis. In arteriosclerosis the hypertrophy is usually less marked and generally affects the left side of the heart only; also it is, as a rule, associated with dilatation, and the second sounds at the base frequently have a ringing quality. In contracted kidney we have marked hypertrophy of both ventricles, an increase in the quantity of urine, usually with some albumin, and possibly the changes in the eye-ground. The character of the pulse and the condition of the blood-vessels must be utilized with extreme caution in making the diagnosis. There is no doubt that in many cases of genuine contracted kidney, especially in younger individuals, the pulse is hard, although the vessel-wall presents no distinct changes. It is quite true that the changes in the vessel-wall, the hardness, and the deposition of the calcareous plates, are the most prominent features in arteriosclerosis. Nevertheless, one is very easily led into error. In the first place, genuine sclerosis sometimes develops as the result of contracted kidney. In the second place, the changes in the

* Compare Senator, Nothnagel's "Specielle Pathologie und Therapie," vol. xix. Krehl, "Pathologische Physiologie," p. 32.

peripheral arteries may be wanting in arteriosclerosis, and it must be remembered that hypertrophy of the heart is chiefly dependent upon sclerotic changes in the splanchnic vessels, while dilatation is most apt to follow sclerosis of the coronary vessels. Finally, even the experienced physician often finds it exceedingly difficult to distinguish between what is really a hard pulse (high blood-pressure) and merely a hard vessel-wall. I have been told by keen observers and experienced physicians that they find this distinction more and more troublesome the more they busy themselves with the subject.

The diagnosis is still more difficult when there is cardiac insufficiency and albuminuria at the same time. Under such conditions the questions to decide are whether the heart or the renal affection is primary, and whether the condition is a nephritis or passive congestion of the kidney. In discussing this question the diseases of the heart may be placed on one side and the changes in the kidney on the other. Of the abnormal conditions of the heart, the ones to be considered chiefly are chronic diseases of the muscle, especially coronary sclerosis and myocarditis, and perhaps also the cardiac insufficiency of beer-drinkers. For the least difficult point to decide, at least if the case can be kept under observation for some time, is whether the fundamental condition is a myocarditis associated with a valve lesion—so-called valvular heart disease. It is by no means always a simple question of differential diagnosis from arteriosclerosis; for in the conditions now under discussion there is, as a matter of fact, quite frequently sclerosis of the vessels, but it is a sclerosis accompanied by some disease of the kidney. It may often have to be decided whether, in a case of cardiac insufficiency dependent on arteriosclerosis, there is at the same time a recognizable disease of the kidneys or not.

In the foregoing pages we have, of course, frequently been compelled to discuss the difficulties of differential diagnosis and have explained at length what deductions are to be made from the urinary findings (see p. 526). The urine in the cardiac insufficiency of nephritic patients is often astonishingly low in specific gravity, although the quantity may be only moderate or even diminished; nor is the secretion dark and cloudy; on the contrary, it is pale and almost clear. Romberg has given a very lucid explanation of this condition. The pulse tension may be normal or even above normal, although the patients otherwise present all the signs of cardiac insufficiency, such as dyspnea and enlargement of the liver. If the heart were stronger in these cases, the pulse would be still harder; hence we are here dealing with exactly the same conditions of relative cardiac weakness as have already been discussed in connection with many cases of arteriosclerosis. The distribution of the edema, the behavior of the urine and of the pulse, the possible changes in the eye-ground, and the demonstration of a perceptible hypertrophy of the heart—all these things will cause the examiner to think of a nephritis; and while they may not be convincing, they are, at least, strongly in favor of such an assumption. The history also has an important bearing, as well as the occurrence of uremic symptoms, be they severe or mild. These are absolutely definite, for it is positively known that they do not occur in simple cases of renal congestion, and they should, therefore, be looked for with special care.

The course, treatment, and prognosis of these conditions, which are matters that belong to the subject of nephritis, will not be discussed here.

CHANGES IN THE HEART ACTION DUE TO DISEASES OF THE LUNGS.

The relations of the pulmonary circulation form the subject of a famous investigation by Lichtheim.* Variations in the cross-section of the pulmonary vessels, provided they do not exceed certain limits, have only a very slight influence on the quantity of blood that passes through the lungs, because narrowing or occlusion of one portion of the pulmonary circulation is neutralized by dilatation of the unaffected vessels, and because diminution of the total cross-section increases the pressure in the pulmonary artery and the force of the right ventricle, so that, although the cross-section is smaller, the same quantity of blood is able to pass through the system in a unit of time. It is only when the cross-section of the pulmonary circulation has been reduced by more than three-quarters that there begins to be a diminution in the quantity of blood passing through it.†

The velocity of the same quantity of blood passing through the lungs in a unit of time is, of course, not without influence on the circulation. If the blood moves slowly, interchange of gases is more complete; hence this is unquestionably the more favorable condition. But elimination of carbon dioxid and absorption of oxygen require a comparatively short time, and for this reason the increase in velocity which accompanies any restriction of the pulmonary circulation is very useful in compensating for the reduction of the cross-section.

It is needless to say that the cross-section of the pulmonary vessels has a very marked influence on the amount of work performed by the right ventricle. The latter increases as the pulmonary circulation is narrowed, a fact which is in entire accord with clinical experience. All those factors which tend to narrow the pulmonary vessels when the strength of the right heart is not impaired increase the pressure in the pulmonary artery, and if this increase persists for some time, produce hypertrophy of the right ventricle. This is most clearly seen in the rare cases of *sclerosis of the pulmonary artery*.‡ If the rigidity of all the vessel-walls in the entire pulmonary system is increased, it always means that the resistance to the right ventricle has been greatly augmented; and if this resistance is overcome, it follows that the muscle of the right ventricle must undergo hypertrophy.

The effect on the circulation is apparent, even in *acute pneumonia*, and, as we have seen in the section on Acute Myocarditis, is sometimes capable of injuring the circulation.

Increased action and hypertrophy of the right ventricle are also observed in *pulmonary emphysema* on account of the obliteration of blood-vessels, which is a feature of this disease. In this condition accentuation of the second pulmonic sound is an integral part of the clinical picture so long as the heart performs its work, and is, in fact, a valuable sign that compensation is preserved.

Practically the same conditions are present in all the other forms of stenosis of the pulmonary tract, such as *chronic pneumonia with contraction, kyphoscoliosis, deformities of the thorax of every kind, pleural exudates, and*

* Lichtheim, "Die Störungen des Lungenkreislaufes," Berlin, 1876.
† Krehl, "Pathologische Physiologie," p. 27.
‡ Romberg, "Archiv für klinische Medicin," vol. xlviii, p. 197.

*even pulmonary tuberculosis.** The size of the right ventricle remained longest in doubt in tuberculosis, and the supposed absence of hypertrophy was usually attributed to a diminution in the entire quantity of blood. But we now know, chiefly through the investigations carried on by Hirsch after the method of W. Müller,† that the weight of the right ventricle is rather large as compared with the actual body-weight; for it must be remembered that edema is apt to cause errors in the estimation of the body-weight.

In Germany it is possible that too little attention has been paid to the clinical behavior of the heart in pulmonary tuberculosis, while the French have busied themselves a great deal with the question.‡ The heart action may be modified in a variety of ways: it may be accelerated, retarded, irregular, or unequal. The patient is often very unpleasantly aware of these changes in the rhythm, and it is possible that they may have an important influence on the course of the pulmonary disease. There is reason for believing that the deficient heart action in tuberculosis has a very unfavorable effect on the circulation. I myself have seen instructive examples of this fact. I cannot find any satisfactory investigation of the reasons why these anomalies in the rhythm and the strength of the heart take place in tuberculosis. It would be well worth while to institute a series of observations on a large number of patients.

Pleural adhesions also cause increased action and hypertrophy of the right ventricle.§ This is explained by the fact that the blood-stream in the pulmonary circulation is, under all circumstances, very much influenced by the respiratory movements. If the latter are abolished, congestion of the pulmonary artery results, and this, of course, leads to hypertrophy of the right ventricle.

Hypertrophy of the heart is also said to follow protracted cases of *whooping-cough.*‖ The explanation in this case is distinctly more difficult. First of all, the cause must be sought in the paroxysms of cough, which are associated with forced expiration and hence an increase of pressure in the pulmonary and systemic arteries. In this way the cough might readily cause an increase in the work of the heart. Other factors are circulatory disturbances, possibly associated with the inflammation in the bronchi, and perhaps also anomalies in the elasticity of the lungs; but before indulging in speculation it would be well to wait for a few more observations.

In general the action of the right ventricle has an important influence on the pathology of numerous pulmonary diseases. So long as the ventricle contracts vigorously—accentuation of the second pulmonic sound is a positive sign that this is the case—there is no danger, because even very great narrowing in the pulmonary circulation is relatively well borne. The respiration is, of course, somewhat more impeded than in health, and these patients by no means have the same power of responding to any extra demands made upon them. But if the patient were to remember this and economize his strength, he might live in tolerable comfort and, within certain limits, might even be able to work.

* Reuter, "Ueber die Grössenverhältnisse des Herzens bei Lungentuberculose," Dissertation, Munich, 1884. Compare Krehl, "Pathologische Physiologie," p. 28.
† Hirsch, "Archiv für klinische Medicin," vol. lxviii, p. 328.
‡ See, *e. g.*, Potain, "Clinique médicale," p. 187.
§ Bäumler, "Archiv für klinische Medicin," vol. xix, p. 471.
‖ Hauser, "Berliner medicinische Gesellschaft," June 17, 1896.

As soon, however, as the strength of the right heart begins to fail, a change takes place; for then the quantity of blood passing through the lungs is diminished. Dyspnea now makes its appearance, even during rest, and is greatly increased whenever an extra demand is made on the heart, especially during muscular exertion. Dilatation of the right ventricle develops, showing that its contractions have weakened, and the accentuation of the second pulmonic sound diminishes. At the same time the flow of venous blood to the right heart is obstructed, the liver becomes enlarged; cyanosis, albuminuria, and edema are superadded. The patients present all the symptoms of weakness of the right ventricle (see p. 494).

These symptoms follow anything that acts unfavorably on the structure and function of the right heart. Thus they may be produced by anything that increases the demand on its strength. A great many patients with obstruction of the pulmonary circulation and compensatory hypertrophy of the right ventricle are tolerably comfortable so long as they keep perfectly quiet and are not required to do anything in particular. But the slightest demand on their strength, such as would make no impression on a healthy person, is capable of bringing on the most alarming heart symptoms. An acute pneumonia is a much more serious matter when the pulmonary circulation is diseased than in a healthy person. Even a relatively slight bronchitis may be serious under such circumstances.* Any infection capable of weakening the heart is to be dreaded in these cases. Inflammatory processes of the heart muscle and sclerosis of the coronary arteries are exceedingly apt to produce signs of insufficiency. And, finally, the significance of a multiplicity of injurious factors, which has been referred to so often in these pages, must be insisted upon once more in connection with these conditions.

In a great many cases failure of the right heart is not accompanied by any special events, and the myocardium fails to show any alterations even on careful postmortem examination.† It is probable that in these cases the obstruction in the pulmonary circulation exceeds the above-mentioned limits and is not equalized even by the increase in power of accommodation which accompanies the hypertrophy of the right ventricle. In such cases the disproportion between the absolute demand on the heart and its functional power would furnish a perfect explanation of the phenomena. Possibly processes similar to those which occur in fatigue are superadded; the cardiac insufficiency, in many cases of chronic nephritis, no doubt presents a complete parallel. In kidney disease also the heart sometimes gives out toward the end without being really diseased, simply because the demands made upon it exceed the limit of what it is able to do. The clinical picture in such a case also presents the well-known phenomena of profound cardiac weakness.

Of the conditions that have been mentioned, *pulmonary emphysema* is the most important; in it, circulatory disturbances play the most important part. And it is just in this disease that one not infrequently finds symptoms referable not only to the right heart, but to the left heart as well—in other words, the patients have all the symptoms of ordinary cardiac insufficiency. In my opinion, the involvement of the left heart cannot be explained by the anomaly in the lungs. It is true that Traube

* Compare Traube, "Beiträge," vol. iii, p. 354.

† Kurzak, "Dissertation," Münich, 1883. Neidert, "Dissertation," Münich, 1886. Forstmann, "Dissertation," Leipzig, 1890. (Not published.)

attempted to do so. He believes that the excess of carbon dioxid in the blood, which is said to produce spastic conditions of the small arteries, is the indirect cause of the cardiac symptoms. I cannot agree with this view. In the first place, it does not rest on a solid foundation, and, in the second place, permanent vascular spasms probably do not occur. The fact, however, that emphysema of the lungs in the end is associated very frequently with weakness of the entire heart is undeniable. In my own opinion the likeliest explanation is that arteriosclerosis and chronic myocarditis represent the intermediate factor; for emphysematous patients, as a matter of fact, very frequently have atheromatous vessels, and, above all, frequently have sclerosis of the coronary arteries.

The condition of the second pulmonic sound is, as has been said, the best evidence of the existence of hypertrophy and adequate power of the right ventricle; in addition, increased pulsation in the epigastrium has some importance. If the apex of the heart is formed by the right ventricle, the apex-beat itself may be abnormally forcible.

Dilatation of the right ventricle and right auricle is indicated by lateral enlargement of the cardiac dulness, especially to the right. But a greatly dilated right ventricle is also capable of displacing the left, and thereby causing enlargement of the cardiac dulness to the left.

It must be remembered, however, that in all these conditions percussion often yields very doubtful results because the emphysematous lungs overlie the heart, and in many cases in which the thorax is distorted it is altogether impossible to determine the relative positions of the organs. That is why weakening of the second pulmonic sound is a very valuable sign. Percussion performed while the patient bends over is often helpful, and good results have also been obtained by means of examination with the Röntgen rays.

The methods of recognizing abnormal conditions in the lungs, especially adhesion of the two layers of the pleura, will not be explained here, because it would necessitate a lengthy consideration of the pathology of pulmonary diseases. It should be remembered, however, that in any disease of the respiratory apparatus in which there is any interference with the pulmonary circulation the physician must take due account of the systemic circulation. For both diagnosis and treatment depend very much more on the state of the circulation than is generally supposed; indeed, I think the circulation is the most important factor in the question. The relations are exceedingly complicated. First of all, the functional power of the right heart must be determined, although in emphysema insufficiency of the left ventricle may also cause a good deal of disturbance through overfilling of the lungs. And, finally, the same symptoms may undoubtedly be produced by peculiar changes in the lungs themselves, the exact nature of which is as yet unknown. At least, we see marked dyspnea in emphysematous patients which we are not justified in attributing to anomalies of the circulation.

Treatment.—In all diseases of the lungs it is most important to preserve the strength of the heart, especially in diseases with a tendency to exert an unfavorable influence on the pulmonary circulation. The patient must not be allowed to expose himself to any overexertion of the right ventricle in the course of his work.

If circulatory disturbances have already developed,—indeed, I am inclined to say if the physician merely gets the impression that the heart is not doing its work properly,—the regular treatment for cardiac

insufficiency, particularly the administration of digitalis, should be instituted at once. In plethoric individuals engorgement of the right heart may at first be very successfully combated by means of venesection and the withdrawal of a large quantity of blood; wonderful results sometimes follow this procedure.

The effect of the procedure will show whether the heart muscle is capable of responding to treatment; if it is not, the next point to determine is what would be done with the patient supposing he were suffering only from cardiac weakness quite independently of his pulmonary trouble. This includes the consideration of such questions as the body-weight and the control of obesity, from which such patients quite often suffer. Training the heart by means of gymnastic exercises, hill-climbing, carbon dioxid baths, half-baths, douches, and cold rubs is sometimes useful, but in every case the condition of the lungs must be taken into consideration. Patience and the combined effect of various remedies sometimes bring success when ordinary treatment has proved fruitless.

Prognosis.—It is, of course, impossible to determine the prognosis as regards the circulatory disturbance alone; for it depends chiefly on the primary condition, which is the pulmonary disease.

So far as the circulation is concerned, the prognosis depends chiefly on the cause of the heart weakness; and as it is usually impossible to say what that cause is, the physician should be very cautious at first. For if the resistance in the lungs increases and the right heart has reached the limit of its capacity, the prospect is extremely bad. On the other hand, I have often seen very good results follow suitable treatment in all sorts of intercurrent diseases affecting the heart.

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A FEW REMARKS ON THE CONDITION OF THE HEART MUSCLE AFTER MEDIASTINOPERICARDITIS.

It is no part of our task to describe all the sequelæ of pericarditic processes, but a few words must be devoted to one aspect of the question because there has been a great deal of discussion whether and to what extent weakness of the heart muscle may occur as the direct consequence of diseases of the pericardium.

The question is an exceedingly difficult one, and it is as yet impossible to give a definite answer, chiefly because the observations in the literature have not been reported with sufficient detail and because, owing to the great infrequency of the process, a single observer's experience is not enough to make amends for the lack of cases in the literature.

We know of a number of clinical histories telling how persons have died with the symptoms of cardiac insufficiency and how, at the autopsy, the most conspicuous finding was pericardial adhesion. What effect have these adhesions on the function of the heart? Have they any at all?

The first difficulty encountered in attempting to give a clear answer

is that the causes of pericarditis frequently attack both the heart muscle and the endocardium (see Carditis, etc., p. 629). Hence, if muscular weakness develops after rheumatic pericarditis, the obvious explanation, even if at the same time the two layers of the pericardium are found to be adherent, is that the weakness was due to a valve lesion or to myocarditis. In addition, Virchow* has made us acquainted with the degenerations in the outer layers of fibers of the heart muscle that develop after pericarditis; hence one must first inquire whether, in an individual case, one of the above-mentioned factors may not have been the cause of a functional disturbance of the heart associated with pericardial adhesion. In the cases contained in the literature this question is not sufficiently considered; indeed, in many cases the whole symptom-complex can quite readily be explained by a simple valve lesion. If it is to be shown that a pericardial adhesion has affected the function of the heart, it is necessary, in an individual case, to exclude any factor that is known to be fully capable of explaining hypertrophy or weakness of the organ, such as endocarditis or myocarditis. Further observation is required to settle this point.

What views have been formed in regard to a possible direct influence of pericardial adhesions on the function of the heart, and what do the above investigations teach us? There is no doubt that simple adhesion between the two layers of the pericardium may take place without causing any disturbance whatever of the cardiac function. This has been proved by numerous cases and coincides perfectly with our theoretic views. Hence it may be said with absolute certainty that adhesions limited to the pericardium as such do not cause or explain disturbances of the cardiac function of any kind.

But inflammation of the pericardium is sometimes associated with processes outside of the sac—in the mediastinum, in the front, to one side, or behind the heart; and these processes are attended by the formation of abundant connective tissue, which becomes hard and contracts and may even become calcified. The causes and clinical course of this so-called mediastinopericarditis, for obvious reasons, need not be discussed here. We have to deal only with its effects on the function of the heart, and these effects may be quite different from the consequences of simple obliteration of the pericardium. From the standpoint of the physiologist it may be said that when the two layers of the pericardium are adherent to one another and to the vertebral column, on the one hand, or to the sternum, on the other hand, the condition may readily influence the action of the heart. For the condition described unquestionably causes some resistance to the systolic contraction of the heart, and the heart muscle reacts to this resistance in one of two ways: either it overcomes the resistance and accomplishes its change of shape (contraction) in spite of it, and in that case does more work and undergoes hypertrophy if the condition lasts any time; or the resistance is too great for contraction to take place, the muscle becomes insufficient, and finally atrophic. Hence from a theoretic standpoint both these effects might be expected to take place.

There is considerable diversity of opinion as to what actually takes place most frequently, and the point has been disputed with more passion than scientific accuracy. So experienced and cool headed an authority as Stokes† asserts that adhesion of the pericardium does not necessarily cause any marked change in the condition of the heart; but if, in associa-

* "Virchow's Archiv," vol. xiii, p. 266.

† "Diseases of the Heart," German translation, p. 11.

tion with the adhesion, some change in the heart muscle takes place, the change is not necessarily hypertrophy, but may be atrophy.

In the literature, which is quite abundant, both conditions are mentioned—cardiac hypertrophy as well as cardiac atrophy after pericardial adhesions. Either condition is conceivable, but it will not do to declare dogmatically that it must be thus or so; for the character of the phenomena depends entirely on the conditions present in the individual case. The questions to be answered in the individual case are whether these conditions are capable of interfering with the contraction of the heart and whether the muscle is capable of responding to any additional demands made upon it or not. But it is by no means easy to give a definite opinion on these points. In discussing them the position of the heart and the character of the external adhesion, as well as the condition of the myocardium and of the valves must be taken into account. Failure to make allowance for these things partly explains the great diversity of views and the bitter partizanship displayed in their defense.

The heart, as has been explained, may not be affected at all; in fact, this is the rule in simple obliteration of the pericardium, and is readily understood. Before it can be said that mediastinopericarditis causes hypertrophy of the heart, it is necessary to exclude valve lesions and changes in the arteries and in the kidneys; and before the same condition can be assumed to cause insufficiency and atrophy, myocarditis must be excluded. Hence new cases must be collected regarding this point. And then, what will be left?

If, however, for the present, the existence of mediastinopericardial inflammation and adhesion are to be regarded as possible causes of insufficiency of the heart muscle, we must inquire whether this form of cardiac insufficiency is clinically distinguishable from other forms.

We have to deal only with the diagnosis of mediastinopericardial adhesions, for simple obliteration of the pericardial sac cannot be recognized clinically. Nor can the former condition always be recognized with certainty, a point that has been explained at length in the section on Pericarditis. Here I shall merely mention that, aside from the patient's statements, the most valuable diagnostic signs are extensive systolic retraction of large portions of the ribs and of the sternum. The mere systolic retraction of a single intercostal space is not conclusive, for it occurs in a variety of other conditions, and, above all, it may be due to some unknown cause.

It is true, on the other hand, that cases of intrapericardial and extrapericardial adhesions have been known without any systolic retractions whatever during life. But after analyzing the reports of these cases I find, as before, that the observers have neglected to investigate whether there were adhesion of the two layers of the pericardium and fixation of the heart to the vertebral column as well as to the chest-wall. For extensive systolic retraction is, in all probability, due to the inability of a strongly contracted heart to attain its systolic minimum (size) without forcing some portion of the chest-wall to which it is adherent to yield to its traction. The symptom, therefore, presupposes vigorous systoles and a definite form of adhesion, conditions that are probably not realized in every case. I am, therefore, inclined to think that extensive systolic retraction of the ribs or sternum points with great probability to the existence of mediastinopericardial adhesions characterized by a certain definite arrangement. This assumption would be strengthened if, at the same time, it were found

that the edges of the lungs as they expand to cover the heart during respiration exhibited impaired mobility. All the other signs are extremely uncertain; even *pulsus paradoxus* occurs in other conditions, as, for example, in simple exudative pericarditis.

It is also said that dropsical effusions developing as the result of pericardial adhesion exhibit certain peculiarities in their course. Thus it is stated especially that the ascites greatly exceeds the anasarca—more so than in other conditions. It was shown, on p. 521, that hepatic congestion may be accompanied by such marked contraction of connective tissue as to cause an effusion into the peritoneal cavity as in Laennec's cirrhosis of the liver. Judging from the statements found in the literature, this phenomenon might be regarded as particularly frequent in cardiac insufficiency due to mediastinopericarditis. But I do not think that as yet we have a right to say so; for the peculiar changes in the liver which occur in association with the pericardial obliteration are not infrequently accompanied by disease of the pleura and of the peritoneum.

Space forbids further discussion of these interesting matters, but the question can by no means be regarded as settled. The character of the peritoneal fluid, whether transudate or exudate, has, in my opinion, not been sufficiently considered; in many cases which are interpreted as cases of ascites due to congestion the specific gravity, as well as the percentage of albumin in the ascitic fluid, seems to me to point directly to an inflammatory origin. As it is stated that the peritoneum was thickened at the same time, the thought of coincident disease of the serous membranes naturally suggests itself. Hence one must be very cautious in basing one's opinion that the dropsy is due to a pericardial adhesion on the distribution of the dropsical effusions.

It follows, from what has been said, that our knowledge of "anomalies of cardiac function produced by mediastinopericarditis" is in a very bad way indeed. We have no positive views whatever to offer, because the statements contained in the literature are too incomplete to admit us to do so. The first thing to do is to collect a good series of cases.

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SUDDEN RUPTURE OF THE HEART MUSCLE.

Rupture of the heart muscle may occur after traumatism, which will be discussed in a separate section. The accident occurs without any external injury only when marked increase in the intracardial pressure is superadded to profound structural changes. That is, part of the musculature must still retain sufficient strength to cause a marked rise of the blood-pressure, and, on the other hand, some grave lesion must have greatly diminished the resistance of a circumscribed portion of the heart-wall. The increase in pressure is often brought about by some special factor, such as violent bodily exertion, for example, although the ordinary systolic contractions may also suffice to bring it about; the essential thing is the disproportion between blood-pressure and resisting power of the heart-wall. The less resistant the latter, the smaller the degree of violence necessary to produce the effect. Nothing is to be gained in a clinical description by enumerating all the causes that may produce increase of intracardial pressure and which, as appears from a study of the extensive literature, have actually produced rupture of the heart. It is an interesting fact, that in one case in which the heart muscle was friable, rupture was brought about by the action of digitalis.*

Rupture takes place most frequently through a so-called aneurysm of the heart in myocarditis, coronary sclerosis, or embolic occlusion of the coronary arteries. The rupture often takes place at the apex; although tears of the heart have been observed in a number of other places, especially in the external wall of the left, and much more rarely of the right ventricle. The pericardium rapidly fills with blood and death ensues from suffocation on account of compression of the venæ cavæ, as Cohnheim proved.† Death often occurs so suddenly that there is no time for any symptoms to develop except those of the most acute anemia and asphyxia; occasionally the patient has time to utter a loud cry or complains of frightful pain.

In other cases rupture of the heart takes place by degrees, and the blood escapes slowly. Sometimes the escape of the blood is delayed by temporary occlusion of the opening with blood-clots or because the blood slowly burrows its way, so to speak, through a diseased heart-wall. This is much more common than is generally believed. The entire process lasts hours and sometimes even days, and the patient dies with the symptoms of slowly progressing hemopericardium; pallor, cyanosis, and shortness of breath gradually develop. The heart action becomes more and more feeble, and the organ is finally arrested in complete asystole. As the change in the heart wall is so frequently due to coronary sclerosis, the clinical picture is often complicated by the symptoms of profound stenocardia. A peculiar feature, and one that is of some diagnostic importance, is that many patients complain chiefly of pain in the abdomen and, as they

* Kroll, "Dissertation," Greifswald, 1895.
† Cohnheim, "Allgemeine Pathologie," second edition, vol. i, p. 29.

often vomit at the same time, ignorance of this peculiarity might easily beget an error in diagnosis.

The above mentioned symptoms are sometimes exhibited by patients in whom the diagnosis of disease of the heart muscle has already been made. In other cases they apparently occur like a bolt from the blue. But on more careful interrogation it is not infrequently found that the accident has been preceded by certain prodromata: a short attack of syncope, an attack of anxiety or pain in the region of the heart, or true stenocardia. In such a case the accident must be attributed to occlusion of a coronary artery or the formation of a large cardiac infarct.

Rupture of the heart is a very rare occurrence, being observed only once in several thousand autopsies. It being the fashion to regard rare cases of disease as "interesting" and to report them, the literature of heart rupture has become quite extensive, as cases have been collected ever since the time when the practice of examining cadavers first began.* I have studied a large number of these cases, but I shall refrain from quoting any of them here. The literature shows that a great variety of different pathologic processes may lead to rupture of the heart, provided the above-mentioned conditions are fulfilled; and, further, that rupture may take place in almost any portion of the heart. Tables showing the relative frequency in the different regions have repeatedly been made, but, of course, the seat of rupture depends on the seat of the pathologic process and certain peculiarities of the contraction of the heart. It does not seem to me that these tables have any place in a clinical discussion.

Diagnosis.—The diagnosis is based on the well-known symptoms of internal hemorrhage and a sudden interference with the entrance of venous blood into the thorax and into the heart. The probability of rupture having taken place is reinforced if the case is such as to render a friable condition of the heart muscle possible, and there is a history of some violent exertion associated with great increase of the blood-pressure. But, as a rule, the physician is unable to determine whether the heart itself or a large vessel in the pericardial cavity has ruptured.

Rupture into the interior of the heart will not be discussed here. These ruptures affect either the valves—more frequently the aortic than the mitral, and the clinical symptoms are described in the section on valvular disease in this volume. Or, especially after rupture of the septum, a communication may be produced between various cavities of the heart. The result of such an accident may be inferred from the description of congenital defects in the septum (H. Vierordt) in the original of this work.†

Finally, a diseased papillary muscle has been known to be torn from its attachment in a few instances, as in the famous courier reported by Corvisart.‡ I am not prepared to admit that the consequences of this accident are altogether clear; for Hammernjk's theory in regard to the function of the papillary muscles, which form the foundation of the views prevailing at the present time, is by no means tenable. One is justified in saying, however, that, so far as the effect on function is concerned, the tearing off of pieces of papillary muscles is equivalent to the laceration of tendon-fibers, as mentioned in the section on valvular disease.

*See G. Meyer's excellent paper, written under Bollinger's direction, "Archiv für klinische Medicin," vol. lxiii, p. 379.

†Nothnagel's "Specielle Pathologie und Therapie," vol. xv, pt. 2.

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SO-CALLED DEVELOPMENTAL HYPERTROPHY OF THE HEART.

Germain Sée made the assertion* that in children and young persons about the time of puberty hypertrophy as well as dilatation of the cavities of the heart occurs with special frequency, and that there is a connection between these diseases and the peculiar mode of development of the infantile heart, which, according to Beneke,† has periods of sudden growth, as well as with the peculiar sensitiveness of the infantile heart to the effects of exercise.

In Germany this theory of developmental hypertrophy never attracted much attention, while in France a lively discussion sprang up about the question, and sides were taken for and against Sée's views.‡

W. Müller's observations § have given us a more accurate knowledge of the growth of the heart in the child and have shown that the growth is essentially proportional to the mass of the body, and that like the latter, the heart increases rapidly in size during puberty. Hence it is quite conceivable that there should be a certain disproportion between the development of the body-mass (or rather, more correctly, the muscles) and the size of the heart, presenting to the examining physician the picture of an anomaly and possibly causing actual discomfort to the patient.

I had the opportunity in the Jena as well as in the Marburg Policlinic to see a large number of children and young people, for I was able to make minute examination of the 200 or more apprentices and young workmen in the optical works of C. Zeiss in Jena twice a year,

*Germain Sée, "Traité des maladies du cœur," Paris, 1889, p. 434. Romme, "Gaz. hebdom.," 1896, No. 34.

†Beneke, "Centralblatt für die medicinischen Wissenschaften," 1879, p. 358.

‡See *e. g.* Romme, "Gaz. des hôp.," 1896, No. 34. Contains the literature. Fiessinger, "De la croissance au point de vue morbide," "Mém. couronné par l'académie de médecine," Paris, 1889.

§W. Müller, "Die Massenverhältnisse des menschlichen Herzens," Leipzig, 1883, p. 122.

and to keep them under constant observation. It is well known that this excellent institution takes very good care of its employees, and Czapski many years ago began the policy of examining new apprentices and all workmen between the ages of fourteen and eighteen twice a year in order to determine how they bear the work. The personnel is composed of individuals from a great many different classes of society, whereas the mode of life and occupation of large groups of these young men are exactly or almost exactly the same. With the exception of a few locksmiths' apprentices, none of these young men really do heavy work, and the hours are very moderate—nine hours in summer as well as in winter, and seven and three-quarter hours on school-days. I was, therefore, able to observe the heart during the period of its development in a large number of young men living under absolutely favorable conditions. In all, I examined 322 young workmen, almost all of them several times,—from three to five,—that is, I watched their physical condition for two years.

Cardiac symptoms were encountered with remarkable frequency. I entirely disregarded all pronounced diseases of the heart belonging to any one of the well-known types, which, of course, occur during childhood as well as at any other period of life. Incidentally, however, it is interesting to note that the boys with ordinary endocarditic valve lesions of moderate intensity were, for the most part, free from any symptoms of discomfort. They knew they had "heart disease," but paid no attention to the fact and were not in the least disturbed by it.

A second group of young men had absolutely normal hearts on objective examination and were free from symptoms.

In the third group of cases, which formed 22 per cent. of the whole, changes were found in the heart and certain symptoms were complained of. The cardiac changes were by no means always associated with subjective symptoms; but, on the other hand, I never observed the latter without finding some abnormality in the heart. The symptoms consisted solely of palpitation, pressure on the chest, and dyspnea on exertion; in other words, the symptoms that are so frequently found associated with a certain loss of strength. In proportion as the changes in the heart disappeared the subjective symptoms also improved.

The *objective changes in the heart* were of various kinds. Sometimes I saw enlargement of the relative heart dulness to the right, with or without accentuation of the second pulmonic sound; quite often both the absolute and the relative heart dulness were enlarged to the left. The apex impulse in such cases was displaced beyond the nipple-line, and sometimes downward to the sixth intercostal space.

The symptoms referable to the left heart were either those of dilatation only—that is to say, the apex-beat was soft, just as the arteries also felt soft to the touch—or the cardiac impulse was high and heaving, and this was not infrequently the case even when there was no enlargement of the heart. Increased resistance of the apex-beat was usually accompanied also by accentuation of the second aortic sound, and often also the pulmonic. In a few instances the second aortic even had a ringing quality, but in such cases the arteries were also found to be tortuous and the walls hard.

A heaving apex-beat and hard arterial walls were not always, although very often, found in association. I sometimes found it exceedingly difficult to determine the character of the pulse (estimate the blood-

pressure) in many young men with hard arterial walls, and I was not always able to overcome this difficulty. It did not seem to me that there was any definite relation between blood-pressure and hardness of the arterial walls. For example, I often found tense arterial walls and even tortuous arteries with accentuation of the second aortic sound when the pulse was really not abnormally hard.

Systolic murmurs at the apex or in the second intercostal space on the left side were not frequent, but were encountered occasionally, with or without accentuation of the second pulmonic sound. Phenomena referable to the left heart may be associated with symptoms of disease of the right; but more commonly one part of the heart is altered independently of any other.

The walls of the arteries accessible to the palpating finger were, as has been mentioned, very often found to be tense, and the vessels themselves in such cases were not infrequently tortuous.

The heart-beat was often quite *frequent*—rarely irregular and unequal. I never observed slowing of the heart action.

None of the young men had nephritis. Small quantities of albumin were found in the urine of some of them; but this was not more frequent in those with cardiac weakness than in others, and on careful study the albuminuria in every instance revealed the characters of accidental (cyclic) albuminuria. Granular or epithelial casts were never found.

Now all these heart symptoms were quite frequently observed in young men who, when examined six months previously, had been found to be absolutely normal, nor was it possible to demonstrate any cause for their development. Infectious diseases may be excluded, as no such diseases occurred. In order to avoid any possible error, I excluded from my statistics one patient with dilatation of the heart following articular rheumatism. I carefully inquired into the character of the work performed by the patients. For the great majority of them it was certainly not severe; the only ones who could be said to have had heavy work to do were the locksmiths' apprentices. But it did not seem to me that even their work was any harder than that of other locksmiths, besides which the heart was not affected more frequently among the locksmiths than among the other young workmen in the factory.

I was also able to exclude any unusual muscular exercises outside of their regular work, as well as imprudent drinking of beer or brandy. Masturbation was also denied.

There was not the slightest reason for suspecting any form of metallic poisoning, such as lead.

In many cases the symptoms disappeared completely in the course of six months; but in most of the young men they lasted longer, being found at two to four successive examinations—that is, they lasted from one to two years.

Even then all the symptoms may disappear, and, in fact, this is the rule. Usually the first retrograde change is the return of the heart to its normal size, while the accentuation of the second aortic sound and of the apex impulse is the last to disappear. Quite frequently these latter symptoms were still present when the young men were examined for the last time after having reached the age of eighteen years.

I would not venture to do more than hint at the cause of these curious findings, and that only for some of them; for the lack of postmortem

observations cannot be supplied from other sources for purposes of discussion.

Some cases were undoubtedly cases of simple dilatation of the right or left ventricle; for, as we have seen, the heart dulness would enlarge for a time and then become smaller again. In these cases the ordinary demands of every-day life were probably sufficient to impair the tone of the heart muscle. So far as our present knowledge goes, it is probable that these dilatations disappear with the same ease as they develop, and this view is supported by other clinical experiences; for dilatation of the heart is not infrequent in children, and its prognosis is quite favorable.

In a second group of cases the hearts of the young men were found to retain the characters of the infantile heart. It will be remembered that in young children the heart dulness is relatively larger and the apex-beat outside of the nipple-line. At the age which the young men whom I examined had reached these infantile conditions are, as a rule, no longer found. On the other hand, however, we know how very variable is the upper limit of the age of childhood in this respect. Among those whom I examined there were boys who still distinctly showed the dimensions of the infantile heart, and subsequent observations showed that the conditions usually found in adults developed very late—between the fourteenth and eighteenth year of age.

But these two assumptions will not suffice to explain a third group of cases in which there was unquestionably a special functional disturbance of individual portions of the heart. This applies, first of all, to subjects with well-marked systolic murmurs without dilatation. As there was not the slightest reason for diagnosing an endocarditis, since the murmurs disappeared again completely, the natural explanation that suggests itself is that they were due to muscular mitral insufficiency.

Accentuation of the second aortic sound and a heaving apex-beat unquestionably point to more vigorous contractions of the left ventricle, but no careful diagnostician will venture to say to what extent hypertrophy of its musculature has already developed. It is true that there is nothing to disprove it, but the more conservative assumption of an increase in the strength of the contractions suffices to explain the signs. How are these increased contractions brought about? In some of the young men marked changes were found in the arteries; the walls felt exceedingly tense. What the meaning of this phenomenon is I will not venture to say, nor do I know whether the condition in the arterial walls is the cause of the increased heart action. It might be the cause, in my opinion, if the change in the arteries had caused an increase in the blood-pressure and, as has been mentioned, I almost always gained the impression that this was not the case. I am more inclined to regard the tenseness of the arterial walls and the increased action of the left ventricle as coincident irritative symptoms, and would, therefore, conclude that, as in the adult, the heart becomes more irritable after certain insults, so in young growing individuals the demands of every-day life suffice to produce a similar state of irritability. It is well known that during this period of development many organs exhibit an increased irritability.

It is possible that in many cases a disproportion between the development of the thorax and the size of the heart is in part responsible for the symptoms; for an abnormal position of the heart might, under such

circumstances, easily simulate some abnormality of size. On the other hand, if the heart fails to keep up with the growth of the entire body, it is not difficult to understand that the organ might have difficulty in doing its work, and this would readily explain many peculiar changes in its action and size. Such an assumption is borne out by the disappearance of the symptoms at a subsequent period.

Treatment.—Young people who exhibit the above-mentioned changes in the action of the heart should be protected as much as possible against anything capable of injuring the organ. Moderation in eating and drinking, abstinence from any form of sexual activity and from smoking, as well as great caution in regard to severe active muscular exertion are necessary. These things have an important bearing on the choice of an occupation, and particularly on the question of athletics, especially bicycling. I would forbid any occupation that necessitates violent muscular exercise, and would prohibit bicycling altogether, because it is difficult for persons with an active disposition to indulge in this form of exercise with moderation. Foot-ball and the other games that are now becoming fashionable fall under the same ban. On the other hand, gymnastics (without apparatus), if carried out carefully and under supervision, I believe to be very useful for preserving the strength of the heart.

A guarded prognosis must be given because it is difficult to draw a dividing line between these affections of the heart and actual morbid changes in the muscle; indeed, in many cases it cannot be done with absolute certainty. But if the physician feels sure of his diagnosis and can succeed in enforcing a suitable régime, the outlook may be regarded as distinctly favorable.

CASE HISTORIES.

1. D. C., helper; born in 1882. Parents healthy; one brother died of heart disease. Always healthy. At the examination in 1897 and 1898 nothing was found. May, 1899: Apex-beat in the fifth intercostal space within the nipple-line, of moderate height and resistance. Absolute heart dulness normal; relative extends one finger-breadth to the right of the right border of the sternum. A systolic murmur, heard in the second intercostal space on the left side, accompanies the first sound. Second pulmonic sound accentuated. Pulse, vessels, urine negative. No symptoms.

September, 1899: Condition entirely normal.

2. E. P., mechanic; born in 1881. Comes of a healthy family. Was formerly well. In 1896 and April, 1897, nothing was found. October, 1897: The apex-beat was high and hard, one finger-breadth beyond the nipple-line in the fifth intercostal space. Absolute dulness extends almost to the nipple-line. No enlargement to the right. Sounds uniformly pure. Pulse, arteries, and urine present nothing peculiar. No symptoms.

April, 1898: The condition is the same.

December, 1898: Size of the heart unchanged, second aortic sound accentuated, not ringing. Pulse and vessels show nothing peculiar. No symptoms.

May, 1899: Running brings on dyspnea. Apex-beat is still just outside the nipple-line and of moderate height and resistance. Otherwise nothing abnormal is found.

3. E. W., mechanic; born in 1884. Comes of a healthy family. Has always been healthy. December, 1898: of weak build, apex-beat in the fourth intercostal space within the nipple-line, high and hard. Heart dulness normal. Second aortic sound accentuated and ringing. Arterial walls exceedingly hard; the pulse presents nothing peculiar. Urine negative. No symptoms.

May and September, 1899: The abnormal signs have disappeared.

4. F. K., optician; born in 1883. Comes of a healthy family and has had measles; otherwise has been healthy. December, 1897: Delicate build; heart

presents nothing peculiar. The urine contains small quantities of albumin, the excretion of which presents the character of cyclic albuminuria.

April, 1898: Nothing was found in the heart, albuminuria as described above.

December, 1898: Urine free from albumin. In the second intercostal space to the left a soft systolic murmur is heard accompanying the first sound; otherwise the heart is negative.

April, 1899: No symptoms; no albumin in the urine. Apex-beat at the normal site and extremely high and hard. Heart dulness normal; sounds impure.

September, 1899: Pallor; no symptoms. Albuminuria has returned. Apex-beat as stated; no enlargement of the heart. In the second intercostal space on the left side a systolic murmur is heard accompanying the first sound. The second pulmonic sound is not accentuated. Pulse and vessels negative.

5. G. K., optician; born in 1882. Comes of a healthy family. Has been healthy in the past. In 1896, 1897, and May, 1898, nothing abnormal was found. December, 1898: Feels quite well; heart shows nothing abnormal; peripheral arteries are very tortuous and the walls exceedingly hard. Urine contains no albumin.

April, 1899: Arteries as stated; pulse not really hard. Second aortic sound somewhat ringing. Urine contains no albumin.

September, 1899: Feels perfectly well. The arteries and the sounds of the heart are now normal. Urine free from albumin.

6. G. W., helper; born in 1883. Parents healthy; one sister died of tuberculosis, one brother of pneumonia. Patient has had measles, scarlet fever, and an attack of pleurisy in his eleventh year. December, 1898: Feels well. Apex-beat in the sixth intercostal space and in the nipple-line, broad and very heaving. No other abnormal findings.

May, 1899: Apex-beat now in the fifth intercostal space at the nipple-line, high and heaving. No other abnormal findings in the heart and vessels.

September, 1899: Feels well; precordia somewhat bulging; apex impulse in the fifth intercostal space within the nipple-line, broad, high, and hard, not quite regular and uniform. No enlargement of the heart. In the second intercostal space on the right side a low, soft systolic murmur is heard, which is not transmitted into the carotids. The pulse is exceedingly soft and small. Nothing found in the urine.

7. G. H., mechanic; born in 1884. Father suffered from a nervous disease, otherwise the family was well. Had measles when a child; was otherwise healthy.

December, 1898: Arterial walls exceedingly hard; otherwise nothing abnormal to be found.

September, 1899: Sometimes he has a stitch in his side on deep breathing. Relative cardiac dulness extends one finger-breadth to the right of the right sternal border. A soft systolic murmur is heard on both sides in the second intercostal space. The second sounds are alike. The arterial walls are somewhat hard. The pulse presents nothing special.

8. G. O., mechanic; born in 1883. Had measles as a child, but was otherwise healthy.

May, 1899: Perfectly well. Apex impulse in the fifth intercostal space inside the nipple-line, high and exceedingly hard. Second aortic sound strongly accentuated, not ringing. No other abnormal signs in the heart and vessels, especially absence of hardness of the pulse. Urine contains albumin.

September, 1899: Feels perfectly well. All the abnormal signs in the heart have disappeared. The quantity of urine is normal; it is often free from albumin, nor is the excretion of albumin typical of cyclic albuminuria. The urine never contains formed constituents.

9. H. C., optician; born in 1881. Comes of a healthy family. Scrofulous. In 1897 and 1898 the examination showed him to be poorly developed. The organs presented nothing peculiar. In the spring of 1899 he had scarlet fever.

September, 1899: Feels perfectly well. Heart is not enlarged. Cardiac impulse at the normal site, high and hard. Systolic murmur in the second intercostal space on the left side. Both sounds at the base are very loud and equal. The pulse is hard. The arterial walls are tortuous. The urine is free from albumin.

10. H. O., optician; born in 1883. Comes of a healthy family. Has always been well.

November, 1898: Feels perfectly well. Second pulmonic sound accentuated; otherwise negative.

May, 1899: No symptoms. Second pulmonic sound still accentuated. Cardiac impulse in the normal site and exceedingly high and hard; otherwise negative.

September, 1899: Perfectly well. Cardiac impulse is now immediately out-

side of the nipple-line, high and hard. No enlargement of the heart dulness. The second sound at the apex is not quite clear; at the base it is very weak. No other findings.

11. E. W., mechanic; born in 1884. Comes of a healthy family; has always been healthy. Examination in December, 1898: Poorly developed, no symptoms. Apex-beat in the fourth intercostal space within the nipple-line, high and hard. Second aortic sound accentuated and ringing; arterial walls exceedingly hard; the pulse presents nothing abnormal. Urine free from albumin.

1899: At the spring and fall examination everything was normal.

THE SENILE HEART.

There are numerous statements in regard to the condition of the heart in extreme old age; by some it is described as enlarged, by others as abnormally small. The careful observations made by W. Müller positively establish the fact that the relative weight of the heart in advanced age is a little larger than in middle age, and Müller himself argues from this finding that the demand on the heart is increased and is met by the organ. The explanation may possibly be found in the frequency of arteriosclerosis. As has been shown above, arteriosclerosis is capable of increasing the demand on the heart, and this explains why, in extreme old age, the heart does not take part in the universal atrophy to the same extent as the remaining organs.

As a matter of fact, I have reached the conclusion, both from a study of numerous papers and from my own experience, that the changes in the heart which we are in the habit of regarding as characteristic of old age are chiefly the effects of arteriosclerosis. Whether the senile changes also include the universal increase of connective tissue described by French authors I will not venture to decide; perhaps this change is due to a senile atrophy of muscular fibers and their substitution by connective tissue.

The size of the senile heart as determined by physical examination has already been discussed; the result of a physical examination depends on the actual size of the heart, as well as on the position of the edges of the lungs and of the diaphragm. The pulse in extreme old age is, as a rule, somewhat accelerated, being about 80 beats in the minute. Disturbances of the rhythm are probably due to coronary sclerosis.

It is well known that the heart in old persons is exceedingly sensitive to the effects of poisons, especially the poisons of the infectious diseases, which have a marked effect on its functional power. This peculiarity may possibly be due to endarteritic processes, and it is also possible that senile changes in the muscle-fibers are in part responsible.

LITERATURE.

[Balfour, "The Senile Heart," Macmillan, 1894.—Ed.]
Geist, "Klinik der Greisenkrankheiten," Erlangen, 1860, p. 73.
Boy-Teissier and Sesquès, "Xerose du cœur," "Revue de médecine," 1899, p. 29.
W. Müller, "Die Massenverhältnisse des menschlichen Herzens," Leipzig, 1883, p. 122.

THE SO-CALLED NERVOUS DISEASES OF THE HEART; CARDIAC NEUROSES.

The term "nervous diseases of the heart" is applied to anomalies of the cardiac function which are due to morbid nervous influence. The question

is: what parts of the nervous system are responsible for these anomalies? Many are of the opinion that it is the nervous structures with which the heart is so abundantly supplied. It is possible that future investigations will justify this view, but for the present we have no positive knowledge of any relation existing between the nervous plexuses situated in the heart and the symptoms now under discussion. For the present all that can be said is that abnormal conditions of the entire nervous system related to or identical with general nervousness and neurasthenia, less frequently hysteria, in a large number of persons produce disturbances of the heart and blood-vessels. As a rule, these patients present, in addition, other symptoms of nervousness, either of a general nature or referable to individual organs. Hence it would be impossible to give an exhaustive account of these things without a general discussion of neurasthenia and hysteria. Nevertheless, there are a number of patients in whom the heart symptoms are either quite prominent or, at least at times, are the only symptoms present. For this reason we are justified in discussing these conditions separately, and they cannot be omitted from a work on heart diseases, if only because they exhibit, from a diagnostic standpoint, many complicated relations with true diseases of the heart.

Everybody knows that the condition of the nervous system exerts a profound influence on the circulation; it has long been a matter of popular belief that psychic excitement may directly arrest the heart and thus cause the individual's death. A few cases have, indeed, been described which appear to support this assertion.* But this is decidedly rare. The disturbances of the heart action by grief, worry, or care in a sense form the transition from health to disease; they may lead to the development of permanent abnormalities.

In most cases of the condition here under discussion the heart action is affected by nervous influences, with the production of peculiar sensations. The causes are almost exclusively to be found in abnormal psychic factors. In some cases the patient is aware of the psychic abnormality; in others he is quite ignorant of it, although a careful examination of the mental state and of the entire mode of life will usually enable the physician to satisfy himself that the symptoms are due to some abnormal psychic condition. Essentially the causes are the same as those which are responsible for the production of nervousness in general. In the case of individuals who have a neuropathic tendency, either on account of their natural disposition or because of some special physical injury, particularly a long period of impaired nutrition, intoxications, or sexual abuse (onanism, *cohabitatio reservata*),—or even healthy individuals in some instances, or persons who cannot properly be accused of possessing a very low resisting power,—the psychologic factors responsible for the production of nervousness are brought about by a combination of extrinsic circumstances. The most important of the latter are great expectations followed by disappointment, a disproportion between one's expectation of what life should bring and its realization. In other words, the individual's power of resistance to the trials and annoyances of life, which no one can escape, is deficient. This deficiency of the resisting power may be brought out by the most insignificant causes,

* Bollinger, "Münchener medicinische Wochenschrift," 1888, No. 20. Pawinski, "Gaz. lekarska," 1887, No. 36, quoted from "Centralblatt für klinische Medicin," vol. ix, 1888, p. 322. Laache, "Revue de médecine," 1895, p. 726.

or may show itself only under exceptional provocation, depending on individual differences of organization and natural disposition. As Möbius points out quite correctly, there is no sharp dividing-line between congenital and acquired nervousness. This is not the place to show that this diminution of the resisting power is a most complicated product of physical and psychic predisposition and physical and psychic injury; that aspect of the question is discussed at length in the works on neurasthenia by Krafft-Ebing and hysteria by Binswanger. [Nothnagel's "Spec. Path. u. Therapie," vol. xii.—Ed.]

Of the various hypochondriac illusions which patients suffer from in regard to their condition, one in particular is to be mentioned, namely, the dread of having heart disease. This illusion may arise in a variety of ways; an accidental examination of the heart and a perfectly harmless remark let fall by a physician may be as potent in arousing this fear as a hypochondriac exaggeration of certain nervous symptoms. The medical reports in current literature and the reading of popular books also contribute their share to the evil. In a similar way an already existing mild affection of the heart becomes exaggerated in the patient's mind and thus there develops the unpleasant association of actual heart disease with secondary hypochondria.

Women are sometimes predisposed to all kinds of nervous symptoms during the period of puberty, and especially at the beginning of the menopause, and these symptoms include such as are referable to the heart. While the menstrual flow is still present but irregular, or soon after its complete disappearance, is the time when the most marked symptoms and the most pronounced phenomena are observed.

The great sensitiveness of nervous patients to influences of various kinds manifests itself in psychic as well as physical disturbances. Nervousness influences the functional activity of the organs in various ways: it may paralyze or stimulate, inhibit or irritate. Here we have to do solely with the heart. Its functions may be modified in any one of the various ways that have been suggested as possibilities in these conditions, and nervous cardiac symptoms may be variously combined with symptoms referable to various other organs, especially the blood-vessels, as well as with the symptoms of general nervousness.

Such is the state of affairs in the great majority of patients suffering from nervous diseases of the heart. These are the cases that are relatively simple; not infrequently, however, the conditions are quite different, for the cardiac symptoms presently to be described sometimes occur in cases in which there is no positive reason for assuming the existence of a neurasthenia. In such a case the local symptoms are exceedingly difficult to interpret.

Symptoms.—The most prominent by far are the subjective cardiac symptoms, and among these chiefly a peculiar sensation of discomfort and palpitation.

Palpitation may be associated with alteration of the heart action, but in these nervous cases particularly it is often observed without such alterations. It may occur quite spontaneously,—that is, without any ascertainable cause,—and then appears at quite irregular times. Very often, however, the cause is found in some psychic emotion; the more carefully one inquires into the patient's psychic condition, the more frequently will this be found to be the case. Individuals vary in the

way they are affected by different things; an occurrence which would have no effect whatever on a person in health is often sufficient to upset a patient of this kind. The physician must be prepared for the most singular discoveries, and may expect to find different conditions in every individual case. I shall not go into details here, because they have to be explained at length in the discussion of nervousness. It is absolutely characteristic of this condition that the unpleasant sensations and the palpitation are not produced by exercise, but chiefly by psychic emotion. In fact, exercise is not infrequently useful in causing their disappearance. This is a point which, in my opinion, is worth careful attention, although it is true there are exceptions.

Quite frequently palpitation, the peculiar sensations presently to be described, and disturbances of the heart action unite to produce true paroxysms. The causes of these attacks may also sometimes be found in some emotional disturbances; in other cases it is impossible to find any cause. As in all these matters, one has to be prepared for the most curious discoveries. Romberg calls attention to the fact that the attacks sometimes occur punctually at the same hour. That is quite right, and I have seen the same thing.

The degree of discomfort which the feeling of palpitation causes in different cases is variable. Some patients suffer greatly, especially when, as so frequently happens, the feeling of palpitation is associated with that of heart-fear. The latter also manifests itself in a variety of forms and varies in degree.

Sometimes it goes on to the condition of apprehension from which nervous people in general, and subjects of melancholia in particular, are apt to suffer. Such patients frequently state that the heart is the seat of the anxious sensations; but not in all cases, and frequently the patients do not mention the heart at all; the anxiety sets in in quite an indefinite way. We shall, of course, omit any further discussion at this point; but it had to be mentioned, if only to show that, from a clinical standpoint, the boundary lines in these processes are merely relative.

On the other hand, the feeling of palpitation and heart-fear presents numerous points of resemblance to some forms of angina pectoris. This has been discussed in a former section in connection with the sensations occurring in coronary sclerosis (see pp. 531 and 688). In nervous conditions, as in coronary sclerosis, any transitional state from the mildest disturbance to genuine severe stenocardia may occur; in fact, the transitional forms are quite frequent in both conditions.

Nervous angina pectoris (pseudo-angina) may closely resemble the genuine disease. As was pointed out on pp. 543 and 692, the distinction between the two forms is very often exceedingly difficult, for the clinical picture in the two conditions is very much the same. The diagnosis is particularly difficult when arteriosclerosis is associated with the nervousness. It is a consoling thought, however, that in a number of cases the picture of angina pectoris described on page 534 exhibits features in nervous individuals which are absolutely characteristic of the origin of the symptoms. These features are the duration of the attack, which often lasts for hours, and the association of purely anginoid symptoms with symptoms of so-called general nervousness (restlessness, excitement, hallucinations). This latter point particularly seems to me exceedingly important, and finally the symptoms depend very much on the patient's

psychic conditions at the time. After all, the differential diagnosis must be based largely on the symptoms of general nervousness and the presence of a cause for the occurrence of stenocardia. The causes of true angina are principally associated with a greater demand on the heart, such as muscular exercise and digestion, as well as sleep; in nervousness the causes are of a psychic nature, that is, processes which affect the psychic condition of the patient and which, as has already been mentioned, may be such as cannot possibly be foreseen and may be downright bizarre. I agree with Romberg that it is much more important to recognize these things than to depend on the clinical symptoms of the attack, as is done by Huchard, for example. In my opinion such a course would be much more likely to lead one into error.

It follows, from the fact that stenocardiac conditions have been referred to in the preceding paragraphs, that nervous individuals also suffer from *heart pain*. Indeed, heart pain is quite common, and is not always of a severe type nor associated with anxiety. The most prominent feature of the pain is a feeling of something troublesome and unpleasant; the sensation worries and annoys the patient. But cases also occur in which the intensity of the pain alone causes the patient a great deal of suffering, sometimes quite independently of a stenocardiac attack.

A great variety of *abnormal sensations in the precordial region* are even more frequent than actual pain and a feeling of great fear. These are often accompanied by painful and anxious sensations, but are also quite frequently observed by themselves. It is impossible to give a general description of their character; every patient gives a different account of them, and in doing so uses the most peculiar expressions, according to the play of his imagination. The purely hypochondriac manner in which the symptoms are exaggerated and adorned is, as a rule, quite characteristic.

Shortness of breath also occurs in patients with nervous heart diseases. As a rule, the general character of the symptoms in these cases reveals the psychic nature of the complaint; the patients may be unable to take a deep breath, or feel compelled to take abnormally deep breaths, or the power of breathing may be suddenly interrupted. All the accompanying sensations, as well as the type of the breathing itself, are described in the most curious ways. Psychic excitement has a most powerful influence in bringing on the dyspnea, while muscular exertion, on the other hand, is without effect in the great majority of cases. In other words, it is exactly the same as with the production of palpitation, and this has an important bearing on the differential diagnosis in these cases; only it must not be regarded as an invariable principle—a practice which must always be avoided in determining the value of an individual symptom. For I know several patients with all the signs of a nervous affection of the heart who positively state that they get short of breath not only after emotional excitement, but after exercise as well. These patients are undoubtedly nervous, and most of their cardiac symptoms are undoubtedly nervous in origin. It is true that in these cases I was unable to exclude with absolute certainty the association of muscular disease—in one case myocarditis; in others, arteriosclerosis; but for all that it is quite possible that dyspnea on exertion may, although very rarely, occur as the result of nervous disturbance of the heart.

Changes in the rhythm of the heart are quite common and occur in a

variety of forms. First, as to *acceleration* of the heart action. In many cases the pulse is always ten to twelve beats faster than the rate that would correspond to the age and the patient's temperament. As has been stated on a previous page (see p. 460), there are wide individual variations even in health. During excitement, especially emotional excitement, the rate is often still more increased, the frequency varying greatly according to the health of the individual patient as well as the specific cause of the rise. In many cases the pulse is normal during rest, and the great instability of the heart betrays itself only in the disproportionate rise of frequency that follows the given cause.

Finally, tachycardia may be paroxysmal and apparently spontaneous, at least, it is often impossible to find a cause. Paroxysmal tachycardia forms the subject of a separate section. It is not always possible to separate and differentiate the acceleration of the pulse-rate which occurs in nervous individuals from the milder forms of paroxysmal tachycardia.

Bradycardia directly dependent on nervousness is unquestionably much more rare in nervous individuals than acceleration of the heart action. Personally, I do not remember ever to have seen it, although I have been on the lookout for the phenomenon. Binswanger, however, describes such cases.* Either there is constant extreme retardation of the heart-beat, or the bradycardia occurs only after excitement.

On the other hand, *disturbances of the rhythm*, irregularity, and inequality of the heart-beat are practically on a par with acceleration. There is no doubt that they make more impression on the observer than changes in the frequency, simply because the frequency is subject to great variations even in health, depending on temperament and other personal peculiarities, while irregularity and inequality of the pulse occur only as a temporary condition in the healthy individual, following some profound psychic impression. On the other hand, these rhythmic disturbances are also found in genuine diseases of the heart, particularly in myocarditis and sclerosis of the coronary arteries. For this reason a careful consideration of the influence of nervousness on the rhythm is absolutely necessary.

Nervous irregularity often presents the peculiarity that the alteration in frequency extends over a large number of beats; in other words, periods of frequent cardiac contractions alternate either regularly, or much oftener irregularly, with periods during which the contractions are less frequent. This is a common occurrence, but it would be altogether wrong to regard it as a positive differential sign between disturbance of nervous origin and the arhythmia of myocarditis and coronary sclerosis which follows no rule whatever. In the first place, the same regular alteration of rhythm may occur in myocarditis and coronary sclerosis; and, furthermore, grave arhythmia is not infrequently observed in cases of simple nervousness in which there is no reason whatever to suspect muscular disease. Even dropping of a beat or the premature appearance of individual contractions † may occur in simple nervousness,—although its occurrence, as well as that of complete arhythmia, chiefly point to changes in the heart muscle. Nevertheless, the regular and, so to speak, wave-like fluctuations in rhythm described above are, to a certain extent, indicative of a nervous condition.

Irregularity in the contraction of the heart also leads to disturbances of the volume of the pulse because it is necessarily associated with var-

* Binswanger, "Die Neurasthenie," p. 212. † Compare p. 465.

iations in the duration of diastole; hence inequality of the pulse is observed as a result of irregularity, both in nervous and in muscular diseases. On the other hand, I cannot remember ever to have seen fluctuations in the size of the pulse not due to fluctuations of the rhythm and as a purely neurasthenic phenomenon, although this occurs in acute myocarditis, for example, and is a most ominous symptom.

Any one of these disturbances of rhythm may be present constantly, or at least for a long period at a time. In other cases the disturbance is paroxysmal, either occurring without any known cause or following muscular movements, the drinking of beer, wine, coffee, or tea, and especially emotional excitement. It is characteristic of nervous heart disease that these factors bring on anomalies of the pulse much more readily and much earlier in such patients than in the healthy; the neurasthenic has a peculiarly sensitive heart.

It is very difficult to determine *whether, and if so to what extent, changes in the size of the heart and in the heart-sounds rest solely on a nervous foundation*. An assertion based on subjective conviction can always be answered by the skeptic as follows: "Whenever there is an enlargement the condition has ceased to be one of mere nervous disturbance; even if the nervous element predominates in the clinical picture, the *enlargement* is, nevertheless, due solely to disease of the muscle itself." How shall such an objection be answered? How very rarely one has the opportunity to perform an autopsy on a nervous subject under conditions that make it possible to determine the changes wrought in the heart by nervousness alone! And even if it were possible to do so and nothing were found after the most painstaking examination of the heart muscle, what would he say? "In spite of the absence of changes there must have been a functional disturbance of the *muscle*." And we could not deny this possibility. Only, the functional disturbance might be regarded as the result of morbid nervous influences.

I do not believe it is possible, at the present time, to give an absolutely positive opinion on this point. But it is my personal conviction that there is much to be said in favor of the proposition that purely nervous influences are capable of affecting the size of the heart. It is, of course, necessary to differentiate between dilatation and hypertrophy. In my opinion, *dilatation unquestionably occurs on a purely nervous foundation*, even aside from the dilatations in Basedow's disease. In fact, the hypothesis that dilatation may depend on the nervous system is usually based on observations made in this disease. This may be correct enough, but I admit the possibility that in Basedow's disease the changes in the heart may be chiefly due to chemic factors. Enlargement of the heart has further been observed in cases of onanism * in association with a variety of subjective disturbances of the heart action, and in these cases the dilatation of the heart disappears and normal conditions are restored as soon as the patient gives up his bad habit. In other cases of neurasthenic heart disturbance also, in which sexual anomalies could not be regarded as the causal factors, I have seen dilatation of the heart and fluctuations in the size of the heart corresponding to the changes in the general nervous symptoms.

From a theoretic standpoint it is difficult to understand the strenuous opposition to the doctrine of changes in the size of the heart being due to nervous influences. It is well known that stimulation of the nerves

* Bachus, "Archiv für klinische Medicin," vol. liv, p. 201.

going to the heart, as, for example, the fibers of the vagus, has a distinct influence on the tone of the heart muscle. Why should emotional excitement not be capable of producing the same effect on the heart nerves? The point is, at least, very well worth noting; for, as we have already mentioned and shall be obliged to explain once more, these dilatations may take place without any marked disturbance of the contractility—in other words, they are due chiefly to a diminution of the muscle tone.

On the other hand, there seems to be very little ground for assuming that cardiac hypertrophy may be due to purely nervous causes, especially if Basedow's disease, in which cardiac hypertrophy undoubtedly occurs, is, for reasons given above, excluded from the discussion. I once thought that hypertrophy occurred and disappeared again in cases of onanism, and it seemed to me that the same thing occurred in other neurasthenic patients. But, after further observation, I am inclined to be extremely cautious in expressing this opinion. It is exceedingly difficult to make a positive diagnosis of a slight degree of cardiac hypertrophy, especially when, as is the case in these patients, accentuation of the apex impulse and of the second sounds at the base may be due to a momentary excitement of the heart.

I am, therefore, inclined to adhere to the theory that enlargement of the heart may rest on a purely nervous basis; but at the same time I must insist most urgently that such a diagnosis must never be made until all other causes that may influence the muscle, particularly the combination of nervous and muscular disease, have been excluded. How the enlargement occurs I am unable to say positively. The general causes of enlargement of the heart are discussed on p. 496. The chief factor in these cases is a reduction of the muscle tone. It has already been mentioned that there is absolute physiologic proof that such a reduction can be brought about by nervous influences. The curious cases of sudden heart failure from fright or fear demonstrate the enormous influence of the nervous system on the heart. But what we should like to know in this connection is what the exact nature of the less severe and more slowly acting nervous factors must be in order to produce enlargement of the heart. And on this point I am unable to give any opinion. Nor is it likely that we shall be able to furnish such information for the present, because the susceptibility of the heart muscle is an important factor in the problem, and we have as yet no means of estimating it. For the present these "nervous" forms of dilatation of the heart are chiefly interesting from a diagnostic standpoint. That is to say, the presence of an enlargement cannot be used as absolute proof that the symptoms are not due to nervous causes, although, of course, the greatest caution is not only advisable, but urgently demanded. We shall return to this point once more.

In a great many cases the *heart-sounds* are not altered. Sometimes impurity of the first sound is noted, and quite often a systolic murmur at the apex or at the base. These murmurs have never impressed me as having anything peculiar about them—anything that would justify the term "nervous" murmurs. In my opinion they are heard neither more nor less frequently than in other individuals who are out of health and somewhat pale. In many cases the murmurs are probably due to anomalies in the contraction of the muscle, which is unquestionably a frequent cause of systolic murmurs in general.

Not infrequently the second sounds at the base are accentuated,

and sometimes they are even somewhat ringing. This phenomenon also is directly dependent on the change, in this case the increased strength of the cardiac contractions. As a rule, these signs are only temporary and occur during excitement. This must be borne in mind, and patients of this kind must, therefore, be examined repeatedly.

The question whether passive enlargement of the heart due to nervous causes occurs may be taken up along with the other question, whether such a thing as *cardiac weakness of purely nervous origin* exists. The characteristic features of cardiac weakness have already been discussed; deficient power of contraction of the heart muscle first claims our interest. Depending on its intensity and its distribution over the various cavities of the heart, the effects of cardiac weakness, consisting of altered blood distribution, may occur during rest or only after a considerable demand has been made on the heart. I do not remember ever to have seen cardiac weakness with altered blood distribution due solely to nervous influences; just as insufficiency of the heart, rendering it unable to respond to an increased demand, very rarely, if indeed ever, rests on a purely nervous basis. In fact, it was pointed out as a characteristic of nervous patients that the majority of them, at least, are not at all affected by muscular exertion, even when it is quite marked. It must, of course, be remembered that many of these patients, owing to general lassitude, take very little exercise and cannot exert themselves at all; while certain others, for psychic reasons, like all nervous patients, are altogether unable to perform certain muscular movements; or their performance is attended by characteristic symptoms. This has nothing directly to do with the heart. It is true that some patients have complained to me of becoming short of breath when climbing a hill. But, as I have already stated, it is in these very cases that I found it impossible to exclude a combination of nervousness and disease of the muscle with any degree of certainty. I, therefore, believe it is quite possible that the heart may lose some of its power of contraction from purely nervous causes; but on this point further observations are urgently needed. The point has never been proved, and there is no doubt that the term "nervous heart weakness" is used much too freely at the present time, and that in doing so the symptoms of general loss of power are confounded with the symptoms of insufficiency of the circulation in particular.

Some physicians during the first half of the nineteenth century upheld the view that nervous heart troubles may develop into genuine heart disease. If that is really the case, the whole question as to what definite symptoms may be found in nervous affections of the heart assumes an entirely different aspect, and the diagnosis in that case would not always be simply between one condition and the other. But I cannot recollect ever to have seen the transition; and although in cases of coronary sclerosis the symptoms may appear to rest on a nervous foundation, it should be remembered that they are much more likely to be the effects of a common cause, for grief, worry, and misfortune figure as etiologic factors in both conditions, and the milder symptoms at the inception of disease of the heart muscle and of the coronary muscles bear a great resemblance to nervous symptoms. However, it is merely a question of mistaking one condition for the other in the diagnosis, although this is very unpleasant and sometimes very difficult to avoid.

All these heart symptoms are very frequently combined in a variety of ways with symptoms referable to the blood-vessels, especially the

very distressing sensation of throbbing in various arteries and fluctuations in the diameter of the vessels in all parts of the body. The latter are associated with curious local effects on the circulation, which in turn give rise to objective and subjective symptoms as curious as they are diagnostically important—for instance, a rapid change of color and the sensation of a hand or a foot having gone to sleep. Unfortunately, these things do not lie within our province, but they are of the greatest importance in the general analysis and recognition of nervous disturbances of the heart.

We have just had occasion to emphasize that this increased excitability and excitement of the heart due to nervous influences is attended by changes in the condition of the vessels in various vascular regions. As for the preservation of life it is necessary that the action of the heart and blood-vessels be graduated to a nicety, and most carefully adjusted to one another to enable the circulation to meet all the complicated demands made upon it by the various tissues, it is *a priori* probable that central nervous stimuli, provided they reach the circulatory organs at all, also stimulate the fibers of the heart and vessels.

But we have reason to believe that there is yet another connection between cardiac and vascular symptoms that rest on a nervous basis. Animal experiments have shown that stimulation of small arterial vessels causes a marked rise in the blood-pressure. In this increase of the blood-pressure contraction of the splanchnic vessels plays an important part; in fact, judging from animal experiments, it is the determining factor, and the cutaneous blood-vessels in a sense bear a reciprocal relation to the splanchnic vessels—that is to say, the former often dilate as the abdominal vessels contract. The exact interrelation between the various vascular regions in man is, of course, not definitely known. We have to resort to reasoning by analogy from animals. Possibly our knowledge may be enlarged in future by a study of the sphygmographic curve when we know more about the latter. It has been mentioned that spasms confined to individual vascular regions quite frequently occur in nervous individuals. Sometimes these spasms are more extensive and, owing to the resulting increase in the resistance, first the left, and then the right, ventricle are also affected. The patient has a chill, the skin is pale and bluish and its sensation is diminished, and he complains* of pain in the chest and a sense of anxiety. The heart is enlarged slightly in both directions; the pulse is slow (down to 50) or greatly accelerated (200), and, at the same time, small and very hard. Occasionally there is dilatation of the pupils. These conditions occur paroxysmally and last hours or days. After the attack is over, the dilatation of the heart subsides; but if the attacks are frequently repeated, the dilatation may persist. I believe it is quite correct in these cases to assume that the symptoms are due to spasm of the vessels, which is also responsible for the rise in blood-pressure and the dilatation of the heart. Whether, and if so to what extent, the splanchnic vessels must necessarily take part in producing this effect one cannot as yet venture to decide, nor do we fully understand the relations existing between the rhythm of the heart and the rise of blood-pressure. At all events, the effect on the labor of the heart of the condition of individual vascular regions in the case of man cannot be directly estimated from the results of animal

* Jacob, "Centralblatt für innere Medicin," 1895, p. 121, and "Zeitschrift für klinische Medicin," vol. xxviii, p. 297.

experiments. It is quite possible that the cutaneous vessels in man, owing to their extraordinary development, have a much greater influence on the general blood-pressure than is the case in animals.

It is obvious that the condition known as *vasomotor angina pectoris* (see p. 542) is closely related to the above-mentioned conditions; for it consists in anomalies of the heart action which may possibly be the result of a functional vascular disturbance, and for this reason must be mentioned here.

In addition to all the symptoms that have been enumerated, there are various *phenomena of general nervousness*, especially of neurasthenia and hysteria. They are not only important, but absolutely decisive, for the explanation of the findings in the heart. For the sake of completeness they ought to be described; but I do not think that is called for at this point, and, therefore, refer the reader to Binswanger's work. [Nothnagel's "Spec. Path. u. Ther."—Ed.]

The course of these *nervous disturbances of the heart* depends altogether on whether the phenomena to which they are due can be removed. In other words, if there is pronounced nervousness, hysteria, or neurasthenia, the outlook depends on the course of these diseases. In the sexual forms everything depends on whether or not the vice is suppressed. If that is done, the alterations of the heart action also disappear. Unfortunately, it is often very difficult to effect a cure in this form, and the question of treatment is by no means a simple one in the remaining forms of nervous heart diseases.

But, as a rule, the patient's anxiety is allayed and his symptoms often show marked improvement as soon as he has been assured that he is not suffering from heart disease.

The theory of cardiac symptoms occurring as the result of nervousness.—Difficult as well as important experimental investigations by prominent physiologists have taught us that the action of the heart is influenced in a variety of ways by the irritation of various nerves, and that its rhythm particularly can be altered in many different ways. Any pathologic discussion of nervous heart symptoms must be based on these observations, but in many respects physicians have been too ready to accept the reconciliation of physiologic and pathologic facts. The effect of electric irritation of the exposed pneumogastric, accelerator, or sciatic nerve in the healthy frog, rabbit, or dog has been utilized directly to explain motor disturbances in the heart, which are produced in disease by the most complicated and, in part, psychic causes. It is obvious that such methods as these cannot really enrich our knowledge. But in this instance the practice was particularly baneful because the theory apparently rested on a "firm physiologic basis." Martius has aptly termed this state of mind "*pseudophysiologism*," which is worse than no foundation at all, because it satifies curiosity and prevents further investigation.

Before we can hope to gain a true understanding of the cardiac symptoms observed in neurasthenics we ought to be properly informed in regard to the many nervous influences that regulate the action of the heart and blood-vessels in the normal individual. But at once we are met by the lack of a "human physiology." It has never been shown by which of the methods discovered by physiologists the activity of the heart is altered in the case of man. Some disturbance of these mechanisms is the essence of the disease.

For the present we must be satisfied with general remarks. In nervous persons the heart reacts with special intensity to ordinary agents; the effect of these agents is much sharper than in health, as we learn by observation of symptoms. The cause is probably to be sought in an increased irritability of the nervous structures, but we are as yet unable to say how much the central nervous system, and how much the cardiac nerves, have to do with the symptoms. In the individual case it may lead to acceleration, retardation, accentuation or weakening, irregularity, and inequality of the heart action. In view of the close relations existing between stimulation and paralysis, irritation and inhibition, these things are not difficult to understand. Whether, and if so to what extent, the above-mentioned agents alone are capable of influencing the heart has been discussed on p. 728.

Any new stimulus, if superadded to the ordinary nervous morbid stimuli, may, and of course will in the same way, lead to reactions that are abnormal both qualitatively and quantitatively. For example, according to Dehio's * observations it is quite possible that some cases of nervous alteration of the rhythm may be due to temporary central paralysis of inhibitory fibers.

There is a final question to be answered: Why are nervous patients more conscious of the action of the heart and blood-vessels than normal individuals? Binswanger and Romberg are of the opinion that nervous individuals are conscious of processes which in the healthy do not penetrate the sensory sphere. This view is undoubtedly correct; for, as we know, the threshold of sensation is at a much lower level in nervous patients. The characteristic increase of sensibility and susceptibility has just been discussed; but certain *peculiar* conditions of excitement also occur, and these, owing to the greater sensitiveness, lead to intensified sensations. It is quite possible that the occurrence of many peculiar sensations is closely associated with qualitative changes in the heart action which are not known to or at least very rarely experienced by, the healthy individual. In support of this might be mentioned the peculiar sensation which often accompanies long pauses of the heart and many irregularities of its action. There are probably numerous finer variations in the motor relations of the heart, and in conditions of increased excitability they may, of course, easily lead to sensory disturbances.

Diagnosis.—The *nervous character of heart symptoms* is determined chiefly by demonstrating nervousness in the patient. As a rule, this is not difficult, but it is also necessary to determine to what extent general nervousness, pronounced neurasthenia, or hysteria is present. It is, of course, no part of our task to show how these diseases are recognized. For that we refer the reader to special works. But if there is reason to believe that a patient who complains of heart symptoms is "nervous," the next step must be to investigate whether the heart symptoms can be fully explained on the ground of a nervous foundation. We have pointed out the peculiarities of "nervous" heart affections. It was shown, in the first place, that the subjective symptoms are characteristic and that the great disproportion between the subjective and the objective symptoms, and among the latter chiefly the changes in the heart action, is no less characteristic. It was pointed out that these changes in the heart action are not directly characteristic of nervous

* Dehio, "Petersburger medicinische Wochenschrift," 1886, No. 31, 32.

heart affections; nevertheless, some of the disturbances occur, if not exclusively as the result of nervous processes, at least much more frequently than under any other conditions. Chief among these are the fluctuations in rhythm extending over considerable periods of time and the association of irregularity with unpleasant sensations. Great caution is, however, desirable in making a diagnosis, especially in the case of persons who have passed their fortieth year, and becomes urgently necessary whenever enlargement of the heart can be demonstrated.

Practically the only conditions that have to be differentiated from nervous disturbances of the heart are sclerosis of the coronary arteries, chronic myocarditis, some forms of beer-heart, and the effects of overexertion. The distinction from coronary sclerosis particularly may be exceedingly difficult, as Stokes long ago warningly pointed out. Even in the diseases of the heart muscle referred to, enlargement of the heart may be entirely wanting at the beginning, while disturbances of the rhythm as well as subjective symptoms are also present in these conditions. The subjective symptoms, it is true, are not so prominent, especially during rest, as in nervous individuals, who, in addition, are given to painting their symptoms in the liveliest colors. To arrive at a conclusion it must first be determined whether the conditions for the existence of nervousness, arteriosclerosis, or myocarditis have been fulfilled. This alone is a great gain. But it must be remembered that the necessary conditions for nervousness and arteriosclerosis, being so frequent and of such a general character, may easily be present in the same individual, and in that case one is no wiser than before, and it becomes necessary to interpret the symptoms in one way or another, in doing which all that has been said about the symptoms in each individual process must be borne in mind. But even under such unfavorable conditions a positive decision may occasionally be possible. The presence of arteriosclerosis especially can be determined with certainty in many cases, and the extent to which nervousness is also present in the individual is a much less important question.

Quite frequently, however, the physician will not venture to decide with any degree of certainty, and occasionally the differential diagnosis must remain open even after extended observation. In view of the many *possible* variations of the clinical pictures in these processes this may at first sight seem remarkable. But the various conditions have a great many points in common, and chance may combine only common properties in one and the same patient. Then, again, even the genuine diseases of the heart muscle, when closely examined, occasionally exhibit new and unexpected features. I have recently made the acquaintance of persons with coronary sclerosis and of others with myocarditis who, although at rest they presented marked subjective symptoms and distinct anomalies of the heart action and even of the size of the heart, were, nevertheless, still capable of performing severe muscular exertion without any discomfort. Such a condition has an important bearing on the treatment, and in every case therapeutic measures must be employed with extreme caution whenever the symptoms are not absolutely clear. Brunner* describes the case of a man addicted to smoking, drinking, and masturbation who suffered from palpitation of the heart and died. At the autopsy a sclerotic myocarditis was revealed.

* Brunner, "Gaz. lekarska," 1885, No. 46, 47. Quoted from "Centralblatt für klinische Medicin," vol. ix, 1888, p. 322.

Another great difficulty, finally, is due to the fact that patients with changes in the coronary arteries and the myocardium not infrequently *develop* nervousness. They begin to worry about the disease, and anxiously watch for every heart-beat. In that way they become hypochondriac and feel every change of the heart action much more keenly than persons of a less excitable temperament. This, again, may have some influence on the rhythm of the heart. Hence a coronary sclerosis may become complicated by general nervousness and perhaps also by conditions which are designated nervous disturbances of the heart. In diagnosing such a case the primary condition must, of course, be determined.

Prognosis.—The prognosis of nervous heart affections coincides in general with that of neurasthenia and hysteria; in other words, the prognosis of neurasthenia and hysteria is not specially influenced by the existence of cardiac symptoms, and even when the latter represent the sole or at least the chief expression of these diseases, the same general rules of prognosis nevertheless apply.

In very nervous individuals the trouble is exceedingly obstinate. Improvement takes place very slowly; complete recovery is not frequent, and if it occurs, is usually deferred until old age or is brought about only by a complete change in the patient's mode of life. The disease, however, has no direct influence on the duration of life, which is a very important point in many cases. In coronary sclerosis, on the other hand, the patient's life is in danger of being terminated at any moment and myocarditis must prove fatal sooner or later. These facts must be duly borne in mind, and if the diagnosis is uncertain, no positive prognosis should be given. In the milder cases of nervous disturbances of the heart, and especially if it is possible to subject the patient to active treatment, the prospect is very much better.

The **treatment** of nervous diseases of the heart is also exactly the same as that of neurasthenia and hysteria. Before instituting treatment I would advise that as positive a diagnosis as possible be made. If this is impossible, it is better, in a given case, to treat the case as one of disease of the heart muscle or, at least, to employ fatiguing measures only with the greatest caution. In this way only can the positive indication of "*non nocere*" be satisfied.

There are no indications for treatment other than those for the treatment of nervousness in general. The essential thing under all circumstances is that the patient should have mental rest and peace. The physician's ability to make his influence felt will vary greatly in different cases. It is rarely in his power to order the patient to live in such a way as to place him under the best possible surroundings. But in any case he can instruct his patient how best to bear his discomfort. In fact, the physician in these cases often performs the office of the minister, and if he tries to avoid doing so he can never expect to accomplish results.

There will always be a number of cases in which positively useful suggestions in regard to the patient's entire mode of life will be quite possible. In these cases the burden of the physician's injunctions must be, "away with every physical or mental exertion that throws an extra strain on the nervous system." It is the physician's duty to find out what is important and what is injurious in the individual case. It is, of course, impossible for me to give detailed instructions, for the possibilities for harm are practically as numerous as the conditions of life them-

selves. Some of the more frequent, however, may be pointed out, such as excess of mental and physical labor performed with haste, excessive physical exertion in the guise of athletics or "relaxation," which those who are not used to exercise are apt to indulge in, and, finally, sexual abuse. All these things must be stopped. Occasionally this is possible, and in such a case alone suffices to bring about improvement. If the patient has no suitable occupation, an attempt should be made to provide him with one, for no normal man can bear constant idleness.

In other cases more indirect ways of influencing the patient must be tried. It is often desirable to get him away from his accustomed work, in the pursuit of which he has contracted the disease, or, at least, to diminish his work so that it may cease to be injurious. But in the place of it the physician should try to provide some other occupation that will stimulate both body and mind.

Cutaneous stimulation takes the patient's attention away from what is going on in his heart, and at the same time strengthens and, it is safe to say, regulates its action. In the presence of distressing sensory disturbances and whenever the patient complains that he feels a certain nervous weakness, simple cold baths, brine baths, or carbon dioxid baths, as well as other hydriatic procedures, if carefully applied, do good service.

It has been shown elsewhere (p. 590) that it is possible to influence the functional activity of the heart and blood-vessels by means of these procedures. In the same place it was mentioned, however, how exceedingly difficult it is, in the present state of our knowledge, to estimate their precise influence on these structures. Since in nervous disturbances of the heart we do not even know how the morbid symptoms originate, every intelligent person will at once admit that actual experience at the bedside must be our sole guide in the prescription of therapeutic measures. Although this is a self-evident proposition, it is necessary to put it into words because in this "therapeutic era" of ours we are not content with having the various forms of treatment, especially hydriatic treatment, employed, taught, and their effects investigated, but also demand that these methods be employed in a rational manner or even treated as an exact science. For the present any serious attempt in that direction is out of the question. On the other hand, it may be confidently expected that in the distant future, when the true nature of morbid conditions and the mode of action of our various methods of treatment are fully understood, therapeutic procedures will follow strict indications.

To resume, therefore, hydriatic procedures are employed in exactly the same way as in other nervous and hypochondriac conditions. In the case of excitable patients, sedative measures are to be employed; when the patient is depressed, gloomy, and melancholy, stimulating and invigorating procedures are indicated. For purposes of treatment there is nothing gained by differentiating separate forms of nervousness; the physician should be guided by the individual patient's behavior and the way in which he reacts to therapeutic interference. Hence my advice always to try various measures at first and select a severe or a mild procedure according to the effect obtained. The following are to be recommended: tepid full baths at 34° to 30° C. (93.2° to 88.7° F.), of from five to thirty minutes' duration; to this may be added oil of thyme or of pine-needles, or common salt in 3 to 4 per cent. solution. Half-baths at 30° to 24° C. (88.7° to 75.2° F.), of from three to ten minutes' duration; the patient is enjoined to exercise while in the tub, and is rubbed and receives affusions over the front and back of the chest with water at the same, or at a slightly lower temperature down to 20° C. (68° F.). Carbon dioxid baths (for direction see p. 590) with or without

salt; faradic baths; cold rubs or ablutions of the entire body or of individual portions with water at 30° to 15° C. (88.7° to 59° F.), equal parts of water and alcohol [proof-spirit—Ed.], or brine in the strength of 3 to 5 per cent.; wet packs either of the trunk or of the extremities with water at 24° to 15° C. (75.2° to 59° F.) and a duration of one-half to one hour; various kinds of douches, which may be applied to the precordial region, but with great caution; ice-bags and various apparatus for cooling the heart (precordial coil, etc.).

Faradization, either general or applied to individual portions of the body, also acts very well in some cases. The greatest caution is necessary in applying electricity directly to the heart; personally I am inclined to advise against it altogether. These procedures are carried out in the forenoon, or between 5 and 7 o'clock in the afternoon, either once or twice a day, once a day, or every other day. The best criterion in this respect is the patient's reaction: he must neither feel fatigued after the procedure nor dread its application. Under no circumstances must suggestion be interfered with.

The treatment of the remaining symptoms of nervousness, such as insomnia, loss of appetite, restlessness, irritability, and lassitude, is exceedingly important. It is to be carried out according to general principles and, if it proves effective, not infrequently influences the heart symptoms most favorably. Special instructions in regard to sleep and waking, eating, drinking and smoking do not appear to be called for in this place, as they would merely be a repetition of what must be said in connection with the treatment of nervousness.

The question whether the treatment can be carried out at home must be very carefully considered in some cases. If the patient is constantly exposed to unfavorable psychic impressions in his own home, removal from his customary surroundings is most advisable. If the conditions are such that the mere removal to a quiet place where he will be under healthful surroundings may be expected to suffice, the patient may be sent to any health resort that happens to suit his mode of life and where there are opportunities for walking without undue exertion. The lower Alps and the mountainous forest regions of Germany, besides some portions of the Baltic coast, are chiefly to be recommended. If constant medical attendance is necessary, the patient should be sent to an institution in charge of men in whom his physician has confidence; here the choice will, of course, depend on personal considerations. [The pine woods of New Jersey, the mountains of the Carolinas and Georgia, the foot-hills of southern California, or the sea-shore of the latter, as well as of the States above-mentioned, correspond fairly well in climatic and therapeutic conditions.—Ed.]

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DISEASES OF THE NERVOUS SYSTEM OF THE HEART.

A great deal has been written about diseases of the cardiac nervous system, but in the present state of our knowledge there is, as a rule, no basis whatever for it. Unfortunately, nothing positive is known as yet in regard to the physiologic significance of the cardiac ganglia and cardiac nerves other than those which form part of the pneumogastric or the accelerators. Many theories may be formed with regard to these structures, and in ascribing to them the function of coördinating the various portions of the circulation and of assigning to these portions their proper places among the organs of the body we shall probably not be far from the truth.

But these are only general conceptions and cannot be utilized for the foundation of a formal theory in regard to symptoms which might with some reason be attributed to changes in these nervous structures.

The other method by which we usually advance in our knowledge, and, in fact, generally advance more rapidly, namely, by observation of the living human subject, to determine whether structural changes of certain portions of organs are present, is of little more value in this case. We know, it is true, that the cardiac nerves are diseased in a great many abnormal conditions of the heart muscle; thus, for example, neuritic processes are encountered in many forms of acute and chronic myocarditis. But I do not believe this knowledge can as yet be utilized for interpreting symptoms, and, so far as I can see, it is equally impossible to explain them by the changes observed in the cardiac ganglia. Such changes have frequently been seen, or at least reported. But, in the first place, one may be permitted to doubt the reality of these findings; for it is exceedingly difficult to distinguish between a normal and a pathologic ganglion-cell. Most observations belong to an earlier time, when even less was known in regard to the minute structure of ganglion-cells than at present, and the changes have been observed in the greatest variety of morbid conditions. It seems to me that investigators will have to make a fresh start in this field. Pathologic as well as clinical investigation has a large field before it, and it is probable that important discoveries will be made by the physiologist as well as by the pathologist.

But what about the conditions which the practitioner includes under the term nervous diseases of the heart? Some, in fact most, of these phenomena belong to neurasthenia and hysteria. Of that I believe there can be no doubt, and I have attempted, in the foregoing

pages, to give a description of all that can be collected under that caption, and at the same time have pointed out what little we are justified in assuming in regard to the origin of these morbid states.

But in doing so I by no means exhausted the field of "nervous heart disturbances." There is no doubt that in the future, as our horizon broadens and we begin to work on altogether different anatomic and physiologic lines, much will be added to our knowledge. Even now we observe many different disturbances of the heart action which we are inclined, by instinct and reasoning by analogy, to include among nervous disturbances, although there is no good reason for assuming that they are based on general nervousness. I am unable to give a comprehensive discussion of such cases because my experience in such matters is inadequate. Hence it is as yet impossible to give the characteristic features of these cases and to point out wherein they differ from disturbances developing only on a neurasthenic and hysteric foundation. The symptoms are like the symptoms of those conditions, which have been explained in the preceding section. The most conspicuous symptom is a peculiar disturbance of the heart action which may or may not be associated with subjective symptoms. But it is positively known that the heart muscle is endowed with considerable automatic function, and this makes the explanation considerably more difficult. Those of us who have become impressed by the wide-spread pathologic significance of changes in the myocardium were perhaps inclined to underestimate the abnormal conditions of the heart nerves, because one is naturally inclined to deal rather with known than with unknown factors.

But if cases belonging to this group are reported with due regard to all the attending circumstances, future writers may find it possible to give a connected account of these things; thus, for example, it cannot be altogether denied that many conditions of overexertion of the heart coming on after severe bodily exertion have some connection with nervous changes.

It is also possible that many cases of tachycardia belong to this group, especially cases occurring in those who are not nervous and present no abnormality of the heart muscle (see later). It must be insisted upon, however, that, as in all these matters, so in this question, we are dealing solely with conjectures.

It may be asked whether some of the so-called *vagus-neuroses* * do not belong to this group. Remarkable cases † have been reported which, in addition to acceleration of the heart action, presented a great many different phenomena that undoubtedly emanated from the heart, and which we are forced to regard as neurotic. When, in a case of this kind, the symptoms of diseases of the central nervous system, especially the symptoms of nervousness, are absent, a morbid change in the cardiac nerves naturally suggests itself. Nevertheless, I shall not hazard an analysis of these cases; for too many points still remain in doubt. Thus Matthes' patient had fever, which naturally suggests some infectious process—and many patients with tachycardia have fever. It is, of course, quite possible for an infectious process to direct its attack chiefly

* Compare Edinger in "Eulenburg's Realencyklopädie," second edition, vol. xx, p. 568.

† See Matthes, "Correspondenzblätter des allgemeinen ärztlichen Vereines von Thüringen," 1891, No. 12.

against the nervous apparatus of the heart, but who would venture to discuss such things at this time?

The existence of vascular symptoms by no means excludes the possibility of a cardiac neurosis. There is no doubt that changes in the blood-vessels may have their origin in some disturbance of the heart, since the closest connection between heart and blood-vessels is absolutely necessary for the maintenance of the circulation, and this close connection undoubtedly exists. Again, both series of phenomena may be coördinate —that is, may be due to a common cause.

It is very probable that the pathologic significance of diseases of the heart nerves will be greatly extended in the future. In the case of reflex disturbances of the heart action, to be discussed in the next section, as well as in those produced by various intoxications (see p. 618), this extension of our knowledge is already close at hand.

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DISEASES OF THE HEART POSSIBLY OF REFLEX ORIGIN.

Disease in various organs of the body may affect the heart action. The disturbances produced in this way have been chiefly studied in France, and our attention in Germany has been called to them largely through the observations of Potain; German physiologists and pathologists created the necessary theoretic basis for their discussion.

Heart disturbances may occur as the result of *painful affections of the nerves, muscles, and bones* in various parts of the body. Under such circumstances there may develop in the heart anomalies of rhythm, palpitation, anxiety, and pain, as in angina pectoris, but in a milder form. Even hypertrophy of the heart muscle has been described.

It is hardly permissible to doubt the statements of good clinicians *a priori*, but I, nevertheless, feel impelled to advise the greatest caution in accepting any existence of hypertrophy in these cases, particularly in the interpretation of the symptoms. It is not quite easy to determine hypertrophy of the heart during life with absolute certainty, and it is exceedingly difficult to explain its presence in these conditions. Before attempting to do so and vainly cudgeling one's brains, a good many more cases must be collected, especially for the purpose of positively distinguishing between hypertrophy, on the one hand, and mere dilatation and simple accentuation of the heart action, on the other.

In the attempt to explain these sensory and motor disturbances of the heart a great many different causes have been thought of; the

effect of an ascending neuritis; processes similar to the reflexes in which, after the irritation of sensory portions and after the coöperation of central cells, other portions of the nervous system are stimulated. Such things are conceivable, but before offering any final conclusions it seems to me that we ought to collect some additional facts and especially, if possible, some further anatomic observations. The observations at present available are, so far as I know, all derived from French physicians, and in France—at least for the present, and among the male population—hysteria is undoubtedly much more frequent than in Germany. In reading many of these reports I simply cannot free myself from the impression that the symptoms were based on hysteric phenomena. Potain himself states that in many of these cases the patient's nervous system is probably exceptionally sensitive.

It is very curious that cardiac symptoms occur chiefly after diseases affecting the left arm; as yet there is, in my opinion, no satisfactory explanation of this fact. It is not without a parallel, however, for the pain of angina pectoris chiefly radiates toward the left arm.

Anomalies of the heart action are also observed in *association with morbid phenomena in the abdominal organs*, especially the gastro-intestinal canal, the liver, and the genitalia. The relation existing between the latter and the heart has already been mentioned in the discussion of the nervous diseases of the heart. The connection is probably exclusively psychic.

On the other hand, the disturbances of the heart occurring in *diseases of the gastro-intestinal canal and of the liver* require special description. Great caution must be observed in establishing causal relations, for we know how frequently both the liver and the stomach become diseased *after*—that is, as the result of—disturbances of the heart action. Of that there can be no doubt. But the converse relation is equally well established. This assertion by no means rests on an uncertain foundation; it is not the result of a luxuriant imagination, for I have seen numerous cases of cardiac disturbances resulting from abdominal disease. It is quite a frequent occurrence and of great practical importance.

One fact has been known for a long time and is now well established. It is that dyspeptic conditions produce disturbances of the heart action, *especially in children.* The disturbance sometimes consists in acceleration, but much more frequently in retardation and irregularity of the rhythm. If headache is superadded, the clinical picture becomes very similar to that seen in the early stages of tuberculous meningitis, and the difficulties of distinguishing between the two conditions have puzzled many a diagnostician.

Exactly the same thing is observed *in adults*, in whom, however, *palpitation, i. e.*, a subjective consciousness of the beating of the heart, is much more frequent and is associated with a violent sensation of fear and "emptiness." The *heart action* may also be *retarded*, but *acceleration* going on to marked tachycardia is much more frequent. *Irregularity* and *inequality* are often present also. These phenomena are particularly apt to occur early in the morning before breakfast, and are often associated in the most extraordinary manner with anomalies of digestion, especially constipation, flatulence, eructations, and distention, to which they are due.

How these phenomena are produced is by no means clear. The various possibilities were given on p. 546, where palpitation is discussed.

The palpitation in these cases is undoubtedly often associated with an increase in the strength of the cardiac contractions, but it is impossible to say what effect increased sensibility has on the contractions of the organ.

Occasionally the right heart is said to be enlarged and the second pulmonic sound accentuated in these conditions. The French investigators attribute these symptoms to spastic conditions in the pulmonary vessels, which, they argue, cause increased action of the right ventricle and a certain degree of dilatation of that cavity. I am unable to give a personal opinion on this question of enlargement of the heart due to abdominal disease, because I have never seen such cases. But I am inclined to think that exaggerated contraction of the pulmonary vessels is the least likely cause of such an enlargement. Gallop rhythm and the occurrence of systolic murmurs, especially at the tricuspid valve, have also been described, and all these symptoms are said to vanish as soon as the abdominal symptoms have subsided.

The primary causes of these disturbances are found in diseases of the stomach, intestinal canal, and liver. There is no doubt, however, that these disturbances are not due to any one lesion in the abdomen; for a variety of conditions may give rise to the same sequelæ in the heart and, on the other hand, the same cause by no means produces symptoms of equal severity in the same, or even in different, individuals. This makes it quite evident that the trouble lies in alterations of the central nervous system or of the heart or of both; for the present it is impossible to arrive at a decision. But, at all events, nervousness is a very important factor in the development of all these conditions.

According to Potain, it is characteristic that the abdominal disturbances which cause the above-mentioned sequelæ in the heart usually do not represent grave diseases, and in this I fully agree with him; I have never seen such effects from cancer of the liver, stomach, or intestine. On the contrary, the milder dyspeptic processes in the stomach and in the intestine, so-called simple catarrhal conditions, constipation, and parasites of various kinds, and gall-stones in the case of the liver, are the conditions which are most important in this respect. The most curious affections of the stomach are cited in this connection, and in studying the clinical histories of these cases one cannot escape the thought that in many of them the most important element in the condition is a "nervous" constitution. For what is one to think when told that every mouthful, or possibly only some special article of diet, produces violent palpitation as soon as it enters the stomach? In not a few of the cases the cardiac attack is not even dependent on the existence of any dyspeptic processes, but is unfailingly induced by some article of food which other individuals find entirely harmless. It is true that in just such cases one might be inclined to assume a peculiar chemic irritation of the peripheral nerves. But if the irritation is followed by such peculiar consequences, it appears to me that it is a sign of an extreme anomaly of the nerves, either in the central nervous system or in the heart. Emotional factors unquestionably play a very important rôle in the etiology of these cases.

Palpitation is a constant symptom in these cases, and occurs only after the exciting causes mentioned. From a diagnostic point of view, and in the total analysis, it seems to me important that special demands on the heart muscle, such as active bodily exercise, are entirely without any effect and, indeed, are very well borne.

The connection between these cardiac symptoms and the abnormal conditions in the abdomen may be explained in one of two ways. Intoxication is one possibility and is most in accord with our modern views. But the theory still lacks foundation; for we do not know what the toxic substances are; all we know is that special forms of dyspepsia are particularly apt to bring on disturbances of the heart action. Another possibility, which is in accord with the view held by Potain and his disciples, is that the disturbances are due to reflexes from the abdominal organs acting on the heart through the pneumogastric nerve. There is undeniably a great similarity between many of these heart symptoms and the symptoms produced by irritation of the vagus. There are, besides, two other things which support this view. One is that irritation in one part of this peculiar nerve is specially apt to be propagated to other portions of the nerve, as numerous observations have shown. And, in the second place, other symptoms are observed after gastric disturbances which it is hardly possible to regard as due to anything but a reflex irritation of fibers of the pneumogastric nerve, *i. e.*, reflex irritation through the lungs. A further description of these reflexes is hardly called for; they consist of peculiar respiratory disturbances which occur independently of abnormalities of the heart action. Finally, the rapid course which often characterizes the conditions directly points to reflex irritation. Thus I have frequently observed most unpleasant cardiac or pulmonary symptoms in dyspeptic individuals disappear immediately after copious belching, and a medical man has observed the same thing in his own person with absolute certainty.* In such cases the disturbances of the heart action can be due only to direct irritation of the stomach, either of a mechanic or toxic nature. It is possible that a stomach greatly distended with gas may directly affect the heart through the diaphragm; but this mode of irritation is probably unimportant, for in many of the cases there is absolutely no tympany of the gastro-intestinal tract.

Sensory disturbances of the heart are also not infrequently associated with diseases of the gastro-intestinal canal. Almost all the symptoms mentioned on p. 531 might be cited except that they usually occur in a milder form: oppression, anxiety, pain in the precordial region. These things are, of course, observed in combination with symptoms referable to various other organs, and it must always be remembered that the clinical picture is usually extremely complicated. A full description belongs to the section on Anomalies of Digestion. Our reasons for describing the heart symptoms separately are, in the first place, comprehensiveness and, in the second place, because of a particular point in the diagnosis.

Conditions more or less resembling angina pectoris may occasionally occur after gastro-intestinal disturbances. Just as true stenocardia develops from a variety of different beginnings and, accordingly, presents many different degrees, so we observe after abdominal diseases combinations of all kinds of sensory and motor disturbances of the heart, and many of these disturbances closely resemble the picture of stenocardia, *i. e.*, the most striking symptom is fear of impending death, with radiating pains to a variable distance from the precordia; alterations of the heart action may also be present. Fortunately, these

*See Jessen's instructive case in "Münchener medicinische Wochenschrift," 1899, p. 1410.

are not frequent. When they are encountered, the physician's first duty is to make sure that the angina has followed some digestive disturbance, and to satisfy himself that the attack is not due to arteriosclerosis, tobacco-poisoning, or some nervous condition. He must determine whether there are any data in favor of the existence of one of the above-mentioned conditions, on the one hand, or of gastro-intestinal disturbances, on the other hand. This is the most important point, and of the French investigators, who were the first to describe these rare conditions, Potain and Huchard lay great stress on the fact that dyspeptogenic angina is always accompanied by dilatation of the right ventricle, accentuation of the second pulmonic sound, and often by gallop rhythm.

The nature of the attack must next be taken into consideration. Stenocardia due to a disturbance of the gastro-intestinal canal is, on the whole, milder, and, as is usual with milder attacks, the duration is longer. Exercise is of no importance whatever as an etiologic factor, the ingestion of food being the only exciting cause. The attacks are never dangerous, and at once disappear when the dyspepsia has been relieved.

Personally I have seen very few cases of dyspeptogenic stenocardia and have, therefore, been obliged essentially to reproduce the descriptions of the excellent French clinicians, especially as the German literature contains very few observations on this subject. From considerable experience I have gained the impression that arteriosclerotic or nervous individuals whose general or cardiac condition predisposes them to the occurrence of cardiac disturbances are also most subject to anginoid attacks following dyspepsia. Being under this impression, I am inclined to be very cautious and reserved in giving a medical opinion of these conditions.

Nothing positive can be stated in regard to the mechanism of dyspeptogenic stenocardia. Some disease of the gastro-intestinal canal or of the liver creates the necessary conditions for the production of angina pectoris; whether this is effected through the action of toxic substances or reflexly is as difficult to decide in the case of angina as in the anomalies of the movement of the heart due to digestive disturbances.

Clinical histories are given by Huchard and Barié.

The **treatment** should be directed solely against the digestive disturbance. When this is relieved, the cardiac symptoms disappear. I would advise the physician to pay great attention to the nervous system, and not to be too free with the diagnosis of these conditions. Coronary sclerosis must always be borne in mind, as it is exceedingly difficult to exclude that condition.

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PAROXYSMAL TACHYCARDIA.

Acceleration of the heart-beat occurs under a variety of circumstances. As a symptom, it has been discussed in connection with the various morbid conditions, and what little is known about the phenomenon theoretically was set forth to the best of our ability on p. 460.

In this section we shall deal with marked increase of the heart's frequency occurring in *paroxysms*. In this form also the phenomenon occurs after a variety of causes; in other words, paroxysmal tachycardia is often a mere symptom. But we must inquire whether the phenomenon always represents merely a symptom or whether it may perhaps, in some cases, be the cardinal symptom of an otherwise obscure disease. Until the properties that characterize the condition are known, it will, therefore, be necessary to continue to speak of "paroxysmal tachycardia" as a clinical entity, and for this purpose the relations or differences existing between the conditions of individual patients suffering from tachycardia must be investigated.

Unfortunately, the advancement of our knowledge was long delayed, because every investigator was bent on solving the question whether paralysis of the inhibitory nerves or irritation of the accelerator nerves was the cause of the tachycardia, and in doing so neglected every other point of view. Clinical medicine wished to share in the great discoveries of physiology relating to the innervation of the heart. But in doing so she left her own domain, in which she is mistress, and became the slave of strange thoughts. Martius * has correctly pointed out the retarding effect which this tendency has had on the progress of our knowledge of these things.

"Essential" paroxysmal tachycardia is characterized by sudden attacks of great acceleration of the heart-beat (150 to 300). The attacks may last a few minutes, hours, or days, or may be protracted for weeks. During the attack the heart, in a great many cases, at least, is unquestionably enlarged in every direction.† Martius was the first to call attention to the frequency of this phenomenon. But I do not think it is correct to regard dilatation as a classic symptom of the entire symptom-complex, or even as the most prominent symptom; for cases of genuine paroxysmal tachycardia have undoubtedly been observed in

*Martius, "Tachycardie," Stuttgart, 1895.

†Martius, "Tachycardie," Stuttgart, 1895. Hochhaus, "Archiv für klinische Medicin," vol. li, p. 1. Freyhan, "Deutsche medicinische Wochenschrift," 1889, No. 19. Bouveret, "Revue de médecine," 1889.

which there was no dilatation.* In some patients the attacks are at times accompanied by dilatation, and not at others.

I would not attach any value to these cases if they had all been reported at a time when but little attention was paid to the frequency of cardiac enlargement; but some of them were observed since that time, and yet the authors failed to see any dilatation. Aside from this, it is distinctly mentioned in many of the earlier cases that the heart was not enlarged.

The pulse, in the great majority of cases, is small and soft during the attack. This may perhaps be directly due to the frequency of the cardiac contractions in some cases. Since, as physiologic observations have shown, the acceleration is effected chiefly at the expense of the diastole, it is quite conceivable that the ventricles at last may be so incompletely filled that, in spite of the great frequency of the heart-beat, the arterial pressure cannot maintain itself at its normal level. It is more probable, as Martius pointed out, that the incomplete filling of the arteries is due to a coincident weakness of the heart.

This *weakness* of the heart might also be regarded as the cause of the dilatation; for it has already been shown that incomplete cardiac contractions usually lead to dilatation of the cavities.

Or the dilatation is the result of a peculiar effect of the morbid process on the tone of the heart muscle. Under no circumstances can the dilatation be regarded as directly dependent on acceleration of the heart-beat; for, since diastole is shortened, it follows that less blood enters the heart, and the volume ought rather to diminish.

At all events, the tachycardiac attack is usually associated with heart weakness, but few exceptions to this rule being known. Thus Nothnagel† describes a case of paroxysmal tachycardia in which the pulse was of good tension and, in fact, remarkably hard. It is true that in this case the rate was not high (150), but the case must, nevertheless, be included in this symptom-group. Brieger‡ also observed a heaving apex impulse in a case of tachycardia without dilatation. From animal experiments we know that acceleration of the heart-beat from irritation of the accelerator nerves at first, and so long as it does not exceed certain limits, increases the pressure in the arteries.

The *heart action* is almost always regular and equal, a point that needs to be specially mentioned because the ordinary acceleration of the heart-beat which accompanies cardiac weakness frequently causes disturbances of the rhythm. In the tachycardiac attack, on the other hand, it is only at the beginning and at the end that a series of irregular pulses is frequently observed.

The *apex-beat*, as a rule, is soft, although there may be concussion and heaving of the entire precordial region. This is readily explained after Martius by the dilatation of the heart, which renders the contractions weaker. It is another positive sign that the heart action is usually weakened at the same time.

The *heart-sounds* are pure unless some special reason for the production

* Loeser, "Virchow's Archiv," vol. cxliii, p. 648. Romberg, "Herzkrankheiten." Kelly, "The Med. and Surg. Reporter," October 24, 1896, No. 17. Abstract: "Virchow-Hirsch," 1896, vol. ii, p. 157. Brieger, "Charité-Annalen," 13. Jahrgang, 1888, p. 193; and a number of other cases. Compare Hoffmann, "Die paroxysmale Tachycardie," p. 15.

† Nothnagel, "Wiener medicinische Blätter," 1887.

‡ Brieger, "Charité-Annalen," 13. Jahrgang, 1888, p. 193.

of a murmur is present; sometimes murmurs disappear during the attack. As a rule, owing to the shortening of diastole, the rhythm of so-called embryocardia is present.

A. Hoffmann observed among his patients abnormal mobility of the heart within the thoracic cage, and suggests that the resulting traction on various structures in the thorax may be in part responsible for the attacks.

Many patients suffer during the attacks, varying greatly in different individuals. Almost all exhibit lassitude, depression, and anxiety. Oppression, pressure, and pain on the chest not infrequently make their appearance. Most patients have a collapsed and cyanotic appearance, though this again is subject to great variations. But in some cases the general condition is not in the least influenced by the attack, even when it is severe. Many patients even go about their usual work, although the tachycardia may attain an exceedingly high degree; while others are compelled to rest even by a very light attack. It is evident, therefore, that the effect of the tachycardia on the strength of the heart is extremely variable.

The *lungs*, in the majority of cases, are practically unaffected, and the curious cases of tachycardia with acute emphysema reported from Riegel's clinic in Giessen are certainly exceptional. In these cases,* which were described by Tuczek, Kredel, and Honigmann, the boundaries of the lungs were suddenly, in the course of the attack, discovered to be unusually low, while the absolute heart dulness was diminished or even disappeared. This, of course, rendered examination of the heart exceedingly difficult, and the size of the organ in these cases is, therefore, not positively known. Tuczek states quite positively, however, that there was no enlargement of the heart in his cases—at least in the beginning. The emphysema would subside more or less rapidly after the cessation of the attack.

As regards the frequency and significance of this emphysema, it seems to me that much still remains to be found out. While the three above-mentioned authors are the only ones who mention any considerable degree of *volumen auctum*, it is, nevertheless, stated by other observers that the lower border of the lung was depressed during the attack.

Stasis in the systemic veins develops quite frequently and is, in fact, the rule when the attack is prolonged. The symptoms are swelling and pulsation in the vessels of the neck, enlargement of the liver, albuminuria, and edema. These symptoms also vary exceedingly in severity, probably depending on the degree of impairment of the strength of the heart or, more accurately, of the functional capacity of the right ventricle. Possibly the weakness of the muscle, as occurs under other circumstances, attacks different parts of the heart in different patients, so that, in some cases, the insufficiency of the left ventricle is more prominent, while in others insufficiency of the right heart predominates. This would explain the differences observed in the respiration. For the latter varies greatly in different cases; some patients are dyspneic and present acceleration or deepening of the breathing, or both. In other cases the respiration is unaffected. One is inclined with v. Basch to attribute the emphysema chiefly to weakness of the left ventricle and stasis in the lungs; but even on that theory we are far from having a clear under-

*Tuczek, "Archiv für klinische Medicin," vol. xxi, p. 102. Kredel, *ibid.*, vol. xxx, p. 547. Honigmann, "Deutsche medicinische Wochenschrift," 1888, p. 919.

standing of the conditions. The theory would work in Kredel's case, for example; his patient had emphysema and dyspnea, as had Freyhan's second patient; but in Tuczek's case there was no dyspnea whatever: in spite of *volumen auctum*, respiration was performed quietly and without difficulty. The *urine* has been mentioned. Owing to congestion, the quantity is usually diminished, albumin is often found, and not infrequently blood-cells and hyaline casts are also present. In a very few of the cases the excretion of large quantities of a pale, watery urine is mentioned. One might be inclined to attribute this symptom to an acceleration of the circulation, but the careful and important observations of A. Hoffmann show that large quantities of urine with a specific gravity of 1003 to 1004 were voided during the attack, although the arterial pressure had fallen. This seems to show that the cause is rather a local increase in the velocity of the blood in the kidneys.

Inflammatory processes sometimes develop *in the lungs*. In such cases we can readily understand the occurrence of fever. But alterations of temperature, even up to 40° C. (104° F.), are by no means dependent on pulmonary complications. The cause of fever in such cases is quite unknown. Possibly it indicates some inflammatory process in the heart. The French investigators get over the difficulty by assuming the existence of a specific "nervous" fever, but I think I am justified in looking upon that explanation with suspicion.

The attacks often occur quite suddenly and subside with equal rapidity. At the instant of onset and of disappearance the patient has a peculiar sensation in the chest, which he describes as a stab or a sudden wrench, and which is a warning to those who have previously gone through an attack that the paroxysm is either beginning or coming to an end. The change from one condition to another, especially the return from the attack to a normal condition, is often extremely impressive.

This sudden appearance and disappearance of the phenomena is undoubtedly what happens most frequently, but cases are also described in which both the beginning and the end of the attack are slow and gradual; these cases have to be mentioned in order to escape the charge of undue generalization. Sometimes the attack is preceded by prodromata, peculiar sensations, or even vomiting.

It is, no doubt, possible to pick out the symptom-complex that I have tried to describe in the foregoing from the great mass of clinical histories with greatly accelerated heart action that are contained in the literature. For this work we are indebted to Nothnagel,* Bouveret,† and Martius. But I am, nevertheless, inclined to think that the classification of these cases is a much more difficult matter and calls for much greater caution than many investigators appear to believe.

The attacks described occur in persons who, on careful observation, present nothing abnormal except during the attack, especially in the heart ‡ and nervous system. It is quite possible that the organ in these cases may be actually sound, although this assertion must be made with a certain reserve, for I agree with Romberg entirely on this point. In the first place, we are learning more and more that our methods of examination are relatively inadequate, especially for the detection of the finer anomalies; we are not even able as yet to determine the functional

* Nothnagel, *loc. cit.*
† Bouveret, "Revue de médecine."
‡ Compare, *e. g.*, the cases of Bouveret and Martius.

capacity of the heart. Persons suffering from essential tachycardia not infrequently give a history of causes which we know not rarely injure the heart action *permanently*. Thus Martius, Hochhaus, and others state that their patients gave them a history of severe bodily exertion previous to the attack. Sometimes the attacks are preceded by emotional excitement, and not only the individual attack, but the general state of the body which favors their occurrence, as it were, is preceded by such emotion. We shall return to this point. Articular rheumatism is also mentioned in the history of some patients with essential tachycardia.

Exactly similar attacks occur in persons with very pronounced diseases of the heart, such as valvular lesions,* coronary sclerosis,† after diphtheria,‡ dilatation of the heart.§ In fact, when we read such histories as that of the first case observed by Hochhaus,‖—tachycardia with dilatation of the heart and high fever,—we cannot help wondering whether the symptoms, as a whole, did not have their starting-point in some inflammatory process in the heart muscle. In fact, Nothnagel actually observed tachycardia with genuine fibrous myocarditis.** Hoffmann made a similar observation. I myself once saw, in a typhoid convalescent, an absolutely typical tachycardia which formed the beginning of a typhoid myocarditis.

The *exciting cause of the individual attack* is not definitely known. Certain factors that have been mentioned as important in the development of the systemic condition which predisposes to the attacks are probably also responsible for the attacks themselves. Of these, muscular exertion and emotional excitement are the most important. Both of these are so frequently given as causes that there is no need of citing examples. In my own opinion the influence of psychic processes is exceedingly important; it has been shown elsewhere that in the so-called nervous diseases of the heart considerable acceleration of the pulse occurs in paroxysms. Most of these cases are undoubtedly different from the symptom-complex now under discussion. But I am also convinced that there are transitional cases; at all events, not a few of those who believe that I go too far in expressing myself thus will have to admit that it may be exceedingly difficult, in a concrete case, to decide whether the patient's symptoms are to be regarded as the expression of acceleration of the heart accompanying nervousness or of essential tachycardia.

Disturbances in the abdominal organs (gastro-intestinal canal, genitalia) may bring on attacks of tachycardia in every respect similar to those just described. In the last section the relations existing between the action of the heart and irritation originating especially in the gastro-intestinal canal and the liver were discussed. A variety of symptoms, including, among others, acceleration of the heart action, occur under

* Zunker, "Berliner klinische Wochenschrift," 1877, No. 48, 49. Honigmann, "Deutsche medicinische Wochenschrift," 1888, No. 45. Bunzel, "Prager medicinische Wochenschrift," 1896, No. 28, 29. Abstract: "Virchow-Hirsch," 1896, vol. ii, p. 156.

† Romberg, "Herzkrankheiten." Brieger, "Charité-Annalen," 13. Jahrgang, 1888, p. 193; compare C. Gerhardt, "Volkmann's Vorträge," No. 209, case 3.

‡ Löwit, "Prager Vierteljahrsschrift," 1879, vol. cxliii, p. 27.

§ Spengler, "Deutsche medicinische Wochenschrift," 1887, No. 38.

‖ Hochhaus, "Archiv für klinische Medicin," vol. li, p. 17.

** Nothnagel, "Wiener medicinische Blätter," 1887, No. 1–3. Compare also Herringham.

these circumstances, and these symptoms may have almost the same character as those here described. Cases of this kind have been described repeatedly.* This appears to me to have an important bearing on our whole conception of these things in several respects. In the first place, it shows the close relation existing between nervousness and the heart action; for this sensitiveness of the heart action to anomalies of digestion is chiefly encountered in nervous individuals. On the other hand, it shows the difficulty of differentiating between different types of cases and proves the occurrence of the transitional cases. The changes in the heart action due to disturbances in the abdominal organs may be of various kinds, and no sharp dividing-line can be drawn between simple acceleration and paroxysmal tachycardia, which is a part of our symptom-complex.

I am, therefore, inclined to believe that there are very curious cases in which acceleration of the heart action occurs in attacks and disappears again, and is usually quite marked. Often the heart is enlarged during the attack and the action is weak; sometimes there is also pulmonary emphysema. To me personally it seems most probable that tachycardia and diminution in the strength of the heart are coördinate phenomena. The dilatation is probably secondary to the diminution in the strength of the heart, or it goes hand in hand with both processes. I do not agree with Martius that the acceleration is secondary to the dilatation, partly because dilatation is absent in a number of cases and partly because even the most severe grades of dilatation do not, as a rule, cause a degree of acceleration that even remotely approaches in severity that observed in paroxysmal tachycardia.

These attacks occur, as has been stated, either as a symptom in various diseases of the heart, or they may represent the sole symptom, and it may be impossible, with our present methods, to detect any change in the heart to which the symptom can be attributed. In these cases we speak of essential tachycardia. Hence we follow exactly the same line of reasoning as in the interpretation of symptoms referable to other organs.

The fact that the tachycardia which occurs in hearts that are actually or apparently sound is exactly the same as the tachycardia observed after genuine diseases of the heart, seems to me a strong argument in favor of a cardiac origin even in the so-called essential cases, and I am also of Romberg's opinion that coronary sclerosis perhaps plays an important rôle.†

There is no doubt that the *tachycardia is intimately related to nervous influences*, a point that has been emphasized repeatedly in the foregoing pages. Pressure on the vagus in some patients suffices to terminate the tachycardiac attack at once. This is a most interesting fact, and may in the future be found to have an important bearing on the interpretation of the phenomena. For the present it cannot be used for this purpose because we still lack the necessary data. For example,

*See, *e. g.*, Preisendörfer, "Archiv für klinische Medicin," vol. xxvii, p. 387. Theilhaber, "Münchener medicinische Wochenschrift," 1884, No. 42. Compare Payne-Cotton, "British Medical Journal," 1867, i, June 1, p. 629. Abstract: "Cannstatt," 1867, vol. ii, p. 80. Bowles, *ibid.*, vol. ii, p. 53. Abstract: "Cannstatt," 1867, vol. ii, p. 81.

† At least a number of transitional cases occur as the result of coronary sclerosis; compare, *e. g.*, Dehio, "Petersburger medicinische Wochenschrift," 1880, No. 48.

it would be desirable to know whether heart failure due to other causes can also be influenced by compression of the vagus.*

As for the causes within the heart which lead to the curious alterations in the action, force, and size, they cannot, in my opinion, be enumerated in regular order because of our utter lack of the necessary data. Personally, I am inclined to assume an abnormal nervous condition. This, it seems to me, is supported by the close relation existing between not a few cases of tachycardia and nervous influences; the effect of compression of the vagus, which has frequently been observed; the sudden appearance and equally sudden cessation of the attack; and the curative influence of factors which act chiefly on the nervous system.

Whether all cases of tachycardia are due to the same fundamental process cannot, in my opinion, be determined at the present time. In this connection it is quite irrelevant that in some cases with an attack of this kind no changes are found in the heart in the intervals between attacks, while in others the organ presents a well-marked pathologic condition; for the latter could not possibly be the direct cause of the tachycardia attacks, since these occur only in a relatively small number of heart patients. Coronary sclerosis, likewise, is not the direct cause of stenocardia; it only creates, more frequently than anything else, the necessary conditions for the development of angina pectoris. It is quite possible that the functional disturbance which brings on the tachycardiac attack, and the nature of which is now entirely hidden, may develop in a great many different diseases of the heart or even in the absence of any heart disease.

Even those who are of the opinion that the attacks are due to some nervous anomaly in the heart cannot, at all events, attribute them to injury of any special nerve-fibers. This has been emphasized by Martius, and I fully agree with him. Instead of trying to explain unknown conditions by the aid of our very limited knowledge of paralysis of the vagus and irritation of the sympathetic nerve, it would be much better to try to build up, from observations on individual patients, a nerve physiology applicable to man. Our knowledge of the influence of the vagus and of the accelerator nerves of the heart is chiefly derived from experiments on the dog, the rabbit, and the frog. Even these animals present certain differences among themselves. In the case of man we have not even laid the foundation, and until that has been done, we should keep clear of speculation,which is absolutely barren of results. The matter is extremely complicated: in the dog the vagus contains a great many different kinds of fibers which influence the number, rhythm, and force of the contractions of the heart in a variety of ways. The sympathetic also has other functions besides that of accelerating the contraction of the heart; the two nerves are by no means antagonists in the strict sense of the term. Aside from these differences, it is quite possible that there may be many others in the case of man. Under such circumstances who cares to use such hackneyed terms as vagus paralysis and stimulation of the accelerator nerves?

A few remarks about so-called *vagus neuroses* are unavoidable in this connection, although a comprehensive treatment of these neuroses belongs to the subject of nervous diseases. The term vagus neurosis should include those symptom-complexes which are produced by anything that acts on the endings or trunk of the pneumogastric nerve and disturbs its function. In such a condition one would expect symptoms in various organs supplied by the nerve, especially the larynx, the lungs, the heart, and the gastro-intestinal canal. A few words must be devoted to this subject because the cardiac symptoms, which are often so prominent in a clinical picture of vagus disease, bear a certain resemblance

*Compare Quincke, "Berliner klinische Wochenschrift," 1875, No. 17, 18.

to the conditions seen in paroxysmal tachycardia, although, as Nothnagel has pointed out,* really the two conditions have nothing whatever in common.

In a number of observations on the human subject lesions of the pneumogastric nerve, which, for other reasons, one is justified in believing capable of disturbing the function of the heart, led to acceleration of the heart-beat. These cases show that there is such a thing as pneumogastric tonus in man as well as in animals, and that its loss causes acceleration of the pulse. The latter is then present during the entire duration of the disease. Palpitation, as well as every other unpleasant heart symptom, is often absent; the organ is not enlarged. In addition, symptoms referable to other organs under the influence of the pneumogastric nerve, especially the larynx and also the lungs and stomach, are frequently present. Hence, aside from the acceleration of the pulse, most of the symptoms are, as a rule, quite different from those observed in paroxysmal tachycardia.

The number of observations is quite inadequate, and I do not believe they as yet enable us to form any definite idea of what the entire symptom-complex in vagus diseases should be, or how it must be limited. It will depend chiefly on the seat of the lesion, which determines the kind and number of organs capable of causing symptoms at all in a given case.

Much will also depend on whether one or both nerves are affected. Animal experiments show that *division* of one of the vagi, as a rule, has no marked effect on the heart action. In man Billroth observed transient acceleration of the pulse after division of one pneumogastric. But stimulation of only one of the two nerves is attended by a marked effect, which is very suggestive when we remember that in by far the majority of clinical cases only one nerve was affected. It is an exceedingly difficult matter to determine whether a nerve lesion is irritative or paralyzing. This one point alone is the cause of great difficulties in nerve pathology. Finally, it is not known with sufficient accuracy what kinds of fibers are contained in the pneumogastric nerve in man, nor at what points they enter the nerve-trunk. Hence one should be very cautious in giving the opinion, "that is impossible if disease of the vagus is present," particularly as regards the possible upper limit of the pulse frequency in that condition.

Nevertheless, it is often exceedingly difficult to draw a dividing-line between paroxysmal tachycardia and the effects of vagus disease. In most cases the above-mentioned factors, and especially their combination, make it possible to distinguish vagus disease from paroxysmal tachycardia, particularly if one has been careful to observe exactly under what external conditions the acceleration of the pulse occurs in the two cases.

Much more difficulty may be experienced in distinguishing the form of acceleration of the heart-beat which occurs paroxysmally in nervous individuals, and particularly after diseases of the abdominal organs. This point has already been discussed. It may fairly be asked whether, under such circumstances, it is possible to speak of any strict differentiation. In these conditions the action of the heart is greatly accelerated through reflex and possibly through toxic influences. How far this acceleration is attended by diminution of its strength remains to be shown by future observations. We know very little about the mechanism of this alteration of the heart action, both in these conditions and in essential tachycardia as above defined, and cannot, therefore, at the present time, pretend to say, under all circumstances, whether two cases are similar or dissimilar.

*Nothnagel, "Wiener medicinische Blätter," 1887.

In my opinion the physician's first duty in making a *diagnosis* of the conditions which are here under discussion is to find out whether there are any factors in the case that can be held responsible for the occurrence of an attack of tachycardia; whether the attack is a symptom of existing heart disease; or whether the attack, as well as the phenomena which, as a rule, accompany it, forms the only symptom—that is to say, whether it is an essential attack according to our present conception of the term. If, in every individual case, an attempt is made to determine these points, a diagnosis in the sense which I have just explained is almost always possible, and, at the same time, the physician will add something to the elucidation of this question, which is as interesting as it is important. To begin with, tachycardia should always be regarded as a symptom, and should be regarded as a nosologic entity, if at all, only after all other possibilities have been exhausted. After that, its persistence as a nosologic entity will depend on the success attending the physician's attempts at greater accuracy in diagnosis. This is the method of defining a clinical picture that proves successful in every department of pathology.

The **prognosis** is determined chiefly by the presence or absence of some grave underlying disease of the heart or of some other organ and by the character of that disease. A single attack of tachycardia is rarely dangerous, although it occasionally leads to fatal cardiac insufficiency. The tendency to the attacks may disappear at any time and, on the other hand, may last for many years. If the patient knows what causes the attacks and it is possible for him to avoid the causes, the prognosis is, of course, very much better.

Treatment.—Since we do not know what constitutes the disposition to the attacks, we have no means of combating it. If the patient knows what causes the individual attack, he must attempt to avoid such causes. Sometimes potassium iodid or carbon dioxid baths seem to have some effect in preventing the recurrence of the attacks.

The treatment of the attack itself is purely symptomatic; digitalis has often been given, but, to judge from reports, it rarely seems to have been followed by any real benefit. The application of cold water or ice to the precordia is advisable under all circumstances. To quiet the patient morphin, opium, or potassium bromid may be given, but, unfortunately, those drugs have but little effect. It has already been stated that pressure applied to the pneumogastric in the neck terminates the attack, and simple suggestion may also be quite effective.* As I have said, these facts both seem to me strong arguments that the condition is due to some nervous cause.

It is probable that the curious remedies with which some of the patients artificially terminate the attack, such as deep inspiration, producing a marked increase of intrathoracic pressure by means of forced expiration after deep inspiration,† also produce their effect by stimulating the vagus.

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HEART DISEASES FOLLOWING BODILY INJURIES.

The heart may be directly involved in any injury to the entire body, such as injuries to the precordia, thorax, and epigastrium by contusion, laceration, or a wound inflicted with a sharp weapon. An injury of this kind may be brought about in various ways,* especially by a saber, bullet, or stab wound or by contusion of the thorax.

If the tissue of the heart itself is injured, the nature of the sequelæ

* Fischer is the author of a careful review of cases of injury to the heart reported up to the year 1867, "Archiv für klinische Chirurgie" (Langenbeck), vol. ix, p. 571. [See also the critical review of Hans Herz, "Centralblatt f. d. Grenzgeb. d. Med. u. Chir.," 1903, pp. 401, 449, 502.—Ed.]

will depend on whether there has been a solution of continuity of the heart-wall. Instant death from cardiac paralysis is very rare in injuries of the heart, only a few well-authenticated examples having been reported. In a great majority of cases, if rupture of the heart occurs, fatal hemorrhage into the pericardium or into the cavity of the thorax takes place. But not always. In cases of stab wounds, the wound may be so narrow as to be occluded by the muscular contraction, and the patient may escape with his life,* although such a result is very exceptional.† Death from hemorrhage is the rule, particularly when the thin portions of the heart-wall are injured; although the end may be delayed for a surprisingly long time.‡

If the heart-wall is merely injured without suffering a solution of continuity, it is wonderful how even a severe wound may heal.§ Hence active surgical intervention is advisable even in the case of penetrating heart wounds. As the chief danger lies in the hemorrhage it is necessary to prevent the filling of the pericardium with fluid under high pressure, which compresses the large venæ cavæ, and, of course, if possible, to guard against any subsequent hemorrhage. The *symptoms produced by injuries to the heart* are extremely variable in degree; they may be utterly insignificant and barely perceptible, or of the greatest imaginable severity. As a rule, the clinical picture is dominated by the general symptoms of the injury—for in many cases other organs are also involved and the accident frequently occurs under conditions of the most intense emotional excitement; but the symptoms chiefly depend on the severity of the hemorrhage. Hence the symptoms of injury to the heart are much the same as those of rupture, the symptoms of which are also chiefly determined by the course of the hemorrhage.

If the injury consists of an *extensive tear of the heart-wall*, the patient dies in a few minutes with the symptoms of acute anemia. But, as in cases of rupture, this is not the rule, the escape of the blood being usually quite gradual even when the opening is relatively large.

On the one hand, this affords time for intervention; but, on the other, the danger persists even after it seems to be past. The prognosis should be given with great caution, for severe or fatal secondary hemorrhage forms an important and most unpleasant feature of injuries of the heart, and also calls for the most active intervention.

In these cases, in which the course is more gradual, the patients exhibit a great variety of cardiac as well as general‖ symptoms. They may be quiet, calm, and apathetic, or, on the other hand, terribly excited. Fortitude in the face of an absolutely certain death is observed side by side with the most frightful terror; in some cases the patient is unconscious; in others, consciousness is perfectly preserved. Dyspnea, oppression, and pain in the precordia and left arm are often observed. Many patients walk up and down or toss about in restlessness and fear; others

*Of these, there are numerous examples reported by Fischer as well as by Hanna, "Occid. Med. Times," July, 1896. Abstract: "Centralblatt für innere Medicin," 1897, p. 12. Reiser, "Wiener medicinische Presse," 1871, No. 39. Abstract: "Virchow-Hirsch," 1871, vol. ii, p. 97.

†Compare the case of Stich, "Archiv für klinische Medicin," vol. xiv, p. 251.

‡Many examples of this have also been reported, as, for instance, by Coats, "Glasgow Med. Journal," vol. xxxvi. Quoted from "Schmidt's Jahrbücher," vol. ccxxxviii, p. 93. Fischer, *loc. cit.*

§Steudener, "Berliner klinische Wochenschrift," 1874, No. 7.

‖See Fischer's excellent description.

lie perfectly quiet. It is a well-known fact that men as well as animals can cover considerable distances after receiving a severe wound of the heart.

To the above picture is added the picture of anemia in other parts of the body; the temperature falls; the pulse becomes soft and small and, as a rule, accelerated; the face is pale and the limbs are cold; symptoms of injury to neighboring organs, especially of the lungs, are also present, and, if the case is protracted, signs of infection make their appearance.

The results of *physical examination of the heart* are variable and often very difficult to interpret because a complicating pneumothorax sometimes completely changes the position of the organs. The presence of blood in the pericardium may produce a large area of dulness. Although percussion, as a rule, is unsatisfactory on account of the severity of the condition, it is, nevertheless, possible, with a little practice, to determine whether hemopericardium is present or not. If air is inspired through the wound in the thorax, hemopneumopericardium results, the clinical picture of which is unmistakable: tympany over the precordia, a metallic note on percussing with the pleximeter, dulness when the patient sits up, metallic heart-sounds, and other ringing sounds synchronous with the heart action. Distention of the stomach lending a resonant quality to the heart-sounds is the only condition for which hemopneumopericardium could be mistaken, and, by taking all the factors in the case into consideration, it should always be possible to differentiate between the two conditions.

The apex-beat varies; it may be diminished or increased in strength; the heart action may be accelerated or retarded. The rhythm is often, but by no means always, irregular, the lack of uniformity in this respect being explained by the variable degree of anemia and by the fact that the injury may either stimulate or inhibit the action of the heart. For the same reasons the heart-sounds are sometimes very feeble, while at others they appear to be exceedingly loud. Systolic murmurs may, of course, occur at any time.

Vomiting and diarrhea sometimes occur, just as in cases of rupture of the heart. If the lung has also been injured, there may be a cough with bloody expectoration.

The force and rapidity with which the blood escapes from the wound in the heart and thorax are quite variable, and again suggest an analogy to cases of spontaneous rupture of the heart. The explanation of these differences is quite obvious: the position, size, and form of the openings; the force of the cardiac contractions; the height of the blood-pressure; and the formation of clots, which may temporarily occlude the wound.

Since the greatest danger in wounds of the heart lies in the severity of the hemorrhage, and since there is danger of severe secondary hemorrhage even when the bleeding at first was slight, wounds of the heart must always be subjected to active surgical treatment. This, of course, requires a positive diagnosis of injury to the heart. The surgeon must know not only whether the injury has involved the organ, but also whether the walls have suffered a solution of continuity.

This point may be very difficult to determine.

A wound in the region of the heart is not a sufficiently accurate diagnosis even when it is associated with profound disturbance of the general condition and of the circulation. The first thing to be deter-

mined in a case of this kind is whether the wound has penetrated the chest-wall. The methods by which this is determined do not come within my province. After it has been definitely ascertained that the pericardium has been opened, two questions remain to be determined: whether the heart has been touched and whether the wall has been torn through.

These points are determined chiefly by the extent of the hemorrhage and possibly by the discovery of a pericardial effusion. The latter may, however, be absent or very slight, even in cases of severe hemorrhage from the heart if the blood can find some outlet to the exterior; and, on the other hand, injury of the mammary or intercostal artery may also cause a profuse hemorrhage. In cases of hemorrhage from the aorta or the vena cava the question of surgical intervention rarely remains long in doubt, for the accident, as a rule, ends in death before a surgeon can be called.

I believe, therefore, that in cases of profuse hemorrhage, whether the blood escapes to the exterior or enters the pericardium, the physician should determine whether there is a laceration of the heart-wall, provided the aorta and vena cava can be excluded as sources of the hemorrhage. But the cases with profuse hemorrhages represent the least favorable group because they nearly always prove fatal. It, therefore, seems to me much more important to close the smaller openings, as it is the only way to guard against severe secondary hemorrhage.

Unfortunately, the diagnosis in these cases is difficult; for how is one to be certain that the heart-wall has been torn if there is not much bleeding? Sometimes inspection may be possible; in other cases the direction of the weapon or instrument that has inflicted the wound may be of assistance. It is also important to find out whether a very large amount of blood was lost at the beginning of the injury. By one of these expedients a diagnosis may be possible in some cases, but in others anything like certainty is out of the question. With regard to the indications for puncturing or incising a pericardial effusion and the advisability of enlarging the wound so as to afford an opportunity for local examination, these are matters to be determined according to general surgical principles, for which I lack the necessary knowledge.

No one but a surgeon is competent to decide whether and when the heart should be sutured. Nor is it my province to speak of the technic of suturing the heart, its technical possibilities and limitations, or, indeed, the justification of resorting to the procedure at all; for I have no knowledge of these matters. A number of cases in recent times have shown that the human heart can be sutured,* although it is true the difficulties are considerable.

All that the internist can be expected to determine in a case of this kind is whether there are any *a priori* objections to the introduction of a suture into the heart; for example, whether the unavoidable manipulation of the organ during the operation would be followed by direct danger to life.

It is obviously impossible to do anything to the heart without some risk, because of the more or less violent movements to which the organ must be subjected in doing so. Physiologists have gained much experience on this point from experiments on animals. From my own observations in numerous operations on the hearts of

* See, for example, Rehn, "Archiv für klinische Chirurgie," vol. lv, p. 315 (1897). Literature.

various animals I believe that a good many things can be done without any serious danger to the action of the heart. But there are exceptions. Sometimes the heart stops beating after what appears to be only a slight touch, and it is found impossible to set it in motion again. Animals vary in this respect. For instance, the dog's heart is much more sensitive than that of the rabbit. Experience gained from animals cannot, therefore, be applied to the human subject without great caution. Under these circumstances it is a fortunate thing that in the human subject the heart can be partially or completely exposed and quite severely handled without doing any great amount of harm. And it may, therefore, be stated that, although it cannot be denied *a priori* that the injury to the heart incident to the introduction of sutures might be followed by alarming symptoms, nevertheless the experience gained from a number of cases indicates that the human heart is endowed with great powers of resistance. Hence in view of the great danger of extensive bleeding heart wounds, and the great advances in the technic of modern surgery, I think one ought strongly to advise exposing the organ and introducing sutures in the case of wounds of the heart. So far the statistics of heart injuries have been very discouraging; barely 10 per cent. of the patients recover.

The expectant treatment of injury to the heart needs no description, as it is based on general principles. Absolute rest must be enforced. The patient is given morphin to lessen cough and relieve pain, and thus keep him quiet. An ice-bag or Leiter's coils should be applied to the precordia. Venesection is recommended by some authorities on the ground that it reduces the blood-pressure and thus diminishes the bleeding from the heart wound. As a matter of fact, it has repeatedly been observed that venesection is followed by improvement in the arterial pulse. This may be because the fluid in the pericardium is more quickly absorbed when the venous pressure is lowered, and the compression of the large veins is thus diminished. But I do not think it is likely; for the rise of arterial pressure is too rapid, and diminution of the amount of blood in the large veins should favor, rather than diminish, compression. However that may be, it has been frequently observed that venesection in the case of hemopericardium is followed by improvement in the circulation.

For all that, I would enjoin great caution in the employment of venesection in cases of this kind because the chief danger from the very outset lies in the loss of blood. A better means to produce an immediate improvement in the circulation would be, in my opinion, evacuation of the pericardium by puncture or incision. It is true that this tends to favor a fresh hemorrhage; but the management of these cases must be determined by experiences at the bedside and not by theoretic considerations.

Foreign bodies not infrequently enter the heart from the outside and remain embedded in the viscus without causing much damage. Isolated cases have been reported of penetrating wounds by needles, swords, or daggers; the patient at first presents practically no morbid symptoms, and the fatal hemorrhage does not begin until the position of the weapon is changed by some movement of the heart or it is withdrawn from the outside.

A bullet embedded in the heart sometimes proves quite innocuous. Still more frequently foreign bodies, such as needles and thorns, for example, which get into the heart by way of the esophagus, may remain without inflicting any damage. This is not difficult to understand, for the progress of the foreign body through the tissue of the heart is so slow that there is time for inflammatory processes to close up the track behind it; thus hemorrhage is avoided, and with it the chief danger incident to the entrance of foreign bodies.

In another class of cases * heart symptoms may develop after more or less grave injuries of the precordia or the adjoining parts, without any solution of continuity in the skin or exposure of the heart. The injury usually is a contusion of the thorax or a momentary pressure of some solid body against the lower half of the anterior surface of the thorax, brought about either by collision with a hard body or by some heavy object falling on the chest. In these cases either the symptoms of impaired heart power develop, or rupture of the heart-wall may take place. The mechanism of rupture is probably essentially the same as when rupture of the thoracic organs is caused by hydraulic pressure. Küster † has made a painstaking investigation of contusions of the kidney; gunshot wounds of various organs have been studied by Kocher,‡ and it seems evident that rupture is the result of bursting caused by great pressure on incompressible organs filled with fluid. Thus Peacock,§ for example, reports a case of rupture of the right ventricle in a drunken man who had received a blow in the epigastrium; Robertson ‖ observed rupture of the left auricle after a fall on the shoulder. It is said that a fall of this kind may cause rupture of a sound heart, whereas so-called spontaneous rupture occurs only in diseased organs, as has been explained on a previous page (p. 712 **.)

These are rare accidents, however. **Contusions of the thorax** much more frequently lead to chronic processes in the heart. I do not here refer to the disease of the endocardium and of the valves to which such injuries give rise, since we have to do only with the heart muscle.

A number of cases have been reported in which an injury of this kind was followed by the symptoms of chronic heart weakness for which no other cause but the traumatism could be found.†† The case histories need not be given in full; the patients presented the well-known picture of chronic heart weakness, such as develops chiefly after myocarditis: dilatation, disturbance of the heart action, passive congestion of the lungs and systemic circulation with general loss of strength. There can be no doubt that chronic cardiac insufficiency may result from an injury, although the mechanism is difficult to understand and explain, particularly as we still lack sufficiently accurate postmortem data. The symptoms observed during life would seem to indicate a probable inflammation of the muscle, which might be thought to be of an infectious nature and to owe its origin to germs contained in the systemic circulation that have found a resting-place in the damaged tissues of the injured organ. Viewed in this general light the process is not without analogy. Another theory is that the injury may be attended by laceration of the heart substance and hemorrhage, around which the well-known reactive in-

* Heidenhain, "Deutsche Zeitschrift für Chirurgie," vol. xli, p. 286. Fischer, Schuster, Stern.

† Küster, "Die chirurgischen Krankheiten der Nieren," first half: "Deutsche Chirurgie," Lieferung 52 b, vol. i, p. 187. *Ibid.*, "Verhandlungen der Deutschen Gesellschaft für Chirurgie," 1895, vol. xxiv, p. 368.

‡ Kocher, "Bibliotheca medica," Part E, Section 2, Kassel, 1895.

§ Peacock, "Trans. of the Path. Society," vol. xxxi, p. 72. Abstract: "Virchow-Hirsch," 1881, vol. ii, p. 138.

‖ Robertson, "Lancet," January 23, 1897. Abstract: "Centralblatt für innere Medicin," 1897, p. 809.

** Other cases are cited by Riedinger, "Deutsche Chirurgie," Liefer. 42, p. 179.

†† Stern, *loc. cit.* Hochhaus, "Archiv für klinische Medicin," vol. li, p. 10. Riegel, "Zeitschrift für klinische Medicin," vol. xiv, p. 328. Heidenhain, "Deutsche Zeitschrift für Chirurgie," vol. xxxix, p. 286.

flammation takes place. As regenerative processes in the heart muscle occur only to a very limited extent, healing takes place chiefly by scar formation.* It is a matter of observation that traumatism may lead to laceration and hemorrhage into the heart substance,† so that the occurrence of compensatory inflammation is readily understood. The difficult thing to understand is that chronic progressive processes are said to develop, and the course of these traumatic diseases of the heart muscle, so far as can be judged at present, is also progressive. This again suggests the idea of an infectious process.

The question is exceedingly complicated. Besides, when a patient suffers a severe contusion of the chest he either purposely, in the attempt to ward off the injury, or involuntarily, makes violent muscular exertions; these efforts may lead to heart strain which, of course, is capable of greatly diminishing the functional power of the heart muscle.

Cardiac disturbances, which the prevailing views include among the nervous affections, may also be brought about by injuries. Among these we may include the nervous excitement which accompanies heart wounds or contusions and which has a special influence on the heart. In many of these cases the traumatism itself produces disturbances of the heart action, although the organ may not have been directly injured in the manner above described. This is inferred chiefly from the nature of the symptoms. The latter are identical with so-called "nervous heart" symptoms, and, like these, are frequently associated with abnormal conditions of the vessels. For a description of these symptoms the reader may, therefore, be referred to the respective section, where it was shown that the nervous diseases of the heart are brought about chiefly by psychic factors. When heart symptoms develop after an accident, they are also chiefly due to the psychic insult—that is, they form a part, and indeed are essentially an expression of, a traumatic neurasthenia, hypochondriasis, or hysteria.

It is unimportant whether the injury has affected the heart directly or not. Any kind of injury may produce nervous heart symptoms if the injury gives rise to the form of excitement which causes such symptoms; and whether or not such excitement is brought on depends very much on the circumstances attending the injury and the constitution of the individual. In many forms of traumatic "neurosis" the heart symptoms are particularly troublesome.

Although we have every reason to attribute heart symptoms of this character to some injury to the nervous system, and although the psychic element is unquestionably the most important factor in the etiology, it would be wrong to deny that the shock to the entire organism which accompanies so many grave injuries is in part responsible for the cardiac affection. The morbid processes which accompany concussion of the brain prove that many portions of the nervous system are sensitive to certain special movements of the body. Some such disturbance may, of course, have to be taken into consideration in the conditions here under discussion, but, unfortunately, our knowledge of these matters is still very imperfect.‡

Before the treatment of this form of neurosis is begun it is desirable to settle the question of damages, according to the laws that regulate [accident—Ed.] insurance. [Very thoroughly arranged in the German

* Bonome, "Ziegler's Beiträge," vol. v, p. 267.

† Such cases are cited by Stern, p. 37.

‡ Compare Riedinger.

Empire.—Ed.] (See the following section.) In other respects the treatment is exactly the same as that of traumatic neurasthenia and of nervous heart symptoms in general.

The statement has often been made that so-called *nervous disturbances of the heart due to a traumatic cause are sometimes converted into organic lesions*, and some cases have, in fact, been reported which appear to support the statement.* Such an interpretation of these cases is, however, hardly justifiable. In two cases Stern showed that arteriosclerosis was present. Indeed, the symptoms of beginning coronary sclerosis are often identical with the symptoms of nervous heart disease. Moreover, the injury itself and the emotional excitement associated with it have a marked influence on the development of arterial changes. Thus the problem is an exceedingly complicated one. The same conditions must be taken into account in making a diagnosis. The great difficulty of distinguishing between myocarditis and coronary sclerosis, on the one hand, and nervous diseases, on the other, has been referred to on several occasions, and all that is known on the subject has been stated. In exactly the same way these conditions have to be differentiated from disturbances due to traumatism.

THE MEDICOLEGAL SIGNIFICANCE OF HEART DISTURBANCES DUE TO INJURY.

In giving a medicolegal opinion on a patient who attributes heart symptoms to an accident, it must first be proved that the symptoms have developed subsequent to and immediately following the accident. The physician can help to decide this point in two ways: by taking an accurate history of the case and by giving his opinion that the accident in question is capable of producing the symptoms and conditions from which the patient is suffering. It is the duty of the courts to determine the patient's condition in regard to the ability to do work before the accident occurred and also the nature of the accident itself. If the physician can obtain the results of a good medical examination made shortly before the time of the accident, a great many difficulties are obviated.

As a rule, it will be found that the individual was in good health before the accident occurred, and in the foregoing pages an attempt has been made to show what kinds of disturbances of the healthy heart can be brought about by external causes. Occasionally, however, the heart is already diseased when the accident occurs, and if it can be shown that the functional power has been still further diminished by the injury, the patient is also entitled to an indemnity; this point has been definitely decided by the courts. It is obviously very difficult to give a medical opinion on this point; it is much easier to decide whether an accident has produced a disease in a previously healthy individual than it is to show that a heart which was already diseased has been rendered worse by the accident. The difficulties are peculiarly great in the case of an organ like the heart, because we are still chiefly dependent on the patient's own statements in estimating its functional power. Such being the case, it will often be found quite impossible to give a positive opinion.

* Compare Oppenheim.

The idea of overexertion introduces certain special complications. According to the prevailing views [of German accident insurance laws —Ed.] accidents are made to include * "abnormal bodily exertions occurring either once or several times in quick succession when accurately and positively proved to have taken place," and which have caused acute dilatation of the ventricles of the heart, followed by immediate disability. This is an important point; for accidents are not infrequently attended by severe muscular exertion, and, on the other hand, it is not always easy to decide from a purely medical standpoint whether heart symptoms are due to violent exertion on the part of the patient or directly to the injury.

Moreover, any excessive exertion occurring in the manner just described is regarded as an accident, even if the heart was previously diseased. This is perfectly obvious, since it is generally admitted that, if it is proved that an already existing disease has been rendered worse by an accident, the patient is also entitled to damages under the law. It is an important practical point because it is positively known that an already diseased heart is much less tolerant of severe muscular exertion than a sound heart. In actual practice it all depends on the scope of the term "overexertion," which is undoubtedly extremely elastic—the question is, how elastic?

This depends upon legal precedent, and Stern quite properly calls attention to the significance of a decision given by the Imperial Insurance Department (Reichsversicherungsamt), according to which a "severe and unaccustomed exertion" in a workman with heart disease was reckoned as an overexertion. This decision shows, once for all, that the idea of overexertion is relative and varies according to the constitution of the "injured" party.

Profound psychic impressions are regarded in very much the same light as violent muscular overexertion. Such impressions are undoubtedly capable of severely injuring the heart, and may even bring on heart failure. In such cases the existence of an "accident" as defined by the law has also been recognized, and it must be remembered that those who suffer from various kinds of heart disease are peculiarly sensitive to psychic impressions. Cases in which heart patients have been injured by impressions of this kind have, in fact, been regarded as due to accidents.† It is, therefore, the physician's duty to make a very accurate examination and to give a clear exposition of the state of affairs, which, of course, is often very difficult to determine. In this way his services may be of great value to the patient.

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Case History.—E. H., thirty-one years old, cooper. Formerly in good health. On the sixth of September, 1898, the patient was injured by a large piece of wood which was hurled against his chest by a machine. When he received the blow, B. made a great effort to hold on to a table, but the force of the impact threw him to the ground. Severe swelling of the breast developed, with pain. Three days after the accident, when B. tried to return to work, he was unable to do so on account of pain in the chest. Palpitation and anxiety were superadded. The patient has had to give up work altogether since the accident; on one occasion he managed to work for a week, but had to give it up again at the end of that time. He says a general condition of nervousness has developed as well as pain in the chest, which he cannot describe.

Status præsens: May 31, 1899: The patient is in a state of anxiety and excitement. Slight cyanosis. Apex-beat in the fifth intercostal space, extending to the nipple-line, soft and not high. There is also pulsation in the fourth intercostal space. Absolute heart dulness extends almost to the nipple-line; the relative dulness is not enlarged (in December, 1898, I found a moderate, although quite distinct, enlargement to the right). First sound at the apex impure; second pulmonic sound accentuated. Pulse 108, regular, uniform, of moderate volume and tension.

The lungs and abdominal organs present nothing abnormal; no edema; urine negative.

A FEW REMARKS ON THE HISTORY OF DISEASES OF THE HEART.

I shall not venture to give a review of the history of diseases of the heart. It is, of course, inseparable from the history of the general views entertained in regard to the functions of the healthy organism and the nature of disease in general, for man is so constituted that his observations depend in a large measure on the mental attitude which he brings to the examination or, in other words, on his previous knowledge and his method of reasoning. If this is true of observation, how much more is it true of interpretation? That is to say, any scientific discussion of any subject whatsoever by a man—be he ever so gifted—cannot be judged as a thing independent and apart; it becomes intelligible only in the light of the period to which it belongs—in the light of *all* the scientific views entertained during that period.

Any one can convince himself of this by observing his own mental processes, and it is also shown conclusively by a cursory review of history. For example, we are unable either to understand the recorded observations of physicians who lived eighty years ago or to make use of them for a modern analysis unless we are perfectly familiar with the general physiologic and pathologic views entertained at that time; and not only these, but the philosophic views as well,—at least in part,—for the old clinicians were naturalists, and naturalists in former times were also philosophers.

How difficult many of us find it to understand the views of naturalists who showed Schelling's ideas! "They are false, and should be ignored," the theorists might object, not without reason. But we clinicians want not only the opinions, but, above all, the observations, of the older physicians. They were clear-sighted. Perhaps more clear-sighted than we are, or at least they were less liable to be led astray, because their methods of examination were much simpler and more limited in number, while the need of helping the patient was equally great. Their observations are unquestionably valuable, provided they can be interpreted. But before we can deduce anything from them, we must understand the ideas on which they were based; and the better we are able to interpret them, the more shall we be able to deduce from them. The difficulty obviously becomes greater in proportion as the observers and the period to which they belong are further removed from us.

My insight into these conditions is as yet too incomplete to permit me to attempt anything like an effective historic treatment of the subject, particularly as it would have to include the literature and prevailing opinions of Germany, Austria, Italy, France, and England, not to mention the lore of the Arabians and the ancients. The writings should, if possible, be read in the original, and with something of the same spirit that has enabled our modern students of history, by a critical study of sources, to make such extraordinary progress in their department, unless we wish to remain a hundred years behind them.

From every hand is heard the demand for historic study, and not without good reason. But it is by no means a simple matter; it is certainly much more difficult than is generally supposed. Unless we go to the original sources instead of forming our opinions from historic reviews, we shall not gain correct ideas. We shall merely deck ourselves out in false jewels, which will deceive none but the inexperienced and will be of no benefit to science.

I trust, therefore, that my apparent neglect of the history of heart disease will not be attributed to my want of appreciation of its importance, but solely to the fear that what I might say would be of no value.

A review of the doctrine of heart diseases from ancient times to the end of the seventeenth century has been written by Testa and Sprengel,* and the development of the subject during the eighteenth century is reviewed by Philipp.† The advances made during the past century are abundantly discussed in the various text-books on heart diseases and in original papers; for it has now become the fashion, and is considered good form, to display historic knowledge and prove one's possession of it by reporting suitable cases. The great majority of these historic

* Testa-Sprengel, "Herzkrankheiten."

† Philipp, "Die Kenntniss der Krankheiten des Herzens im XVIII. Jahrhundert," Berlin, 1856.

studies do not appear to me to emanate from any true knowledge of the matter or to reveal any great judgment in the writer. It is to be hoped that a comprehensive review for the nineteenth century will appear at some future time.

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DISEASES OF THE PERICARDIUM.

BY

L. v. SCHRÖTTER, M.D.

DISEASES OF THE PERICARDIUM.

ANOMALIES.

Among the diseases of the pericardium, inflammatory affections occupy by far the most prominent position, as I shall show in detail further on. The anomalies of the pericardial sac present but little of clinical interest.

Absence of the pericardium has been noted in a number of cadavers, including some bodies of old persons (in one case, over sixty years of age), but in no instance did this anomaly as such produce any symptoms. The heart was found simply to lie in the left pleural cavity. Possibly inflammatory processes may be demonstrable in some cases. An additional anomaly, observed in such a case by Chiari, consisted in an abnormal position of the phrenic nerve on the left side, which was found in front of the heart. Even in this case no symptoms were observed.

The *formation of diverticula* in the pericardial sac is of greater importance. Hernial protrusions of the serous layer of the pericardium through apertures or defects in the fibrous layer may occur, may assume various sizes, and may be either single or multiple. I have observed such an anomaly but once; namely, a diverticulum of the size of a hen's egg, which protruded into the right lung and was filled with serous fluid. This anomaly had not caused any symptoms during life, as it was covered by the lung, but the patient had been suffering from a pericarditis which had followed Bright's disease. The specimen may be found in the Museum of the Pathologic Department at the Rudolf Hospital.

These diverticula are, doubtless, congenital in most cases, but it is possible that they may be formed in the course of an old pericarditis with a moderate degree of exudation as the result of strong pressure upon gradually yielding walls.

PERICARDITIS.

Causes.—The two varieties of this affection which we still distinguish at the present time are the primary, or idiopathic, and the secondary form. Among the *primary cases* there have been rare instances in which the pericarditis was the only discoverable lesion in the body, and in which even a careful investigation of the patient's history gave no hint as to the persistence of lesions of other diseases, nor as to the presence of a predisposition to other maladies; such as, for example, tuberculosis. The mode of origin of such a primary pericarditis cannot be stated with certainty at the present time. It is doubtful whether exposure to cold, so frequently cited as a cause, plays a rôle of importance in most of these cases, and the presence of an infection cannot be proved in all instances. In fact, in some cases on record no germs whatever could

be isolated by the methods at our command at the present time. In contrast to this, we may cite the observation of Foureur, who reported a case of purulent pericarditis in which the pus contained the Streptococcus pyogenes, although no other purulent focus could be found in any part of the body. In this instance, the Streptococcus pyogenes must be regarded as the sole cause of the primary pericarditis. It is possible that a primary pericarditis may be due to the invasion of the pneumococcus, all the more so as a secondary pericardial inflammation due to the same germ has been observed, and as the researches of Weichselbaum have shown that meningitis and pleurisy may be due to the germ of pneumonia.

The cases of primary pericarditis which I have had occasion to observe were characterized by a moderate amount of exudate. According to Weichselbaum, the pneumococcus produces an exudate of rather fibrinous character, and, therefore, in my cases it is probable that other germs were at fault. That traumatism may produce inflammations in the pericardium cannot be denied. I may cite especially those forms which give rise to the so-called *maculæ tendineæ*, and it is possible that we may include here also certain ill-defined affections in which no other explanation can be given for a heart murmur than the presence of a pericarditis. A definite statement cannot be made as regards these (traumatic) types of pericardial inflammation, inasmuch as there are as yet too few reports of autopsies in such cases. Personally, at least, I have never had the opportunity to see such a case at autopsy.

Pericarditis is much more frequently seen *as the sequel of some other disease*. No definite statistical data as to the frequency of this form of pericarditis can be given at present; for, in the first place, we must agree as to whether we shall count the cases seen at autopsy, or those seen during life. Another difficulty is the question as to whether or not the maculæ tendineæ should be regarded as lesions of pericarditis. In general, however, I think the figures given by Duchek are approximately correct; namely, that there are 15.1 cases of pericarditis among every 100 cases. [Duchek's results were drawn from an autopsy experience of 590 cases. Pericarditis was present in 89. Among the living the proportion is smaller. Lockhart Gillespie found pericarditis clinically in 5 per cent. of 2368 patients with heart disease.—Ed.] The disease occurs at all ages, and is probably more frequent in men than in women.

If we consider *the causes* in the order of their frequency, we must name, first of all, *acute articular rheumatism*. Ever since Pitcairn showed the connection between acute articular rheumatism and pericarditis, in 1788, we have known that a pericardial inflammation may occur in any form of this disease, and at any stage thereof. In fact, as others as well as myself have observed, rheumatism may begin with a pericarditis and gradually develop the changes in the different joints characteristic of the disease. The conclusion which may be drawn from this is that pericarditis, like endocarditis, belongs to acute articular rheumatism—in other words, that the changes in the pericardium are produced by the same cause as rheumatism—by the hypothetic micro-organism which has not yet been found. The objection against this theory, that if this were so, pericarditis would occur more frequently than it actually does in rheumatism (it occurs in 30 per cent., as I have also found) is not valid, because we find in many other diseases that not all the symptoms must necessarily be well marked, and because in many cases an endo-

carditis, which may be said to be the equivalent to the pericarditis often takes the place of the pericardial inflammation.

The exudate (in these rheumatic cases) is in most instances serous or serofibrinous in character, but it may be quite purulent, and in this form it may be very abundant, as I have seen in a robust young woman of twenty-four years of age, in whom the exudative processes which developed as the result of a severe rheumatic polyarthritis necessitated the puncture of first one pleural sac, then of the other, and finally also of the pericardium. In all three cavities the exudate was quite purulent. A mixed infection is probably responsible for the exudate which consists of pure pus.

Next in frequency is that form of pericarditis due to the extension of changes which accompany *tuberculous lesions in the lungs* and the pleura. I am here speaking of the purely inflammatory processes only, and shall refer later in detail to tuberculosis proper of the pericardium. In these forms the exudate may assume any of its subtypes, but it is very frequently purulent.

Next in order of frequency, in my opinion, is the pericarditis following a *pneumonia* or *pleuropneumonia;* for these cases are far less common than is generally supposed. The results of autopsies, which alone should be regarded as a criterion in this connection, show that a pericarditis occurs but rarely in the wake of the diseases mentioned. [Osler found pericarditis clinically in 5 per cent. of cases; at autopsy in more than 15 per cent., in pneumonia.—Ed.] Clinical observations must be given that interpretation which is warranted by our present knowledge of this subject. We must remember that not every friction-sound which is heard over the heart after a left-sided pleuropneumonia necessarily indicates a pericarditis, but may depend upon the pleurisy itself, as we shall see more in detail later on. For this reason the official statistics in which all these things are confused have no value, and such questions can be solved only by special studies, as I have shown elsewhere.

In contrast to this we must cite the statement of Weichselbaum, who believes that pericarditis occurs as the sequel of pneumonia much more frequently than is generally supposed. This author supports his opinion by the fact that even in cases in which the pericardial fluid was perfectly clear, he could demonstrate the presence of the pneumococcus in cultures prepared from the exudate. In spite of this, I am inclined to adhere to the opinion which I have expressed above.

According to Weichselbaum's investigations, a pericarditis which occurs during the development of a pneumonia is due to the pneumococcus. The pericarditis may be said to belong to the pleuropneumonia, or may be regarded as a less frequent localization of the pneumococcus-infection, which, as a rule, produces inflammation of the lungs. On the other hand, Banti has found nothing but the Staphylococcus pyogenes aureus and albus, and no pneumococci in the pericardial exudate of pleuropneumonia, although the pneumococcus was found in the lungs in these cases. The exudate in these (pneumonic) cases is usually serofibrinous or seropurulent, rarely entirely purulent.

Among the other infectious diseases which may cause pericarditis we must mention, first of all, *scarlet fever.* It is probably true that pericarditis occurs in those forms of this disease in which the joints are involved; and yet, pericardial inflammation may be observed without any joint-involvement, and, for the present, we must leave undecided

the question as to whether the pericarditis in these patients arises as the result of the scarlatinal infection or of other factors. According to the investigations of Kolisko upon the large material of the Children's Hospital, the etiologic factor in these cases is an invasion of the streptococcus which enters by way of the angina.

In *measles* pericarditis is very rarely seen; the same is true of *typhoid fever* and *erysipelas*.

In *small-pox* pericarditis may occur, probably not from the action of the specific poison of the disease, but as the result of the pyemic processes which accompany this affection. An inflammation of the pericardium, with seropurulent or purulent exudation, is observed in general *in all pyemic conditions*, especially in those of *puerperal origin* [and including gonorrhea—Ed.].

It is well known that various types of pericarditis not infrequently occur in the different forms of Bright's disease. The exudate which occurs in these cases is not always a serous one, and I have seen a number of instances in which it was fibrinous or serofibrinous.

In the so-called *hemorrhagic diathesis*, especially in *scurvy*, I have seen moderate amounts of bloody exudate, and the same type of effusion occurs in debilitated persons, especially in drinkers with cirrhosis of the liver, in general, after severe and prolonged diseases.

Pericarditis may occur through a direct *extension of the inflammation from neighboring organs*, especially from the heart, in diseases of the myocardium with superficially situated pyemic or mycotic abscesses. A striking case of the last-mentioned type has been described by Eberth.

Localized or diffused pericarditis may occur as the sequel of multiple or single *aneurysms* of the ascending portion of the aorta or of aneurysms of higher portions, which exercise pressure against the heart. We must be cautious in interpreting the manner in which such pericardial inflammation develops, for in these cases we have to deal surely not only with the phenomena of pressure and stasis in the vessels of the heart, but in some cases we must undoubtedly also ascribe the origin of this pericarditis to the same cause which has led to the formation of the aneurysms—namely, infectious processes. This seems to be supported by the fact that we not infrequently see an almost complete obliteration of the pericardial sac, while the origin of the aneurysm appears to be more recent.

In a similar manner pericarditis may arise as a sequel of *endocarditis* by the extension of the inflammatory process from the endocardium. But here, also, both lesions may be due to the same cause, and the same view must be taken if we admit that an endocarditis may develop from a pericarditis.

We have already mentioned the development of pericarditis by *extension from the pleura*. The greater frequency of pericarditis in left-sided pleurisy shows that in these cases we are actually dealing with a direct extension, which is favored by the greater surface in contact with the heart on that side.

Caries of the sternum, of the *ribs*, and less frequently of the *vertebræ* may lead to a pericarditis which is then accompanied, as a rule, by a purulent exudate. Among the diseases of the mediastinum, cancer of the esophagus is the most frequent cause of pericarditis, as I have observed in several instances. In these cases the exudate was serofibrinous or purulent, and in one instance, in which a perforation was about to take place, it was gangrenous. Other tumors of the mediastinum, arising

from the lymph-glands or from the remains of the thymus gland, may lead to pericarditis.

I have also seen several instances in which the severe *phlegmonous inflammations in the neck* extended along the cellular tissues of the mediastinum to the pericardium, giving rise to a purulent exudate in the latter. In view of the numerous communications between the thorax and the abdominal cavity, it is not astonishing that inflammatory processes from the various organs of the abdomen may extend to the pericardium. Thus we find such a series of events in *abscess of the liver*, in *ulcer of the stomach*, in *new-growths*, in *echinococci*, etc., in which there is either a direct extension of the inflammation, or indirectly from multiple encapsulated foci. Any form of *peritonitis* may lead to a pericarditis, and the communications are very often exceedingly complex in these cases; as, for example, in the peritoneal changes produced by cholelithiasis.

Finally, a variety of *injuries* may lead directly or indirectly to a primary or secondary pericarditis, according as to whether the source of the inflammation penetrates directly into the pericardium, or gets there in a roundabout manner. The former event takes place in a direct wound; the latter, when the foreign bodies which penetrate into the organism wander into the pericardium from elsewhere, especially from the esophagus. I fear that the latter form occurs more frequently than is generally supposed; for it is not very rare to see, even today, that injudicious manipulations upon foreign bodies which have become lodged in the esophagus produce a mediastinopericarditis, generally with a fatal termination.

Pathologic Anatomy.—The pathologic changes which occur in pericarditis and which are interesting from the clinical viewpoint include the alterations on the surface of the pericardium, the presence of the fluid exudate, and, finally, changes in the condition of the heart muscle.

Furthermore, it is of clinical importance to remember that the anatomic process in pericarditis is fairly complex. It may involve only the serous layer, either diffusely or over a small portion; or else the fibrous layer as well, or the latter alone, or this and the neighboring pleura, or it may extend from the epicardium upon the heart and the large vessels. Finally, just as is the case in pleurisy, the inflammation may pass from the fibrous layer of the pericardium to the wall of the thorax or to the cellular tissue of the mediastinum, and in these circumstances may give rise to both suppuration and to those fibrous changes which are known as pericardiomediastinitis. One can understand the varying pictures and the occurrence of certain special phenomena in the complex of symptoms of pericarditis only by remembering that so many different forms of this affection are possible. Thus, for example, is explained the prominence of the thoracic wall; the varying symptoms observed on the part of the heart, etc. All degrees of inflammation may occur in pericarditis, and I even have no doubt that very often there is only a congestion, as, for instance, in articular rheumatism. In such cases there can be only a distention of the vessels, a diffuse reddening of the pericardium, with scattered ecchymoses.

The next degree is characterized by a loss of endothelium, which may be recognized by a dull, lusterless, and "dusty" appearance of the serous membrane. Then there comes a delicate film of easily detachable fibrinous exudate, which increases in thickness until it becomes a layer

of considerable depth, which may cover the entire heart and the entire inner surface of the fibrous layer. The appearance of the surface of the heart at this stage varies, as we may easily understand, according to the movements of this organ against these masses, which are still soft. A variety of designations have been adopted for these appearances, according to the presence of warty, shaggy, or furrowed formations: cor villosum, hirsutum, tomentosum, etc.

The classic comparison of these lesions with the appearance of two pieces of bread and butter torn apart cannot be improved upon. I may state, as a matter of literary interest, that the comparison in this form did not originate with Laennec, as has been generally and erroneously asserted. This author, who described pericarditis so well that scarcely anything of importance remained to be added, said:* "The appearance of the surfaces resembles completely that which would be presented by two slabs of marble which had between them a pretty thick layer of butter, and which then had been forcibly separated from each other by the process employed in the experiment with the Magdeburg hemispheres."

I shall only add a few words concerning the horizontal furrows: These presuppose a special movement of the heart which has been described by Kornitzer, who was confirmed by Skoda. According to Kornitzer, the heart during each systole turns upon its vertical axis, as the result of the lengthening of the two large vessels, which produces an unwinding of the spiral twist which these two structures form around each other. Skoda, with his exceedingly sensitive ear, confirmed this theory by the observation that, in these cases, he could not hear the pericardial friction in the ordinary manner, moving up and down, but purely transversely. The fact that this type of friction is relatively rarely heard is only calculated to confirm the correctness of this explanation; for Kornitzer has already drawn attention to the fact that the twisting motion of the heart is not equally great in every individual. Therefore, in a person in whom the twisting motion is greater, all the other mechanic conditions for the development of horizontal furrows must be fulfilled also.

The microscopic examination of vertical sections also shows a variety of degrees in the inflammatory process. After the endothelium has been shed, the serous membrane is coated with a layer of fibrillary fibrin, which contains numerous leukocytes and some red blood-cells, the vessels showing marked hyperemia. Or there is an abundant accumulation of round-cells between the thicker layers of fibrin; or there is young, newly formed connective tissue, which grows from the serosa, with the formation of new capillaries. In this the round-cell infiltration is more or less deep, and may penetrate into the muscles—a fact which is of special importance from the clinical viewpoint.

While a complete restitution to the normal is possible in the milder degrees of the inflammation, the healing of the last-mentioned type of the process is followed by the formation of a more or less uneven scar-tissue. We shall mention more in detail further on the possible adhesion of the opposed surfaces of the pericardium which may take place under these conditions.

The last-mentioned form of plastic inflammation must also develop in a chronic and insidious manner. The remains of pericardial inflamma-

* "Traité de l'auscultation médiate," etc., tome iii, Paris, 1837, p. 366.

tions, and even extensive adhesions between the layers of pericardium, have been found on autopsy in the bodies of persons whose histories were well known and who had never shown the signs of an acute pericarditis. In such cases the process was very slow and developed without any clinical signs, just as chronic inflammation in the pleura will do at times.

With this is associated the formation of the so-called "tendinous spots." More or less extensive patches of milky cloudiness, consisting of cicatricial connective tissue in the serosa and subserosa, are observed upon both surfaces of the serous pericardium, especially upon the anterior surface of the right portion of the heart. Their origin has been attributed to a variety of causes. Where they show unevenness or thread-like fringes it is very probable that they are the product of an inflammatory process —residues of an acute or an insidious pericarditis. But inasmuch as in the majority of cases they present a smooth surface and do not show any other characteristics of an inflammatory process, it is possible that they are pure hypertrophies of tissue, which possibly are due to local congestions along the vessels, just as such thickenings have very often been observed in various parts of the peritoneum, especially on the spleen. It is possible that in some cases slight injuries affecting the chest-wall may be the cause of these spots, and in favor of this may be advanced the frequency with which they are observed upon the anterior surface of the heart, which is not protected by the lungs. These tendinous spots have no great clinical interest, as we shall see later, because their surface is perfectly smooth. [See Herxheimer, "Ueber Perikardknötchen u. Sehnenflecke," "Virchow's Archiv," 165. "Ziegler's Beitrage," 32. "Zentr. f. path. Anat.," 1903. Ribbert, "Virchow's Archiv," 147.—Ed.]

In addition to the forms of pericarditis described thus far,—which are known under the names of "fibrinous" or "dry" pericarditis, because the lesions which characterize them are very similar to those which occur in inflammation of the pleura,—there are also types of pericardial inflammation in which the exudate is fluid. This fluid exudate is often present in considerable amounts; is rarely purely serous and perfectly clear, but usually contains larger or smaller flocculi of soft or tough fibrin, or else is clouded to a greater or lesser extent by desquamated endothelia or by the admixture of pus-cells, so that the exudate is called serofibrinous or seropurulent. But a quite purulent exudate occurs also in very large quantities at times; amounts up to one liter are not rarely seen, and I have personally observed a case, in a patient with articular rheumatism, where there were two liters of wholly purulent exudate. It is astonishing how the pericardium can resist the pressure of this enormous amount of fluid, although, it is true, in most of these cases, the parietal layer of the pericardium is thickened and coated with a fibrinous exudate.

Ulcerations or small abscesses have also been noted in the fibrous portions of the pericardium, and may lead to a perforation outward, as in "empyema necessitatis," although very much less frequently than in the latter.

As the result of the rupture of very small, usually newly formed vessels, a hemorrhagic exudate is formed, which may contain a slight admixture of blood or may be composed of almost pure blood. In these cases the solid portions of the inflammatory product, especially the deeper layers of fibrin, are saturated with blood.

Gangrenous exudates occur under quite particular conditions. Apparently the simple contact with gangrenous foci, without any actual perforation, suffices to produce a gangrenous change in the exudate. The origin of this change is perfectly clear in cases where there are open communications with gangrenous foci in the lungs, with sloughing cancers of the stomach, etc. Opinions are still divided at present concerning the possibility of a spontaneous development of gas from an existing exudate.

I have already discussed in detail the particular diseases in which the various forms of exudate are most frequently met with.

But we must consider a little more closely the behavior of the heart muscle, inasmuch as this is of special clinical importance. Virchow was the first to show how extensively the heart muscle may take part in a pericarditis, and not only in the chronic form, but also in a perfectly acute case. A fatty degeneration of the muscle-fibers of the heart may occur, either localized in large or small foci, or involving the entire organ, beginning at the surface and passing inward. In the superficial layers there may also be a purulent infiltration of the interstitial tissues. It is clear that the contractile power of the heart muscle must suffer through these changes and that, therefore, exhaustion is readily brought about, as the result of which a dilatation of the heart develops. At the beginning of the disease, however, when the process has not progressed very far, the muscle may regain a considerable portion of its power and may even react with a partial or total hypertrophy.

We have already mentioned that the inflammation may possibly pass on even to the endocardium. But, in addition to this, the large vessels at the base of the heart may also take part in the disease, the exudate soaking through their walls, or inflammatory changes penetrating clear into the media. These pathologic processes are also of considerable clinical importance.

Symptoms.—There is scarcely another disease which presents so variable a picture as pericarditis. At times it runs its course without any symptoms whatever; in other cases it leads to the most severe clinical manifestations, while in still other instances it may begin with mild symptoms, and only in its later stages dangerous accidents appear, often with great suddenness.

It is just on account of this variable picture that one finds it impossible to present a satisfactory classification of the various forms of this disease. Even the distinction between an acute and a chronic form is rather worthless clinically, as the transition is, in general, very gradual, without being marked by any special phenomena. The symptoms of the chronic form may also be marked in the acute stage, so that we cannot obtain objective data for the distinction of the two forms. One of the chief causes for the great variability of the manifestations of pericarditis lies in the fact that, as I have stated, the disease is usually secondary to other affections with which it is combined and which may give similar symptoms, for example, coëxisting affections of the heart, the pleura, etc. The variations in the clinical picture depend chiefly upon three factors: The amount of exudate; the condition of the heart muscle (especially), and, finally, the possible primary disease. The quality of the exudate is only of secondary importance in its effects upon the clinical picture. While there is an entire series of symptoms which are met with in many of the cases, yet only those which are brought

out by physical examination are characteristic of the disease; only with the aid of these is it possible to make the diagnosis with certainty.

For these reasons I refrain from giving a general description of the disease, but shall begin immediately with a discussion of the individual symptoms, and, on account of its especial importance, shall take first the physical examination.

Inspection of the anterior wall of the chest will in most cases show no deviation from the normal. Precordial prominence (*Hervorwölbung, voussure*) is certainly a very rare sign, although it is very generally described. Even in cases with fairly abundant exudates I have seen at times nothing but a slight obliteration of the affected intercostal spaces. We must not forget that at first room is made for the increasing exudate by the easily retracting lung. I must believe that a marked prominence of the chest-wall occurs only under very peculiar circumstances, as, for example, in those very rare cases in which the disease has spread to the fibrous pericardium, the neighboring cellular tissue, and finally to the wall of the chest itself,—in other words, has passed, as it does in pleurisy, to the deeper layers,—and now, under the increased pressure of the large amount of fluid, has compelled the paretic intercostal muscles and other structures to yield. Rendu describes, among other cases, one in which a moderate amount of exudate was present in the pericardium, while the chest-wall was prominent and there was edema of the skin around the heart. Later on there developed in this case adhesions between the heart and pericardium, circumstances which point to an intense affection of the deeper layers.

Naturally, the prominence over the affected area is more easily brought about in persons with delicately constructed chests, especially in young people. When the lung is, furthermore, adherent to the thoracic wall nearest to the pericardium, we can also imagine that an intrapericardial increase of pressure would more easily produce a prominence of the chest, especially in the conditions described above.

The prominence in the epigastrium which has been described by some authors is possible as the result of the pressure of a massive amount of exudate upon the diaphragm. It is certainly not a matter of indifference whether or not the pressure of two liters of fluid rests upon the diaphragm. But such exudates are very rare, and in all probability the alleged prominence over the epigastrium has been misinterpreted or was due to complications. It is very possible that the congested liver which is present in high degree in many cases of pericarditis may lead to this error, and, as regards the complications, I may mention one case in which there was a large amount of exudate (over two liters) in the pericardium, and at the same time an abundant effusion in both pleuræ. On the other hand, there are cases in which a large amount of pericardial exudate led to the opposite error. Thus, I remember in one instance that a marked, somewhat fluctuating prominence under the left costal arch gave rise to an erroneous diagnosis of echinococcus of the left lobe of the liver.

The quality of the apex-beat has always received a certain amount of attention. The phenomena connected with this can only be utilized in pericarditis under certain definite conditions. A weak or imperceptible heart impulse is by no means a sign of pericarditis. For this may occur under entirely normal conditions, or may be due to disease of the heart muscle itself. It is different when one knows the

patient from previous observation, or has watched him from the beginning of the primary disease. A patient with acute articular rheumatism may have a heart-beat of normal strength in the usual position. A few days later the impulse is much weaker, and on the following day it can no longer be felt. There is then every reason to think of a complication in the heart—endocarditis or, more probably, pericarditis. But even in this case the weakening of the heart impulse is not necessarily caused by a large amount of fluid exudate, but may depend merely on the condition of the heart muscle. For this reason I call attention to the importance of the frequent participation of the myocardium in the pericarditis.

A large amount of exudate will certainly have a distinct influence upon the movements of the heart, in virtue of its pressure and weight. If the heart has preserved its normal power of contraction, however, it will still be able to force itself in systole through the fluid, and to beat against the chest-wall, in spite of the considerable pressure upon it. When the disease has lasted for some time, however, and when it has reached a certain degree, especially when the heart muscle has participated in the malady, the heart impulse will grow faint. That these views are correct I shall prove later on, when I come to speak of friction-sounds over the heart. In cases in which the diaphragm is pressed downward under the weight of a large amount of exudate the apex-beat may appear lower by one, or even by two, intercostal spaces; but all such statements must be accepted with great caution, and one must not believe that this phenomenon is frequently met with, still less that it is typical.

I should like to emphasize, finally, that even the absence of the heart impulse, when the radial pulse is strong, does not constitute a sign of pericardial exudate, for this combination of symptoms may be due to other conditions.

The examination of the heart impulse may also be of importance in other directions. When the patient is on his back, and when there is an exudate, no heart impulse may be felt. When he leans over forward, or when he cautiously assumes the knee-elbow position, the impulse may be felt, inasmuch as the heart then lies more closely to the chest-wall after having pushed back the fluid. The same may take place when the patient assumes the lateral position.

I shall speak later of this behavior of the apex-beat, to which Skoda long since called attention, when I come to the description of the areas of dulness in the differential diagnosis of pericarditis.

When an increased heart's action is observed at the beginning of a pericarditis, the tones of the heart may be sufficiently loud and clear. But weak heart tones are in themselves by no means signs of the presence of a pericarditis, for they may occur under normal circumstances, as well as in other pathologic states.

Matters are entirely different when we have an opportunity of observing a diminution in the intensity of the tones, or when percussion demonstrates an unusually large fluid exudate. As regards the former circumstance, the sequence of events may be as follows: Let us again take the case of an acute articular rheumatism. Today we hear clear heart-sounds of sufficient loudness. In the afternoon of the same day we hear these much less distinctly, and on the following morning we hear the first sound over the aorta remarkably weak and dull, and in the

afternoon, possibly, do not hear it at all. In these conditions one is perfectly right in thinking that a pericarditis has developed, and it is very possible that within the next few hours the characteristic friction-sound will bring a proof for the correctness of this assumption. In these cases the disappearance of the tones is certainly not produced by the accumulation of fluid, but in virtue of the loss of the normal power of contraction of the heart, due to the infiltration of the aortic wall with the exudate, resulting in a lessened elasticity of that structure. I may refer here to the similar occurrences which may be produced by endocarditis. When there is a large amount of fluid exudate, the heart tones may, of course, be weakened; but even here the fluid is only responsible in a very slight degree for the weakening of the tones; for, aside from the fact that, when there is sufficient contractile energy, the heart will always get close to the chest-wall at the moment of systole, I need only recall the fact that we can hear distinctly the heart tones of the fetus through a layer of amniotic fluid much greater than any that has ever been present in a case of pericarditis. Therefore, here again it is the lessened power of contraction of the heart which is chiefly responsible for the weakening of the tones, not considering now the fact that coëxistent endocardial changes may produce a partial or total disappearance of the tone.

In this connection I should like to call your attention to a very interesting acoustic phenomenon which is not infrequently heard at the beginning of the development of a fluid exudate, and less frequently later on during the absorption of the fluid. I refer to the reduplication of the second heart-sound at the apex or above it. This phenomenon, which may last only a few hours, and may immediately precede the appearance of the friction-sounds, is probably due to a certain degree of soaking of both pericardial layers at the very onset of the exudation. The surfaces, which have become sticky, adhere more closely to each other at the moment of systole, and during the diastole the detachment of the heart from the parietal layer produces a short sound which, in combination with the diastolic tone that comes from the interior of the heart, gives rise to this reduplication. [Josserand and Warthin independently called attention to an accentuation of the second sound in the pulmonic area. It may occur early and last a long time, or may disappear with the increased exudate.—Ed.]

As regards the shapes of areas of percussion dulness which are observed over a pericardial exudate, there are a great many contradictory statements. These areas are said to be very characteristic; to assume the shape of a triangle, to be triangular at the beginning with the base upward, and during the further course with the base downward, and so on. Let us, then, in the beginning understand clearly what circumstances can influence the form of the area of dulness.

In the first place, the amount of exudate, and, in the second, the varying resistance of the pericardium, will have distinct influence upon the form of dulness. The pericardium is most yielding, as a rule, at the base of the heart, because it is there less closely adherent about the large vessels than anywhere else. Another factor determining the area of dulness is the more or less extensive overlapping of the left border of the lung over the heart, the degree of which, as is well known, is not always the same. Furthermore, the varying degree of resiliency of the lungs will influence this phenomenon, and, finally, the position of the

heart will not be entirely indifferent—I mean according as to its more or less vertical or horizontal position, for this varies in different individuals. If we consider all the circumstances, we shall see that the shape of the area of dulness cannot possibly be always the same, even if one or two of these conditions remain constant.

Skoda long ago called attention to the fact that, at the beginning of a pericarditis with an effusion of fluid exudate, the latter gathers at the base of the heart, for the reason that at that point there is the least resistance on account of the conditions described above, and because the exudate is specifically lighter than the heart. Accordingly, at this stage, we find an area of dulness high up at the base of the heart; when there is little exudate, at the third rib; when there is more, at the second rib, and, therefore, the area of dulness of the heart is increased vertically along the long axis of this organ, possibly to a slight extent also horizontally toward the left side at the base. I must emphasize once more that we are not here dealing with exceptional yielding of the wall of the pericardium at another point than the base, nor with the presence of diverticula of the pericardium into which fluid could pour, nor with adhesions between the parietal and visceral pericardium, etc. I must also call particular attention to the fact, in order to avoid errors, that I speak always of the area of absolute dulness. When the amount of fluid is increased to a certain degree, it no longer finds room above at the base, but flows downward along the sides and along the posterior surface; and now we get those enlarged areas of dulness at the anterior surface of the chest which characterize the several forms of the disease. To a certain extent these areas assume the shape of a triangle whose rounded apex is situated at the manubrium of the sternum and whose base is at the diaphragm. But the sides of this triangle are not perfectly straight lines. As an example of the shape of the area of dulness in a case with a large amount of exudate I give the following: A rounded apex of the triangle near the manubrium, at the level of the upper border of the second rib, bounded on the right by an oblique line running downward to the upper border of the sixth rib, and crossing the latter slightly internally to the right mammillary line. This right boundary line is not straight, but shows a slight concavity to the right.

The left boundary is much more oblique from within outward, and is still more curved than the right, the dulness exending to an equal distance at the lower border of the second, the third, or even the fourth rib, to the left of the sternum, and only at that point sweeping to the left, even as far as the mammillary line and beyond, and then falling in an easy curve down to the diaphragm. I have also noted this boundary line on the left side in those cases in which I have been able to convince myself at autopsy that there were no adhesions between the pericardium and the lung, or between the lung and the chest-wall—that there was, therefore, actually only a variation in the power of retraction of the lung. The size of the area of dulness just described varies with the amount of exudate, and I have seen such areas occupy the greater portion of the anterior aspect of the chest down to the seventh rib; to the right, a few centimeters beyond the mammillary line on that side, and to the left a still greater distance beyond the mammillary line and beyond the site of the apex-beat.

There remains to be discussed more in detail a question which is considered of great importance by many authors, especially by Oppolzer,

in regard to the percussion sounds. This clinician examined all his cases of articular rheumatism with a view of detecting the presence of pericarditis, and looked especially for the change in the percussion-note to be described. It is frequently found that a tympanitic percussion-note, often exquisitely tympanitic, is heard on the left side, more or less extensively around the area of dulness described, but extending further upward or further to the left. This is due to the retraction of the lung under the pressure of the exudate. Such a retraction is the physical condition which obtains at first from the presence of fluid in the pericardium, and it is only when the contractility of the lung becomes exhausted that compression of this organ is observed. When the amount of exudate is sufficiently large, the posterior and lower portion of the lung is compressed. But, as Oppolzer has correctly pointed out, we must be very cautious in interpreting the tympanitic percussion-note, because it is very easily possible that such a note may arise as the result of the presence of infiltration in the lung, or as the result of a pleurisy. Therefore we must formulate a rule as follows: A tympanitic percussion-note over the area described is only then characteristic of a pericarditis when a careful examination has excluded positively all other causes of such a change in the percussion sounds.

Pins has lately called attention once more to the compression of the left lower lobe in pericarditis, which is shown by the presence of dulness and bronchial breathing over that area, and has especially emphasized the fact that these phenomena vary when the patient changes his position. It is astonishing that this change is said to take place within a few minutes.

I must call attention also to the fact that there are two other matters connected with the percussion-note, and which were thoroughly appreciated long ago by Skoda: First, that the area of dulness extends beyond the site of the apex-beat on the left side, and, second, that the area of dulness varies in its boundaries according to the position of the patient. The pericardial sac behaves like a yielding bag filled with fluid. If the patient bends forward, the area of dulness over the anterior chest-wall becomes larger; if he lies on his left side or upon his right, the corresponding boundary of the area of dulness is moved from its former position in the corresponding direction. This shows also how rapidly the lung retracts under these conditions, yielding to the pressure of the exudate.

Probably the most important symptom of pericarditis is the friction-sound known as the "pericardial rub." It is interesting to find how closely Laennec came to the correct understanding of this symptom. He stated that, at first, he had regarded the "squeak of the leather of a new saddle under the rider," with which he was evidently well acquainted, as a sign of pericarditis, but that he had later on changed his view. Collin, in 1824, was the first positively to declare that there was a connection between pericarditis and the friction-sound, but he attributed this phenomenon to the dryness of the parts. We know now that the friction-sound is produced by the gliding upon each other of surfaces which have become roughened by the exudate, and that the various characteristics of this sound, such as faintly blowing, rubbing, scratching, creaking, and scraping noises, depend partly upon the intensity of the heart's action and partly upon the physical qualities of the roughness. One thing I must especially emphasize at once: While a pleural friction-sound

can only occur where the rough portions are in direct contact with each other and while the smallest amount of fluid between the pleura will make this sound disappear, a pericardial friction-sound may still be heard in the presence of a certain amount of exudate. This is because a strongly beating heart at the moment of systole is able to penetrate the fluid, and thus to bring the rough surfaces in contact with each other, while during the diastole they are again separated.

The pericardial friction may be so marked that the physician, or even the patient himself, can feel it.

Let us now look into the question as to whether the dryness of the exudate is sufficient to produce a friction-sound. Various data exist in literature which tend to show that friction-sounds occur after losses of large amount of fluid; as, for example, according to Pleischl, in cholera, and it cannot be denied that the ear at times receives the impression as though the surfaces which rub against each other were very dry. Even the well-known phenomenon of the reduplication of the second pulmonary tone sometimes produces the same impression. And yet, whenever I attempted to convince myself as to the conditions present in such cases at autopsy, I have been unable to find this dry condition of the affected tissues. It may be objected that the parts have become more moist after death, but I believe that, if the friction-sound were due to dryness of the serous surfaces, it could be much more frequently demonstrated.

The friction-sound may be heard over any part of the heart, but is most frequently heard at the base. As may be understood from the manner in which it arises, it may be heard with varying intensity, and even with different qualities, at various parts of the cardiac area. It may be heard during systole and during diastole, or only during one of these two phases. The consensus of observations shows that it is louder, as a rule, during inspiration than during expiration, although there are some observers who have found the opposite to be true—in cases where the friction-sound was heard during expiration only, or more loudly during the latter. A number of experiments have been made in order to explain the reason for these differences in behavior, but it is very difficult to do this satisfactorily, inasmuch as the anatomic conditions are neither very well known as yet nor perfectly connected with the clinical manifestations. As opposed to the contention of those observers, like Lewinski, who attribute the greater loudness of the sound during expiration to the fixation of the anterior border of the lung, we must note that this increasing intensity may also occur when the lung is perfectly free. This increase in loudness may also be much more easily explained, under conditions such as are described by Lewinski, by assuming that the pericardium is under a different tension during expiration, and this theory is also supported by the fact that the phenomenon in question is not equally marked during every expiration.

I return to a subject to which I have already called attention in discussing etiology, inasmuch as it has given rise to a variety of mistakes. A friction-sound may be heard which is synchronous with the heart-beat, but is not at all produced by the diseased condition of the serous pericardium. Roughnesses on the fibrous pericardium, or on the mediastinal pleura, or on the neighboring pulmonary and costal pleura, may be pressed against each other by the movements of the heart, and thus an extrapericardial friction-sound may be produced. In addition, such

a friction-sound may occur during the systole, as Walshe has observed, from the rubbing of a swollen and uneven upper surface of the liver against the diaphragm, or, according to Emminghaus, in peritonitis.

We must next pass on to the consideration of the differences between the pericardial and endocardial murmurs, a subject which is of special diagnostic interest. It is generally admitted that the pericardial sounds may have all the qualities of tone which are found in the endocardial murmurs; and yet the character of the murmur is often so well marked that the ear at once gains the positive impression that it is caused only by the rubbing of rough surfaces. When the character of the sound is not so well marked, other means must be employed for differentiating their origin. We have several such means, and we shall discuss them now at length.

It very rarely occurs that endocardial murmurs have the same characters in both systole and diastole, while in pericarditis the rubbing character is expressed in the same manner with the same quality of tone in both phases of the cardiac cycle. On the other hand, we cannot rely upon the impression gained by the ear that the pericardial murmurs sound nearer than the endocardial. The difference in the distance between these sounds is too small to enable us to make this distinction; and we must also remember that the pericardial murmur may be faint, and that the endocardial may be loud, and also that the pericardial sound may be transmitted from the posterior surface of the heart—in other words, through the entire substance of this organ.

It is said that pressure with the stethoscope makes the pericardial sound louder. In some cases this actually occurs, provided the chest-wall is sufficiently yielding, inasmuch as the interlocking of the roughnesses is increased by this pressure. But this method fails us in other instances, for very often the chest-wall is too sensitive to bear a marked pressure, and possibly the heart can evade the increased pressure and thus cause the experiment to fail.

The variations in the friction-sound as regards time, place of occurrence, and position of patient are much more important for its differentiation from other similar sounds. While the pericardial friction-sounds are connected with the two great phases of the cardiac cycle, they are not so absolutely synchronous with these that one cannot hear the endocardial sounds between the pericardial murmurs, so that the pericardial sounds are continuations or appendages of the intrinsic heart-sounds. Or the pericardial friction may be systolic in the forenoon; may be present during both systole and diastole or during diastole only in the afternoon, and may change again in a short time as regards its time of appearance. In a similar manner the character of the murmur may change at different times.

In some cases the pericardial friction-sound is heard very distinctly in a certain place, but even the slightest movement of the stethoscope to another position shows a remarkable alteration in the intensity or even in the quality of the sound.

As regards the place where the friction-sound is observed, we must note the following: If we hear a systolic or diastolic sound only over the right ventricle, it is very possible that the murmur is pericardial; for if it were endocardial, it would indicate an insufficiency and stenosis at the tricuspid valves, and the latter occurs but very rarely. Besides, we should have to look for other symptoms to prove the presence of a tricuspid lesion.

Finally, we must mention a very important differential point: We hear the murmur when the patient is on his back only at the front of the heart, and there it is heard very clearly. With the patient in the left lateral position, we find that the murmur has become more faint at the place where it was heard distinctly before, or even that it has disappeared, while now a murmur may be heard at the left border of the heart. This is due to the fact that either a clump of fibrin which had been caught previously had changed its position, or that intimate contact had been created at another place as the result of the altered position and heavier pressure of the heart, in obedience to the laws of gravitation. A similar sequence of events may take place when the patient changes his position to the right side. It may occur also that we suspect pericarditis in a case of acute articular rheumatism, and hear only muffled sounds when the patient lies on his back, but perceive a distinct murmur when he lies on his side. We know that such variations do not occur with endocardial murmurs.

I have already stated that pericardial friction-sounds may have all the various qualities which are observed in endocardial murmurs. It is a question, however, whether this is also true of the whistling, musical noises. There are cases on record in which a whistling sound was heard temporarily and in which other symptoms of pericarditis were present. I have personally observed such cases. It is very difficult to conceive how such a sound can develop within a pericardial sac. In analogy with the musical endocardial tones, one involuntarily thinks of a string or thread-like structure which is made tense and produces the aforesaid tones, and one can fancy that when this thread breaks, the symptom may suddenly disappear. Mátrai very recently has asserted that a whistling tone may occur in pericarditis in virtue of the vibrations of several threads found at autopsy in the pericardial sac in one case, the increased tension of which, at the moment of systole, accounts for the sound. I cannot admit the possibility of such a sound being produced by this mechanism in the pericardium, with or without the presence of an effusion, and I think that this is especially impossible in the case reported by Mátrai. Inasmuch as he states that the cardiac impulse could not be felt from the start, the systole could not have caused such a marked tension of the threads in question that a sound would be produced. As a number of threads were present, their simultaneous vibration would have given rise to interference phenomena which could scarcely permit the development of a musically pure tone. It is evident that we are still in the dark as to the exact mode in which the whistling sound is produced.

Can a differential diagnosis be made between the so-called endopericardial friction-sounds, which come from the serous layer of the pericardium, on the one hand, and the extrapericardial or pleurocardial sounds, on the other? Skoda long since expressed his opinion on this subject, and gave us all essential data for this differentiation. If the friction-sound is heard over the sternum or lower down, near its left edge, the murmur arises in the serous pericardium; for at this point the fibrous pericardium is adherent to the thoracic walls by means of that cellular tissue which, according to Luschka, becomes thickened into the superior and inferior cardiac ligament. Therefore, the possibility of a friction-sound in this region is excluded. If the area of dulness is enlarged, that is, if a fluid exudate is present in the cavity of the pericardium,

then the friction-sound is also of serous origin. If it is heard only at the borders of the heart, and is present without increased area of dulness, especially on the left side, then its origin is doubtful.

On the other hand, if friction-sounds are heard synchronously with the respiration, or if other signs of pleurisy are present, the sounds are probably extrapericardial; for if there were an exudate in the mediastinal pleura, a friction-sound could be heard synchronous with the heart's motion and yet, on account of adhesions, not be heard also with the respiration.

If the friction-sound continues to be heard when the patient holds his breath, it can naturally arise just as well within the pericardial sac as outside, inasmuch as a roughness on the pericardial pleura may be pressed against a similar roughness on the pulmonary, or even better the costal, pleura during the movements of the heart. If, on the other hand, the friction-sound ceases with the arrest of respiration, then it is undoubtedly due to lesions on the outer surface of the cardiac envelop.

Certain sounds which may arise in the neighborhood of the heart in some special conditions of aspiration cannot be mistaken for friction-sounds on account of their usually blowing character. The possibility must be admitted, however, because some indistinct and ill-definable murmurs may arise in this manner.

As a rule, the pericardial friction-sounds are heard in a more or less vertical direction. I have already called attention to the fact that Skoda distinguished also a horizontal friction-sound. Personally, I have only been able to observe this phenomenon in a few cases, and I have already stated what significance is to be attributed to it.

Friction-sounds may last only a short time,—only a few hours,—but they may be heard during a number of weeks. I have had under observation a woman, thirty-three years old, in whom friction-sounds existed in varying intensity for four months. I may mention, in passing, that no cause could be found, either for the pericarditis or for the coëxisting pleurisy and peritonitis in this case. Repeated examinations of the sputum and cultures of the aspirated fluid always gave negative results.

In some cases friction comes and goes, sometimes changing its character, etc. This can easily be understood from the production and absorption of the fluid exudate, and the varying character of the plastic exudates.

Does the friction-sound also occur in cases which are chronic from the start, and does it also occur from the presence of "tendinous spots"? At times one hears sounds over the heart which must needs be considered pericardial, although the patient is otherwise perfectly well and has no fever. I have seen such cases, and Skoda also has mentioned the subject, and doubtless other authors. I have never seen a positive proof of such a case at autopsy, but, on the other hand, I must state that I have never yet heard a friction-sound in a case in which the autopsy demonstrated the presence of a "tendinous spot." Bozzolo is of the opposite opinion. [Babcock and Gibson, among others, admit the possibility, and H. Eichhorst mentions a case in which an unusually thick but smooth "milk-spot" caused friction-sounds.—Ed.]

There is no pulse which is characteristic of pericarditis. In various types of the disease the pulse may not show anything abnormal. The conditions for alterations in its character may be found in disturbances of the innervation, in a coëxisting disease of the heart muscle, and, finally,

in the amount of exudate. I exclude, of course, all other complications, such as valvular disease, etc. As regards the first factor, one sees not infrequently an increased pulse-rate at the beginning of the disease, without any fever or without relation to fever. Whether this is the result of irritation or of depression must remain undecided, although the former is more probable. There may also be the various types of irregular pulse, and such disturbances of innervation will astonish us less when we come to learn of others of more serious character. As a rule, the increased pulse-rate disappears after a short time, but it may persist, as I have observed in several cases, during the entire disease, and even after convalescence. In such cases we must suspect that a more serious lesion exists in the myocardium and in the ganglia in the heart muscle. The suspicion becomes stronger when the pulse becomes less tense and smaller. A trifling excitement or a slight physical exertion is sufficient to interfere with the regular heart's action, especially to increase the rate of the beat. If the amount of exudate exceeds a certain degree, or if it appears very rapidly, the fluid must exercise pressure, which affects the more yielding structures first. Thus, the superior vena cava, the pulmonary veins, and then the auricles are compressed first of all; the diastolic filling of the ventricles is interfered with, and stasis occurs in the systemic veins—less markedly so in the pulmonary veins. The result is a sluggishness in the renewal of the blood-stream passing through the lung, therefore, a lessened gaseous exchange, and, what is most interesting to us, a less active filling of the arteries and a smallness of the pulse. As we have pointed out before, these phenomena are due not only to the amount of exudate and the rapidity of its formation, but also to the condition of the heart.

I have never observed a slow pulse at the beginning of the disease, and when this is noted at the end, I do not think any other interpretation necessary than that which is given to this sign in the convalescence from other severe diseases; for example, pneumonia.

I shall speak later on of the *pulsus paradoxus* which may also occur in simple pericarditis.

Although Traube has observed a narrowing of the left carotid and left radial arteries in a case of pericardial exudate, these anomalies were probably accidental, or indirectly related to the pericarditis. I have seen such a case in which there was no sign of pericarditis, although no other causes than an anomaly in the size of these vessels could be found. I think there is no reason to assume the existence of an obliterating arteritis or an embolism. The same may be said of two cases of pericarditis in which Gerhardt observed a pulsation over the spleen. This sign is certainly only indirectly connected with pericarditis, and, as Gerhardt believes, through the diminished tension of the blood-vessels resulting from the fever.

If we remember the results of pressure upon the venæ cavæ which have been spoken of above, we will at once understand that pericarditis may be accompanied by a stasis in the systemic veins, with all its consequences—more or less marked cyanosis; swelling of the liver; congestion of the kidneys, etc. These phenomena are further increased because diminished filling of the left ventricle causes a lessened *vis à tergo*. Hence edemas result and all the phenomena are increased if the myocardium becomes affected at the same time, not to mention the possibility of other complications. The stasis will be most clearly marked in the

cervical veins, which may be very much swollen, may undulate and show both systolic and, less frequently, diastolic pulsations, which arise through the contractions of the right auricle.

Pericarditis may influence respiration and may cause dyspnea in a variety of ways. It is possible that respiration may be affected through a simple irritation of the vagus, and that a large amount of exudate is not even necessary. But the amount of fluid may also play a part. A large exudate produces difficulty of breathing in virtue of the compression of the auricle and the pulmonary veins described above, and the stasis of the blood in the pulmonary capillaries; and also, in the case of large exudates, a stasis occurs in the branches of the pulmonary artery from the compression of the left lung; and, finally, a bulky exudate, generally speaking, crowds all the other structures in the thoracic cavity. That such conditions actually occur is clearly evident from the fact that the patients themselves instinctively seek to create more favorable conditions of respiration; they avoid the dorsal and left lateral positions; sit up and bend forward, in order to lessen the backward pressure upon all the structures. A marked pressure upon the trachea or, as has often been mentioned, upon the left bronchus, does not take place or is exceedingly rare. I have given special attention to this subject, and have looked for evidences of compression of the above-mentioned portions of the air-tubes in cases of pleuritic and pericardial exudates, but am obliged to report only negative results. I think, also, that it is doubtful whether the difficulties in swallowing which are at times observed are caused by the compression of the esophagus, and I believe, rather, that these signs are of nervous origin, just as others are which also occur—vomiting, not rarely a very troublesome hiccup from the direct irritation of the phrenic nerve, and, finally, disturbances or abolition in the function of the recurrent branch of the vagus nerve on one side, as I myself, Landgraf, and Riegel have observed, or even bilateral paralysis of the vocal cords, as Bäumler has reported. In these cases there may have been an edematous inhibition, inflammatory processes, or simply compression of the nerve due to an unusually large exudate.

There is no doubt that many cases of pericarditis run their course entirely without fever, or at least with such a slight febrile movement that the patient does not even notice it. In this category may be included the very mild and the chronic insidious forms. On the other hand, a primary pericarditis, just like other infectious diseases, especially pneumonia, may begin with a sharp febrile movement, or even with a chill, and may be accompanied with a regular *fébris continua*, ending with a sudden or gradual defervescence. In the secondary forms it is not always possible, of course, to decide what particular rôle the inflammation of the pericardium plays in the febrile manifestations. In acute articular rheumatism, for example, the course of the fever may remain the same in spite of the advent of pericarditis, while in other instances a lively rise of temperature may follow. Just as, in the course of an articular rheumatism, the recurrent affection of a new joint is sometimes accompanied by renewed febrile movement, and at other times without the latter, so also does the temperature vary in a complicating pericarditis. Very high temperatures, very severe febrile manifestations in general, are undoubtedly rare in pericarditis, and when they occur we must be careful to determine whether they are not due to other complications. The defervescence is not infrequently accompanied by a profuse perspiration.

In the milder forms the urine does not show anything abnormal, but when it does show changes, they may belong to one of two types: either the urine shows those peculiarities which are characteristic of fever, or else it assumes that form which is attributed to stasis. In the former event we have a concentrated dark urine of high specific gravity, and it is not infrequent to find an abundant lateritious sediment. In the latter case the urine is diminished in quantity, also dark, but contains a more or less marked amount of albumin and possibly a few casts. In both cases, however, we must determine what elements in the urinary findings are to be attributed to the pericarditis and what to the primary disease, especially, in the latter case, how much of the urinary disturbances must be credited to a coincident disease of the heart muscle. It appears, therefore, that we cannot gain any essential diagnostic information from the examination of the urine. Whether the chlorids are constantly diminished, or whether any prognostic significance may be attributed to their behavior, cannot at present be told.

Among the subjective symptoms, pain is to be mentioned first. I may say, unhesitatingly, that the greater number of cases of pericarditis run their course without any pain. On the other hand, one must admit that sometimes a severe spontaneous pain may be present, or the least touch, the slightest pressure with the stethoscope, may indicate great tenderness. We cannot trace this very marked difference to certain forms of the inflammation, nor to varying amounts of exudate.

There is no doubt that this pain can only be caused by the pericarditis. But inasmuch as it may not merely be limited to the cardiac region, but also may radiate to other parts of the chest, to the neck, the shoulder, and even the arm, we must determine very carefully in a given case by examination whether we are dealing with a rheumatic pain of the chest-wall or possibly with a disease of the shoulder-joint or of the pleura accompanying the pericarditis. The decision will be the more difficult because the pain is not infrequently increased by deep inspiration, both in pleurisy and in pericarditis. My own observations do not place me in a position to state whether, as Bäumler claims, the pain is more frequently felt in the region of the epigastrium than in other places. The observation of Bäumler as to the presence of a painful spot on the left side of the larynx also seems to be unique. While, as has been said, an intense tearing or stabbing pain is quite rare, patients often complain of other painful sensations, such as a feeling of weight or pressure.

They often speak of a troublesome feeling of palpitation of the heart, especially at the beginning of the disease—a symptom which may be accounted for by the increased cardiac activity, and which generally disappears when the heart's action becomes more quiet.

I must call special attention, however, to still another sign, which has also been spoken of by other authors. I mean the very marked feeling of anxiety and oppression which accompanies the shortness of breath. It is true that these signs are observed especially in cases with large exudates in which the amount of fluid rises rapidly, but these are certainly not the only causes; for the intense feeling of anxiety and fear which is distinctly expressed upon the features of the sufferer, and which excites our fullest compassion, occurs in the presence of comparatively small amounts of exudate. Large pleuritic effusions, valvular lesions of the heart, also produce dyspnea—and yet the picture of peri-

carditis is different. I should like to go so far as to declare that in this there is something characteristic of the disease, and I have several times actually made the correct diagnosis simply from the picture of the patient sitting erect on his bed, breathing heavily, slightly cyanotic, and showing all signs of the most intense anxiety.

Diagnosis.—Without a physical examination pericarditis would very frequently be overlooked—in fact, it is no exaggeration to say that it would not be noticed in most cases. We can make the diagnosis only by examining the heart of every patient, especially by examining it particularly in all those diseases in which we know pericarditis occurs. In this way collating all the clinical data we can, in most cases, arrive at the proper diagnosis.

The most important factors to be considered in this connection are the area of dulness, the friction-sounds, and, as I shall show later, the course of the disease. As regards the dulness on percussion, this sign may be overlooked even in the presence of a large amount of exudate, when a marked emphysema of the lung is also present. The enlarged area of relative dulness may possibly call attention to the existing pathologic condition, but this sign alone cannot bring us to a definite decision, and, as a matter of fact, when no friction-sounds are present, it makes a correct diagnosis scarcely possible unless there be some special manifestations to assist us. The difficulty is still further increased by the fact that, in both emphysema and pericarditis, the same degree of dyspnea may be present.

We have already stated that a dulness on percussion at the base of the heart is characteristic of a small amount of exudate. Error is not readily possible if we consider the history of the case, the mode of onset, and, especially, if we examine the lung repeatedly, particularly as regards its mobility. The triangular area of dulness which occurs in cases with large amounts of exudate, as we have already described in detail, requires a more minute examination. In these cases, also, there is no possibility of a mistake when the other symptoms of pericarditis—the loud and characteristic friction-sounds—are clearly marked. What about those cases in which these are absent? If the area of dulness is not too large, there might be, in the first place, a perfectly normal heart, the enlarged area of dulness being attributable to a preceding pleurisy, after which the retracted lung has formed adhesions to the thoracic walls. The same retraction may occur when the lung has become fixed after the absorption of a large pericardial exudate. In such instances a correct solution should be found in the normal position of the apex-beat, in the absence of change in the boundaries of the dulness during deep inspiration, and in the possible absence of symptoms of heart disease.

Another source of error in connection with an increased area of dulness is the so-called *cor bovinum*, in which the heart is enlarged considerably in all its dimensions, so that there is a very extensive area of dulness. In these cases the diagnosis must be based upon the location and the character of the heart impulse and the mode of origin of the dulness. If the apex-beat is felt at the boundary of the area of dulness, we are probably dealing with a hypertrophied heart, for we have already seen that, in the presence of a pericardial exudate, the area of dulness overlaps the site of the apex-beat to the left to the exent of several centimeters. In the circumstances described, a clearly defined apex-beat

also speaks in favor of a hypertrophic heart, for the presence of even a moderate amount of exudate generally lessens the vigor of the impulse. If an enlargement of volume has taken place in the course of months, then this alone, aside from other symptoms, would speak in favor of hypertrophy.

The question of differentiating from a marked dilatation may arise only in acutely developing enlargements of the area of dulness. This is, however, quite rare. Therefore, if the increased area of dulness has developed under our eyes, pericarditis is certainly more probable than enlargement of the heart. The latter also would certainly produce more severe sequelæ, such as congestion of the liver, swelling of the ankles, etc. Furthermore, severe hypertrophies of the heart are usually caused by valvular disease. Therefore, wherever the manifestations of a valvular affection are absent one would be justified in thinking rather of a pericarditis. This is reversed, however, when Bright's disease is present, and when we have not previously known the patient, or in those rare cases of valvular disease in which there are no murmurs. A differentiation may be made even in these cases when alterations in the extent of the area of dulness occur within short intervals, for such changes would undoubtedly speak in favor of a pericardial exudate. If fluid collects about a heart which is enlarged at the same time, the presence of the exudate can only be detected when it develops under our eyes, or when the variability in the area of dulness just mentioned is observed. In these cases the remarks made above will also apply.

The differentiation between pericarditis and hydropericardium will be discussed later on. Aneurysms of the ascending aorta, those of unusual site, as in the concavity of the arch, or even of the thoracic aorta, may also give rise to an enlarged area of dulness which may be distinctly triangular in form. This is due especially to the fact that the heart is displaced to the left and downward by these aneurysms. If other symptoms of aneurysm are present, the differentiation will not be difficult, but it will be puzzling, or even impossible, in those instances in which these symptoms are absent. It must be borne in mind that there are aneurysms which lie close to the anterior thoracic wall and still do not produce any pulsation.

Mediastinal tumors may also produce enlarged areas of dulness in a very similar manner. The mode of development, the presence of extensive networks of veins upon the anterior aspect of the chest, the auscultatory phenomena, the swollen lymph-nodes, the course of the disease, and the age of the patient are all to be regarded as diagnostic criteria in such cases.

When distinct friction-sounds are present, and when the disease has developed under our observation, it is not difficult to distinguish a pericarditis from a unilateral or bilateral free pleuritic exudate. On the other hand, the opposite is the case when a small unilateral or even bilateral exudate is sacculated close to the heart, or when the adhesions which have formed about the sacculation are so situated that they produce a triangular area of dulness over the heart. If the dulness has developed steadily under our eyes, there can be no doubt as to the diagnosis, especially when friction-sounds are also present. If the completed process is present, however, and if no other signs of pericarditis are manifest, then the differentiation becomes very difficult. In such cases we must rely again chiefly upon a very accurate history, upon the variations

in the position of the margins of the lung with the respiratory movements; the change of location of the apex-beat when the patient changes his position, or the appearance of the apex-beat after changes of posture, when it could not be felt before.

I have already discussed in detail the modes in which the friction-sounds may be employed in the diagnosis of pericarditis, especially in what manner they are to be distinguished from endocardial murmurs. I have also outlined the features in the course of the disease which are important in its diagnosis; but for the sake of clearness, I shall sum these up briefly again. When I find that in a given case the apex-beat has become weakened, and that the first sound over the left ventricle or over the aorta has grown muffled, I am justified in thinking of a pericarditis. The diagnosis is made certain when dulness appears over the base of the heart or when friction-sounds occur.

It seems useless to discuss the question whether the exact character of the exudate may be determined by physical examination, after I have already called attention to the fact that the variety of exudate may be approximately foretold in secondary cases from the character of the primary disease. I say approximately, because we cannot be absolutely sure. Thus, in a case of articular rheumatism or of pleuropneumonia the pericardial exudate is usually fibrinous or serofibrinous, but it may also be purulent. The fever does not give us a decisive clue. Prolonged fever speaks in favor of a purulent exudate, but the latter may also occur without any febrile movement. Serofibrinous exudates may also be present as the result of the proximity of a carcinoma in the neighborhood. They need not always be purulent, but under special conditions they may be gangrenous.

Course, Duration, and Termination.—The course of pericarditis may vary greatly, and the different cases observed range through a large series of transition-forms from an acute type, running to its end within a few days, to one which stretches over many months. The lower boundary of this series cannot be accurately defined when we consider such observations as that of Weichselbaum. This author found that pneumococci were present in a perfectly clear pericardial fluid in a case of pleuropneumonia; in other words, that everything had been prepared for the outbreak of pericarditis. It would not be correct to say in this instance that only the death of the patient as the result of the coëxisting disease in the lung had prevented the development of a pericarditis. Other influences certainly had some bearing, otherwise pericarditis would more frequently follow pneumonia.

Some symptoms are often only transient. Thus, for example, a pericardial friction-sound may be observed only during one or two days, and it is certainly improbable, in these instances, that the pathologic process, as such, did not disappear with the cessation of the friction-sounds, although it is likely to have been only of short duration.

The usual termination of pericarditis, as has already been mentioned, is in recovery. The fever subsides; the pulse, which has been small, grows stronger and fuller; the amount of urine passed increases; the area of heart dulness becomes smaller, while the friction-sounds which were present at the beginning, but then disappeared, may reappear, and finally permanently disappear, and thus apparent recovery takes place. I say apparent recovery; for true recovery is not by any means indicated by convalescence. It takes a considerable length of time until all the sequelæ

of the disease disappear, until the exudate is entirely absorbed, and, especially, until the heart muscle regains its full power. Recovery may be apparent only also on account of the formation of adhesions between the two layers of the pericardium, an event which may be designated as the end of the disease, but certainly not as a true recovery therefrom.

A fatal termination takes place in the acute form, as a rule, at the height of the disease, through heart failure—either as the result of the pressure of the massive exudate, especially when the fluid accumulates very quickly, or as the result of a diseased condition of the muscle itself, which leads to paralysis of the heart. The feeling of fear and the shortness of breath grow more and more threatening; the cyanosis increases; the temperature falls; the pulse grows smaller, and the disturbances in the pulmonary circulation finally lead to edema of the lung. A transient improvement is still possible, or a second attack of collapse may lead to the fatal end. The primary disease itself is of great importance for the prognosis in such cases, and also the constitution of the patient, his general condition, and whether or not he was addicted to alcohol.

Some authors attribute the rapid death which occurs in some cases of pericarditis accompanied by hemorrhagic exudate to the loss of blood. Although the possibility of such an occurrence cannot be entirely denied, I believe that in these cases death is due chiefly to the paralysis of the heart muscle. A fatal termination through other causes scarcely needs to be considered. As I have already mentioned, abscesses with perforation which burst outward very rarely occur in the course of the disease, and are more frequently seen in connection with the subacute or chronic type.

The chronic type may develop very slowly from the acute form of the disease, and we have no means of detecting whether or not the disease is destined to become chronic from the start. The acutest manifestations may cease; the fever may disappear, and yet nothing is changed in the physical signs over the heart, or the area of dulness may possibly diminish somewhat temporarily, but only to increase again, even to a greater degree than at first. In other cases, while the physical signs partly disappear, the patient does not seem to recover fully; the appetite does not increase, and therefore nutrition lags; the patient is weak, and the slightest exertion produces palpitation of the heart and shortness of breath. At this stage the patient may still recover completely at any time, even after the disease has lasted for weeks, the fluid exudate being absorbed, the solid exudate undergoing fatty degeneration, and the molecular débris being absorbed. Even this termination may only be an incomplete one, because there may be partial adhesions inclosing encapsulated pus or deposits of calcium salts, or the diseased heart muscle may not recover, but, on the contrary, the walls of the heart may yield more and more to the intracardial pressure, thus leading finally to dilatation of this organ. As a sequel of these anatomic changes, and as a result of stasis in the pulmonary circulation, persistent bronchitis may develop and gradually there may appear the general phenomena of dropsy; at first slight, later more marked, until finally the patient dies of marasmus.

In still another class of cases the course of pericarditis is influenced by that of the primary disease, or the fatal termination is due to this primary condition.

We must always be cautious in making a prognosis in a case of pericarditis, because we can never tell which course the disease may take, and because we have no means of detecting with certainty the exact condition of the heart muscle. I remember two cases in which the patients seemed to be convalescing, but died suddenly after a slight physical exertion, as the result of a sudden failure of cardiac activity.

The mild forms of pericarditis with a small amount of exudate, without complications, and with satisfactory heart's action, as a rule, justify a favorable prognosis. We may say, however, that the prognosis depends upon the quality and quantity of the exudate, and, as we have already stated, upon the rapidity of its development, as well as upon the condition of the heart, the constitution of the patient, and, finally, upon the primary disease which may be present.

Cases of primary pericarditis, and such as develop as a sequel to acute articular rheumatism or to pleuropneumonia, as a rule, permit a favorable prognosis, but even here, as we have said before, exceptions are by no means rare.

An unfavorable result may be foretold when Bright's disease is added to a pericarditis or when the latter occurs in an alcoholic subject. The consequences of injuries in the region of the heart must always be regarded with caution. Hoffman speaks of a case in which the point of a knife had penetrated through the anterior mediastinum into the arch of the aorta in such a manner that the opening in the intima was only one millimeter long. As there were no severe symptoms, the injury was declared to be slight, but the patient died sixteen days later from pericarditis. Serous or serofibrinous exudates present in not too large amounts permit a favorable prognosis, while purulent, hemorrhagic, and, of course, gangrenous exudates indicate an unfavorable termination, because they are connected with diseases which in themselves are of unfavorable prognosis. Finally, the prognosis is rendered more unfavorable the longer the disease lasts, as may be easily deduced from the description of the course of the malady given above.

Treatment.—Our most important aim should be to prevent the development of pericarditis. I mean, of course, not the primary form, for there we must admit our helplessness; but only where it is not possible to prevent the occurrence of pericarditis in those diseases in the course of which pericardial inflammation may be expected, especially in acute articular rheumatism. The discovery of the salicylates, which may be regarded as specifics against the rheumatic process, brought the hope that they would enable us to avert a development of endocarditis or pericarditis. Unfortunately, experience has shown that this is impossible, and that specific treatment has not lowered the percentage of pericarditis occurring during acute articular rheumatism. Our task then must be to combat the disease, whatever may be its cause, when it has developed, and to seek to limit its spread. It is true that this will only be necessary in those cases in which pericarditis appears as an independent disease, and is not masked under the symptoms of the principal affection. We must admit at once that we have today no remedy with which we are able to check the disease with certainty after it has once broken out. And yet we may console ourselves with the idea that even this admission is a considerable stride forward, and it is only to be wished that this viewpoint may be generally adopted.

Skoda was the first to show, on the basis of extensive and thorough

scientific experiments, that blood-letting and the use of mercurial preparations are to be rejected as perfectly worthless. On account of this declaration he has been anathematized as a therapeutic nihilist, but it is not difficult to prove that he did not deserve this epithet, and it is well to put an end to the uncontrolled and schematic application of these remedies. It is certainly a scientific acquisition when it is demonstrated that certain remedies are of no use, and possibly may do harm. This is the case as regards general blood-letting, for the patient with pericarditis is in constant danger of sudden heart failure. How much will he then need his blood!

Our treatment, therefore, will necessarily be chiefly symptomatic. Above all, such a patient needs perfect rest, preferably in a somewhat raised position, and must be kept from all mental and physical excitement.

The diet must vary according to the appetite of the patient. At the beginning, while the disease is developing and high fever is present, only liquid diet is to be given, and acid drinks are to be ordered to relieve the thirst. The bowels must be regulated, and easy evacuations secured in order to prevent, on the one hand, a distention of the abdomen, and, on the other, to prevent all physical exertion. The application of cold offers one of the most important means of treatment in pericarditis and fills a whole series of indications. It may be applied either in the form of a light ice-bag or of cold compresses, or, what is very useful, by means of the Leiter coil. Cold is applied to combat directly the inflammatory process, to lower the temperature, and to diminish the excitable heart's action, which is often very much increased in frequency early in the disease. These applications are particularly beneficial in relieving palpitation, even when other modes of treatment fail. They also diminish the shortness of breath, and may be tried even as the most convenient method of relieving pain. As we have said before, the fever in pericarditis is not, as a rule, very high. If it reaches a higher degree, for example, as the result of the primary disease; if the heart muscle is threatened by the high temperature; and if cold, applied as mentioned above, does not suffice, then we can give, with due caution, a few doses of antifebrin [acetanilid—Ed.] (0.3 gm.). Baths in any form are not to be recommended in the acute stage of the disease, for the simple reason that the patient requires rest.

If the pain becomes more severe, and if the cold does not suffice to relieve it, it is not advisable to change to hot applications, inasmuch as the latter easily increase the heart's action. If the heart-beat is vigorous, then there is no objection to the use of morphin in small doses, internally or by subcutaneous injection. It is more advisable, however, to omit the use of morphin, and, at all events, it is best to use it only in those cases in which the physician is able to watch the patient carefully. The employment of leeches is perfectly useless, although they relieve the pain temporarily in some cases. The remedies mentioned above are certainly more useful.

When there is marked dyspnea, sodium bromid in doses of 1 gm. repeated several times gives, usually, great relief. If the shortness of breath is very severe, morphin may again be used with caution. The increased heart's action and the palpitation are best combated by means of digitalis in the form of the infusion of the leaves (1.5 gm. in 15.0 c.c. of water per day). If the pulse is weak, or if one is timid, one might first try 30 or 40 drops

of tincture of strophanthus per day. This is also excellent and less severe in its effects than digitalis. Inasmuch as such a patient must always be carefully watched, medication can be changed instantly at the proper moment. If the strong, large pulse should become small and weak, the digitalis should at once be stopped. For the irregularity of the pulse, I know today no better remedy than quinin, which can be given as a useful tonic in doses of from 0.5 to 1 gm. per day, even when there are no other indications for its employment.

If the absorption of the exudate does not take place in the expected manner after the acute stage has passed, the nutrition of the patient should be improved at once, and more nutritious food gradually be given. Rest is still to be maintained in the same manner because at this stage the danger of an affection of the heart muscle is rather increased than otherwise. For the same reason baths, which are ordinarily so useful in promoting the absorption of exudates, are to be avoided, or are to be employed only with the greatest caution.

I must return to what I have said about Skoda. He showed, in the most convincing way, that blisters, Authenried's ointment, horsehair issues, and moxæ, do not promote the absorption of the exudate, and when we see how remedies which have often been rejected, and which have sunk into oblivion, may again come to the surface and be used for a time, we must remember that this is not an evidence of their real efficiency, nor of a scientific way of thinking, but only a fashion which arises from our therapeutic helplessness, always striving to improve itself.

By what means, then, can we bring about the absorption and excretion of a large exudate? The diaphoretic method by hot packs is not to be recommended, because it increases the heart's action. Pilocarpin in subcutaneous injections is of but little use, according to my experience, and is an uncertain remedy. Often it produces only a great perspiration at the head or a disagreeable salivation. The old diuretics are of but little use; but, fortunately, we have at present an almost certain medicament in diuretin, the sodiosalicylate of theobromin, which is almost invariably well borne. We employ it in increasing doses of from 2 to 6 gm. per day. As a rule, a marked diuresis occurs on the third day, and does not cease for several days even after the remedy has been discontinued. I have not had sufficient experience with calomel in the treatment of this disease to offer an opinion; at all events, it must be employed with care on account of its after-effects, and it is certainly better to use the harmless diuretin first. In the treatment of a severe case of pericarditis the physician must be ready for attacks of heart failure, syncope, and collapse at any moment in the acute or chronic stage. Energetic cold frictions, injections of camphor or of ether, and the use of champagne or of brandy are at present indicated. I say at present advisedly, for I believe that the last-named remedy has been used entirely out of proportion to its true indications, and we hear of the use of brandy as a stimulant far too often even in ordinary life. One of the best stimulants in my experience has been strong tea with some good red wine or with a little rum. Tea acts particularly well in practice among the poor, because they are less used to this beverage.

Finally, we must speak of the surgical treatment—paracentesis of the pericardium. The idea of performing this operation had arisen several times, but in 1819 Romero, of Barcelona, actually punctured

the pericardium for the first time. The method was introduced more generally, and has been performed more frequently, however, since Schuh and Skoda, of Vienna, have studied it in detail. It was a very fortunate circumstance that the skilful and bold surgeon of the Vienna school, Franz Schuh, who was intimately acquainted with Skoda, gave himself with zeal to the study of the achievements in physical diagnosis attained by Skoda; himself acquired skill in the then new methods of examination, and then tried to make the successes of exact diagnosis applicable to surgical procedures. Since Schuh and Skoda performed the operation in 1840 a large number of cases have thus been treated. In the same year Kyber, in Russia, operated on a large series of patients with hemorrhagic pericarditis due to scurvy, but only three recovered, and when they died of intercurrent disease later, the autopsy showed the presence of adhesions between the heart and the pericardium. Later on the operation was performed several times in the same subject, as, for example, by Moor in a boy thirteen years of age six times within two months, and by Bouchut as often as eight times in a girl twelve years old.

No matter what we may think of the surgical treatment, we must admit, upon reading the reported successes and upon seeing such cases ourselves, that in some instances the operation has been directly the means of saving life, or, at least, of prolonging life, and all authors agree that the patient obtains the greatest relief from this procedure. Even in the first case operated upon by Schuh it was reported that the patient, who had not been able to sit up nor to lie down on account of the great dyspnea, felt perfectly well after the operation and was discharged cured twenty-one days afterward. When the patient died six months later of cancer of the mediastinum, the autopsy showed that the operation had fully served its purpose, for the heart was found adherent to the pericardium.

Although the operation has now been performed in about 100 cases, the results cannot be said to be uniform, and it is perfectly natural that they should differ, according to the presence of one or the other primary disease. Rendu claims to have obtained improvement or recovery from pericarditis in 32 per cent. of his cases of paracentesis. Of 10 patients with uncomplicated pericarditis, 8 recovered under this treatment.

Von Weismayr has taken the trouble to look over the entire series of cases reported thus far. There were 99 cases, and if we add the case of Mader, soon to be reported, there are just 100. Of these, 47 recovered and 53 died.

A favorable result is possible, as Mader has shown, even in the cases with hemorrhagic exudates. In a man aged forty-one years 70 c.c. of pure bloody exudate were removed. Not only was the immediate relief considerable, and the secretion of urine increased to four and a half liters, but the patient improved so much that he was able to leave the hospital five months later. The final result was not favorable because there was probably tuberculosis at the bottom of the affection.

What are the indications for the performance of the operation? They do not differ essentially today from those which were given by Schuh and Skoda:

1. When a large exudate does not show any tendency to absorption after a prolonged observation and after the employment of the proper means described above, and when the patient is more and more markedly affected by the disease.

2. The vital indication, when the patient is threatened with death as the result of severe dyspnea.

While the operation was almost without danger in those days, provided it was properly performed, we may say that it is positively safe today. The puncture is best made in the fifth, or, at all events, in the fourth, intercostal space on the left side, and it is quite indifferent whether we puncture directly or cut through the separate layers one by one. If the diagnosis has been made correctly, there can be no possibility of injuring any other structure. The lung must, of course, be widely retracted over the affected area, in the presence of a moderate exudate, and it is easy to avoid the mammary artery. It is, as a rule, best to operate in the dorsal position, although one may assume that in the presence of an exudate indicating an operation the heart will have been pressed so far back as to preclude the possibility of its being wounded. Mader inserted a fine needle into the sac as a matter of precaution before making the final puncture, and as he found that he could introduce this needle for some distance without meeting resistance, he concluded that the heart must lie still more deeply. The amount of fluid which will flow out of the puncture at first depends upon the elasticity of the distended pericardial sac and the weight of the column of fluid. A very slow aspiration is possibly advisable, but in all cases it must be made with great caution.

My excellent results with puncture and injection of iodin in cystic goiter, and the favorable results which I obtained with the same method in echinococcus cysts, lead me to advocate the injection of iodin after puncture also in pericarditis. This method has been followed, and with favorable results, and I do not doubt that in most cases it will secure the desired end—the adhesion of the pericardium to the heart. Yet it is very doubtful whether this is really desirable. As we shall see in the next section, adhesions between the heart and the pericardium may be followed by unpleasant consequences, and this termination can no more be regarded as a true cure of pericarditis than the amputation of a limb.

Under any circumstances the patient, even after a successful paracentesis, must take the best care of himself.

[*Paracentesis Pericardii.*—Since this section was written much has been added to the literature of paracentesis and incision of the pericardium. For diagnostic purposes, in order to show the character of an exudate, as well as, in some cases, to confirm its presence, careful and, if necessary, repeated exploration, under aseptic conditions, is generally recommended. There is no single point of election for either the exploratory or the therapeutic aspiration. Until recently most authors advised the points mentioned by von Schroetter—the fourth and fifth left interspaces. Two factors are concerned: the position of the heart with reference to the chest-wall, and the shape and position of the pericardium. The internal mammary artery and the pleura are important factors also.

Position of the Heart.—It has generally been supposed, since the time of Skoda, that the heart sinks down in pericardial exudates, owing to its superior weight. Schaposchnikoff, however, has shown, in a series of articles published during the past ten years, that this is not so. Friction-sounds may be heard, the apex-beat be present, or a needle may touch the heart at once upon puncturing the pericardium, even in cases with large exudates. Thayer has reported a tuberculous case in which,

"with over 1200 c.c. of fluid, the visceral and parietal pericardium were still adjacent anteriorly." Schaposchnikoff showed experimentally that the position of the heart in such cases is due to the elasticity of the great vessels. He admits that in cases where the great vessels lose their elasticity the heart sinks. Doebert suggests that inflammatory processes in and around the great vessels help to fix the latter. Schaposchnikoff's view has been accepted by Romberg and many other clinicians. It was combated by Damsch, on the ground of experiments the technical faults of which have been shown by Aporti and Figaroli, whose experimental results were, however, slightly divergent from Schaposchnikoff's.

Numerous clinical observers have followed Schaposchnikoff's teachings, notably Heiking, Albert Fraenkel, Doebert, and Peters and Rudolf. Schaposchnikoff advises puncture in the third or fourth right interspace near the sternum, or, in the case of large exudates, the sixth left interspace. Several operators have successfully punctured in the fifth right interspace. Romberg, following Kussmaul and Curschmann, advises the fifth or sixth left intercostal space outside the mammary line, at a point where there is dulness, but no pulsation or friction. The danger of opening the pleural cavity is not to be considered, especially as pleural effusion is often associated, in the left side, and can be drained after emptying the pericardium by partially withdrawing the cannula.

A number of recent American and English authors,—Shattuck, Fitz, Osler, Ogle and Allingham, Pendlebury, Latham, Coutts and Rowlands,—and Mintz and Heiking in Russia, recommend the left xiphocostal space. This was proposed by Larrey nearly one hundred years ago, but looked upon as visionary until lately. In cases with large exudates the pericardium may be entered at this point without danger of injuring the liver, peritoneum, or pleura, and it offers excellent facilities for drainage by incision following aspiration.

Good results have been obtained by operations at all the various points described. In some cases the choice may be limited by peculiarities of the outline of dulness, and I should recommend exploratory aspiration in the order mentioned. The operation is usually done with the patient lying down, but if this fails, the patient should be partly raised, or even made to lean forward. Exploratory aspiration should always be made with a sharp, small, but not too fine-calibered needle. No anesthetic is necessary for this operation. The needle must be put in slowly, so as to feel the structures (as, in fact, all exploratory punctures should be made). It is usually possible to feel when the needle enters the pericardium, but in some cases—occasionally involving errors in diagnosis—the heart muscle has been punctured. The right ventricle cannot always be avoided in puncturing in the left side, but in general the accident is not serious. Much more to be feared is puncture of the right auricle. Having found fluid by aspiration, it is next necessary to learn its character and the distance from the surface at which it can be reached. Clear serous exudates may be removed through the fine needle by attaching a siphon tube. Thicker exudates require a trocar and cannula or large aspirating needle of 2 to 3 mm. diameter. Curschmann uses a flat trocar and cannula 3 to 5 mm. wide. The larger instruments can be used with an aspirating apparatus, but a simpler method is by the siphon tube, a long rubber tubing, sterilized, full of water, attached, while still full, to the end of the cannula; the other end, placed

in a sterilized vessel on the floor, being opened, sets up a siphon action which is usually efficient.

In cases with thick exudates or masses of fibrin, incision and drainage must be practised. These operations can be done under local anesthesia, even in children, though if resection of costal cartilage is necessary, chloroform may be advisable. The locations for incision are the same as for aspiration, though most recent authorities prefer the xiphocostal region. This may be done by means of a vertical or transverse incision, or one parallel to the seventh costal cartilage. After laying back or removing entirely a section of the seventh costal cartilage, the internal mammary vessels are pushed toward the middle or divided; the xiphocostal space broken into; the triangularis sterni, intercostal muscle, and membranes divided. The pericardium can then be seen, incised, and drained. Coutts and Rowlands give a good account of the various operations recommended. (For further details and bibliography see Dock, G., "British Medical Journal," 1906, vol. ii, p. 1026.)—Ed.]

ADHESIONS BETWEEN THE HEART AND THE PERICARDIUM.

Pathologic Anatomy.—Adhesions are not at all infrequently the result of pericarditis, for they are often enough found at autopsies, although they are very rarely recognized clinically.

When a pericarditis has produced a certain amount of delicate or strong granulation tissue and newly formed vessels, at opposite surfaces of the pericardium, the two layers may come into contact either immediately, or upon the absorption of the intervening exudate, the vessels of one side may unite with those of the opposite side, and thus adhesions may develop. These may occupy small or large areas, or even the entire surface of the heart, and so cause a complete obliteration of the pericardial cavity. At first these adhesions may be so delicate that they may be easily separated; later they become tough, resistant, and of considerable thickness. In some cases there may be one or more cord-like formations, more or less long and tough, which pass through the pericardial cavity. At times one finds a long projection of connective tissue on one side, and at a corresponding place on the other surface a shorter process, so that one gains the impression that there has been a solution of continuity in the adhesion. Remains of the fluid exudate, of fibrin, and of inspissated pus may be found in the interstices between adhesions. A deposit of calcium salts may take place over considerable areas, even over the entire surface of the heart.

Thus, in a case reported by Rivet, in a woman aged seventy-four years who had had general dropsy, regular but weak heart's action, muffled sounds, but no murmurs, the autopsy showed a hypertrophied heart which was inclosed in a veritable lime cuirass 1 cm. thick within which the pericardial layers had become fused. The incrustation was incomplete in only a few spots.

One condition for the development of adhesions is a certain amount of quiet in the heart's action, and we have already seen that, in the course of pericarditis, the heart-beat is less vigorous, whereby the formation of adhesions is undoubtedly promoted. If the inflammation disappears and the heart gradually grows stronger, so that by its vigorous beat there may be a loosening and stretching of the adhesions, these may

be completely destroyed by the increased motion of the heart. Possibly the reappearance of a friction-sound may be dependent upon this circumstance, for I have noted this, but only for a short time, in a number of cases at the end of a pericarditis when the patient had become practically well. The adhesions may not only develop as the termination of an acute pericarditis, but also as the result of the insidious form, so that the time of their appearance cannot be determined in any way at our command, just as similar chronic adhesions very frequently also form in the pleura, less often in the peritoneum, and there too may lead to the complete obliteration of these cavities.

A consideration of the condition of the heart muscle is also of importance. This tissue may be found to be perfectly healthy in some cases, but, as a rule, it is degenerated and the heart cavity is dilated when the disease has lasted long, and has been accompanied by extensive adhesions. At times there is also a partial or complete hypertrophy of the heart.

Recalling what has been said about the condition of the heart muscle in pericarditis, it is easy to understand that in the presence of adhesions there is a more or less wide-spread affection of the myocardium from without inward, in the form of fatty or fibroid degeneration. The latter may in turn produce disturbances in the circulation which influence the nutrition of the heart muscle. But even in cases in which no such severe changes have occurred in the heart muscle, this tissue often appears remarkably soft and friable at autopsy. It is easily seen that such a heart muscle would readily yield to pressure upon its walls from within outward, and that, therefore, a dilatation of the heart cavity could take place.

Why is cardiac hypertrophy relatively rare in these cases? In determining its existence one must, first of all, ascertain whether or not a hypertrophy had been present previously, and especially whether it had been caused by a coincident valvular affection. The fact that adhesions, as such, do not easily produce a hypertrophy is explained when we remember that adhesions produce an entirely different set of conditions from that in valvular disease. In the latter the heart is obliged to overcome an abnormal resistance, and, therefore, to develop a greater propulsive power. But when there are adhesions, the heart being hampered in its motion, is simply obliged to exert itself more in order to be able to contract. This increased labor will easily weaken it, and, therefore, a dilatation will be more apt to take place, especially because in most instances there is, at the same time, a disease of the heart muscle itself. If the adhesion is so wide-spread that the heart cannot contract completely around its contents, that is, cannot drive out all the blood at each systole, the ventricle is obliged to increase its action in order to overcome this difficulty. But on account of the condition of the heart muscle and the difficulty in movement due to adhesions, heart weakness and the severe conditions which we shall discuss later will usually occur in such cases. If adhesions also exist between the pericardium and the pleura and between the costal and pulmonary pleura, they need not necessarily be due to the same cause nor appear at the same time, but may be independent of the pericarditis which has produced pericardial adhesions and may have appeared either earlier or later. It is important to remember this, for it explains the fact that the signs over the heart may be different at different times of observation.

Symptoms.—Pericardial adhesions do not present a definite clinical picture. At least we have not as yet been able to erect such a picture, for the reason that our knowledge of this condition is by no means complete. Although Lancisi removed the essential errors which had been held concerning the pathologic condition underlying these adhesions, and although Morgagni added to our knowledge of the pathology and devoted himself to the study of the clinical manifestations, it is only recently that this subject has been thoroughly studied. The numerous observations on living subjects and the comparisons with autopsies, with the remarkably logical conclusions which Skoda has given us, constitute the chief basis for our present knowledge of the subject. Since Skoda, however, important observations by Traube and by Friedreich have shown how complicated these conditions are, and have proved that a satisfactory explanation of all the clinical signs is, as yet, impossible. The chief difficulty lies in the difference of opinions as to the movements of the heart. Although all observers are at present agreed that during systole there takes place not only an alteration in the shape and form of the heart, but also a change in its position, yet opinions are still widely divergent on the question as to whether this displacement takes place in virtue of the recoil or through the straightening of the vessels. I cannot discuss this in detail at present, but remark that I still adhere to the former opinion, because it enables us to explain with satisfaction all the phenomena observed during the movements of the heart. Another difficulty lies in the fact that, while there are many very ingenious theoretic arguments upon this question, there is still a lack of comparisons of that which has taken place during life with the corresponding autopsies, and, therefore, that the subject has not been thoroughly studied. Still another difficulty, finally, is found in the fact that in the present state of our knowledge when we speak of adhesions of the heart and pericardium we include not only the adhesions between the serous pericardial layers, but also those between the outer pericardium and the surrounding tissues, and even those in the mediastinal pleura and the costal pleura, and even certain changes at the edges of the lungs.

Observations have shown—and all authors agree upon this—that there may be partial or total, loose or dense adhesions, which do not produce any symptoms during life, even if the patient is watched for a long time, and that, in other cases, even slight adhesions may be followed by distinct symptoms, or that, finally, the entire clinical picture may correspond exactly to that seen in a case of uncompensated valvular disease or with purely myocardial changes. It is then necessary, therefore, to analyze carefully the individual symptoms which appear in cases of adhesions between the heart and the pericardium, and to select those which are characteristic for this disease in order to determine to what extent a definite diagnosis may be made in such cases.

Skoda called attention to the fact that the prominence in the cardiac region may be looked upon as a symptom of adhesions between the heart and the pericardium, or between the latter and the wall of the thorax, provided this prominence is not caused by a fresh pericardial exudate. I have spoken of this sign in the discussion on the symptoms of pericarditis, and called attention to the fact that this prominence in the cardiac region can only take place in those cases in which there is a diseased condition of the thoracic walls in addition to an affection of the pericardium. It is very probable that permanent adhesions between the tissues men-

tioned will remain as the outcome of severe inflammatory conditions. It is possible that this form of pericardial inflammation may disappear after it has produced a prominence, that the exudate may become absorbed, and that the prominence may remain, but we have as yet no definite observations to determine this accurately. If we could exclude such very improbable cases, the prominence of the cardiac region might be regarded as a sign of pericardial adhesions.

No matter what view we take of the cause of the apex-beat, it is clear that the heart may be so hampered by adhesions between it and the pericardium that its impulse is weakened and finally entirely disappears. Yet this sign is by no means characteristic for the condition in question, for we know that the apex-beat may be absent even in perfectly healthy persons, without any discoverable cause, and also in a variety of diseases which I cannot discuss here at length. The condition of the myocardium is especially important. Conditions are different when one has known the patient for some time and when a distinct apex-beat had formerly been present, or when the latter has disappeared during the course of, or upon the termination of, a pericarditis. If we do not feel any apex-beat, and if we can prove with certainty that a pericarditis had been present, the absence of the impulse may also be of some value in diagnosis. I have had under observation a case in which a young girl, who came from abroad with a cirrhosis of the liver, showed absolutely no apex-beat, but whose physician answered our inquiries by saying that he had made a diagnosis of pericarditis some time previously from the presence of loud friction-sounds over the heart. This alone would not, of course, be sufficient to prove the presence of adhesions; for a pericarditis may also be followed by disease of the heart muscle, resulting in a weakening of the apex-beat. The decision must be based in such cases upon the consideration of other symptoms, to be discussed further on.

In spite of adhesions, a distinct apex-beat may be present when there is also a hypertrophy of the heart, either as a sequel of the adhesions, or, what is more probable, a hypertrophy which existed before the adhesions developed.

Under normal conditions the heart, as we know, changes its position with the position of the patient, according to the laws of gravity, as we can show by the change in the position of the apex-beat or the alterations in the area of dulness. The displacement of the heart to the left is especially clearly marked under normal conditions. When the heart is fixed by adhesions, it is evident that it cannot take part in changes in the position of the body. The apex-beat will remain in the same place, and the area of dulness will be unchanged. However, this is not a sufficient proof of the existence of adhesions between the heart and the pericardium, because this absence of a displacement of the heart may be seen in the same manner when obstacles against a change of position exist in the neighborhood of that organ, especially adhesions between the mediastinal and costal pleura, adhesions between the lungs and thoracic wall, etc. It is at times possible to prove the presence or absence of these conditions, but it is not always possible to be absolutely sure of their existence, and we shall discuss this particular phase of the subject somewhat more in detail later on.

It has been stated, furthermore, that with adhesions between the heart and the pericardium the area of dulness remains unchanged during

inspiration. It is well known that under normal conditions we can often show very distinctly, by percussion, how the resonant note moves downward with every inspiration. It is said that this does not take place in the presence of adhesions between the heart and the pericardium. This statement is evidently not correct as expressed here, for there is no reason why, in case of a simple adhesion of the serous layers of the pericardium, the lung should not be able to pass between the heart and the thoracic wall during inspiration just as easily as in normal conditions. If there is an adhesion between the outer layer of the pericardium or between the mediastinal pleura and the costal pleura, the lung will certainly not be able to cover the heart in the manner described during each inspiration. But the same conditions will obtain when the border of the lung is adherent to the thoracic wall, or when there is an obstacle to the distention of the lung from any cause whatever. Such changes in the pleura and in the lung are by no means rare, and they may be demonstrated very easily in many cases by the history of the case and by percussion and auscultation.

Tuczek, and especially Riegel, have also called attention to the fact that a diagnosis of adhesions may be made even when the adhesions are relatively delicate and stringy and the borders of the lung are still movable. They say that, in the presence of adhesions between the border of the lung and the external pericardium, the apex-beat is weaker during expiration, while under normal conditions it is weaker during inspiration, although not markedly in every case. Both authors assume that the motility of the heart is hindered by such adhesions and their greater tension at the moment of expiration. This is, indeed, possible, but only on condition that the lung is movable. The suspicion of this condition is rendered more probable when the weakening of the apex-beat during expiration appears after a disease in the tissues concerned, which has been made evident through the history of the case or through our own observation.

It is obvious, from these explanations, that no symptom can be considered by itself, but that the phenomena observed when the patient changes his position must be compared with the result of percussion during respiration.

Systolic retraction is considered the chief symptom of adhesions between the heart and the pericardium. I have left the consideration of this sign to the last because I thought it important to make clear the views regarding the other symptoms. We must emphasize, to begin with, that the symptom under discussion consists of a retraction in the neighborhood of the heart apex, and that only in specially severe cases there is, in addition, a retraction of the neighboring intercostal spaces, and even of the lower portion of the sternum. It is necessary to remember the true significance of systolic retractions at the base of the heart in the second, third, and even fourth intercostal space on the left side, and even in the epigastrium when, at the same time, the heart apex is felt distinctly and vigorously beating in its proper intercostal space. Such retractions may occur under perfectly normal conditions; they may, in fact, be the expression of a completely free mobility of the heart and of a marked change in position of the heart from above downward, from behind forward, and from right to left, but for some reason the space which is abandoned by the heart is not filled with lung and, therefore, a vacuum is created which demands the retrac-

tion of the intercostal spaces and the epigastrium. These are, therefore, normal systolic phenomena of retraction.

It is different when a systolic retraction takes place at the apex instead of a systolic impulse. We have already spoken in detail concerning the significance of a weakening of, or an absence of, the systolic expansion. The systolic retraction is to be looked upon as an expression of impeded locomotion of the heart without a compensating occupation of the space to be filled.

Skoda has expressed himself most accurately on this subject, and when one has had an opportunity to observe his acumen; when one remembers with what collosal industry he studied every case for years, and made comparisons between what he observed during life and the findings at autopsy, one must needs place great value upon the conclusions of this clinician. For a long time Skoda's views prevailed until they were placed in doubt by the observations of Traube, Friedreich, Riegel, and Galvagni. Let us hear, first of all, what Skoda has to say: A simple adhesion between the heart and the pericardium is not capable of so fixing the heart that a sufficient interference in the commotion of the latter will be produced, for the pericardium will, to a certain degree, take part in the movements of the heart, and even in the presence of adhesions the diminution in the volume of this organ during systole will not prevent the lung from filling the space formed when the heart apex nears the base of the heart. However, matters are reversed when there are adhesions between the outer surface of the pericardium and the thorax, or between the mediastinal and the costal pleura. It makes no difference whether the lung is adherent at the same time or not. What occurs, now, during systole? The heart must become smaller in this phase of the cycle; it must approach the base of the heart, for only in this manner can the blood be driven out—provided, of course, that the heart possess a sufficient degree of contraction. If the heart is fixed through the existence of the adhesions described, the apex will tend to approach the base, but cannot do so unhindered, owing to its fixation. Therefore, as the space which the heart apex leaves cannot be filled out by lung (inasmuch as the lung now cannot pass between the heart and the chest-wall), there will be a retraction or depression of the thoracic walls opposite the apex. I shall come to speak of this distinction again.

Then came two observations by Traube, later one by Riegel, another by Friedreich, and finally some by Galvagni, which were intended to controvert the theory of Skoda. These authors claimed that the systolic retraction at the apex is not a sign of adhesions between the heart and the pericardium, or at least not in the sense accepted by Skoda, for retraction may take place with a perfectly free heart (Friedreich), or in the presence of a marked aortic stenosis, or even in old people as the result of arteriosclerosis (Galvagni), or (Traube) on account of a certain amount of fixation of the heart, but in a much more simple manner than has been described by Skoda.

It is necessary to discuss these statements more in detail, although very briefly. I shall do this in the order which seems to be the simplest.

According to Galvagni, a sclerotic condition of the aorta prevents the stretching of this vessel during the systole of the heart—the latter organ cannot move downward and from its contraction causes a systolic retraction. It is impossible for me to imagine that the mechanism is so simple. One must remember how many cases of arteriosclerosis occur among

old people in whom there is no retraction whatever. Something else must be present, and this, in my opinion, is the adhesion of the lung in such a manner that it is no longer able to fill out the space left by the heart apex during systole. I must maintain the same view in opposition to the observation of Friedreich: In a case of marked aortic stenosis and hypertrophy of the left ventricle he noted a distinct systolic retraction in the intercostal space corresponding to the heart apex. At the autopsy the heart was found to be perfectly free from adhesions with the pericardial sac. Friedreich assumed that the normal stretching and lengthening of the aortic arch did not take place in a sufficient degree or with sufficient force on account of the smaller amount of blood slowly streaming into this vessel, so that the hypertrophic heart did not move downward and to the left to a sufficient extent, and therefore a retraction took place during the systole. In my opinion, in such a case there could only be an absence of the apex-beat, and not also a retraction, provided the lung was able to enter into the space created by the contraction of the heart. Unfortunately, I do not find any data in Friedreich's report concerning the condition of the lung, and we must assume that some other cause existed for the systolic retraction which he observed. How often we see the most marked cases of aortic stenosis without the retraction over the apex which he described?

The same may be said of the important observation of Traube. He believed that no extensive adhesions are necessary to produce a systolic retraction, but that a single strand between the parietal and visceral pericardium suffices, or merely an abnormal fold between both layers. He admits, however, that the locality of these anomalies is an important consideration, and I, therefore, shall give you his first case in full. Traube* says: "A band-like strip of old connective tissue, three-quarters of an inch wide and three-eighths of an inch long, and very tough, passes close under the point of origin of the pulmonary artery, at the level of the second left intercostal space, from the conus of the right ventricle to the anterior wall of the pericardium. If the pericardium be fixed in this region, we can convince ourselves that the conus of the ventricle can only move slightly downward and to the left. It is here only that the heart and the pericardium are closely adherent to each other."

In the second case reported by Traube an abnormal fold passed between the upper end of the pulmonary artery and the left auricle to the posterior wall of the pericardium and was said to have prevented the systolic movement of the heart forward, downward, and to the left, and thus to have produced a systolic retraction. We cannot doubt the correctness of Traube's observation, but are we not entitled to question the explanation which he gives? When we consider what extensive adhesions are often present, even at the base of the heart, without producing any such disturbances of motion that a systolic retraction takes place, we must ask ourselves whether other causes did not contribute in this instance to the development of the retraction. Assuming that the fixation of the base of the heart was complete in the first case, we still must believe that the space created by the contraction of the heart was so well filled by the lung that no retraction could have occurred. Traube says, in fact: "The right lung is closely united with the parietal pleura over a large area by connective tissue infiltrated with serum. The left lung, on the other hand, is adherent only in a few places." In the second

* *Loc. cit.*, p. 344, *et seq.*

case, also, he says: "The left lung is adherent to the thoracic wall in a few places, but easily detachable." It seems to me, therefore, that the condition of the lung in these cases was of great importance. As regards the second case, it is really very difficult to admit that such a fixation of the heart could have taken place from the fold alone, that a sufficient disturbance in the motility of the heart could be produced. If one takes the trouble to open a thorax from behind and to expose in this manner the posterior wall of the pericardium, one can see very distinctly how small the area is where the fibrous pericardium is adherent to the diaphragm, and how readily, therefore, the pericardium must yield to the movements of the heart. Admitting, therefore, the correctness of all the observations, I still believe that the anatomic findings which have been thus far disclosed do not offer a sufficient explanation for the systolic retraction, and that it will be necessary to study this question much more thoroughly on the basis of a much larger material more completely investigated. The effect of these strands of connective tissue, their length, and their direction must be very cautiously considered in deciding the question. If such a strand runs from right to left and from above downward and is 2 cm. long, as in the case reported by Tuczek, it is evident that a considerable amount of contraction of the left ventricle is still possible, for the heart can certainly contract more than 2 cm., and inasmuch as, when the contraction exceeds that limit, the strand will be turned about, finding, so to speak, its fulcrum in the parietal pericardium. Summing up the matter, I can only say that I agree with Skoda that an adhesion at the base can only produce a systolic retraction when, the apex being free, the lung is so fixed that it cannot take the place of the contracted part.

A case which I had occasion to observe corroborates the correctness of my remarks concerning the insufficiency of the anatomic material for a decision of the question under consideration, and shows something of the difficulties in interpreting the observations made in this connection. In a case of pleurisy with an enormous amount of exudate on the left side the heart had been pushed 12 cm. beyond the right sternal margin. A systolic expansion was seen and felt distinctly in the fourth intercostal space in the right axillary line. The explanation of this may be very simple: It may be assumed that this expansion is due to the prominence of a portion of the right ventricle beating at this place as the result of the displacement of the heart. However, a distinct systolic retraction was seen in the epigastrium—a sign which I have never seen before, in spite of my varied experience in these cases. I must admit that I am not able to explain this phenomenon. We must assume that the apex was situated behind the junction of the costal cartilages with the sternum. Was the displaced heart adherent to the thoracic wall? I did consider such a possibility, but after paracentesis had removed 2500 c.c. of purulent exudate, the heart had returned toward the median line, and now neither a pulsation nor a retraction could be observed.

It is impossible to understand why an adhesion of the heart at the apex should not also produce a retraction of the intercostal spaces, provided that there are also adhesions with neighboring structures. We cannot regard the validity of the explanation that in this case the base of the heart was drawn downward during contraction and thus the space filled, which is created during systole; for retraction must necessarily follow above at the base, as the stretching of the vessels would certainly

not suffice to fill this space again, else we would observe a retraction of the jugular fossa corresponding to the flattening of the aortic arch, a phenomenon which no one has as yet observed. After all, I do not think that the stretching of the vessels which is so often invoked is of great importance. I should like to call attention to the fact that in such cases we might just as well hear a systolic murmur over the aorta as in those instances in which this vessel has been distorted, but such a murmur no one has ever reported in cases of this class.

In the very rare instances in which there are extensive adhesions between the heart and the anterior chest-wall, and at the same time a close adhesion of the former to the spinal column, the mobility of the heart must obviously be very markedly affected, and the systolic contraction of the organ must show itself in the most striking manner. In these cases *the lower portion of the sternum is actually drawn inward* together with the neighboring ribs, more or less forcibly in proportion as the heart has retained its power of contraction.

For the present we must admit that we do not know what effect extensive adhesions of the lower surface of the heart with the diaphragm alone have upon systolic retraction, and especially the high degrees of that observed by Friedreich. It is scarcely possible to conceive that the diaphragm can be so strongly drawn upward with the contraction of the heart—that its attachments to the anterior chest-wall are drawn inward. It seems probable that in these instances the space created by the contraction of the heart will be filled by the lungs on both sides, and that whenever this is impossible the outer air-pressure will press the thoracic wall inward. The more marked the systolic retraction is, the more energetic is the return of the thoracic wall to its old position at the moment of diastole, and, therefore, we find that the intercostal space corresponding with the apex of the heart, and in some cases also the surrounding thoracic surface, is pushed outward with the diastole and is drawn in with the systole. The retraction of the thoracic wall at times occurs with a very marked jerk, so that the hand which is placed over this region feels a distinct shock, and even a sound is heard which, immediately following the second heart-sound, may produce a splitting or reduplication of the latter. [A similar disappointment applies to "Broadbent's sign," a systolic retraction of the lower (eleventh and twelfth) ribs of the left back, synchronous with the heart-beat. This has been shown to be a comparatively frequent phenomenon, occurring not only in pericarditis, but also in hypertrophy of the heart.*—Ed.]

The inspection of the *veins of the neck* may be of great value in the diagnosis of the condition under discussion, as was first pointed out by Skoda, and more completely described by Friedreich. The veins of the neck, which gradually reach a marked distention at the moment of systole, collapse so suddenly at the moment of diastole that they disappear completely, or even that the tissues over them are depressed. It is evident that the diastolic collapse is due to the sudden diastolic rebound of the thoracic wall. The more vigorously this return of the chest to its full distention takes place, the more markedly and more suddenly does the phenomenon at the veins of the neck appear, so that this sign is seen only in the presence of well-formed and firm adhesions of the heart to the anterior chest-wall. The expansion of the thorax at the moment when the chest-wall returns to its full distention after it has been retracted

* Alice W. Tallant, "Boston Med. and Surg. Journal," vol. cli, October 27, 1904.

by the systolic contraction produces such an aspirating effect upon the column of blood in the venæ cavæ and in the veins of the neck that the blood-pressure is suddenly lowered in these vessels. Friedreich, evidently under the influence of his opinion about the adhesions of the heart with the diaphragm, believes that with the sinking back of the thoracic wall the diaphragm is also flattened, and that the superior vena cava is thereby lengthened. This does not appear possible when we remember that during the systolic contraction of the heart its base is drawn down, and therefore a lengthening of the vessels takes place. The movement of the chest-wall alone is certainly sufficient to explain the phenomenon.

The examination of the *pulse* gives us a clue to the power of contraction of the heart. But as this depends not only upon the presence of adhesions around the heart, but also upon the condition of the heart muscle the pulse phenomena cannot have a direct relation to the adhesions.

Riess had occasion to observe several cases in which *metallic heart tones* were heard as the result of adhesions between the heart and the pericardium, and explains this occurrence by the consonance of the heart-sounds in a dilated stomach. He regards this phenomenon as a certain sign of adhesions between the heart and the pericardium. But when we consider that this peculiar metallic sound occurs at times simply as the result of a markedly distended stomach, as a pure phenomenon of consonance, or as a splashing sound which arises through the blow of a hypertrophic heart against the stomach, we can at most admit that this manifestation is more frequently heard when there is an adhesion between the heart and the pericardium, provided the other conditions which are necessary for its development are also present.

Finally, a series of symptoms may be observed in these cases, which are in no way characteristic of the disease under consideration: *Palpitation* of the heart, a feeling of tightness and *oppression* in the chest, a more or less marked *dyspnea*, *cyanosis*, and, finally, *dropsical phenomena*. These conditions may, it is true, be produced by the adhesions alone, which diminish the contractile power of the heart and the perfect emptying of its cavities, but they may also be the result of disease of the heart muscle alone or of uncompensated valvular disease.

It is not always easy to interpret the dropsical symptoms, and at times it becomes very difficult. Thus it is not at all impossible that the case of the young girl which has been mentioned several times should have been interpreted in the same manner as an observation of Bamberger and a similar case reported by Weinberg, namely, that the ascites which occurred before the general dropsy had developed was due to adhesions between the heart and the pericardium, which produced a kink in the inferior vena cava, whereby stasis was first induced in the hepatic veins, followed by changes in the liver substance, and thus by stasis in the portal veins.

[This condition has become known as Pick's syndrome.* The striking feature of the dropsy is that it begins in the peritoneum instead of the dependent parts, as in heart weakness. Just how much local or widespread serositis is present in these cases is still unsettled.—Ed.]

Diagnosis.—Extensive adhesions between the heart and the pericardium may occur without giving any symptoms whatever. The diagnosis in such cases can only be made at the autopsy. In other instances the dis-

* Friedel Pick, "Ueber chronische unter dem Bilde der Lebercirrhose verlaufende Pericarditis," "Zeitschrift für klin. Med.," vol. xxix.

ease may be so masked by the manifestations of other maladies that nothing reveals its presence. Among the diseases which may mask the symptoms of cardiac adhesions are found not only affections of the heart itself, especially of the valvular apparatus, but also diseases of the respiratory tract. We have already made clear that the majority of the subjective symptoms, as well as a great many of the objective signs, are common to disorders of both systems. Furthermore, it is possible that a series of symptoms may be present which may correspond to several diseases without presenting a definite picture. Let us assume, for example, that the patient is brought to us with a somewhat increased area of dulness; with impure heart-sounds, edema of the ankles, and with complaints of palpitation of the heart and shortness of breath. We would be justified in thinking of adhesions between the heart and the pericardium only after having excluded all other possibilities which might produce a similar picture, such as, for example, renal disease, disease of the heart muscle, valvular affections without any murmurs, aneurysm, and arteriosclerosis. We know, however, how difficult it is to exclude these diseases. Even a careful history will not enable us to draw a positive conclusion; for, let us assume that the patient has gone through a pericarditis in connection with an attack of acute articular rheumatism. This pericarditis may have been complicated with an endocarditis, on one hand, and with a disease of the heart muscle, on the other. Possibly we can make the correct diagnosis even in such a case when, in the further course of the disease, more definite symptoms develop. Thus we have heard, when we discussed the individual symptoms in detail, that a weak heart-beat, or one which cannot be felt, is not in itself a sign of adhesions. But if, in the presence of the above-described symptom-complex, the heart impulse is at first very marked, or if we have heard from a reliable source that it had previously been marked, the diminution in its intensity or the disappearance of the apex-beat would justify us in assuming the presence of adhesions. However, we must not forget that even in this contingency the same sequence of events could be the result of a gradual degeneration of the heart muscle. We, therefore, shall only be able to make a probable diagnosis, or to say that one of the two conditions may be present.

It is another matter when, with the same history and symptoms, the apex-beat gradually becomes less marked, although the pulse remains vigorous. This should be regarded as a proof that the contractile power of the heart has remained good, but that its mobility has been lessened. Such a combination of signs might be observed even in the presence of valvular disease, and, in fact, such cases have been reported. Let us assume that an aortic insufficiency has developed as the result of a rheumatic polyarthritis, and also a pericarditis, the symptoms of which have gradually disappeared, and instead of the vigorous heart impulse which corresponds to the valvular disease, the apex-beat gradually becomes less vigorous. In such a case we can draw the same conclusions as in that mentioned above, and the same may be said when the pulse grows weak. We can say, therefore, in general, that a weakening of the apex-beat or of the pulse when it occurs gradually may be regarded a sign of adhesions between the heart and the pericardium, provided no other causes can be found for these phenomena, especially when there are other causes which may be expected to strengthen the apex-beat and the pulse.

It is of different import when we see a retraction of the intercostal space corresponding to the apex of the heart. We shall at once be justified, as we have explained above, in thinking of the presence of adhesions between the heart and the pericardium. For, even if those objections which we have discussed in detail in the foregoing paragraph be admitted, we could easily prove the presence or the absence of a high degree of aortic stenosis. The question remains to be answered, what form of adhesions is present when there is a simple retraction of the region of the apex? I have shown in detail that adhesions between the visceral and the parietal pericardium alone are not sufficient to produce this sign. I have expressed my doubts about the view that this sign may be produced by a fibrous band or by an accidental fold between the two layers, or by a simple adhesion at the base of the heart, and I have emphasized the fact that in order to produce apical retraction a more intimate fixation of the heart (therefore a more extensive adhesion to the chest-wall, or an adherence of the border of the lung) is necessary. But if the retraction is wide-spread, and if there is a systolic retraction of the lower part of the sternum and the corresponding ribs, there can be nothing but an adhesion, and an intense one at that, of the heart with the pericardium, the latter with the thoracic wall, the vertebræ, and, most probably, with the neighboring lung also. No other condition could cause these signs. Another support for the correctness of this diagnosis in the more severe cases is the diastolic collapse of the veins previously mentioned.

I should like to call attention again to the fact that, for purposes of diagnosis, we should not only estimate the distinctness with which these signs appear, but also that we may, to a certain extent, estimate the presence of a weakened or diseased heart muscle from the degree of the systolic retraction.

I do not think it is necessary to enter more in detail into the differential diagnosis between adhesions without characteristic symptoms and a case of myocardial disease. Such a differentiation is impossible, so long as no characteristic symptom is present, as both diseases have the same signs and the same causes and histories. The same may be said of the differentiation between adhesions and chronic pericardial exudates. If the reader will recall what I said of the cardiac signs in the girl with cirrhosis of the liver, he will at once clearly understand that there could have been a chronic pericardial exudate, just as well as adhesions between the heart and the pericardium, and that these two conditions could not be distinguished from each other unless we could demonstrate the occurrence of a systolic retraction instead of absence of the heart-beat to prove adhesions or until we could distinguish a friction-sound or a variability in the areas of dulness to show the presence of a pericardial exudate.

The mere uncovering of the heart by retracted, infiltrated, or adherent borders of the lung will produce an enlargement of the area of dulness, but this sign can only be employed in differentiating adhesions between the heart and the pericardium when the apex-beat is at the same time impalpable. In such cases we must take into consideration the history of the case, the presence of a disease of the lung, the altered mobility of the heart, and all those factors which we have mentioned above, in speaking of cases of adhesions which are not characteristic.

On the whole, we may conclude that the diagnosis of this condition constitutes one of the most interesting problems in internal medicine; that it always requires a very careful examination; that, possibly for

this reason, adhesions between the heart and the pericardium are so rarely made out during life; and that we may say truly that the subject has by no means been completely worked out, but requires a great deal of impartial observation upon a large amount of material.

Course and Prognosis.—Although adhesions between the heart and the pericardium, as such, may in many cases run their course without any symptoms, and, therefore, it may be said that their presence has no influence upon the prognosis, this is not always true of the whole course, for the adhesions may, after a time, become more dense and firm; the heart muscle may become affected, and may no longer have the necessary contractile power. When these stages are reached, there appears at once the entire series of symptoms which we have already discussed, resembling those of a non-compensated disease of the heart, and which gradually grow more and more severe. In such cases much depends upon the ability of the patient to regulate his life. If he is in a position to place but slight demands upon his heart, the prognosis will be very different from that in the case of a poor man dependent upon the labor of his hands for his livelihood, and so is early doomed to that general invalidism which threatens all patients with heart disease. It is necessary, therefore, in forecasting the further course of the disease, to take account of the strength of the apex-beat; of the extent of the systolic retraction; of the peculiarities of the pulse, and of the possible presence of circulatory disturbances or of murmurs over the heart. The heart murmurs in these cases depend upon degeneration of the heart muscle, gradually giving rise to a dilatation of the cardiac cavity, and this, in turn, may lead to a relative insufficiency of either the mitral or the tricuspid valve. It is easy to see what difficulties we may encounter in interpreting the significance of murmurs when these are present at the time the patient first comes under observation.

Treatment.—Let us recall the difficulties which we met in treating pericardial exudates. On the one hand, the effort to secure the absorption of the exudate and to produce the obliteration of the pericardial cavity, even by the employment of irritating substances; but, on the other hand, the fear that the adhesions between the pericardial layers might extend beyond these structures. It certainly would be desirable to prevent this, but I need not say that, unfortunately, this is impossible.

Our object, therefore, can only be to treat the condition after it has developed. In the first place, we must seek to limit the demands made upon the heart to the very lowest possible degree. We must secure for the patient physical and mental rest, appropriate diet, and the avoidance of stimulating beverages. We must administer appropriate drugs in case there is excited heart's action or cardiac weakness, especially digitalis or the tincture of strophanthus, in order to regulate the work of the organ and to render its contractions as effective as possible. It may be necessary to administer these drugs at various times during the course of the disease, but in the intervals we may give quinin in moderate doses with considerable advantage.

When severe circulatory disturbances occur, or, still more, hydropic manifestations, the therapeutic measures mentioned above must be increased, and cardiac stimulants must be systematically administered, while appropriate remedies are to be employed for the other symptoms. Thus, for example, the dyspnea may necessitate the use of a narcotic; the dropsy, the employment of a diuretic, etc.

INDURATIVE MEDIASTINOPERICARDITIS.

I have mentioned at various times how a pericarditis may not only involve the fibrous pericardium, but may also spread to the neighboring tissues, especially to the mediastinal, costal, and even the pulmonary pleura, to the diaphragm, and to the large vessels. I have also discussed in detail how such a mediastinopulmonary or mediastinocostal pleurisy may simulate an ordinary pericarditis, and have shown what confusion has arisen through this similarity. I have also dwelt upon the symptoms and the difficulties in the diagnosis of adhesions between that pulmonary pleura which lies immediately adjacent to the heart, on the one hand, and the mediastinal or the costal pleura, on the other. I have emphasized all the points which could possibly be of importance in differentiating these conditions. There remains to be discussed another condition which is very rare, at least in its well-marked form—the extension of a pericarditis upon the more remote portions of the mediastinum.

In circumstances for which we as yet have no definite explanation, indurative inflammation, in severe forms of pericarditis, may spread from the base of the heart to the cellular tissue of the mediastinum and the adjacent pleura, and may give rise to other phenomena, as first described by Griesinger (reported by Wiedemann) and later by Kussmaul, who reported several cases of this kind and carefully analyzed the symptoms.

As already stated, definite causes for this extension cannot be specified. It is possible that, as in the case of Griesinger, an injury, such as a vigorous pressure of the chest against a tool in working, may lead to this form of inflammation, or that it may be due, in other instances, to tuberculosis. It is also possible, as Kussmaul remarks, that the milder forms of this affection which do not present such distinct symptoms occur more frequently than is generally supposed.

The changes in the pericardium itself are those which have already been discussed, except that they are especially well marked. When the fibrous pericardium is most markedly thickened, there is a complete obliteration of the sac, together with extensive changes in, and thickening of, the serous pericardium, extending more or less deeply into the subserous connective tissue of the heart. Between the layers of the pericardium there may be sacculated pus-cavities, or places filled in with friable yellowish fibrin. The heart muscle itself is flabby and easily torn; its cavity dilated. Intimate adhesions may be present between the fibrous pericardium and the sternum, along with layers of fibrin and a considerable thickening of the connective tissue. Tough fibrous bands may pass upward from the base of the heart toward the ascending aorta, even to its arch and to the three vessels which pass out from it. These structures may be partly compressed, partly constricted, or at times even twisted upon their axes or drawn downward by the adhesions. Similar bands of adhesions may pass to the pulmonary artery and may produce the same changes in this vessel, and also toward the vena cava, the innominate, and the azygos veins, producing marked narrowing of their lumens. In a similar manner extensive adhesions may occur between the fibrous pericardium and the diaphragm, and between the neighboring mediastinal, pulmonary, and costal pleura.

There are, at present, too few cases on record to give a complete

picture of this disease. The chief symptoms are, at any rate, those of the pericarditis which is always present with this condition. But pericarditis alone does not suffice to explain some of the symptoms which have been observed, even at the earlier stages of the mediastinal affection —as, for example, the occurrence of hemoptysis. Further observations are necessary to explain the reasons for the various symptoms which have been observed, and it is only astonishing that such severe changes in the mediastinum do not induce more marked disturbances in the organs contained in this region, as, for example, in the trachea or in the esophagus.

The feeling of oppression, the shortness of breath, the weakening of the apex-beat, together with the feeling of palpitation, the rapid and irregular pulse, the cyanosis, and the gradually developing dropsical phenomena are all symptoms which belong to pericarditis, as such. The same may be said of the usually irregular febrile movement. The intermission of the arterial pulse during inspiration is a remarkable sign which was observed in the case reported by Griesinger (reported by Wiedemann), and also in the three cases so well described by Kussmaul. The latter designated this irregularity of the pulse by the name of "*pulsus paradoxus*" and regarded this symptom as characteristic of the disease in question. It should be discussed somewhat more in detail.

In these cases, without any alteration in the regularity of the heart's action, and without any change in the heart-sounds, every inspiration coincides with a lessened volume in the arterial pulse, or even a disappearance of the same. The explanation of this phenomenon is simple when we remember the intense changes which take place about the great vessels and the manner in which these structures were compressed and surrounded by fibrous bands in the cases reported by Griesinger and Kussmaul. There can be no doubt that these structures produce such traction, especially upon the aortic arch, with every inspiratory depression of the diaphragm, that the lumen of the aorta, which has already been narrowed, must be almost obliterated. I think that it is not of importance what particular forms of adhesions exist, and whether they unite the arch of the aorta with the sternum, or the former with the base of the heart. The question is only whether the adhesions have assumed such directions and attachments that they impede or check the regular flow of the blood-stream during inspiration. The more closely the pericardium is adherent with the diaphragm, the easier it is for the phenomenon in question to develop, provided, of course, the other factors mentioned also come into play.

An intermittent pulse will, therefore, be observed during inspiration when the changes described are present; but, is it seen only in this condition? Hoppe, who first observed this pulse, saw it in a case in which there was only a complete obliteration of the pericardial sac, and other authors, such as Traube and Bäumler, have observed this phenomenon in the presence of simple pericarditis. The case reported by Rendu is of importance; for here this peculiar pulse was observed along with a large pericardial exudate and disappeared after the removal of 970 c.c. of fluid. Here, perhaps, also belongs the case of the young girl with cirrhosis of the liver which I have already mentioned several times. The tracing of her pulse on p. 812 (Fig. 81) shows how distinctly it became smaller with every inspiration.

An intermittent pulse, therefore, only gives us the right to think of a simple pericarditis which has gone on to complete obliteration of the

pericardial sac, but does not necessarily imply the existence of indurative mediastinitis. Finally, inasmuch as this peculiarity of the pulse has been observed in cases of cardiac weakness as the result of other diseases, in consequence of altered pressure in the thoracic cavity, we must say that it cannot be regarded as a perfectly trustworthy sign of the condition under discussion. It may, however, be considered, not only as a very prominent symptom of this disease, but also may be an important diagnostic factor, inasmuch as it is not so well marked in the other conditions mentioned above, which may also give rise to it.

The question has also been raised, whether a *pulsus paradoxus* may not occur in mediastinal tumors. I have had occasion to see a number of such cases, but have never observed this phenomenon in these patients. Theoretically, no objection can be made to the possibility of the occurrence of this pulse in mediastinal tumors, for it may be easily imagined that a tumor may become adherent to the aortic arch in such a manner that the depression of the diaphragm may produce the narrowing of the aortic lumen, just as a corresponding band would do, provided, of course, that the aortic arch is sufficiently movable.

Kussmaul has also observed another sign which is intimately connected with the pathologic condition in question; namely, the swelling of the



Fig. 81.

already distended jugular veins at each inspiration, and even a recurrent wave in these vessels. This phenomenon is also evidently dependent on the retraction which the vena cava or the innominate undergoes during inspiration and the constriction of the lumen of these vessels at that moment. The occurrence and the distinctness of these phenomena are dependent upon the development and arrangement of the adhesions.

Pins describes "a rare musical mediastinal murmur," and believes that this occurs as the result of an adhesive mediastinopericarditis through traction upon a pathologic band passing between the aorta and one of the large bronchi. It is difficult to understand the origin of such a sound in this manner, but Pins may be right in relating the phenomenon in question to the mediastinal adhesions; for the murmur became more distinct during inspiration, and there was paradoxic pulse. But for this very reason it is far more probable that the sound arose through traction and narrowing at the arch of the aorta, or possibly the left subclavian. In further observation of such cases it will be necessary to pay attention to the occurrence of murmurs.

[Wenckebach* calls attention to a sign that he thinks is likely to occur in all cases of adherent pericardium with adhesion to the diaphragm and chest-wall. This is an arrest or even retraction of the lower part of the sternum with its cartilages in inspiration.—Ed.]

* "British Med. Journal," January 12, 1907.

As we see from the symptoms described, the chief diagnostic weight is to be placed upon two phenomena—the *pulsus paradoxus* and the inspiratory swelling of the veins of the neck. Especial importance should be attributed to the coincidence of these two symptoms, for they have not been observed together in a case of simple pericarditis. They may both be present in a case of mediastinal tumor, but here a series of other differential factors would be present—increased area of dulness on the anterior chest-wall; altered position of the heart; swollen lymphatic glands. I do not lay any stress upon the irregularity and the frequency of the pulse, inasmuch as these occur in the most diverse diseases, nor upon the *pulsus differens*, because the latter may occur in both conditions and certainly is very rare, even in tumors. The differentiation will be doubtful only in those cases in which we are still unable to explain the occurrence of *pulsus paradoxus* alone. At all events here, too, the history of the case and the course of the disease will have to be considered and may possibly give important clues.

As regards the course of the disease, it does not differ in any way from that observed in severe cases of pericarditis or of non-compensated valvular disease. Everything that we have said before on this subject applies in these cases, including the prognosis, which is always unfavorable. As we may easily imagine, slight adhesions may become broken down and even disappear, but the severe forms which we have described cannot do so.

The treatment must, of course, be a purely symptomatic one, and must be directed against the individual symptoms, among which the shortness of breath is, again, the most important.

[L. Brauer has made an important addition to the treatment of adherent pericardium, and his operation of "cardiolysis" has been practised by several surgeons, with results that fully justified the treatment. The operation consists in resecting enough of ribs, costal cartilages, and sternum to get access to the heart and then loosen the adhesions. The operation is difficult largely on account of the tumultuous action of the heart, but also from the danger of wounding or tearing veins. The immediate relief to the heart and respiration is very great, and in a number of cases in the five years since Brauer began the operation improvement was most gratifying.*—Ed.]

HYDROPERICARDIUM.

As very often happens in medicine, we cannot give a fixed definition for this lesion. It is easy enough to say "by hydropericardium we mean an increased amount of serous fluid in the pericardial sac which has developed without any inflammatory manifestations." But what constitutes the normal amount? While some observers regard a small amount of fluid, which is not expressed by any definite measure, as physiologically present in the pericardium, others deny this, and claim that the two layers of the pericardium should merely be moist. Hammarsten states that the amount which is found normally, as, for example, in cadavers of executed criminals, is sufficiently large to allow of a chemic examination. Others again consider the fluid which is found at autopsy to be

* L. Brauer, "Archiv für klin. Chirurgie," vol. lxxi, "Annals of Surgery," vol. xxxviii, p. 907. K. F. Wenckebach, "British Medical Journal," January 12, 1907.

the result of the processes which go on during the agony. We can, therefore, only employ the vague expression that a *larger* amount of fluid should be considered as pathologic. In this there may be a number of degrees, and while we have already spoken of the lower limit, the largest amounts which have been found exceed one liter.

The fluid is alkaline in reaction, clear yellow or yellowish-green in color, and is scarcely different from those effusions which are found as transudates in other serous membranes. It contains a large amount of albumin, and, according to Hammarsten, contains more fibrin than other transudates. It contains the ordinary mineral salts and a small amount of urea. Whether sugar is present in it only as an accidental constituent (Grohé), or whether it is generally found, must remain undecided for the present. This is the composition of the transudate, in the majority of cases, but it may be clouded through the presence of cast-off endothelia; it may be colored red or reddish through the admixture of blood-pigment, and it may contain here and there isolated flocculi of fibrin. Distended veins appear upon the pericardium, especially on the epicardium, which is generally pale; the subserous cellular tissue, especially in the grooves of the heart, is usually edematous; the fibrous pericardium may become very thin when there are very large exudates, but when the disease develops slowly and lasts for a considerable length of time, it may also be considerably thickened. In these cases there may also be more or less extensive clouded patches on the visceral pericardium, giving it a certain resemblance to the condition which we have described as the termination of pericarditis with the formation of "tendinous spots."

The large accumulations of fluid are undoubtedly formed during life. It is impossible to say how many of the smaller transudates are to be placed to the account of the agony, and this is especially difficult because no symptoms accompany the development of these pericardial transudates.

The heart muscle may remain entirely unaffected, but it may be hypertrophied as the result of the primary disease, or it may be found weak and flabby or more intensely degenerated, according to the condition which preceded the transudation.

Recklinghausen found accidentally in the body of a person who died of suffocation about 22 c.c. of a whitish milky fluid which he, and also Hasebroeck, after a chemic analysis, considered to be chyle. They assumed that a chylous vessel had burst or that a capillary transudation of chyle had taken place as the result of stasis.

Hydropericardium is not a primary, independent disease, but occurs as a sequel of other affections. The conditions under which it develops may be either local or general. The former are present when there is difficulty in the emptying of the coronary veins or a great distention of the right auricle with blood from any cause whatever, but especially as a sequel of the various valvular diseases, of affections of the respiratory apparatus, such as emphysema, induration of the lung, etc. Further, compression, either by tumors from without or those which arise from the heart muscle; through changes which are produced by tuberculous processes, and finally through endarteritis of the coronary arteries which cause a deficiency in the *vis a tergo*.

All the causes which lead to a general dropsy, which produce a change in the mass of the blood and a defective nutrition of the walls of the blood-

vessels, are responsible for the large transudates in the pericardium. These include, in addition to diseases of the circulatory and respiratory tracts and to renal diseases, a variety of prolonged severe maladies, especially tuberculosis or cancer. As a rule, the accumulation of fluid occurs rather later in the pericardium than in the other serous cavities, although local causes may contribute to the more rapid development and to the greater volume of a transudate.

The so-called *hydrops ex vacuo* can only occur in the pericardium when the space created through the contraction of the heart and the fixation of the pericardial sac cannot be filled in any other way—an event which is extremely unlikely.

Symptoms.—In the greater number of cases hydropericardium runs its course without any symptoms, along with the primary disease, as we have already seen in discussing the causes of this affection. It is only when larger amounts of fluid accumulate in the pericardium that their quantity will be expressed clinically in a purely mechanic manner. The condition of the heart muscle must receive special consideration. With the exception of the phenomena which depend upon the inflammatory process,—such as fever, pain, friction-sounds, the development of adhesions,—the symptoms of hydropericardium are the same as those of pericarditis.

The shape of the area of dulness, on the whole, is found to be the same in this affection as in pericarditis, save that it tends to vary rather more frequently from the typical form which we have described, for the reason that adhesions are very often present, not only in the pericardium itself, but also in the surrounding tissues as the result of those primary diseases which are so frequently noted in cases of hydropericardium. This last circumstance may also be of influence upon the respiratory mobility of the adjacent lung tissue.

The apex-beat is often normal and vigorous, in spite of large accumulations of fluid, and this normal heart-beat is more frequently observed here than in pericarditis, because in the latter disease the heart muscle, as a rule, is more rapidly affected. In hydropericardium the heart impulse may also be found within the left boundary of the area of dulness, and may become distinct when the patient lies on his side or bends forward, while it is not perceptible in the dorsal position.

The heart-sounds may remain unchanged, but in the presence of large amounts of fluid may become muffled, or even disappear. Large pericardial transudates may give rise to compression of the lung and to depression of the diaphragm. The difficulty in filling the heart cavities with blood and the interference with the work of the heart finally result in an engorgement of the venæ cavæ and of the veins of the neck, or of the rest of the body, and thus may lead to cyanosis, which is all the more marked because the blood-supply to the systemic arteries must also suffer, as shown by the small pulse. In this manner hydropericardium itself may lead to an increase of the intensity of the general dropsy. In addition to a feeling of more or less marked oppression, the patients complain chiefly of shortness of breath.

The urine is scanty, concentrated, and may show other changes which are caused by the primary disease.

Diagnosis.—Given the physical signs appropriate to these conditions, it is important to differentiate hydropericardium from pericarditis. But this question can only arise in those cases in which no friction-sounds

are to be heard. A careful history of the case; a consideration of the other symptoms, including the order of their occurrence, the presence or absence of fever, will all contribute to a decision. At all events, the further observation of the patient, noting the course of the disease and the occurrence of friction-sounds with diminishing dulness, will lead to differentiation.

The distinction of pericardial effusions from aneurysms of the aorta, from mediastinal tumors, and from sacculated pleuritic exudates has already been discussed under the heading of Pericarditis. The differentiation of hydropericardium from a hypertrophic heart may offer considerable difficulties in those cases in which the apex-beat is not perceptible—an event which is rare enough in hypertrophied heart. In these cases we are obliged once more to rely upon the consideration of the accompanying conditions, especially upon the examination of the mobility of the area of dulness in changes of position. If the apex-beat is felt within the limit of dulness when the body is bent forward, we are dealing with a hydropericardium. Muffled sounds indicate hydropericardium; endocardial murmurs, hypertrophy of the heart. If these signs are negative or uncertain, then the differentiation cannot be made for the moment; but a clue may be obtained from further observations when the dulness rapidly diminishes, etc. Hypertrophy of the heart with a simultaneous accumulation of a large amount of fluid cannot usually be diagnosticated.

The differentiation may be extremely difficult when there is a markedly dilated right auricle. In a case of this kind which I observed a careful consideration of all the symptoms and physical signs could not lead to a conclusion, although it was very important to make the diagnosis, for therapeutic reasons. The correctness of the interpretations of the physical signs was afterward proved at autopsy. On account of extreme dyspnea, the question arose whether a puncture into the sacculated effusion, which was probably present in the pericardial sac, might give relief. In order to decide this question I undertook an exploratory puncture with a hypodermic syringe and found that there was nothing but an enormously distended right auricle. The operation was perfectly devoid of danger, as I had foreseen, and may be employed for the differentiation of these two conditions.

The **prognosis** of hydropericardium must always be considered as unfavorable, if only for the reason that this affection usually develops when other severe diseases are already present. The addition of a hydropericardium must always be regarded as an unfavorable complication of the primary disease. On the other hand, there is today a possibility of improvement, either temporary or of some duration, even after the development of a dropsy of the pericardium. For, just as modern treatment has attacked and successfully combated accumulations of fluid in other cavities of the body, so it has also been successfully applied to the pericardial cavity.

In addition to the primary disease, the prognosis must take into consideration chiefly the condition of the heart muscle. If we succeed in improving this condition, then the prognosis of the secondary disease becomes more favorable.

The object of treatment in hydropericardium is to combat the primary disease, after which comes the treatment of the dropsy. In this we must have recourse again to all those methods which I have recommended for the absorption of chronic pericardial exudates. Besides milk

diet, the diuretics are of the greatest importance here, as we have stated above. It is also important to keep up the strength of the heart, and for this purpose we should employ the drugs already mentioned. If these prove insufficient; if the heart grows dangerously weak and shortness of breath threatening, then paracentesis is absolutely indicated in order to obtain at least a temporary improvement and to give the weakened heart an opportunity to recover.

HEMOPERICARDIUM.

We have already discussed the occurrence of hemorrhagic exudates in the pericardium. Hemopericardium may be defined as the accumulation of pure blood in the pericardium. This may be caused by injuries from without as well as from within; for example, by sharp objects which have been swallowed. We are interested only in those cases in which the accumulation of blood reaches a high degree, and these are caused chiefly by the rupture of aneurysms of the ascending portions of the aorta which is inclosed in the pericardial sac; by rupture of the heart as the result of fatty degeneration or of inflammatory changes in the muscle, and, more rarely, by the bursting of an aneurysm of one of the coronary arteries. The case of hemorrhage into the pericardium which resulted fatally and which was reported by Eichhorst as a sequel of a tuberculous abscess is so rare that it is probably to be considered as a curiosity. The amount of blood is not in direct proportion to the size of the tear, for the larger this opening is, the more rapidly will the blood gush out and the more quickly will the fatal result occur, while a smaller opening allows of a slow and gradual trickling and thus permits a much greater accumulation of blood.

The danger of these conditions is twofold: interference with the functions of the heart and anemia of the brain. These two factors are intimately connected with the suddenness of the hemorrhage and the amount of blood effused.

The symptoms are so striking that the diagnosis may be easily made: The rapidly developing collapse, the extreme pallor of the skin, the smallness of the pulse, the loss of consciousness, the slight or even more severe convulsions—in a word, those phenomena which are characteristic of severe internal hemorrhage—will easily lead to a correct diagnosis when there are added to them an increased area of heart dulness, the disappearance of the apex-beat, and the muffling of the heart-sounds. A knowledge of the preceding affections from which the patient had been suffering, and which might have led to hemorrhages, will facilitate the recognition of the cause of the attack.

Small amounts of blood may undoubtedly be absorbed, but when larger amounts have become effused, the prognosis is absolutely fatal, for reasons which have been mentioned above. No treatment can be hoped to have any effect, but it should be the same as that employed in other cases of internal hemorrhage.

PNEUMOPERICARDIUM.

The rarity of an accumulation of air or gas in the cavity of the pericardium may be gathered from the fact that, although I have studiously examined the enormous material of our hospital for thirty-five years, I have never yet seen a case of this kind, and that my old master Skoda had also failed to find such a case. I can, therefore, say only what I know from the literature, and call attention to the diseases that simulate the condition in question. Of such cases I have seen a sufficient number.

As regards the *origin* of pneumopericardium, it has been for a long time believed that a spontaneous development of gas may arise from a purulent or gangrenous exudate, but this question has been widely discussed, and this assertion is almost universally denied at present. The circumstance that no opening outward and no communication to a neighboring air-space can be found in some instances speaks in favor of the old theory, and when a spontaneous development of gas may be demonstrated in abscess cavities and other organs in which absolutely no communication with air-spaces can be proved,—as, for example, in the thyroid gland,—then I do not see why this should not be possible also under similar conditions in the cavity of the pericardium.

It is impossible to say, as yet, how much weight should be placed upon the fact that in the few published investigations the percentage of gases in such cases was found to be the same as regards the relation of oxygen and nitrogen as that found in the atmosphere. Whether or not this fact can be employed in the argument against the spontaneous development of gases must remain undecided; for there is a lack of material for observation that could lead to a decision in this question.

Air may also enter through injuries from without which occur under certain conditions. If, for example, a dagger is simply thrust into the chest-wall, no air need enter into the pericardial cavity. On the other hand, if its point is moved in various directions after it has penetrated into the chest, then air may also enter with it. The process which takes place in these cases is one of aspiration, both in virtue of the systolic contraction of the heart and of the constant efforts of the lung to retract.

The same results may follow after injuries from within—that is, from sharp objects which have penetrated into the esophagus. A very rare occurrence is the bursting of a suppurative process within the pericardium into a neighboring pulmonary cavity. By far the most frequent event is a perforation of an abscess in the respiratory organs into the cavity of the pericardium, as, for example, the perforation of a pyopneumothorax, of an ulcerating cancer of the esophagus, of an ulcer of the stomach, or even of an abscess of the liver, which, at the same time, communicates with the stomach. In these perforations the air in the pericardium may be under a considerable amount of pressure, so that the walls of this sac are considerably dilated, as is seen by the fact that the lung is compressed, the diaphragm depressed, and the air issues with a hissing noise on puncturing the pericardium. Inasmuch as pure air does not usually enter the pericardium, but bacteria enter at the same time, a simple pneumopericardium does not remain so for a long time, but soon becomes a pyopneumopericardium.

The **symptoms** which occur vary according to the conditions under which the air has entered the pericardial sac. The chief symptoms are

those which appear on physical examination. I am not astonished to find that most authors describe a more or less marked prominence of the cardiac region, for we can readily imagine that a paresis of the intercostal muscles rapidly develops in these cases and thus allows a projection to occur even with a relatively moderate pressure from within. The apex-beat may be weakened or absent, but it may appear more distinctly when the patient sits up or bends forward.

The phenomena observed on percussion and auscultation are of special value. If air alone has accumulated in the sac, a uniform tympanitic or metallic percussion-note may be heard over the cardiac region and beyond it. This sound, as Gerhardt observes, may vary in its pitch when rapid percussion is performed on the same spot over the heart, inasmuch as the changes in the shape and position of the heart create variations in the arrangement of the air-cavity. The air always occupies the uppermost portion of the sac. If the patient lies horizontally on his back, it may happen that if there is a large amount of fluid present, when we percuss over the left parasternal line from above downward, we find first a zone with dulness on percussion, then one with a high tympanitic note, and, finally, a third extensive area of dulness, corresponding to the remaining portion of the exudate. The air changes its position when the patient changes his posture, and this is one of the most important symptoms. If a cracked-pot sound has been observed in a closed pericardial sac, I can only explain this by the assumption that adhesions between both pericardial layers had developed as the result of a preceding pericarditis, and that a communication had been formed between the two portions of the pericardial cavity in such a manner that, given a different pressure of air in both air-spaces, the air from the one is forced into the other during percussion.

All authors consider the phenomena of auscultation as the most striking in this condition. These manifestations are very marked, whether pneumopericardium or pyopneumopericardium is present, whether the walls are smooth or have been roughened by the successive inflammations, and they are not only appreciable to the physician, but often even to the patient. The heart-sounds assume a metallic tone, and even the apex-beat may have a metallic clinking. The presence of fluid produces a mixed sound, which is sometimes splashing, sometimes gurgling, and may be compared to the noises of the paddles of a water-wheel as it strikes the water. It is always distinguished by a special intensity or a metallic tone. The latter may even be noted in the friction-sounds that may be present.

The other symptoms may vary to a great extent and will depend upon the presence of a complicating disease, or upon changes in other parts of the cardiac apparatus. Thus, there may be fever of very irregular type or fever may be absent. The quality of the pulse depends upon the fever, or its smallness and lack of tension may be due to the compression of the heart and the paralysis of the heart's action. Dyspnea and a more or less marked cyanosis may accompany the latter changes, together with the compression of the lung. Pressure upon the esophagus will produce difficulty in swallowing.

The **diagnosis** will not be difficult when the symptoms described are carefully considered. The conditions to be differentiated include sacculated pneumothorax, a cavity in the neighborhood of the heart, and, under certain conditions, a markedly dilated stomach.

A sacculated pneumothorax close to the heart can be recognized on

gentle percussion—for when we percuss vigorously we communicate the vibration to the entire neighboring air-space—by the area of dulness of the heart, usually in an altered position. Palpation of the apex-beat may also give a clue, especially in changes of the patient's position. It is also possible that amphoric breathing may be clearly heard, in addition to the metallic heart-sounds. The differentiation can only become difficult when a pyopneumothorax develops from a sacculated pneumothorax. In these cases we are obliged again to rely upon changes in position.

Careful percussion will delineate the limits of the cardiac dulness when a cavity is present in the neighborhood of the heart. If the cavity is suitably located, the well-known changes in the percussion-note will be heard, and, finally, the quality of the râles will be of value in the diagnosis.

When the stomach is dilated, the heart-sounds may normally assume a metallic tone, but this change is only transient. The apex-beat, the position of the heart, and, finally, the introduction of a stomach-tube will give sufficient evidence for a diagnosis.

As regards the **course,** it is possible, and has actually been seen, that the pure air which enters may become reabsorbed, just as it may disappear under similar conditions in pneumothorax. If there is an inflammation of the pericardium, the course may be a very rapid one, but it may terminate even then by the gradual increase of the exudate, the absorption of the air, and thus the formation of a chronic pericarditis with exudation, which, in turn, may end in recovery. In most instances, however, the termination is unfavorable, on account of the primary disease, and also on account of the secondary changes described above.

The **treatment** will necessarily be directed toward the primary disease, and, in general, will be symptomatic. The use of cold will be the most practical method of relieving the subjective symptoms. Narcotics should be avoided as much as possible. When there is severe dyspnea, we may be permitted to puncture the pericardium under the precautions already described. If we have reason to assume that the exudate is decomposed, a more extensive opening of the pericardium may be employed, followed by antiseptic treatment of this cavity, according to the principles of surgery; this is certainly permissible provided we take into consideration the condition of the rest of the body.

TUBERCULOSIS OF THE PERICARDIUM.

We have already mentioned the fact that pericarditis not infrequently occurs in tuberculous subjects. This inflammation of the pericardium does not differ in any way from the forms which occur as the sequel of other diseases, except that the exudate is often hemorrhagic. We have now to consider genuine tuberculosis of the pericardium. The tubercle bacilli doubtless enter the pericardium from some neighboring structure—either from the adjoining pleura or lung, by way of the lymphatics, or from the glands of the mediastinum. It is possible that the bacilli may spread from the mediastinum by way of a direct perforation, as Kast has shown in one case in which the pus obtained from the pericardium by puncture originated from the perforation of a cheesy mediastinal gland. But infection of the pericardium may also take place by way of the circulation, as, for example, in a case in which the infection came from the lung, and which I had occasion to observe lately. Here a severe tuberculosis

of the pericardium and even of the heart muscle itself had developed, but only a single tuberculous focus of long standing was found in the right apex of the lung. The patient was a woman aged forty years. The same course of infection may be possible by transmission from distant organs, such as the bones.

The rare cases in which tuberculosis of the pericardium occurs as a primary disease, and remains the only tuberculous affection until the death of the patient, are certainly curiosities. But it is probable that in such cases traces of a tuberculosis of the lymph-nodes, especially of the bronchial nodes, may be demonstrated. We are tempted to assume that, even in these instances, the tuberculous process was added to a simple pericarditis, because this diseased portion of the organism presented the most favorable opportunity for the localization of the tubercle bacilli. The occurrence of tubercles in the adhesions, according to Orth, speaks in favor of such a secondary invasion. But if we consider the fact that these cases are often protracted for a very long time, and yet are markedly developed, it seems more probable that they were tuberculous in character from the start.

Pathologic Anatomy.—Tubercle bacilli may induce an infection of the pericardium in two distinct ways: First, they may produce a tuberculous pericarditis, and, second, a true tuberculosis of the pericardium.

1. As regards the first form, it is accompanied at the beginning of the process only by a serofibrinous or a hemorrhagic exudate. It is only when the organization of the fibrinous exudate gives rise to the formation of new connective tissue which covers the pericardium that the characteristic gray nodules develop in the latter, and that the disease may be recognized as tuberculosis. Very frequently, therefore, the true nature of the disease can only be recognized after a careful examination, and after the removal of the superficial layers of fibrin, in order to inspect the deeper layers which have already become organized.

This form may occur either as an acute or a chronic affection. When it is chronic, it appears either immediately after the acute stage, or it may be chronic from the beginning. It is characterized by the organization of the exudate; by the formation of tubercles in the new connective tissue; by adhesions of the pericardial layer with the formation of more or less extensive cicatrices, which result from the organization of fibrin, in which the remains of a fibrinous or hemorrhagic exudate may be inclosed. In these masses of cicatricial tissue more or less extensive layers of constantly developing and caseating nodules may be found inclosed, so that a uniform layer of tuberculous material may be found therein, which in some cases may reach such a size that it swells the adhesions to the magnitude of a tumor.

2. In the second form the development of tuberculous nodules takes place within the tissue of the pericardium itself. Here we find miliary nodules which are scattered, especially along the sulci and along the pericardial vessels, or else, more rarely, caseous nodules which may reach a considerable size. Orth observed a caseous tuberculous node about the size of an almond, which was situated upon the pericardial portion of the aorta, while the remainder of the pericardium was perfectly free from disease. Nodules of still larger size have been seen in our Pathologic Institute, but these were always present in connection with tuberculous processes in the neighborhood, especially in the mediastinal lymph-nodes. This form is at times found in combination with a tuberculous pericarditis,

which has been excited by it, and less frequently it occurs as a sequel to a chronic tuberculous pericarditis.

Still another very rare occurrence is the destruction of the superficially located tubercles and the formation of ulcers, such as have been observed by Eichhorst in a case of primary tuberculosis, and in other cases by Riegel and Orth. In the case reported by Eichhorst there was a fatal hemorrhage as the result of such an ulceration.

The **symptoms** of the tuberculous forms are the same as those of other types of pericarditis, but they may be modified, according to the presence of a primary tuberculous pericarditis or of an affection of the pericardium, in addition to a tuberculous inflammation of the other serous membranes (the so-called *tuberculosis serosarum*), or an involvement of the pericardium as the complication of tuberculosis of other organs, especially the lung. In these cases the question is largely as to what stage of the primary disease becomes complicated with tuberculous pericarditis. The clinical picture may also be variously influenced, but the characteristic element of pericarditis will always remain the same. As the course of the disease is usually a protracted one, it may easily happen that its manifestations may vary from time to time. Thus, for example, the friction-sounds may disappear with the development of a large amount of fluid, while they may reappear when this fluid is partly absorbed.

As regards the **diagnosis,** we can only make out the presence of a pericarditis from the symptoms present. The existence of a tuberculous process elsewhere, usually in the lung, does not entitle us to assume that the same disease is present in the pericardium, any more than we can say that the tuberculous nature of the affection is excluded in every case of pericarditis. We know that the disease may occur, in the primary form, or as the sequel of a primary tuberculous affection which does not give any symptoms, and, therefore, we must consider the question of a tuberculous origin in every case of "idiopathic" pericarditis.

A protracted course may point to tuberculosis under certain conditions, but, as we have already remarked, this is not a positive criterion, for a pericarditis due to other causes may also show such a course. Only a few weeks ago I discharged a patient who showed friction-sounds for four months and who developed dropsy and bilateral pleurisy in the course of the disease. Inasmuch as all other methods of examination failed to determine the nature of the disease, the fluid removed from the pleural and abdominal cavities on various occasions was examined bacteriologically, but the results were negative. You may judge from this the difficulty in this portion of the diagnosis. But, as the exploratory puncture of the pericardial sac is perfectly devoid of danger, provided it be carefully executed, this operation may be employed for diagnostic purposes in such doubtful cases.

The **prognosis** is always unfavorable. The only question is the duration of the disease, which may be judged according to the degree of the various symptoms.

The **treatment** must pursue the same aims as that of pericarditis. The first purpose should be to support the strength of the heart and of the body in general, and the remainder of the treatment must needs be purely symptomatic, according to the requirements of the individual case. [See Riesman, D., "Primary Tuberculosis of the Pericardium," "American Journal of the Medical Sciences," July, 1901. Norris, G. W., "Tuberculous Pericarditis," *ibid.*, July–August, 1904.—Ed.]

SYPHILIS OF THE PERICARDIUM.

Although syphilitic affections of the myocardium are very rare, the primary occurrence of syphilis in the pericardium is certainly exceptional, and but a few cases have been reported in literature (Lancereaux; Orth). Lancereaux describes a gumma of the size of a cherry-pit on the parietal portion of the pericardium in a patient who also showed gummata in the lung. The epicardium takes part somewhat more frequently in syphilitic affections of the myocardium because it may become distended over gummata or over scars of the same, and may be infiltrated in its subserous cellular tissue by small round-cells. In addition, there also may be a formation of new connective tissue which produces cicatricial thickening in the pericardium, or adhesions of the two layers to each other.

According to Mraček, who gives the most detailed description of this affection, and who has collected the reported cases, inflammatory infiltrates and thickenings of connective tissue may occur over the gummata, but only the cases with most markedly degenerated cardiac walls show the formation of adhesive membranes. Complete adhesion of the pericardial layers seems to be rare. The coronary arteries may be so narrowed through the inflammatory or productive changes in their vicinity that transudates may appear in the pericardial sac.

Wanitschke has shown a case of hereditary syphilis which occurred in Chiari's institute in Prague, in a newly born child, in which syphilis of the pericardium was the result of a very large syphilitic tumor in the upper lobe of the left lung, which had become adherent to the parietal pericardium, had penetrated through this structure, and had led to a pericarditis with a serofibrinous exudate. Round-cell infiltrations and, in places, delicate homogeneous or net-like layers of fibrin containing a few round-cells were found over the congested visceral pericardium. The noteworthy feature in this case was the fact that, although syphilis usually leads to plastic inflammations, the form observed here was a serofibrinous exudative process.

There is no question that the pathologic changes just described give rise during life to symptoms which we have described as resulting from pericarditis. The syphilitic nature of the disease may be at most suspected, but never definitely determined during life.

NEW-GROWTHS.

Primary new-growths in the pericardium are extremely rare. Orth mentions small multiple warty tumors of connective tissue, which often contain fat, and then there are combinations of lipomata and fibromata. Förster and Guarnieri have reported primary carcinoma, and Redtenbacher primary angiosarcoma. In the last case the pericardial sac remained only around the apex of the heart, and was filled with 100 c.c. of a dark, bloody fluid, while the remainder of the cardiac surface showed intimate adhesions through loose connective tissue which could be easily detached. The parietal layer was transformed into a cicatricial structure from 2 to 5 mm. in thickness. A flat, fluctuating prominence of the size of the palm of the hand was situated over the anterior and outer surface of the right auricle and over the cone of the pulmonary artery.

When the two layers were separated, this prominence was torn and discharged some reddish-black clots. Corresponding to this prominence the visceral layer showed a cavity filled with clots, and after removal of the latter a delicate fret-work of strands with nodular swelling of sizes varying from that of a poppy-seed to that of a pea. Similar nodules of the size of a hazel-nut were found over the posterior and upper surface, in the pleura, and in the parenchyma of the lung. On microscopic examination these proved to be masses of capillaries with intervening spindle-cells.

Secondary new-growths of the pericardium are much more frequent, and may be derived from the neighboring tissues, such as the lung, the pleura, the mediastinal glands, or the thymus. These affect especially the parietal layer, and may either engulf the latter, or may be transplanted into it by metastasis. The latter form is especially frequent in the various types of sarcoma and carcinoma. Thus, there is a specimen in the museum of the Vienna Pathologic Institute of a pericardial carcinoma following a cancer of the penis, in which the heart was surrounded by a cuirass 3 cm. in thickness, arising from a cancerous infiltration of a mass of adhesions uniting the two pericardial layers. The examinations of these infiltrations showed the characteristics of a medullary epithelial carcinoma.

When a patient presents new-growths which are inclined to metastasis in the neighborhood of the heart, or even in one of the organs further removed, and when he develops signs of pericarditis without any other discoverable cause, one is justified in thinking of a transmission of the neoplasm, all the more when the disease in the pericardium has a specially protracted course. Conversely, the occurrence of pericarditis with concealed new-growths is generally overlooked, so far as its connection with the tumor is concerned. We have already discussed in detail the influence of the affection upon the course of the primary disease.

ANOMALIES OF THE CONTENTS.

For the sake of completeness I may mention *actinomycosis* in addition to the diseases described. Several cases have been observed in which actinomycosis existed primarily in the lung, and was transmitted to the heart and the pericardium, with considerable changes in both (Münch, Redtenbacher). It is possible that in those cases in which a diagnosis of actinomycotic disease of the lung or of the skin has been made, a pericarditis which supervenes may be due to the same parasitic cause.

Pathologic curiosities which, for the present, are not of great clinical interest are *echinococci*, *cysticerci*, *trichinæ*, and *foreign bodies* of various kinds, including the so-called *cardioliths*, which may occur in the pericardium.

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