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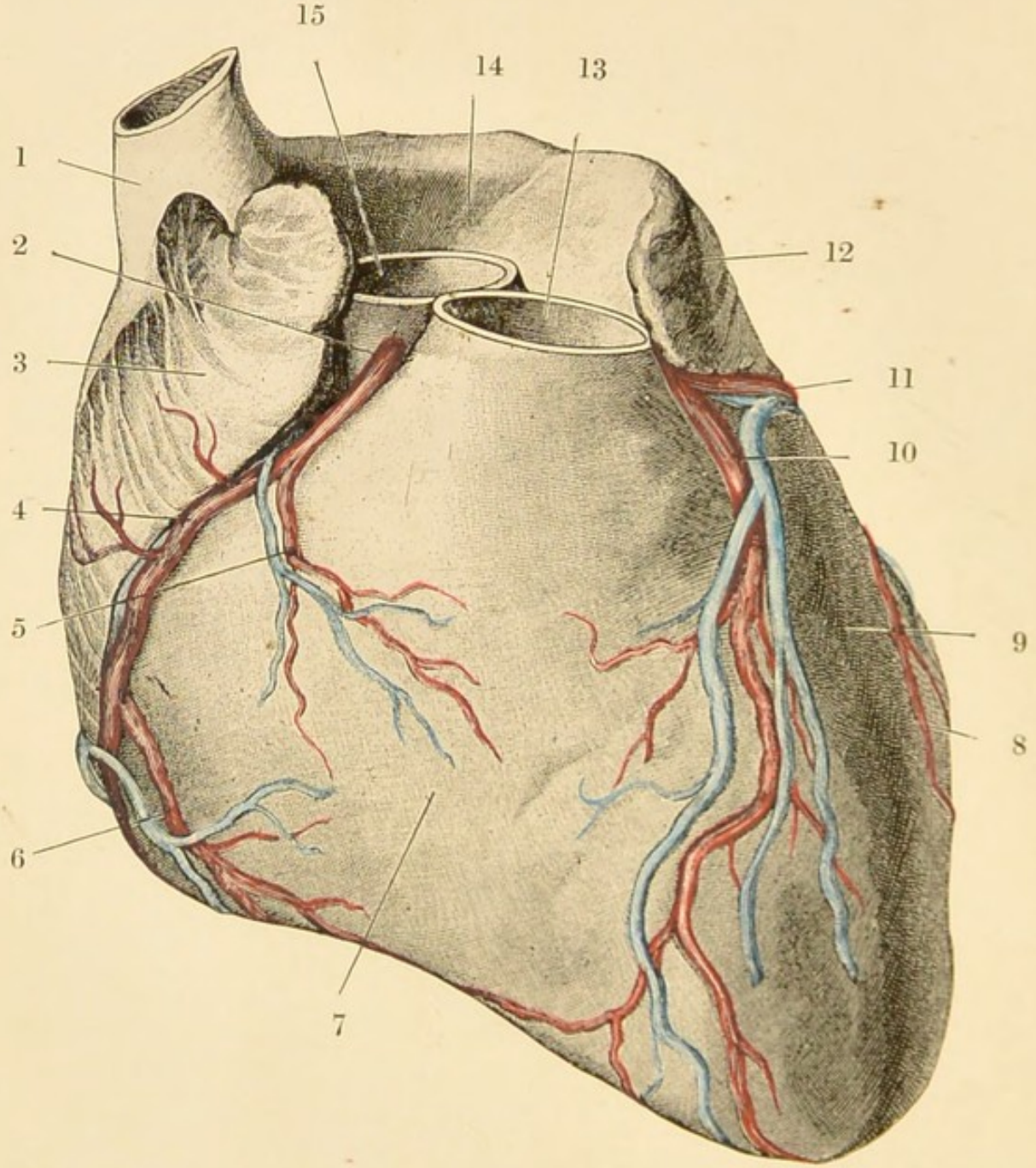


Fig. 1.

1. Superior vena cava; 2. right coronary artery; 3. right auricular appendix; 4. right coronary artery; 5. anterior ventricular artery; 6. right marginal artery, 7. right ventricle; 8. left marginal artery; 9. left ventricle; 10. interventricular branch of left coronary artery; 11. transverse branch of left coronary artery; 12. left auricular appendix; 13. pulmonary artery; 14. left auricle; 15. aorta.

From Cunningham's "Text-book of Anatomy."

Text Book

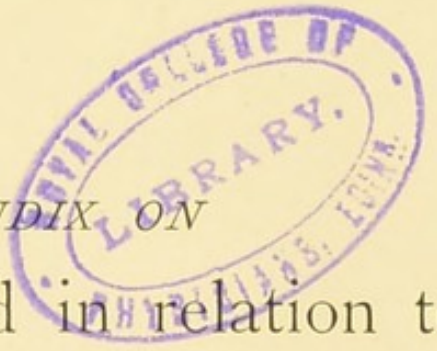
ON

Diseases of the Heart

BY

GRAHAM STEELL, M.D., F.R.C.P.,

Senior Physician to the Manchester Royal Infirmary.



WITH AN APPENDIX ON

The Volume of Blood in relation to Heart Disease

BY

J. LORRAIN SMITH, M.A., M.D.,

Professor of Pathology.

MANCHESTER
AT THE UNIVERSITY PRESS
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PREFACE.

It has been the endeavour of the writer to give in this book an accurate account of the clinical aspect of heart disease, along with a description of the principles of the treatment of the disease. With regard to the former object, it is claimed that the account given is drawn "from life," and that the influence of "authority" has been excluded as much as possible. With regard to the latter object, it is claimed that the statements made do not go beyond the facts of actual experience at the bedside.

The book is published at a time when there is immediate prospect of a great advance in our knowledge of the anatomy, physiology and pathology of the heart, which promises to lead to much improvement in the application of available remedies. That there has been such advance is the result of laborious investigations of scientists and clinicists alike, among whom may be mentioned Keith, Gaskell, and especially Mackenzie and Wenckebach.

Thus it became necessary to indicate, in the text, the lines along which knowledge has recently progressed, and is likely in the future to progress, and the work of Mackenzie and Wenckebach has been specially drawn upon for the purpose.

It must not be supposed, however, that the advance in our knowledge referred to had been preceded by a prolonged period of stand-still. This is very far from being the case: though progress had been slow, it had been steady.

In 1873 the writer had the good fortune of being House Physician to the late Dr. G. W. Balfour, in the Edinburgh Royal Infirmary, and he well remembers how the views of his master were, then, regarded. The great work done by Balfour can hardly be realised now: suffice it to say that up to that time the prevailing idea had been that "mitral disease" and "mitral incompetence" were practically equivalent terms. That this was so seems all the more strange in view of the fact that every physiology text-book of the period taught the so-called "safety-valve action" of the tricuspid-valve apparatus. The application of the same principle to the mitral-valve apparatus was not made earlier no doubt because of the great proclivity to disease of the valves of the left ventricle in comparison with the practical immunity of those of the right ventricle. "Curable mitral regurgitation" was the term used by Balfour to call attention to the then new doctrine, the cases he took for illustration being chiefly examples of chlorosis. Unfortunately, all cases of mitral regurgitation the result of muscle failure are not curable, inasmuch as the cause of the muscle failure cannot be removed as it can be in chlorosis.

Balfour, moreover, recognised the fact that when

mitral incompetence had been associated with structural change in the valves—"mitral disease"—the condition found *post-mortem* in chronic cases was commonly mitral stenosis—NOT STRUCTURAL DAMAGE TO THE VALVES, such as might explain their incompetence WITHOUT STENOSIS OF THE ORIFICE.

Several years ago Dr. Kelynack, at the time Pathologist to the Manchester Royal Infirmary, was good enough, at the request of the writer, to look over his Records with reference to the point under consideration, with the following result. He writes: "At your suggestion I have gone over the *post-mortem* notes of your cases for the past few years. It is remarkable that out of the very large number of cardiac cases examined—a very considerable number being examples of mitral stenosis—I have hardly been able to select a single case of what I might call straightforward mitral incompetence from mitral lesion pure and simple. From the list of cases I have sent you, you will have noticed that several were to a great extent 'muscle' rather than 'valve' cases." As is fully explained in the following pages, it is impossible to consider the action of the mitral valves apart from *muscle-contraction*, which is an essential complement of the valve apparatus. No mitral valves can be competent if they fail to receive that assistance from the heart-muscle they were designed to receive in the performance of their function. Thus it would happen in rheumatic cases that altogether trifling lesions of the mitral valves were found from time to

time in a case that had been associated during life with evidence of free regurgitation. It is idle in such cases to attribute, as used to be done, all the failure in the circulation that preceded death to such trivial lesions. The essential condition was "muscle-failure," but the diagnosis of "mitral disease" was technically correct. Dilatation of the left ventricle was usually present in cases of the kind, and ingenuity was displayed in explaining why mitral incompetence should lead to dilatation of the left ventricle. It was not, however, the mitral incompetence that had led to the dilatation, but rather the other way about. The right way of regarding the case would be to look upon both conditions—the mitral incompetence and the dilatation of the left ventricle—as coincident results of "muscle-failure." Such destruction of the mitral curtains as would *alone* cripple their function (without there being mitral stenosis) is almost limited to cases of septic endocarditis, comparatively a rare disease and a condition that in all probability is never the cause of chronic heart disease.

In 1882, Dr. Donald MacAlister gave, in the *British Medical Journal*, a lucid description of recent experimental investigations on the Continent, proving the importance of the muscle-contraction complement of the mitral-valve apparatus, and supporting the views that had been advocated by Balfour on clinical grounds.

Another very important consideration, teaching, again, the lesson of the importance of the heart-

muscle, is that afforded by those cases of cardiac muscle-failure that throughout their course never yield evidence of valve incompetence. Such cases are far from rare. A condition of imperfect contraction on the part of the ventricles seems to be the only feasible explanation of the disturbance of the circulation that results, though the question of a "silent regurgitation" is not easily set aside. Indeed certain clinical facts concerning tricuspid incompetence might seem to answer it in the affirmative. The great sign of cardiac muscle-failure *per se* is the *bruit-de-galop*, which may be associated with evidence of mitral and tricuspid incompetence, or exist alone. In either case the mechanism of the production of the sound still awaits explanation, and that, too, in spite of the fact that the auscultatory sign is often associated with a corresponding palpation sign at the apex of the heart.

Mitral stenosis, aortic stenosis and aortic incompetence are the great valve *lesions*, and their effects upon the heart-muscle and upon the chambers of the heart, *i.e.*, the heart-muscle, have been long recognised and fully appreciated.

From the foregoing considerations it is evident that the trend of thought with regard to heart disease had been in the right direction, and for thirty years and more, before the advance of knowledge, referred to early in this preface, began: the muscle of the heart had been receiving the chief share of attention. Thus the ground was being prepared for the advance.

Disease is a great experimentalist, and the careful

observer of its working may gain knowledge fraught with suggestion to the physiologist, not to speak of the anatomist. To Dr. Mackenzie belongs the great merit of demonstrating by mechanical means the nature of movements of the circulatory system, that, without such means, must have remained matters of endless controversy. The writer cannot express in too high terms his appreciation of Mackenzie's work.

As regards the treatment of heart diseases, the predominance of the heart-muscle receiving confirmation from recent research should bring increased hopefulness and encouragement. The impossibility of restoring a damaged valve or dilating a narrowed orifice has no doubt contributed to the gloomy aspect with which heart diseases in general are usually regarded. Certain it is the heart-muscle that is most susceptible to therapeutic influence and in a large number of heart cases it is the muscle that is mainly concerned, while in all valve cases it is by the myocardium that a *modus vivendi* is established.

In the near future probably a great deal will be learnt as to the action of digitalis and like drugs upon the heart-muscle, with the result that their application will be much more discriminate than heretofore. As the different functions of the heart-muscle become better understood it will at the same time become more evident that no single drug can be of universal application in heart disease. Moreover, the drugs employed in the treatment of heart disease are for the most part very active drugs—powerful for good or

ill—and when manifestly they fail to do good it is only too likely that they will prove injurious to some extent in some direction.

While we have still to learn much as to the action of these active drugs in the different forms of disease and disorder of the heart, the weighty words of the late Sir William Jenner with regard to the treatment of enteric fever arise in one's mind as singularly applicable to the treatment of heart disease: "My experience has impressed on me the conviction that that man will be the most successful . . . . who, when prescribing an active remedy, weighs with the greatest accuracy the good intended to be affected against the evil the prescription may inflict, and if the possible evil be death and the probable good short of the saving of life, holds his hand."

As regards therapeutic achievement concerning heart disease during the latter part of last century, the writer would single out as specially prominent the use of morphine—best given hyperdermically—for dyspnœa and the other distresses that cause so much suffering to the victims of the disease. Both patients and their medical men owe Professor Clifford Allbutt an incalculable debt of gratitude for the introduction of the remedy. The inhalation of nitrite of amyl and the internal administration of various nitrites have also contributed largely to the relief of cardiac suffering, into the cause of which arteriole spasm enters largely, and for the introduction of these remedies Sir Thomas Lauder Brunton and the late

Professor D. J. Leech have laid the therapeutist under obligation.

The writer has to confess his belief that there is a great deal to be learnt yet with regard both to cyanosis and dropsy, especially in relation with blood conditions, and it is satisfactory to him that Professor J. Lorrain Smith has been so kind as to accede to his request and give as an Appendix a short account of his researches having a bearing on the subject.

It remains for the writer to express his obligations to Dr. Albert Ramsbottom, Medical Registrar, and late Resident Medical Officer to the Manchester Royal Infirmary, for the great assistance he has afforded him in the preparation of this book and in many ways besides that of the construction of an Index, which is his own work.

Grateful acknowledgment of permission to use illustrations is due to the following publishers:— Messrs. Macmillan and Co., Messrs. Smith, Elder and Co., Messrs. Longmans, Green and Co., Messrs. W. Green and Sons, and Mr. Young J. Pentland.

CONTENTS.

Diseases of the Heart

ANATOMICAL CONSIDERATIONS.

A knowledge of the anatomy and physiology of the heart and vessels is assumed in the reader of this book, and therefore no sketch, however rough, of these subjects will be attempted. Nevertheless, with a few figures, culled from the best sources, before him, the reader is asked to give heed to a very few considerations, concerning these subjects, that have a bearing on clinical medicine.

The "apex-beat" is an object of supreme interest. Unfortunately it is not always present, and this even in health. When present, its position, extent, force, rhythm and certain vibrations that may accompany it in disease—thrills—as well as abnormal added shocks, that usually accompany the *bruit-de-galop*, all require attention and will be referred to in detail later. The apex-beat is to be associated with the contraction of the left ventricle, which alone forms the apex of the heart. The apex is, as it were, held up against the fifth intercostal space while the ventricle is being emptied. In this relation it is curious to note that the wall of the ventricle at the apex is peculiarly thin, so that this part might be almost fancied to bulge when the thicker portions of the

ventricular wall are in contraction. Probably this thinness of the apex of the left ventricle has something to do with the well-defined impulse under consideration. The position of the true apex-beat marks the extension of the left ventricle to the left and downwards. The right ventricle has no impulse comparable to the "apex-beat" of the left ventricle. It has usually been stated that the diffuse impulse—amounting in cases of great enlargement of the cavity and thickening of the walls of the right ventricle to a heave—is systolic in rhythm; but Dr. Mackenzie, by the aid of his polygraph, has adduced evidence showing that the expansion movement is diastolic, while the systole is associated with retraction. Indeed, it may be said that Dr. Mackenzie has demonstrated that the surface movements are as indicated. The sensation conveyed by the hand placed over the epigastric region is, however, often difficult to reconcile with this instrumental demonstration. The writer admits this difficulty in his own experience, and has tried to explain the discrepancy between his sensation and the result of instrumental investigation as follows: the ventricle is distended when its wall enters into contraction, and the seeming impulse felt is the *hardening of the full ventricle*. But, as systole proceeds, the ventricle diminishes *in size*, its con-

tents being expelled. It is this diminution in size that Dr. Mackenzie's lever will record in systole, just as during the diastole and filling of the ventricle it will record a movement in the opposite direction. It is seldom that a well-marked apex-beat and much epigastric impulse are found associated, so as to be the objects of simultaneous observation. The fact was long ago described by Dr. Stokes, who observed in severe typhus the failure of the apex-beat and the development of the epigastric impulse as the fever proceeded. In the course of heart disease Dr. Mackenzie has shown how the right ventricular impulse may throw into the background the impulse of the left ventricle—the apex-beat—when the latter chamber fails in contractile power and the right ventricle is dilated. In cases of great enlargement and hypertrophy of the left ventricle, especially as the result of aortic incompetence, an impulse may appear in the epigastrium which is due to the huge left ventricle and not to the right ventricle (*vide* Mackenzie: "Study of the Pulse," p. 37, par. 28).

Above the third left costal cartilage a pulsation may be visible that is little palpable. At one time, this pulsation was the object of much controversy —the writer would be inclined to add—and of a great heresy. It was contended that this visible

pulsation represented the movements of the left auricle under circumstances that entailed its enlargement. It has to be remembered, however, that it is only the *appendix of the left auricle that comes to the front*, the part of the chamber least likely to become dilated and often found plugged with clot when the rest of the chamber is dilated. The left auricle lies essentially indeed posteriorly, and its position no doubt explains the free conduction of mitral regurgitation murmurs to the back. The visible impulse in the second space referred to, is evidently not due to the auricle at all, but to the infundibulum of the right ventricle which is apt, of course, to become enlarged under the same circumstances as the left auricle—for instance in mitral stenosis. In the cardiac muscle-failure of chlorosis the visible impulse under consideration is often seen, and in such cases the right side of the heart is primarily and specially involved, as is proved by the pronounced venous pulsation in the neck that occurs in the subjects of chlorosis. A glance at the illustration Fig. 2 shows how readily the impulse of the infundibulum will become visible when that portion of the right ventricle is enlarged.

The superficial position of the infundibulum and the pulmonary artery which springs from its summit, is of importance by often enabling the

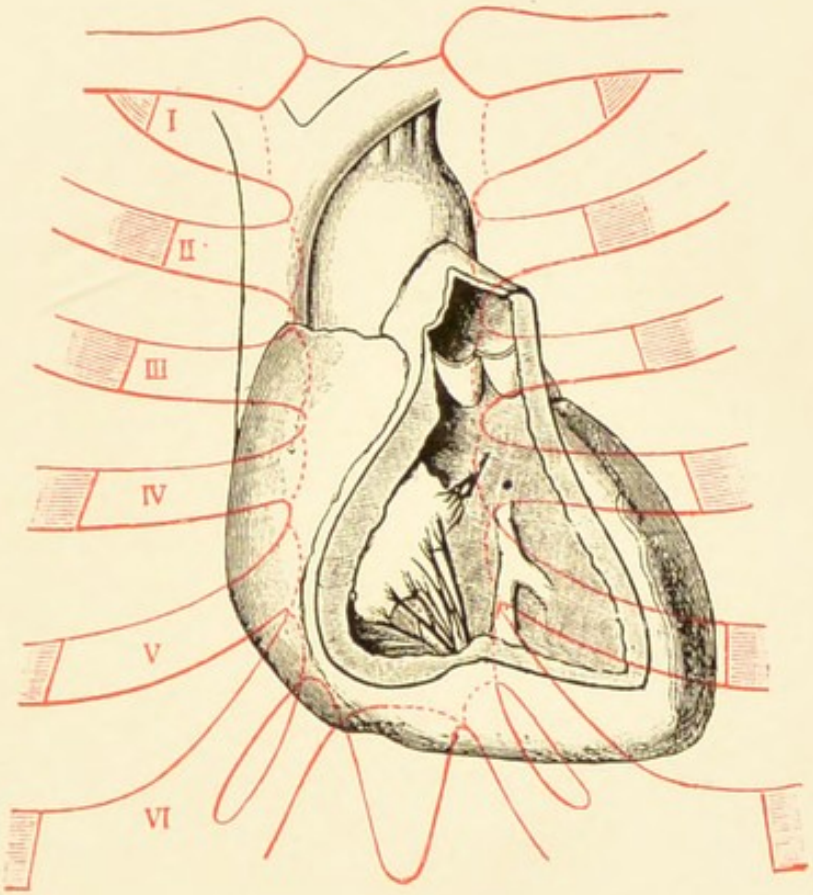

FIG. 2. The relation of the heart to the anterior wall of the thorax. I, II, III, IV, V, VI, the upper six costal cartilages.

From Cunningham's "Text-book of Anatomy."

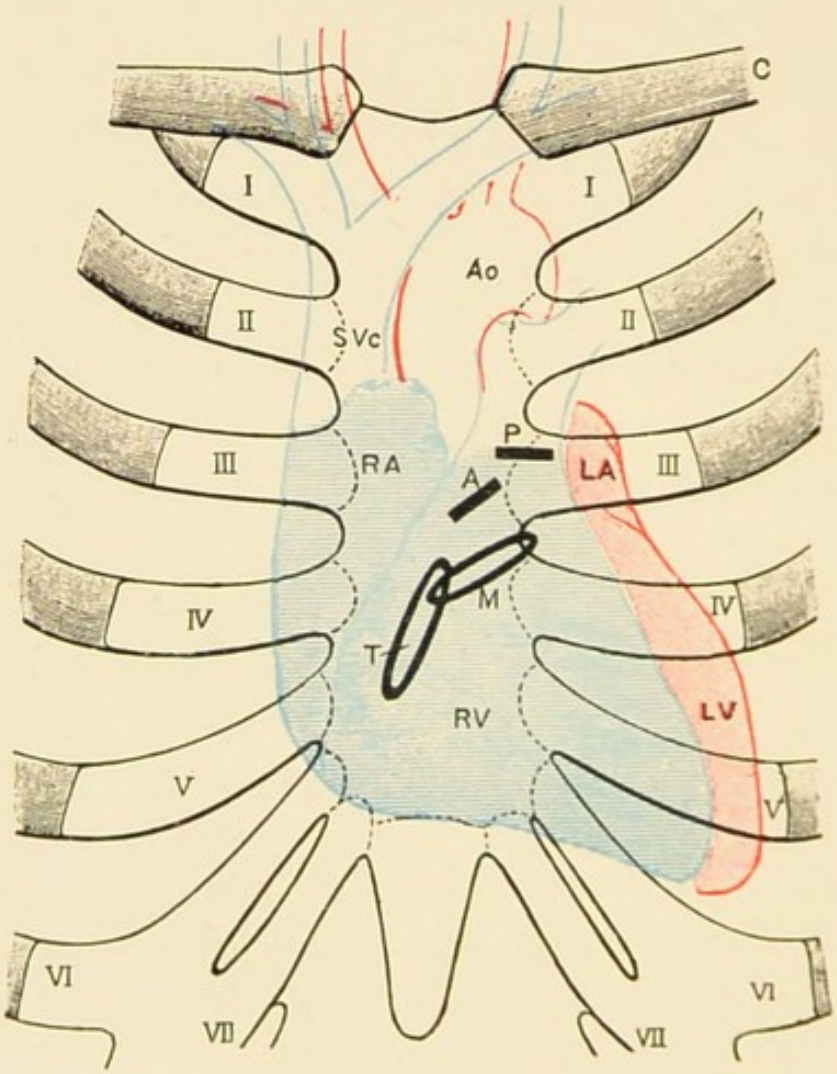

FIG. 3. The relations of the Heart and the auriculo-ventricular, aortic, and pulmonary orifices to the anterior thoracic wall.

I to VII, costal cartilages.
A, aortic orifice.
Ao, aorta.
C, clavicle.
LA, left auricle.
LV, left ventricle.
M, mitral orifice.
P, pulmonary orifice.
RA, right auricle.
RV, right ventricle.
SVc, superior vena cava.
T, tricuspid orifice.

From Cunningham's "Text-book of Anatomy."

clinicist to *feel* the shock of the closure of the pulmonary semilunar valves, when they are closed with undue force.

The right auricle is the chamber of the heart that alone lies to the right of the sternum. It may become greatly enlarged so that its right border reaches the right nipple line—a condition ascertained by percussion. When so enlarged, the chamber has probably always become a passive reservoir, and any impulse seen over it is the result of a backward wave from the right ventricle through incompetent tricuspid valves.

The close proximity of the different sets of valves is shown in Fig. 3, and the impossibility of distinguishing sounds produced at any one of them by placing the stethoscope over it will be evident. The right orifices are the superficial ones, and abnormal ausculatory signs produced at them are listened for directly over their site. As regards the aorta its orifice is seen (Fig. 4) to be in the centre of the heart, while this vessel forms, as it were, the great stem of the heart. As will be seen later the spot selected for isolating, as far as possible, abnormal sounds produced at the aortic orifice, is the sternal end of the second right costal cartilage, where the vessel is most superficial. A comparison between the second sounds produced respectively at the aortic and pulmonary orifices is often considered

to be of great importance, but it must be borne in mind that in the case of the pulmonary sounds the stethoscope is placed directly over the valves, while in the case of the so-called "aortic area," it is placed far from the orifice which lies deep in the heart, the site being chosen in the hope of getting the aortic sounds or murmurs specially conducted along the vessel to the spot where it lies nearest the surface—in other words *isolated.*

With regard to aortic murmurs there is another anatomical consideration worthy of note. In Fig 4, it is seen that while the aortic orifice is centrally placed immediately in front of the mitral orifice, it is interposed between the two orifices of the right side of the heart—the pulmonary and tricuspid orifices. This fact results from the infindibulum of the right ventricle passing in front, and it is possible that the clinical fact of the ready conduction of aortic diastolic murmurs over the sternum may bear relation to this arrangement of parts.

Recent observations on the muscular structure of the heart point to important differentiation of its fibres, and future research may throw much light on peculiar functions of the infundibulum calculated to explain clinical phenomena that have long been familiar as simple facts of observation.

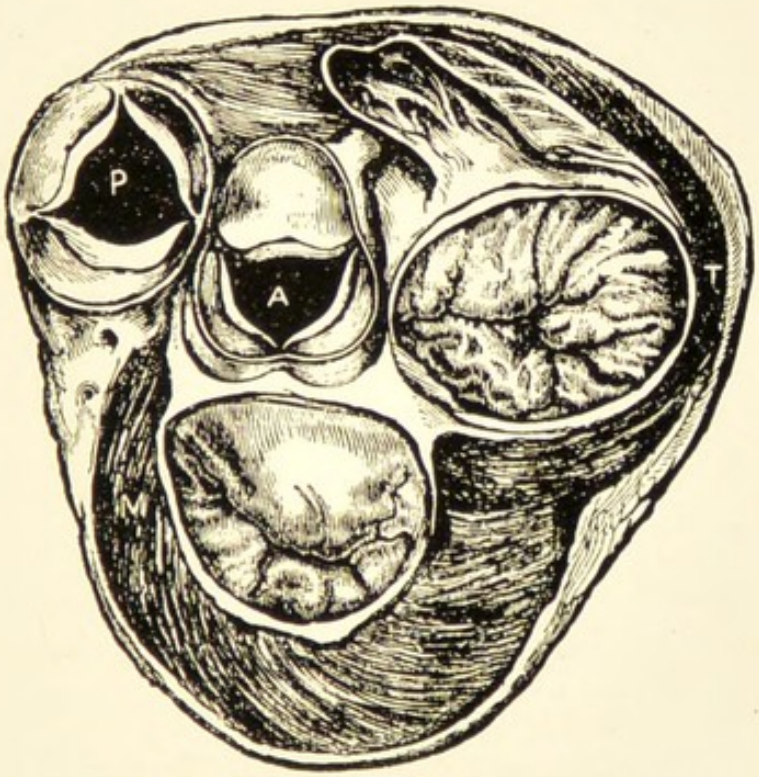

Fig. 4. (from Heath's "Practical Anatomy") shows the relative position of the different cardiac orifices. It will be evident why aortic regurgitation murmurs are so well heard over the sternum, as they have only to pass through the infundibulum of the right ventricle, where, during the diastole, there is no movement of blood. Having once reached the sternum, such murmurs are well carried along the bone

CLINICAL EXAMINATION OF A HEART CASE.

In the examination of a case of heart disease it is customary to divide the details of evidence to be obtained into *symptoms* and *signs*, the former being *subjective* and afforded by the personal experience of the patient; the latter *objective*, and ascertained by the physician. This division is, however, an arbitrary one, and is made for purposes of convenience merely. For instance, the condition of the liver often gives valuable evidence of the state of the circulation, which we call "venous stasis," and which is the common result of slow failure of the heart to maintain the circulation. The condition of the liver, referred to, is constituted by the organ becoming *enlarged* from excess of blood within it, the liver, however, not only becomes *enlarged* but also *tender on pressure*, and sometimes even spontaneously painful. Now, the enlargement, as discovered by the physician—though the patient himself may be aware of it too,—is a *sign*, while the tenderness is a *symptom*, though usually elicited by a manipulation of the physician. It is convenient, however, to speak of

the "enlarged and tender liver" of heart disease, the *combination* is of so much value, and so to regard the combined condition as a *symptom*—nay, it forms, as we shall see, one of the three symptoms of heart disease that may be considered cardinal. Again, the appearance of the neck in a patient suffering from heart disease is of great value with regard to visible pulsation: either the carotid arteries show exaggerated pulsation or there is venous pulsation. Such conditions are usually described among *symptoms*, but are entitled to be classed among inspection-*signs*. They strike the eye of the observer, however, as he obtains the history and symptoms of the case from the patient's statements. In the following pages the appearances observed by the physician, as he inspects his patient generally, will be considered under *symptoms*, though many of the same phenomena, studied instrumentally or otherwise in detail, will be considered under *signs*. Utility is placed before exact classification.

THE THREE CARDINAL SYMPTOMS OF HEART DISEASE.

The *Three Cardinal Symptoms of Heart Disease* are *Dyspnœa, Enlargement and tenderness of the liver* and *Dropsy.*

Dyspnœa.

Among the symptoms of heart disease, there can be no question as to the right of *dyspnœa* to rank first both in importance and in time of occurrence. It is usually "*dyspnœa on exertion*"—for example, on ascending a stair or incline or hurrying to catch a train—that usually first attracts the attention of the patient. Such dyspnœa will commonly have manifested itself before other types are developed, though the patient may have given it little heed. These types of dyspnœa may be described shortly as more or less *paroxysmal*, and they are not necessarily determined by exertion, though the sufferer will invariably be found to have deficient "wind" for any *exertion.* A common example of paroxysmal dyspnœa is seen in the advanced stage of Bright's disease ("granular kidneys") associated with an element more or less pronounced of

relative cardiac muscle-failure, notwithstanding that the arterial tension is usually high. Such patients go to bed comfortably, and perhaps sleep for a few hours: they then wake up in a state of great respiratory distress, and have, as they say, a true "struggle for breath." They are compelled to sit up in bed—*orthopnœa*—and it is often difficult and occasionally impossible for them to remain in bed at all. In this struggle for breath they often perspire freely, and their lips and countenance generally are *cyanotic* or bluish. After a time, whether under treatment or not, the paroxysm subsides and the respiration becomes again easy, sleep soon supervening. Dyspnœa of this type occurring notoriously in cases of Bright's Disease led to such breathlessness being at one time regarded as occurring apart from disturbance of the circulation, and as being the direct result of a toxæmia of renal origin. Careful observation will usually, however, afford ample evidence of the circulation being involved—of there being, in fact, some degree of cardiac muscle-failure, and that notwithstanding the maintenance of a high degree of arterial tension. Similar characters of dyspnœa are met with in cases in which there is no albuminuria (no obvious evidence of kidney disease), though the condition of the heart and arterial system closely resembles that associated

with disease of the kidneys. It will be found later that the presence of albumen in the urine is an exceedingly common condition in cases of heart disease in general, resulting from the kidneys being involved in the systemic venous stasis in which all varieties of cardiac failure sooner or later express themselves, provided the patient survives long enough. Another type of dyspnœa often met with in cases of granular disease of the kidneys, though first described as occurring in cases of "fatty heart," is that known by the name of the physicians, who first recognised its peculiar features —"Cheyne-Stokes Respiration." These features consist of the occurrence of periods of complete cessation of respiration—*apnœa*—and periods of *dyspnœa*, very shallow respirations with which breathing is resumed, gradually increasing in depth and frequency till the deepest inspirations culminate in a paroxysm of dyspnœa, which again gradually subsides, the respirations becoming shallower and less frequent till another period of complete cessation of respiration supervenes, and so the cycle is repeated. "Respiration of ascending and descending type" is another designation of such disturbance of respiration, which will receive further consideration. In old people during sleep respiration of this type may occur without evidence of cardiac failure, but the period of apnœa is short

or absent and the patient does not awake. Again, in patients suffering from apoplexy, tubercular meningitis, and other grave diseases of the nervous system, respiration of this type is often observed, though the element of subjective dyspnœa is absent. On the other hand, a distressing variety of dyspnœa met with in rare cases of heart disease, is that in which the stimulus to the respiratory movements fails as soon as the patient goes to sleep, and he awakes in great distress. Twitching movements of the hands are common before the patient awakes. This condition occurs specially in cases of the type of "muscle-failure." A peculiar and also rare variety of dyspnœa—if such it can be called—is that which causes the patient in repose to take occasionally a very deep breath or "sigh." This occurrence was described many years ago, and was then associated with fatty degeneration of the heart-muscle. The patient is hardly aware of any feeling of dyspnœa, and the long-drawn breath is largely automatic, but there is probably generally some slight subjective sensation of "air-hunger."

The common type of cardiac dyspnœa is that *on exertion*, and the rare types, briefly referred to, are usually accompanied, or have been preceded by it in some degree. The man whose heart is failing cannot have "good

wind," but it is remarkable how differently patients regard their disability in this respect. In rare cases patients will have their attention called to their dropsy, instead of to their dyspnœa, as is usual. It is hard to believe in such cases that the dropsy had not been preceded by dyspnœa, and it is probable that had the patient come under direct observation before the dropsy appeared his "wind" would have been found more or less at fault on exertion.

Enlargement and Tenderness of the Liver.

One of the most important indications of the disturbance of the general circulation which results from heart failure, and which we have termed *venous stasis*, is the *swelling and tenderness of the liver* already mentioned. When this condition of the liver has been once developed, the variations it undergoes under observation form important indications of the state of the circulation. Even when the swelling and tenderness of the liver are pronounced, if the heart recovers and the circulation thereby becomes restored so that the venous stasis is lessened or removed, then the tenderness rapidly diminishes and soon ceases, while the liver as speedily shrinks. The tenderness would seem to be produced under the influence of tension of the capsule of the liver. It may diminish or cease in very chronic cases while the enlargement re-

mains. If the finger of the observer be placed below the edge of the organ and parallel with it, and is then passed upwards, it will be found that the moment it impinges on the swollen liver the patient will complain of pain. Another manœuvre is to keep the finger still, while the patient is asked to take a deep breath: when the liver-edge impinges on the finger, pain is experienced by the patient. It has been held that the tenderness is cutaneous and "referred" and indirect, and no doubt in some cases an area of superficial cutaneous tenderness is found, most characteristically in the distribution of the tenth dorsal spinal segment, the upper border of which is marked by the umbilicus. The deeper tissues of the abdominal wall may also become tender. As a rule, the *spleen*, whose blood is returned by the portal vein through the liver, is not enlarged in ordinary heart cases: at all events to such a degree as would render it palpable or cause a great increase in its dull area. When, however, the obstruction to the circulation exists primarily in the liver as in interstitial hepatitis (cirrhosis), enlargement of the spleen, on the other hand, is common. Probably in heart disease, the ready distension of the liver under the influence of venous stasis accounts for the spleen escaping similar distension and enlargement in most cases. In a few cases of heart disease—

generally of long standing and of mitral stenosis—the spleen is considerably enlarged from venous engorgement, and its dull area becomes increased in size, whether or not its lower end comes within reach of palpation. (*Vide* pp. 41 & 299 for other causes of enlargement of the spleen in heart disease: Embolism and Septic Endocarditis). Often the engorgement of the liver, that results from venous stasis, produces a certain amount of jaundice, so that the patient's conjunctivæ are yellow and his skin is also yellow. In the final stage of many cases of heart disease, jaundice becomes pronounced, though there is seldom actual cessation of the flow of bile into the intestine as a result of swelling of the mucous membrane from catarrh of the bile ducts. Jaundice is most commonly observed in pronounced degree in cases of long-standing mitral stenosis, in which cases it is a curious fact that the liver seems to suffer more severely and earlier than in other forms of heart disease. The mingling of lividity (cyanosis), resulting from venous engorgement, with the yellowness of the bile-stained skin gives a peculiar greenish hue to the countenance of the patient that is very characteristic and of exceedingly bad omen. The special tendency to disturbance of the hepatic circulation, displayed in certain cases of mitral stenosis, is exemplified

by the occasional early development of engorgement of the liver, with enlargement, tenderness and even jaundice, while as yet there is no trace of dropsy. Moreover, in cases of this lesion, ascites may be the first dropsy developed, and may remain the only effusion of serum for a considerable period. It is in long-standing cases of the same lesion—mitral stenosis—that the phenomenon of expansile pulsation of the liver is most commonly observed (*vide* p. 153).

In ascertaining the presence of enlargement of the liver *palpation* is by far the most useful physical method of diagnosis, although percussion should never be neglected, and for defining the upper border of the liver or ascertaining the complication of fluid in the right pleura, percussion has to be depended on entirely. Engorgement of the liver accounts not only for the frequent presence of actual bile pigment in the urine in heart cases, but also for the deep pigmentation of the fluid, and especially of its urate deposits, which is not altogether accounted for by the concentration of the secretion as the result of the scanty flow (p. 26). It must be remembered that, in heart cases, fluid accumulation in the peritoneal cavity may rarely arise as the result of a more or less chronic inflammatory affection of the serous membrane, as well as from obstruction to the portal circulation. The peri-

toneum covering the liver and spleen often shows a greater degree of thickening and change than elsewhere, and it has been supposed that the thickened capsule contracted so as to compress the liver and impede the portal circulation within it, in this way leading to ascites. It is more likely, however, that the mechanism of effusion is essentially a direct inflammatory one, such as is observed in the case of other serous sacs—pericardium, pleura, etc.

In cases of a more evenly distributed chronic peritonitis, which may possibly complicate heart disease, the effusion is very great, and the much enlarged abdomen of a male may yield a dull note on percussion all over. This puzzling condition is explained by the mesentery of the intestine having become shortened so that the intestines are bound down to the posterior abdominal wall.

Dropsy.

Dropsy, or the exudation of serum from the capillaries and venules into cellular tissue or serous sacs, may seem to be a very natural result of the disturbance of the circulation, that follows heart failure and takes general expression in accumulation of blood on the venous side of the circulation and reduced tension on the arterial side. Clinical observation shows that the matter is less simple than at first sight it might appear,

and in every local dropsy there is probably a "tissue-element" if we may so say: that is a change undergone by the dropsical tissues, especially by the vessel-wall.

The recognition of dropsy is usually made by inspection, and its presence is confirmed by palpation—pressure with the finger, which leaves a depression of the surface easily felt even through a stocking. As one might expect from theoretical considerations, the dropsy of heart disease usually develops first of all in the lower extremities, beginning in the feet and about the ankles, and spreading upwards more or less. It may occur, however, first of all elsewhere than in the lower extremities, and sometimes in apparently capriciously chosen sites. This is specially apt to happen in certain varieties of heart disease, as will be pointed out later. The fact of such occasional caprice of the distribution of dropsy renders it necessary that one should admit into one's idea of dropsy an element of "tissue-predisposition" in which probably the walls of the minute vessels of the part are largely concerned, and, it may be, mainly concerned. Moreover, the amount of chloride of sodium in the serum would seem to exert an influence, excess promoting effusion, and *vice-versâ*. Capricious distribution of dropsy is specially apt to occur in cases of the cardiac muscle-

failure of beer-drinkers and of the disease known as Beri-Beri, of both of which diseases, it is curious to note, peripheral neuritis is a clinical feature. Under the circumstances, not only is the distribution of the dropsy apt to be capricious, but it is apt to be also very extensive. The sub-cutaneous tissues of the trunk are specially likely to become involved, and in cases of the kind, pressure with the finger over the sternum, for instance, will often leave a depression of great depth in the œdematous pad that overlies the bone. Curious special localizations of œdema met with, in cases of the kind, have been the scrotum, and together the upper trunk, upper extremities, and scalp and neck, so that the œdema simulates that resulting from mediastinal tumour. A common local pad of œdema, not rarely found in ordinary heart cases as well as in the cases just considered, is that over the sacrum—a region that should always be examined in heart cases even in the absence of œdema of the lower extremities. In the production of the combined affection of the peripheral nerves and the heart met with in beer drinkers, arsenic has been shown to play a conspicuous part. Pigmentation of the skin, of various distribution, in arsenical cases often affords an indication of the influence of this toxic agent. It would seem that the peripheral element, that is probably present

more or less, in all dropsies is, in these cases, specially developed. Vaso-motor influences, that is to say, the control of the arterioles by the sympathetic nervous system, are often clearly at work in the production of œdema, as may be observed in hemiplegic limbs, and especially those of cases of Bright's disease with secondary cardiac muscle-failure. The patient has a "stroke" or hemiplegic seizure, and the palsied limbs show the first œdema ever witnessed in the case.

In ordinary chronic heart cases, the œdema, as already stated, occurs in the lower extremities, and slowly travels up the limb if treatment be neglected and the patient do not rest in the recumbent posture. The lower portion of the trunk is apt to become early involved, especially the sacral region, and in unfavourable cases the subcutaneous tissue of the abdomen and lumbar regions are infiltrated with serum and enormously thickened. The external genitalia are very commonly involved. Above the pubes, in chronic cases specially, the lymphatic vessels become obviously dilated. In heart cases without albuminuria, that is to say in cases in which there can be no kidney complication, the upper extremities and even the face may be involved in the œdema. In most heart cases, however, when œdema has become so developed and distributed, there is albuminuria, due to the state of

venous stasis, if indeed this state has not predisposed to inflammatory or sub-inflammatory parenchymatous changes in the kidneys, so that it is impossible to exclude a nephritic element in the production of facial or brachial œdema. But, rarely such general dropsy may be found without albuminuria. The posture of the patient often has a great determining influence in the localization and development of cardiac dropsy. Thus the limbs, trunk, and face of the side on which the patient lies, may be much more swollen than on the other side, while pleural effusion may be influenced in the same way. Associated with the infiltration of the subcutaneous cellular tissue, the serous sacs of the viscera are often the seat of effusion, and the pleuræ are implicated with special frequency. If both pleuræ are healthy, hydrothorax is usually bilateral, but one pleura may have become adherent previously, and then œdematous infiltration of the lung is apt to take place on that side, while fluid is effused into the pleural sac on the other. Again, a certain amount of inflammatory action in the pleura is very common in the course of chronic heart disease, and is apt to determine a unilateral or bilateral effusion according as the affection is of one or both sacs.

The convex upper border of the dull area of effusion and the practical immobility of the same

in changes of the patient's posture must be borne in mind, especially with regard to right pleural effusion. These physical conditions are identical in cases of effusion of inflammatory origin, and in those, in which the effusion results from venous stasis only and forms but a part of more or less general dropsy. Serous accumulation in the pericardium is also common. It likewise may be only a part of a general dropsy or be determined by inflammatory action even of slight intensity. The subject of pericarditis, however, will be considered in detail later.

With regard to dropsical effusion into the pleuræ and pericardium, it is important to remember that a large accumulation is apt to take place rapidly, and yet insidiously. Ascites or effusion into the peritoneum, has been specially referred to in association with hepatic and portal-venous stasis, and the possibility of the effusion being, in whole or in part, of inflammatory or sub-inflammatory origin has to be remembered. Peritoneal friction sound may be occasionally heard under the circumstances, but too much importance must not be attached to it, seeing that little more than a slight nutritive change in the serous membrane may determine the requisite condition for its occurrence, as ovariotomists have proved.

SYMPTOMS OF GENERAL VENOUS STASIS.

Pulmonary Symptoms:—Among the symptoms of heart disease that depend on the disturbed general circulation—venous stasis—there are none more important than those that have their origin in the lungs. The pulmonary circulation is early disturbed in all varieties of chronic heart disease, but in none earlier or more seriously than in cases of mitral stenosis. There is the unyielding narrowed orifice in front and the usually more or less hypertrophied right ventricle behind, so that quite early in the case the pulmonary circulation suffers and symptoms result. A tendency to bronchial catarrh is often the way in which congestion first takes expression. Occasionally after violent exertion hæmoptysis occurs. Such a hæmoptysis may, indeed, be the first indication of the lesion in a young subject. In that case it has not the significance of the hæmorrhage (to be considered later, *vide* p. 36) that arises from *embolism* and implies profound failure of contractility in the walls of the right auricle, and it may be of the right ventricle also, and clotting of blood in the recesses of these chambers,—especially the former.

Simple œdema of the lungs gives rise to a small-bubble-sound which, with the like sound met with in the incipient stage of pneumonia, constitutes *the vesicular bubble-sound* of Skoda or *true crepitation.* The best examples are found in the cases already referred to (*vide* p. 21), in which the pleura of the base of one lung has become previously adherent and the potential sac obliterated, so that hydro-thorax is rendered impossible, and, instead, serous effusion takes place into the vesicular tissue of the lung. Such effusion does not replace all—or anything approaching all—the air contained in the lung structure, so that the percussion-sound does not, as a rule, differ from that over healthy lung tissue—at most some degree of tympanitic quality may be assumed by the resonance, the "fulness" of which is little, if at all, diminished. Œdema of the lung does not give rise to dulness because the fluid does not displace all the air. A much more common adventitious sign heard over the bases of the lungs in heart disease is the sound due to the bursting of bubbles of much larger size than those of "crepitation" or the vesicular bubble. Such bubble-sounds are probably produced by the passage of air through fluid in the smaller tubes. They are similar to the bubble-sounds that often linger for some time at the bases of the lungs

after a severe bronchitis has subsided. Preceded or not by bronchitis, they are among the most frequent auscultatory signs met with in heart cases, and they are significant of decided disturbance of the pulmonary circulation, whatever be the cardiac lesion at work in their production. In the earlier stages of heart disease their presence indicates that at least the lesion, whatever it may be, has begun to disturb the pulmonary circulation seriously, and further developments are to be apprehended. Venously congested organs and tissues are ever prone to take on inflammatory action: thus the bronchial mucous membrane becomes "catarrhal," and as regards the lung tissue itself, the pneumonic process is one of the most common and fatal complications of heart disease and often accomplishes the end of the case. (For lung complications due to embolism *vide* p. 36.)

Urinary Symptoms:—Among the symptoms of chronic failure of the circulation, changes presented by the urine are of much importance as indicative of the state of the kidneys. The quantity, specific gravity, colour, proportion of normal ingredients, and the presence of abnormal ingredients, not to speak of their amount, are all-important data to the clinicist in estimating the gravity of a cardiac case. Although the secretion of urine is not to

be regarded as entirely dependent on the state of the circulation, there can be no doubt that, speaking generally, a rise of blood-pressure on the arterial side promotes the flow of urine, and on the venous side diminishes the same, hence it is that the urine is generally diminished when the kidneys are subjected to venous stasis and is increased when the arterial pressure is raised on the heart recovering power, but secretion depends on several factors, including the vital activity of the protoplasm of the tubular and glomerular cells, and the degree of contraction of the muscular fibres of the arterioles, whether under the influence of vaso-motor nerves or independent thereof, while the saline ingredient of the serum in the tissues has its influence. As a general rule there is no better indication of improvement in a heart case than an increased flow of urine. On the other hand a diminished flow and rising specific gravity are unfavourable indications, along with which, urate precipitates, and increased pigmentation—over and above that due to mere concentration,—and even the presence of bile pigment are frequent associates. Under such circumstances albumen is very likely to make its appearance, and when it is in considerable amount, careful search or centrifugalisation will often reveal a few casts—generally only

hyaline, occasionally granular and even cellular. The tendency of venously congested surfaces to take on inflammatory action, which was found in the case of the bronchial affection of heart disease, is also observed in the tubular apparatus of the kidneys. It is often hard to draw the line where the one condition (venous stasis) passes into the other (inflammation). A large quantity of albumen may have been present in the urine, and yet the pathologist will pronounce the kidneys to be only "venously congested," though microscopical examination will usually reveal indications that this state was being transgressed and nutritive changes in the direction of inflammation were being established. Then there is the great class of cases, in which heart failure supervenes on chronic kidney disease—Bright's disease. The history of the case is important under the circumstances, and deficiency in the pigmentation of the urine with a low specific gravity in the absence of polyuria are suspicious facts, when the quantity of albumen is considerable. Casts are usually easily found, but in a few cases of granular disease of the kidneys they are very difficult to find owing to their scarcity. Centrifugalisation of the urine is then important.

A wide survey of the whole case is essential in estimating the relationship of the heart and

kidney symptoms respectively. The direct kidney symptoms are almost entirely derived from an examination of the urine. Curiously enough, as we shall find later, decisive evidence of actual Bright's disease may be derived from an ophthalmoscopic examination of the eye, characteristic "albuminuric retinitis" being found. When albuminuria depends on mere venous congestion of the kidneys, and the patient has not been confined to bed, it will often be found that rest in the recumbent posture greatly diminishes, and often removes it. On the other hand, when the albuminuria is in considerable amount and remains persistent, although the patient be at rest in bed, while casts—hyaline, granular and especially cellular—are plentiful in the deposit, it may be assumed that there is more than mere "congestion" of the kidney present. Again, in a few cases of granular kidney the symptoms and signs are those of heart disease entirely, although there is a certain amount of albumen in the urine—it may be little more than a trace in a urate-depositing high-coloured urine—and casts may be few and difficult to find, if present at all. In such a case there is great risk of the profound kidney disease being overlooked, unless it should be found that the patient has albuminuric retinitis.

Under the circumstances the specific gravity of

the urine is often rather low, considering the urate deposition of the secretion, while its pigmentation is normal, or even higher than normal, because the liver is congested, and there may even be biliary pigment present from the same hepatic condition (p. 15). It will often require the application of all the observer's knowledge of the features of heart and kidney disease respectively, to enable him to recognise the existence of actual kidney disease, as it were "behind" the heart disease. It is in cases of simple muscle failure of the heart, without valve disease, that the recognition of the combined morbid conditions—cardiac disease and Bright's disease—is most often required. But all forms of heart disease in their advanced stages are liable to become associated with nephritis. Apart from embolism of the kidney, the symptoms of which will be described later, hæmaturia points to nephritis, especially when fairly persistent. It cannot be denied, however, that hæmaturia may result from mere passive congestion, venous stasis of the kidney, but the quantity of blood that may find its way into the urine under the circumstances is probably never large. When the presence of blood in the urine is recognised by the naked eye, acute nephritis or embolism is its probable cause.

Gastric Disturbance:—Although the readiness

with which the liver swells under the influence of venous stasis saves the spleen to a certain extent from like swelling, the whole portal venous system must suffer more or less, as soon as cardiac failure has led to disturbance of the circulation and systemic venous stasis. The stomach is thus venously congested, and its mucous membrane is apt to pass into a catarrhal state, according to the pathological law so often affirmed: that the venously congested mucous membrane is apt to take on inflammatory action—the law found to be so early in operation in the bronchial mucous membrane. In the case of the stomach, acute attacks of catarrh are specially apt to occur, which during their existence seriously interfere with alimentation, for the food taken into the stomach is almost at once rejected, or, if it be suffered to remain, fails to be properly digested in the stomach, although should it succeed in passing the pylorus, it may be dealt with more efficiently lower down in the alimentary canal. Along with the food that is rejected, the vomited matter usually contains much mucus, and minute quantities of blood are not uncommonly present.

Sickness and vomiting, while of much importance in themselves as impediments to nutrition and depressors of cardiac energy, are of special importance with regard to therapeutics. For it

may be said that most drugs which are used in the direct treatment of heart disease, exert an action upon the stomach too. In the first instance this is exerted possibly on the muscular coat of the organ, giving rise to pain of spasmodic character, and later to vomiting. Great care must be taken in estimating the influence of any drug, that is being taken, in the production of sickness, and there must not be too great tendency to blame the drug in the knowledge that the catarrhal process is apt to arise in the gastric mucous membrane, as in other like membranes, simply as the result of venous stasis, along with possibly some slight accidental irritation produced by the contents of the viscus.

Disturbances in Intestinal Canal:—Perhaps, on the whole, the influence of venous stasis, as exerted on the intestinal canal, is towards constipation, with sluggishness of movement and deficiency of secretion. Now and again, probably owing to the formation of irritating substances in the contents, diarrhœa is set up. As a matter of fact, however, opportunities of observing the intestinal functions uninfluenced by drugs are not often forthcoming, so common is the practice of giving purgatives with the object of depleting the portal circulation and diminishing its venous stasis (*vide* Treatment, p. 338). Hæmorrhage from the mucous membrane

of the intestine occurs occasionally, apart from embolism (*vide* p. 38), and seemingly as the result of congestion of the mucous membrane sometimes purely mechanical (venous stasis) and at other times partly of inflammatory origin.

Cerebral Symptoms:—Mental disturbance is occasionally associated with heart disease. Most commonly it is only the "mental wandering" that so often attends slow dissolution, irrespective of its form.

Dr. Head has called attention to hallucinations of ill-defined spectres that stand by the bedside, and distress the patient, who is apt to be peculiarly reticent as to the cause of his depression. Writers on insanity have gone so far as to ascribe certain varieties of mental aberration to certain forms of heart disease. Such a belief has probably little foundation in fact. The delusions of cardiac sufferers are almost always of a painful description: not rarely they involve suspicion and apprehension of evil intention on the part of attendants and friends. Under the influence of delusion patients may perform acts that might have been thought impossible to them, and in so doing may cast away the last chance of temporary recovery that remains to them. While the view that certain forms of insanity accompanying certain cardiac lesions is deprecated, it may be

admitted that the brain probably suffers to some extent differently in cases of extreme cyanosis and venous stasis, in the circulatory disturbance resulting from great aortic incompetence, and in cases of muscle failure of alcoholic origin. Cases of pulmonary emphysema with cardiac dilatation and of mitral stenosis furnish examples of the first type, while in the third type there is usually a direct toxic influence exerted on the brain itself by the agent that led to the weakening of the heart muscle. Patients with emphysema are specially apt to vociferate in their sleep, although they are rational when awake. In the heart failure of Bright's Disease, again, besides the disturbed cerebral circulation and some degree of cyanosis, the influence on the brain of various toxic bodies—varying even in the course of the same case—has to be admitted. In septic endocarditis, with the development of pyrexia and the "typhoid" state—dry brown tongue, twitching of the hands, etc.—the delirium may be of the type characteristic of typhus fever, and so also in the last stage of Bright's Disease, usually without fever and occasionally with subnormal temperature. Active delirium often accompanies the hyperpyrexia of acute rheumatism so often associated with endocarditis and pericarditis.

For the most part, if not altogether, dependent

on the state of the cerebral circulation are probably the syncopal and epileptoid "faints" and convulsions associated with extreme infrequency of the heart's action, or "*bradycardia*," though the infrequency of the pulse is believed by some to have its origin in the nerve centres.

Thrombosis of cerebral arteries, owing to their local disease, may account for certain "strokes" occurring in the course of heart disease. Cerebral hæmorrhage of ordinary type may likewise be met with in association with heart disease. It must not be assumed that because a patient has heart disease he is exempt from the ordinary local accidents that depend on vascular degeneration in the brain. Moreover, given vascular degeneration, feeble action of the heart may contribute to thrombosis and too forcible action to rupture of a vessel, with consequent apoplexy and hemiplegia. The most characteristic cerebral conditions met with in heart disease, however, depend on the embolic process (*vide* p. 39).

Thrombosis of Large Veins, *especially of the legs*:—Thrombosis of large veins is by no means of rare occurrence in heart disease, especially in its later stages, though it is apt to be overlooked in the presence of general dropsy. Local pain in a limb should always call for careful examination of the part, as should also the special development

of dropsy in one limb, which suggests thrombosis of a vein. The most important symptoms due to arterial conditions are described under embolism, but it may be here repeated that the effects of local disease of an artery are apt to be intensified by any failure of the general circulation, and so the occurrence of thrombosis is promoted.

SYMPTOMS OF THE EMBOLIC PROCESS.

Pulmonary Embolism:—One of the most common symptoms of advanced heart disease is "*hæmoptysis*" due to embolism of the lung—the "hæmorrhagic infarct" of pathologists. "Pulmonary apoplexy" is an old-fashioned name for the accident, though an utterly unsuitable one, seeing that it connotes the symptoms of hæmorrhage into the brain instead of into the lung. In "pulmonary apoplexy" the patient usually spits up pure, dark-coloured blood, often in considerable quantity, but never in sufficient quantity to render necessary* the mere loss of blood being taken into consideration. The mass of consolidation formed by the infarct is usually small, so that dulness cannot usually be detected unless several infarcts occur in close proximity, or a pneumonic process be set up around. Pleurisy, again, is often set up over an infarct, and may result in effusion. The most

* As this is passing through the press, a case occurs in the hospital practice of the writer that would make him slightly modify this statement—the case being one of mitral stenosis.

characteristic auscultatory sign of "infarct" is a small-bubble-sound, the bubbles being much larger than those of true crepitation, and they may accompany expiration as well as inspiration. Friction sound may complicate the intra-pulmonary adventitious sounds. Bronchial breath-sound is seldom heard, and occurs only when there is a large mass of consolidation formed, and even then much more seldom than might be expected. The bronchial tubes may become blocked with blood. Usually the breath-sound is *indeterminate* (Skoda), *i.e.*, neither vesicular nor bronchial, and it is often obscured by the abundance of the râles. The physical signs, however, afford less valuable evidence of infarction than the *hæmoptysis*, for similar signs occur commonly enough in cardiac cases from other causes than infarction.

Embolism of the Kidney:—An embolism in the kidney may be revealed clinically by vomiting and the appearance of blood in the urine. Pain in the back has been complained of, and shivering may occur when the temperature rises abruptly. These symptoms usually soon pass off, and no doubt embolism of the kidney often occurs without clinical recognition, the presence of blood in the urine having been overlooked or misinterpreted, while subjective symptoms are trivial or even absent. Mention need only be made of such

conditions as embolic obstruction of the renal artery or the rarest of accidents: a block in each renal artery resulting in suppression of urine.

Mesenteric Embolism:—A decidedly rare embolism is that of the mesenteric arteries. The patient has usually sudden abdominal pain, followed by shock and collapse, and later by a profuse hæmorrhage from the bowel.

Embolism of Arterial Trunks and Branches in the Limbs:—Any of the arteries in the limbs may become the seat of embolism, and according to the size of the vessel and the amount of anastomosis, the consequences vary. Severe pain at the site of the obstruction and pain and numbness or tingling in the limb beyond, may indicate the accident. In every case, as already stated, when pain is complained of in a limb by a patient with heart disease, the affected part must be carefully examined. Rarely gangrene of the limb, within the distribution of the obstructed artery, is the result of the obstruction, but when this happens the case is usually far advanced and grave, quite apart from the immediate accident. An *aneurysm* may form later at the site of obstruction, the coats of the artery being damaged locally, and consequently yielding before the blood-pressure. Either the lesion of embolism or the presence of a consequent aneurysm may be found on examination, without

the patient having made any complaint, but usually there has been some suffering, at least sufficient to indicate the time when the artery was obstructed.

Cerebral Embolism. The vessels most commonly the seat of embolism of the brain are the middle cerebral, posterior cerebral, and the vertebral. The first is by far the most commonly plugged artery, being most directly the continuation of the internal carotid, while the left artery is the one more usually affected, probably owing to its origin lying more directly in the course of the circulation. Right hemiplegia results and is often associated with aphasia. The vertebral arteries are much less frequently the seat of embolism, and here again the left artery is the one usually involved, owing to its coming off from the sub-clavian while that artery is almost vertical, instead of horizontal as on the right side. The posterior cerebral arteries do not show any predisposition, a fact explained by a plug necessarily having had to pass along the basilar artery before reaching them. The basilar artery is rarely plugged, as the plug in its case would have had to pass through the smaller vertebral artery. The anterior extremity of the basilar is rarely, however, the seat of embolism.

Curiously enough the middle cerebral artery may

be the seat of embolism *on both sides*, but the accidents are usually not simultaneous. A rare result of cerebral embolism is the local formation of *aneurysm*, owing to arteritis being set up with consequent yielding of the affected portion of the arterial wall. Such an aneurysm may finally rupture, the patient dying of apoplexy. The sudden occurrence of hemiplegia in a young person, known to be the subject of heart disease, is almost certainly of embolic origin. Such a seizure is often right-sided, and associated with aphasia, the left middle cerebral artery being the obstructed vessel. The special symptoms that may result from special arterial branches being blocked by embolism, it would be inappropriate to discuss here, they will be found described in treatises on diseases of the nervous system. In its common form, and due to the middle cerebral and its branches being obstructed, the hemiplegia that results is ushered in abruptly and without the premonitory symptoms that often precede the attack when due to the local disease of a cerebral artery (atheroma and syphilis).

The simple, convulsive and apoplectic modes of onset of hemiplegia may all result from embolism, but the simple is most common when the lesion is embolic. Convulsion often attends implication of the branches supplying the convolutions of the motor area. Headache and vertigo may be experienced,

when there is no loss of consciousness. Rarely there is slight transient delirium. Along with the loss of power, numbness or tingling of the affected limbs is common. As in other forms of hemiplegia, symptoms due directly to the lesion which do not, or only partially pass away, must be distinguished from those that are only indirectly due to it and depend on inhibition, or other influence exerted upon neighbouring or otherwise related parts of the cerebrum. The latter symptoms are usually transient only.

Retinal Embolism:—A rare variety of embolism is that of the retinal artery causing interference with vision and special ophthalmoscopic appearances.

Splenic Embolism:— The spleen may swell and become tender on palpation below the ribs, when the seat of embolism, and friction may become audible over it, owing to the capsule being inflamed. More or less pyrexia is usually present.

CLINICAL PATHOLOGY.

By clinical pathology it is meant to imply the knowledge of the processes of disease, which it is necessary that the observer—be he student or physician—must have in mind in his interpretation of the clinical facts of any heart case which comes before him. The patient may have told the history of his health from childhood onwards, and described his recent symptoms from their outset, while objective physical examination may have revealed many important departures from the normal state of the organism, and yet without the light that pathology alone can throw upon it, the case can only be partially appreciated.

A junior student wanders prematurely into a medical ward, in which a case of aortic incompetence is being demonstrated; his knowledge of anatomy and physiology should enable him to appreciate a large number of the objective signs—the jerking pulse and even the displaced and exaggerated impulse of the apex beat; the shortness of breath

and dropsy he can even understand, as consequences of the disturbed circulation, that must follow the lesion, but when the history is being enquired into, he can hardly be expected to appreciate the emphasis laid upon certain questions as to rheumatism, syphilis, hard physical work, the experience of pain in the arm, etc. To fully appreciate these he must have some knowledge of the processes of disease: of "*pathology*." To supply him with a very rudimentary knowledge of the kind, concerning heart disease in general, is the object of the following paragraphs, and first of all those processes that are concerned in the production of aortic disease will be considered as they are perhaps the most simple, inasmuch as there is no "contractile" element to be considered in the case of the aortic valves as there is in that of the mitral valves.

The aortic valves may be rendered incompetent in two different ways:—

(*A*) The valves may be deformed and so rendered incapable of perfectly sustaining the column of blood, it is their function to support, and

(*B*) They may be rendered incompetent while sound in structure, because the orifice they have to close has become enlarged.

The enlargement of the orifice indicated, results

from the walls of the aorta having lost their elasticity because of a chronic inflammatory and degenerative change, and yielded before the blood pressure. The ring to which the valves are attached long resists, but it too may finally yield, and so the valves become incompetent without themselves undergoing change.

(*A*) Structural damage in the valves themselves is by far the more important mode of production of incompetence. The pathological processes that bring it about are several:—(1) Rheumatism. (2) Septic endocarditis. (3) A low inflammatory process, the result of the valves having been habitually exposed to excessive strain, in men whose occupation has been laborious. (4) Implication of the valves in the ordinary atheromatous—inflammatory and degenerative—processes of the aortic wall. (5) Implication of the valves in syphilitic disease of the aorta.

(1) Rheumatic fever is apt to be associated with an endocarditis specially affecting the valves, as a result of which, deformity and incompetence are produced. The disease Chorea or Saint Vitus's Dance may be regarded as of the same significance as rheumatism (with which it is often associated), in so far as an attack is likely to have led to structural damage of valves, but as a matter of practical

experience disease of the mitral valves is much oftener the result of chorea than disease of the aortic valves. Rheumatic endocarditis seems to affect the mitral valves by preference, too, and when the aortic valves are damaged in a rheumatic case, the presumption should be that the mitral valves have not escaped, even though obvious evidence of their implication is absent at the time of observation. Rheumatic endocarditis acquires its chief importance in clinical medicine not at the time of its occurrence, but years afterwards, owing to the deformity of the valves, that results from chronic shrinking occurring in the damaged tissues of the affected valves. This process generally entails incompetence of the valves and often some degree of stenosis or narrowing of the orifice as well. The latter event is specially common, as we shall see, in the case of the mitral valves, but it is not very rare in the case of the aortic valves.

(2) Septic endocarditis is comparatively a rare disease, due to micro-organisms, but the damage wrought by it in the valve structure, is infinitely greater than that usually wrought by rheumatic endocarditis. Moreover, the disease is generally, if not invariably, fatal within a comparatively short period of time. We may presume, then, that septic endocarditis practically never eventuates in chronic disease of the heart. The illness through-

out is an acute or sub-acute febrile one, apart from the symptoms and signs that have their origin in the damage wrought in the valves and heart generally, and its clinical features will be described later.

(3) The third form of aortic valve disease resulting in incompetence is the very chronic thickening and shrinking process that results from exposure of the valves to frequent strain. Such changes are not necessarily accompanied by atheromatous changes in the aortic wall: in any case these latter are comparatively incipient.

(4) Fourthly, the atheromatous process may involve the valves and deform them, though essentially one attacking the aortic walls.

(5) Syphilis possibly promotes the ordinary atheromatous process, rendering its appearance premature, but the affection here referred to is of very limited extent and well defined, especially above. The valves are not affected primarily, though they may become implicated and incompetent as they may do in the ordinary atheromatous process.

Syphilitic aortitis, if such it may be called, is of great importance, not only as a cause of aortic incompetence in the comparatively young, (and it may be, female subjects) in whom there has been

no exposure to strain, but also as a cause of angina pectoris (*vide* p. 201), the patch of disease having involved and narrowed the coronary orifices, commonly both, more or less.

B. Affection of the wall of the aorta leads to incompetence of its valves by the process of dilatation, that results from the loss of its elasticity. Such dilatation is usually widely spread, involving the arch more or less, but there is neither *saccular* expansion nor indication of *pressure-symptom*, though the condition of so-called *fusiform aneurysm* may be produced. The ring of valvular attachment is often finally implicated, and yields, so that the sound valves are no longer capable of covering the orifice in diastole.

The physiological second sound of the heart, in cases of the kind, early undergoes a modification—accentuation—which persists later, when the leakage that results has produced the murmur of incompetence.

(1) The ordinary atheromatous process leads essentially to loss of elasticity, and consequent yielding of the first part of the aorta, but ultimately to dilatation of the aortic orifice. In its common form the dilatation of the vessel is diffuse, and the yielding of the valvular ring is of very gradual production.

The low inflammatory process, almost from its inception is linked with degenerative changes which, beginning in the vicinity of the valves, spreads more or less diffusely, unlike the well-defined small patch of the syphilitic process. This common type of disease, atheroma of the aorta, is almost a natural feature of the later periods of life, even lived without strain. Under a variety of conditions the same process seems to be precipitated and exaggerated, and such conditions are apt to occur in combination. Gout, with its high arterial tension, and often associated kidney degeneration, seems to promote atheroma. Heredity has also to be reckoned with, even apart from gout with its declared disturbance of metabolism. Some individuals have—as is said *constitutionally*—high arterial tension, just as others have low. Hard work, and even syphilis, may find an easier victim, as far as the aorta is concerned, in the former subjects.

(2) True saccular aneurysm of the aorta of the ordinary type and often associated with pressure-symptoms, frequently leads to incompetence of sound aortic valves. The modification of the physiological second sound already referred to (accentuation) long precedes in this case the murmur indicative of the valves having become incompetent. Aneurysm, to produce this effect,

must involve the first or ascending portion of the arch.

A curious consideration is that the mere presence of aortic valve incompetence tends to promote and render premature the ordinary type of atheroma. This fact is illustrated by the occurrence of such atheroma prematurely in rheumatic subjects, whose aortic valves have been rendered incompetent by rheumatic endocarditis.

In the case, then, of aortic incompetence revealed by the presence of certain physical signs, the observer, after having heard the patient's statements as to his symptoms, by which the amount of actual disturbance of the circulation is to a large extent estimated, must form some conception of the lesion, *i.e.*, the precise condition of the aortic valves and adjoining aorta—these two being considered together. Are the valves themselves implicated in structural change? Are the valves themselves normal, and only rendered incompetent by dilatation of the orifice, sequential to that of the channel of the aorta, the whole process depending on disease and loss of elasticity of the aortic wall?

The condition of the second sound is of much value in answering these questions:—

(A) *The second sound is absent or defective, and*

there is diastolic murmur in the aortic region (*vide* p. 105). Actual valve disease is rendered likely:—What are the pathological processes, one of which is likely to have been at work in the production of the incompetence? The observer has to think of no fewer than the five conditions already referred to:—

(1) Rheumatic endocarditis.

(2) Septic endocarditis.

(3) Exposure to habitual strain in a patient in or under middle life.

(4) Ordinary atheroma involving the valves: often there will have been exposure to gout, plumbism, worry, and other causes of high arterial tension in a patient under or in middle age, while in later life ordinary wear and tear suffice to produce the condition.

(5) Syphilis, especially when the patient is under middle age.

B. *The second aortic sound is not only present, but accentuated, in spite of the diastolic murmur.*

Disease of the wall of the aorta, with consequent loss of elasticity, and yielding of the same is rendered probable; moreover the structural integrity of the valves themselves is likely. The observer has to think of the following conditions:

(1) Ordinary atheroma. The patient being in or over middle age; the aorta dilated.

(2) Aneurysm. There are the signs and symptoms of true saccular aneurysm involving the ascending portion of the arch (*vide* p. 180).

An important consideration in diagnosis is the presence of a combination of causes in the same case. Thus a man may have had rheumatic fever say, at the age of 18; have contracted syphilis after 20; been exposed to strain after middle age, and present the signs of aortic incompetence when examined for the first time at the age of 55. In such a case the presence of an accentuated 2nd sound would be in favour of the ordinary atheromatous process, in spite of the history of rheumatism, which, be it remembered, did not occur till adolescence, when the aortic valves might well have escaped endocarditis.

Numerous as are the possible conditions that have to be considered in explanation of incompetence of the aortic valves, they are much simpler than those to be considered in the case of the mitral valves. The aortic valve apparatus is comparatively simple, and acts in purely mechanical and passive fashion. In the case of the mitral valve apparatus, a vital and contractile element has to be reckoned with.

No mitral valves are competent without the assistance of the heart muscle. Muscle contraction is a necessary complement of the mitral valve apparatus.

In a very large number of cases of mitral incompetence there is no structural disease of the valves; they are rendered incompetent only on account of their failing to receive, in the performance of their function, the assistance from the heart muscle, that physiologically they are entitled to.

In two ways is muscle contraction necessary for the perfect function of the mitral valve apparatus: it diminishes the orifice to be closed by the valves, and by shortening the musculi-papillares it keeps the spread-out curtains taut. In an enormous number of cases of mitral incompetence, the incompetence is due to muscle failure of the heart only, and is independent of any structural change in the curtains. The causes of cardiac muscle failure are extremely numerous, and often two or more work in combination. Only a few of the more common can be mentioned here. The condition of anæmia is one of the most common, and is curious as seemingly affecting the right side of the heart primarily and, as it were, specially, though by no means only. The left side sooner or later is inevitably affected, unless the case is speedily cured.

Chronic Bright's disease offers, again, common examples of mitral incomptence due to cardiac muscle failure. Hypertrophy may have been long predominant, but sooner or later, if the patient escapes cerebral or toxic accident, such failure sets in, and mitral (and it may be tricuspid) incompetence is one of the most common results.

The association between the gouty state and granular disease of the kidney is notorious; but before the kidney has become seriously damaged the high arterial tension, so common in the gouty, may have determined mitral incompetence. In cases of the latter kind it must, however, be remembered that the muscle failure of the heart may be only relative and strictly comparable with the so-called "safety-valve action," long ago described by physiologists as occurring on the right side of the heart under the influence of temporary obstruction to the circulation in the lungs. A similar occurrence takes place in the case of the mitral valves. Thus it is no rare thing to hear the systolic apical murmur of mitral incompetence in gouty and allied states of the constitution, while the arterial tension is still maintained at a high degree. When the vast importance of the muscle complement in the mitral-valve-apparatus is realised, it will be obvious how difficult it is to assess the amount of incompetence that may be

referred to it, and the amount that may be referred to coincident valve change wrought by rheumatic or septic endocarditis, for in the worst forms of the former, which occur specially in young subjects, the muscle of the heart is apt to be implicated—*myocarditis;* while in every case of the latter the heart muscle has been exposed to the depressing influences of pyrexia and toxæmia.

Although the consideration of mitral incompetence is so much complicated by the introduction of the "muscle element" into the valve apparatus, it is simpler, in so far as the causes of structural damage to the valve curtains are fewer. Thus the exposure to habitual strain as a cause of thickening and contraction of the valve curtains may be dismissed as improbable in the case of the mitral valve apparatus with its "safety-valve" possibilities, and as much may be said of the effects of habitually high arterial tension and for the same reason, while syphilitic affection of the mitral valves is practically unknown. Atheroma probably never plays an important part in damaging the mitral valves.

There remain:—

(1) Rheumatic endocarditis.

(2) Septic endocarditis.

(1) The importance of *rheumatic endocarditis*

depends chiefly upon its being *the great source of chronic valve lesions, and especially of stenosis of the mitral orifice.* It is in this last that the majority of cases of rheumatic endocarditis eventuate, and the victims are almost always young at the time of the endocarditis. In the cases of acute rheumatism in which recovery of the general health takes place, progressive structural changes are set up in the valve and end in the production, it may be years later, of a narrowed mitral orifice. In the cases in which recovery of general health does not take place and the heart undergoes rapid dilatation, the structural damage to the valve curtains is often only trivial, and it is evident that the predominant factor in bringing about the disastrous result has exerted its baneful influence essentially on the heart muscle—the myocardium. Pericarditis, under the circumstances, is a common antecedent, and its result—adherent pericardium—was at one time supposed to exert a peculiarly mischievous effect upon the myocardium. This view was soon modified, however, by the frequent observation of the same disastrous dilatation of the heart in the absence of adherent pericardium and the no less frequent observation of adherent pericardium without dilatation of the heart (*vide* p. 280). These cases of rapid progressive muscle failure, which are

essentially limited to the young and characterised clinically by the greatest amount of mitral regurgitation (if loudness and free conduction of murmur to the back can be accepted as evidence of freedom of regurgitation), are far from rare, while the valve changes found *post-mortem* do not bear comparison for a moment with those that result from septic endocarditis in the damage done.

The importance of endocarditis during a first rheumatic fever attack then is remote in the majority of cases: it depends upon the likelihood in years to come of the formation of mitral stenosis. This is indeed the great mitral *lesion* associated with rheumatism. Mitral incompetence is no necessary indication of *mitral disease* at all.

(2) But if the lesion left by rheumatic endocarditis is in the first instance generally trivial, the case is otherwise with regard to septic endocarditis, which often structurally damages the valve curtains to a very serious degree—to such a degree, in fact, as to render them incapable of performing their function, and that quite independently of coincident cardiac muscle failure. This disease being almost inevitably fatal within a year or a little more, does not derive its importance, however, from the gravity, great as it is, of the valve lesions, for the too simple reason that the fatal effects to which they would mechanically lead are anticipated

by those of the toxæmic and septic illness, and it may be of embolism in such important organs as the brain.

As already indicated, mitral stenosis is the goal at which rheumatic endocarditis of the mitral valves usually arrives. The different types of mitral stenosis have not been utilized in practical medicine sufficiently to give them clinical importance, and the same lesion may often be regarded as of one or other type according to the point of view from which it is inspected.

A history of acute rheumatism or chorea then is of great importance, from the foregoing facts, in the diagnosis of mitral stenosis without characteristic murmur. Still, in a considerable proportion of cases the diagnosis has to be made in spite of the absence of an attack of rheumatism from the patient's past history. Especially is this the case in young women. In a much smaller number of instances conclusive evidence of mitral stenosis is found in characteristic murmur, while the patient denies all history of rheumatism and chorea. In a proportion of these cases in both sexes, mild rheumatism in childhood* has been undoubtedly overlooked, and it has happened that the young woman with conclusive evidence of mitral stenosis, who denied all

* Often regarded as "growing pains."

history of rheumatism and chorea, has actually, while under treatment for heart symptoms, developed her *alleged first attack* of rheumatism.

The fact that in childhood rheumatic arthritis is apt to be very trivial and easily overlooked, while endocarditis may be at the same time pronounced, though latent as regards symptoms, is of immense importance, and should call for the frequent examination of the heart in children suffering from various—especially febrile ailments. On the other hand during the course of acute rheumatism the absence of murmur must not be taken to guarantee absence of endocarditis.

In the consideration of the pathology of heart disease, morbid physiology, as well as morbid anatomy, demands attention. Especially is this the case with regard to what may be called the vital reaction of the heart muscle in the presence of a valve lesion or valve lesions.

To take the simplest example: aortic stenosis without incompetence of the valves. The chamber, on the part of which reaction may be expected, is of course the left ventricle, and the reaction takes the form of hypertrophy of its walls. It has long been recognised that this lesion is often borne for a long series of years almost without symptoms—certainly without suffering—and that the left ventricle retained its size, or nearly so. The lesion by its

nature is a very slowly produced one, and moreover all that can be required from the left ventricle on account of it, is increased driving power supplied by growth of the contractile structure. Any increase in the size of the cavity would render the burden imposed upon the ventricle much greater, inasmuch as the larger the cavity the greater contractile force required from its walls for its emptying. Dilatation of the ventricle is thus an unmixed evil, and hypertrophy of its walls is all that is necessary to compensate for obstructive lesion at the aortic orifice. Towards the end of such cases, symptoms—dyspnœa, congested liver, dropsy, etc.—are, however, apt to manifest themselves, and dilatation of the left ventricle having once set in, proceeds rapidly. There is now "muscle failure" on the part of the left ventricle, and its cavity as it increases in size requires more and more power on the part of the already failing muscle of the wall. Disaster under such circumstances is inevitable, but let it be remembered how long these circumstances may be delayed in the case of aortic stenosis. Aortic incompetence, again, reacts very powerfully on the left ventricle, but in a different way. A moment's consideration, in its case, shows that the evil effects of the lesion must be experienced by the ventricle during its diastole. Hypertrophy, then, cannot be a result of the lesion in

the first instance. Between the lesion and hypertrophy there stands the "unmixed evil of dilatation"—increase in size—of the left ventricle. The filling of the left ventricle, from the aorta backwards, as well as from the auricle forwards readily explains the dilatation that ensues, for how otherwise are the increased contents to be accommodated?

It is no wonder then that cases of free aortic incompetence afford the best examples of huge left ventricles.

CARDIAC MUSCLE-FAILURE.

There has next to be considered that great class of cases of heart disease in which there is no disease of the valves, and in which disturbance of the circulation is brought about as the result of "muscle-failure," as it is conveniently termed. However produced, cardiac muscle-failure is apt to take expression in dilatation of the chambers—auricles and ventricles. Sometimes the chambers on the right side are primarily and principally dilated, at other times those on the left, but whichever side takes the lead in the pathological process, the change does not remain limited to that side. That the right side should follow the left seems only natural, but the opposite sequence is less easy to explain. For instance, in cases of pulmonary emphysema, in which the pulmonary circulation is primarily obstructed, and in cases of mitral stenosis, the dilatation does not as a rule involve the right chambers only, for the whole heart ultimately becomes enlarged, though the right chambers retain

the lead. There would seem to be a natural law, which for lack of a better name, we may call that of the "solidarity" or unity of the heart, and by which the ventricles keep pace in expansion to a certain extent. A good example of the right chambers becoming dilated in sequence to the left is found in cases of Bright's disease—"granular kidney" especially—in which the walls of the left ventricle have been long hypertrophied, and ultimately the cavity becomes enlarged as a result of muscle failure. When the latter event has occurred, the enlargement involves the right chambers in greater or lesser degree. Cases of free aortic regurgitation also furnish good examples of the left ventricle being primarily and principally affected—and so it may remain,—but the right ventricle, with its auricle, ultimately undergoes similar change in most cases, though it may lag a long way behind. In the case of aortic incompetence, dilatation would always seem to precede hypertrophy, while in aortic obstruction hypertrophy precedes dilatation. Chronic pulmonary emphysema and mitral stenosis furnish the best examples of dilatation of the left ventricle following that of the right.

One of the most common and least serious forms of dilatation of the heart, is that due to the anæmia of young women (chlorosis), and the right chambers are specially involved, and a difficulty in the pass-

age of the blood through the lungs may indeed be imagined. At all events the right chambers manifest the earliest changes. The infundibulum of the right ventricle is specially affected, as is shown by pulsation appearing above the third left costal cartilage, while the implication of the right auricle is revealed by the development of venous pulse in the neck. Nevertheless, provided the determining condition be sufficiently prolonged, the left ventricle (and no doubt the left auricle too) becomes involved and suffers dilatation. *Muscle failure tends to dilatation of the heart,* and with regard to the principal chambers—ventricles—this result may be theoretically considered as explicable in two ways:— (1) Muscle-failure is likely to accomplish incompetence of the auriculo-ventricular valves by insufficiently diminishing the auriculo-ventricular orifice, which has to be closed, and insufficiently shortening the musculi-papillares. Incompetence of the valve implies a backward current of blood into the auricle, but the regurgitated blood has to be returned to the ventricle *plus* the normal quantity next systole if the circulation is to be maintained. Room for the regurgitated blood, in addition to the normal amount, can only be provided by dilatation and enlargement of the cavity. (2) A much more important consideration, however, is

imperfect contraction on the part of the left ventricle—*asystole* or *systole catalectic*. By which occurrence residual blood remains in the ventricle at the end of systole over and above the physiological amount, which is limited to the supra-papillary space between the mitral valves and the apices of the musculi papillares. As Roy and Adami showed, however, the evil effects of imperfect contraction are not limited to systole, for the "increase in the residual blood does not, however, under normal circumstances (they are speaking of physiological experiment) lead to a diminution of the amount of blood expelled by the heart in a given time, seeing that *it is compensated for by an increased expansion during diastole*." Moreover, the larger a cavity of the heart becomes the more contractile power is required of its walls for its complete emptying, and if failure of this very contractile power be the cause of the dilatation, it is obvious how serious is the condition. It is a true saying that as regards the heart, while hypertrophy has its merits, dilatation is an unmixed evil.

The immense importance of the blood supply to the heart muscle must be realised. When the coronary main branches are narrowed by local disease angina pectoris is apt to be produced.

When minor branches are obstructed so-called "cardiac aneurysm" may result, the portion of ventricle-wall that has its blood supply more or less cut off, losing its contractility and "degenerating," so that local bulging results, or there may be rupture of the ventricular wall and sudden death.

In a syphilised subject of cardiac muscle-failure the possibility of gumma of the myocardium must be borne in mind.

THE PHYSICAL METHODS OF DIAGNOSIS,

INSPECTION.

It is taken for granted that we have the patient in the recumbent posture with the chest bare, but, when possible, it is well to examine him in the upright posture as well. The various points to be attended to *on inspection* may be arranged in the following way:—

A.

1. Bulging of the whole cardiac region.
2. Bulging of the intercostal spaces in the cardiac region (rare).

B.

Displaced cardiac impulse (*i.e.*, when the whole organ is displaced as from pleuritic effusion, shrinking of the left lung, etc.).

C.—*Areas of Visible Pulsation.*

1. The apex-beat or impulse of the left ventricle.
2. So-called epigastric impulse or movement of the right ventricle.
3. Movement seen over a dilated infundibulum (conus arteriosus).
4. Movement seen over dilated right auricle.
5. Thrust forward of heart as a whole.
6. Irregularity of beats.

D.—*Vascular Pulsation.*

1. Aneurysm of aorta, etc.
2. Venous pulsation in neck.
3. Abnormal pulsation of carotids, etc.
4. Capillary pulsation.
5. Irregularity of arterial and venous pulsations.

E.

Systolic depression (1) of intercostal spaces over ventricles above the apex-beat and (2) of ribs, sternum, and space between xiphoid cartilage and ribs, with absence of apex-beats (rare).

F.

A double arch of varicose venules is not rarely seen extending from the sternum along the costal arches. It marks the division of the venous currents passing upwards and downwards respectively and is often exaggerated in heart disease, though it does not necessarily indicate heart disease at all.

A.

1. *Bulging of whole Cardiac Region.* This rarely occurs in great and generally chronic pericardial effusion. It is common in the cardiac enlargement associated with valvular disease, myocarditis, and often pericardial adhesion, in early life. Sometimes it is found as a consequence of rickets and interference with the due expansion

of the lungs. Œdema of the integuments, limited to the cardiac region, may occur in pericarditic effusion of some standing.

2. *Bulging of Intercostal Spaces in Cardiac Region.* This may result from pericardial effusion when of inflammatory origin and considerable duration (rare).

B.

By displaced cardiac pulsation is meant pulsation of the heart, *away from* the normal cardiac region, *with absence of pulsation in the normal area*, not merely pulsation due to extension of the normally situated heart beyond its legitimate limits. Displacement of the heart is most commonly observed in left pleuritic effusion, in which case it may lie quite to the right of the sternum. It is usually difficult to identify the apex-beat in such cases, and the tendency to engorgement of the right side of the heart under the circumstances must be remembered. Retraction of lung substance may draw the heart to the right or left, but generally upwards, inasmuch as it is the upper portions of the lungs which chiefly undergo phthisical contraction. Ascites, ovarian cystic disease, and tympanitic distension of the bowels, elevate the whole heart, besides causing it to lie more horizontally. Hydatids growing from the

upper surface of the liver may likewise displace the heart. Aneurysms of the arch of the aorta may again lower the heart, while mediastinal growths may push it aside according to the direction of their development, and they are often associated with pleural effusion.

When fluid accumulates in the pericardium the walls of the sac yield probably to some extent, and it would seem that it is the upper portion of the sac which yields first: at any rate, the fluid distends the sac upwards from the first (*vide* Percussion, p. 93). Whatever be the explanation of its occurrence, apparent elevation of the apex-beat is often observed in pericardial effusion, but it is difficult to identify the abnormal impulse as the apex-beat. Effusion into one or other pleura displaces the heart to the right or left, but the displacement is greater in the case of left effusion.

C.

1. *Displacement of the Apex-beat.* Enlargement of the left ventricle displaces the apex-beat downwards and to the left, and increases its area. Enlargement of the right ventricle eventually tends to displace it to the left, but in this case the true apex-beat is apt to disappear, a more or less diffused undulatory movement between the sternum and the apex, accompanied by epigastric movement

indicating the cardiac action, which movement no doubt represents the action of the right ventricle. In health even, if the breath be held, the apex-beat will disappear, epigastric pulsation becoming developed. This results from engorgement of the cavity of the right ventricle. In dilatation of the heart in excess of hypertrophy, the true apex-beat may be replaced by a diffused impulse to the left of the sternum, while the downward displacement of the impulse is less than in cases in which the left ventricle, although enlarged, retains its shape. In such cases the possibility of mistaking the movement of the right ventricle for that of the left must be borne in mind.

2. *Epigastric Impulse or Impulse of the Right Ventricle.* Epigastric pulsation, which, we have seen, may occur physiologically, is a common phenomenon in all diseases in which the pulmonary circulation is embarrassed. In such cases the liver is frequently enlarged from passive congestion, tending to render such pulsation more visible; while in emphysema the lowered diaphragm may be a further factor in its production. Functional palpitation of the abdominal aorta, the pulsation of an abdominal aneurysm, and that of the liver itself (p. 153), can hardly be confounded with the epigastric pulsation under consideration, and need only be mentioned here.

The question of the rhythm of the epigastric impulse due to heart disease is reserved for discussion under palpation (*vide* p. 82).

3. *Impulse in second left intercostal space due to an enlarged infundibulum.* A pulsation above the third left costal cartilage may be seen, especially in cases of the cardiac muscle failure of anæmia and in long-standing cases of mitral stenosis; it represents the movement of the dilated infundibulum (conus arteriosus) of the *right* ventricle.

4. *Pulsation of Dilated Right Auricle.* Pulsation to the right of the sternum, auricular in origin, is rare. It occurs only in cases of extreme enlargement of the right side of the heart. The right auricle, which in the majority of cases alone passes to the right of the sternum, usually suffers dilatation in excess of hypertrophy, and in cases in which it is shown by percussion to be much enlarged and even to have pushed aside the superimposed lung, no pulsation may be visible. When the right auricle has become so much enlarged that its movements become visible a profound degree of asystole has usually been established, so that the chamber is practically a passive reservoir. Such being the case, any impulse seen is presumably systolic and due to a backward wave from the right ventricle.

5. *General and forcible pulsation over the cardiac surface, which pulsation does not correspond to the impulse of the ventricles nor to the situation of the auricles,* occurs in cases of intra-thoracic tumour—aneurysm or neoplasm—in the posterior mediastinum, in which the heart is pushed forwards from behind. The cardiac impulse is observed in an area almost co-extensive with that of the whole organ, and the usual impulses of the right and left ventricles escape recognition in the diffuse general impulse. The heart during its diastole accommodates its shape to surounding parts, but during systole it asserts the circular outline presented by its ventricles in a state of contraction, hence the forward impulse when the posterior mediastinum is encroached upon by tumour.

D.

1. *Aneurysms of the arch of the aorta* often occasion visible pulsation only, or pulsating tumour above the third rib, to the right or left of the sternum or in the middle line, this bone having been corroded by pressure. Great care should be taken to have the patient in a good light and to observe the chest from different points of view, especially looking *along the surface* of the suspected area. "Tracheal tugging" can be seen

in some cases (*vide* p. 193 Palpation) of aneurysm. In certain deformed chests, even though the deformity be not extreme, an abnormal position of the arch of the aorta to the right of the sternum may closely simulate an aneurysm by the visible pulsation it occasions. In contraction or collapse of the left lung, a deceptive appearance of aneurysm may also be given, from exposure of the pulmonary artery and infundibulum, frequently in such case dilated. Again, a simply dilated aorta may occasion visible pulsation in the second right intercostal space. Aneurysm of the descending aorta may occasion visible pulsation at the back.

2. *Venous Pulsation at Root of Neck.* This is an important feature of cardiac disease. It is almost necessarily accompanied by abnormal fulness of the veins, the position of the valves being indicated when they remain at all competent. The veins of the right side are usually the more, it may be only, affected. It must be remembered that respiration exerts a powerful influence on the return of blood to the thorax. Thus, during an expiratory effort, we see the veins of the neck swell up; while, during a deep inspiration, the circulation in them is accelerated, and they collapse. In cases of adherent pericardium, sudden collapse of the veins during the cardiac diastole has been described,

and has been supposed to be due to stretching of the intra-thoracic veins by the heart adherent to the diaphragm and chest-wall, during its systole and subsequent recoil during the diastole.* In chronic mediastinitis the cervical veins, instead of collapsing during inspiration, may swell up. Venous pulsation is auricular-systolic or ventricular-systolic, and often both, so that an undulatory movement of the vein is visible. The whole subject of venous movement will be considered later with the aid of instrumental observation. Distension of the veins, if habitual, as in cases of chronic cardiac disease, must render the valves of these vessels incompetent. Much care is necessary on the part of the beginner to distinguish venous from arterial pulsation. *Light* pressure at the *root* of the neck at once obliterates a venous pulsation. Moreover, by pressing a finger of the other hand upwards and emptying the vein from below, on removing the finger from the root of the neck, the blood will be seen to rush up according to the degree of incompetence of the valves of the veins and tricuspid orifice.

3. *Abnormal visibleness of arterial pulsation*

* The phlebograph will no doubt throw much light on such conditions, and the statements made probably require modification.

accompanies one cardiac valvular lesion as a peculiar feature. This valvular lesion is aortic regurgitation. Mere exaggerated visibleness hardly adequately expresses the peculiar jerk which characterises the pulse in the affection named. As this subject, however, will again require careful attention later, it may be briefly dealt with here, *vide* p. 162. In all cases in which the left ventricle is enlarged, carotid pulsation tends to become unduly visible, and especially when there is an abnormally great variation between the maximum and the minimum blood pressures within the vessel. So also in cases without cardiac enlargement, whenever the circulation is excited temporarily by emotions, etc., or permanently, as in Graves' disease. Morbid changes (degenerative) in the arteries themselves likewise may simulate in some respects the pulse of aortic reflux; but with the precaution to fix our attention on the larger trunks, the sign under consideration is of great diagnostic value.

4. Another phenomenon, which, when well marked, is almost characteristic of aortic reflux, is the so-called *capillary pulsation*. By rubbing the finger nail over the skin, say of the forehead, a red mark is produced, and after a few seconds, if this be carefully watched (it is often most evident some little distance off), a distinct wave of deepen-

ing and paling in colour is observable over the surface of the artificial erythema.

5. Irregularity of arterial and venous pulsations will be considered later.

E.

1. *Systolic Depression of the Intercostal Spaces in the Præcordial Region.* This is not uncommon in the fourth space above the apex-beat, whenever the heart is enlarged, or at all events comes into more extensive contact with the chest-wall. There is in such cases still an apex-beat, and while the apex protrudes, the portion of ventricle above it is depressed. It may be noticed over a normal but unusually exposed heart.

2. *Systolic Depression of the Ribs, Sternum, and Space between Xiphoid Cartilage and Ribs, accompanied by absence of the Apex-Beat.* This indicates universal pericardial adhesion, the præcordial pleural layers being likewise united, but it is a *very rare* phenomenon. Most cases of simple pericardial adhesion cannot be recognised by physical signs, but this is less to be regretted, inasmuch as dilatation has been often attributed to this lesion, when it really depended on coincident muscle-failure from myocarditis (*vide* p. 56).

PALPATION.

Signs ascertainable by palpation:—

A.—*Cardiac Impulse.*

1. Impulse, as it were, of whole heart thrust forwards in systole in cases of tumour or aneurysm situated behind and between the heart and spine.

2. Situation, extent, and strength of the apex-beat.

3. Epigastric impulse.

4. Closure of pulmonary semilunar valves with undue force perceptible to hand.

5. Pulsation above third left cartilage.

6. "Diastolic backstroke."

7. Double diastolic impulse at the apex accompanying the bruit-de-galop (*vide* p. 98).

B.—*Vibrations perceived by means of the hand applied over Cardiac Region.*

1. Thrills—presystolic, systolic, diastolic.

2. Friction fremitus.

C.—*Pulsations, thrills, etc., perceived by means of the hand placed over areas other than that of the heart.*

1. Aneurysms.
2. Dilatation of aorta, or normal aorta unduly in contact with chest-wall, owing to deformity of spine and framework of the chest.

In the use of palpation as a method of cardiac examination, it is important that the whole hand should be laid flat upon the surface, and this is especially necessary when the apex-beat has to be sought for.

A.

1. Impulse, as it were of the whole heart thrust forwards in systole, in cases of tumour or aneurysm situated behind the heart in the posterior mediastinum is the phenomenon already referred to under Inspection. The forcible, often "heaving," character of the pulsation is appreciated by palpation. The pulsation of an aortic aneurysm is heaving, but such pulsation is not præcordial.

2. *The apex-beat,* or *impulse of the left ventricle,* is a subject well worthy of some little

consideration physiologically. If a vertical section of the left ventricle is made, the thinness of its wall at the apex is at once noticeable, and it may be almost wondered that this portion of the ventricle does not yield and become bulged after the manner of a "cardiac aneurysm" (p. 177). Physiologically it is this portion of the left ventricle which impinges against the 5th intercostal space, and which hardens and gives to the hand of the observer the impact he calls the "apex-beat." During the emptying of the left ventricle its apex is, as it were, held up against the intercostal space. That this is so, is explained to a large extent by the lengthening of the aorta that occurs as the ventricle contracts and forces its contents into the vessel. The student should take pains to familiarise himself, by frequent practice, with the "apex-beat" in health and disease. By practice he will soon learn that this impulse is *sui generis*, and different from all other impulses the heart may afford. The so-called impulse of the right ventricle is of an entirely different nature from the "apex-beat" of the left ventricle. (*Vide* p. 82.)

If the apex-beat be invisible, its position must be ascertained, if possible, by palpation. The normal situation of the apex-beat has already been defined, and some of the changes it undergoes in

disease indicated. When inspection and palpation alike fail to reveal the presence of an apex-beat in the recumbent posture, the patient should be asked to sit up, inclining forwards (*not inclining to the left side*, if we would determine the *position* of the apex). Absence of the apex-beat may become an item in the data for diagnosis, due allowance being made for unusually thick chest-walls, emphysema, the apex beating under a rib, etc., and time spent on the investigation is not lost, though the object of our search be not found.

When the left ventricle undergoes enlargement, the apex-beat is necessarily altered in situation, and it becomes more extensive. The presence of a true well-defined apex-beat implies the retention by the ventricle of its normal pointed form in greater or less degree. When the chamber becomes dilated and rounded, the true apex-beat is apt to be lost, and only a diffuse impulse remains to represent the systole of the heart, or there may be no impulse perceptible, in spite of the ventricle being enlarged. The vigour of the heart-muscle, the degree of effort with which the left ventricle accomplishes its contraction, and the distension of the chamber at the outset of systole are elements that exert a great influence on the production of the apex-beat. In severe fever, for instance, the apex-beat may be watched failing day by day

until it is entirely lost. In Bright's disease, with cardiac hypertrophy well maintained, the apex-beat is slow and heaving, while in palpitation of nervous origin the apex-beat is quick and "slapping." In the case of extra or premature systoles (p. 134) the impulse may be well marked because the contents of the aorta are at high pressure, though the filling of the ventricle is incomplete when systole occurs. Certain valve lesions are accompanied commonly by peculiarities of the apex-beat. Thus aortic incompetence is generally accompanied by a vigorous apex-beat so long as the heart-muscle remains vigorous. The apex-beat may fail or be ill-marked in great aortic obstruction, although the left ventricle is hypertrophied and its muscle sound. Many cases of mitral stenosis are accompanied by a well-marked apex-beat, which corresponds with the sharp abrupt first sound, that is characteristic of the lesion. In this case it may be supposed that the ventricle begins to contract before it has been distended to the full amount—before, in fact, the current from the auricle has ceased.

The strength and definition of the apex-beat are matters of much importance, both in cardiac and general diseases. Among the former fatty metamorphosis and the compensatory hypertrophy of aortic valvular disease offer good examples. The

weakening of the heart in typhus, so admirably described by Stokes, and the hypertrophy consequent on Bright's disease, furnish illustrations in essentially non-cardiac diseases. In both these cases it is the left ventricle that is chiefly affected, and consequently it is the apex-beat that undergoes alteration. Pericardial effusion usually diminishes the force of the cardiac impulse—apex-beat, or otherwise—but the concomitant weakening of the heart-muscle must be borne in mind, and the result not attributed entirely to the mere presence of fluid.

3. The impulse of the right ventricle is altogether different from the apex-beat, and is delivered by the broadside of the chamber. Moreover, it is *diastolic* in rhythm and coincides with the filling of the chamber (*vide* pp. 2 & 147). Epigastric impulse is always diffuse and ill-defined. The apex-beat and the epigastric impulse generally vary inversely—when the one is pronounced the other is defective or absent. Thus, when there is much enlargement of the right side of the heart, the left ventricle is pushed back, as it were, by the enlarged chamber, and in long-standing cases the right ventricle may reach the apex of the heart. In certain cases of aortic regurgitation, the left ventricle is able for long to cope with the difficulty entailed by the lesion, so

that the embarrassment of the circulation does not pass backwards through the lungs to the right side of the heart. Such cases commonly have a much exaggerated and displaced apex-beat.

It is seldom that both impulses—if such they can be called—are well marked at the same time. In severe fever (as typhus), we have seen that epigastric impulse is apt to be established, as the result of the obstruction in the pulmonary circulation, invariably more or less present under the circumstances. When the left ventricle becomes dilated and rounded, its systole is probably invariably imperfect—*systole catalectic* or *asystole*—and whether there be or be not mitral regurgitation, the right side of the heart becomes involved in the obstruction suffered by the circulation. Thus there are two causes at work in obliterating the apex-beat and establishing epigastric impulse. While the apex-beat is a normal phenomenon, epigastric impulse is generally, though by no means always, abnormal, for in a few healthy individuals there is always some epigastric pulsation present, as if from the sternum being unusually short.

4. *The Closure of the Pulmonary Semilunar Valves* is frequently perceptible as a shock transmitted to the hand placed on the surface of the chest over the appropriate area (Fig. 3), in cases in which

there is obstruction to the pulmonary circulation from any cause. It will be rendered more distinct in proportion as the pulmonary artery is exposed by retraction of the lung. The pulmonary orifice, it will be remembered, is situated near the surface of the chest. A similar phenomenon in the case of the aorta may be felt over aneurysm or even dilatation of the vessel.

5. When the foregoing sign is present there will sometimes be seen a pulsating area in the second left intercostal space close to the sternal margin (p. 71), and such pulsation will be more or less perceptible by palpation. At one time this pulsation was attributed to the left auricle, but the latter chamber lies behind, and only its appendix reaches the front. The pulsation represents the movement of the infundibulum of the right ventricle.

6. *Diastolic Back-Stroke.* This is held to be a sign of adherent pericardium and pleura with cardiac enlargement. The writer's experience of this rare phenomenon would lead him to regard it as resulting from the rebound of the dilating heart after the systolic depression already noted under "Inspection" (p. 76), but his opportunity of observing the sign was a single one and occurred a long time ago.

7. When the Bruit-de-galop is audible a double shock corresponding to the 2nd and 3rd portions of the triple sound may be felt, diastolic in rhythm.

B.—*Vibrations perceived by means of the hand applied over the Cardiac Region.*

These naturally fall into two classes—according as the cause is endocardial or exocardial.

1. *Endocardial Thrills.* Thrills are produced at or rather beyond the cardiac orifices by the *veines fluides* which give rise to the corresponding murmurs. A thrill is most common when there is obstruction to the blood-current at a rigid and contracted orifice, as in aortic and mitral stenosis.

The aortic obstructive thrill is systolic, the mitral obstructive thrill is presystolic or diastolic in rhythm. In cases of dilatation of the aorta, a systolic thrill may be perceptible over the vessel, although there is no actual constriction of the orifice, but in this case a perceptible shock may be felt to accompany the closure of the aortic valves and second sound. The presystolic thrill of mitral constriction, which accompanies the systole of the auricle, is usually limited to the apex, and possesses the peculiar feature of increasing in intensity up to the apex-beat, which at once cuts it short. The diastolic thrill of the same lesion accompanies the expansion of the left ventricle, and diminishes in intensity towards its end. It also is an apex thrill. When they co-exist, as they often, do, these two thrills can be easily distinguished.

Sometimes mitral regurgitation is accompanied by systolic thrill at the apex, but this is comparatively rare, and occurs generally in cases of mitral stenosis. Aortic regurgitation is very rarely attended by diastolic thrill felt at the apex of the heart, to which it may be limited. The incompetence of the valves is usually considerable in cases of the kind.

2. *Friction Fremitus*, due to exudation on the pericardial surfaces, need be here only mentioned, as the subject of friction is fully considered in the section on Auscultation. This sign usually gives a most distinct impression of the mode of its production: by the rubbing of two surfaces upon one another.

Emphysematous bullæ on the edge of the left lung possibly account for peculiar thrills that assume cardiac rhythm.

C.—*Pulsations, thrills, etc., perceived by means of the hand applied over Pulsating Areas other than that of the Heart, viz., Aneurysms and Dilated Aorta.*

1. When an impulse, other than that of the heart, yet synchronous with its systole, seems to be at least as forcible as the cardiac pulsation, very reliable evidence is afforded of the presence of an aneurysm. Murmurs audible over

aneurysms are sometimes represented in palpation by thrills. Again, as already indicated, when the semilunar valves are closed with abnormal force, as is always the case in aortic aneurysm when these remain competent and the first part of the arch is involved, a distinct "thud" may be felt by the hand placed over the tumour. Aneurysm of the descending aorta may occasion pulsation behind.

2. When the aorta is dilated and in abnormally extensive contact with the chest-wall from any cause, a thrill may be perceptible over the area of contact. Such thrills accompany murmurs, and their causes will be considered under "Auscultation." The rare phenomenon of "pulsating empyema" does not belong to the subject of this book, so that mere mention of it must suffice.

PERCUSSION.

The heart being surrounded in front, except inferiorly, by air-containing lung, we are enabled to estimate its size or an amount of pericardial effusion, by means of percussion, inasmuch as the heart or the pericardium distended with fluid yields no resonance over that portion of the chest-wall with which it is in contact, and modifies the resonance of the lung beyond that area, by render-

ing the layer of resonant lung-tissue, superficial to the heart, or distended pericardium, less thick.

Percussion thus determines for us, with more or less accuracy, the size of the heart or pericardial sac distended with fluid, also the area in which aneurysmal tumours come into contact with the chest-wall. In health the large vessels cannot be recognised by percussion. If they could, they would form a more or less circular dull area, super-imposed upon the area of cardiac dulness proper, *i.e.*, above the level of the third rib.

Percussion is employed to distinguish the size of the heart or distended pericardium according to two different methods. One aims at determining the so-called "superficial dulness." *This really means mapping out the area in which the heart comes into contact with the chest wall.* The anterior border of the left lung separates from the anterior border of its fellow at the fourth rib, and passes outwards and downwards to about the union of the fifth rib with its cartilage; it then extends downwards and inwards more or less along the sixth left costal cartilage, thus leaving a rudely triangular space in which the heart (chiefly the right ventricle) comes into contact with the thoracic wall. If this space formed a regular triangle, the superior or external side would measure about three inches, the internal side corresponding to the

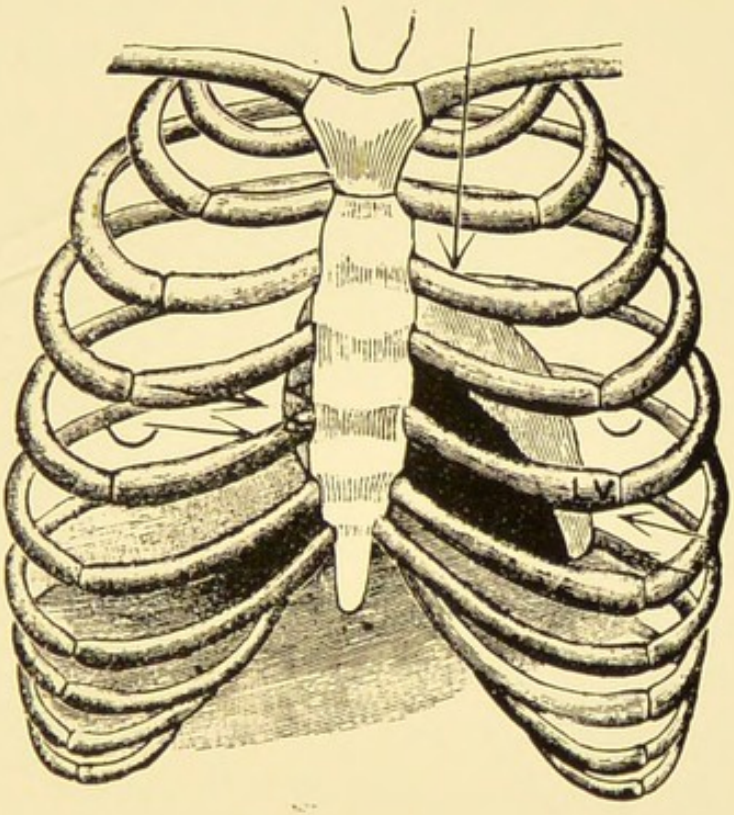

Fig. 5.

middle line, two inches, and the inferior two and a half inches, but it is only approximately of triangular form, and by some authors has even been described as circular. Except at the right side, where *sternal* resonance modifies the percussion note, this dulness is easily defined by light percussion. The peculiar resonance of the sternum interferes to some extent with our determination of the right border of cardiac dulness, unless the right auricle be engorged, and the dulness extend to the right of the bone.* The information acquired by this mode of percussing the heart, though alone relied upon by many physicians, is open to the objection that it does not tell us the absolute size of the heart, but indicates only the size of the cardiac surface in contact with the chest-wall.

The other method of estimating the size of the heart by percussion depends upon the modification which the organ produces on the resonance of the lung overlying it: it determines approximately the actual size of the heart. In performing percussion according to this method, we have, as it were, to percuss *through* a certain depth of lung-tissue. A few experiments in the *post-mortem* room will make abundantly plain the fact, that a thin layer of lung tissue gives a "less full" or

* Anatomically the right auricle extends about half-an-inch to the right of the sternum, but such extension is too deep to be detected by percussion.

"more empty" note than a thick layer. In percussing from well beyond the cardiac region towards the triangular area of superficial cardiac dulness, the gradual transition from the "full" pulmonary note elicited beyond the heart, to the complete "emptiness" or "dulness" of the percussion note in the triangle referred to, is readily appreciated. Until the heart comes into immediate contact with the chest-wall, the lung will yield its clear and resonant note, though one becoming more and more "empty." By practice, the power of determining sufficiently for all clinical purposes the line at which the heart begins to modify the pulmonary note is soon acquired.

In cases of "large-lunged emphysema," thick masses of emphysematous lung-tissue spread over the heart, so as to render this method of percussion useless. But, in this case, the other or "superficial method" of percussion is of no greater value, inasmuch as the voluminous left lung encroaches upon the triangle.

The determination of the size of the heart or of a distended pericardium by percussion, may obviously be interfered with by pleuritic effusion, or consolidation of the neighbouring portions of lung, but these conditions usually present no difficulty of recognition, as characteristic pulmonary physical signs are pronounced. Truly dishearten-

ing, however, to the beginner, are the results of what Dr. Walshe well called "horizontal conduction." For instance, in a case of cardiac disease with enlargement of the organ, the heart is seen pulsating over a wide area, yet over the very pulsating area a tympanitic note is elicited on percussion—a distended stomach lying beyond, being the cause of the anomaly.

For the purposes of practical diagnosis, the essential points to be ascertained by percussion are the following:—(1) The size of the right auricle; (2) the size of the left ventricle; and (3) the presence or absence of pericardial effusion or dilatation of the conus arteriosus. This may be done by a few very simple manipulations.

Percuss downwards an inch, or more if necessary, (to get beyond the right auricle) to the right of the sternum until the liver dulness (the lung-margin) is reached. Mark the spot with a dermograph pencil. Then percuss towards the sternum about an inch above the level marked, and note the spot at which the modification (if any) of the lung sound, due to the auricle beneath, becomes appreciable. When absolute dulness to the right of the sternum is detected in this way, it signifies so great a degree of distension of the auricle, that the lung-margin has been displaced. Such distension probably implies imperfect systole

—*systole catalectic*—on the part of the auricle. It is a good habit to compare the percussion sound to the right of the sternum above and below the third cartilage, when any modification of the lung-sound has been noticed below the latter level, although the approximation of the lower cartilages below may contribute to the result.

We next turn our attention to the left of the sternum and endeavour to determine the size of the left ventricle. Inspection and palpation having preceded percussion, the situation of the apex-beat will probably be known. If so, the spot should be marked and the intercostal space in which it is situated noted. This spot will correspond pretty closely to the level of liver dulness already marked on the right side, or it may be a little lower. Let percussion next be made from the left mid-axillary line towards the heart, choosing the level of the apex-beat if that has been determined by inspection or palpation, or if not, the level corresponding to about an inch above the liver dulness to the right of the sternum. Whether the situation of the apex-beat has or has not been revealed by inspection or palpation, the cardiac dulness, reached at the level indicated, will represent the apex of the heart closely enough for all practical purposes, for the apex is the part of the heart that extends most to the left. Outwards (*i.e.*, to the left) and down-

wards are the directions in which the apex of the heart is displaced, when the left ventricle is enlarged, and it fortunately happens that when the downward displacement is most pronounced, the apex-beat is usually easily discerned — the ventricle retaining more or less its normal form instead of becoming rounded—so that any error that might arise from our getting *above* the apex is prevented. It is to be remembered in relation to this method of determining the size of the left ventricle, that approaching the apex of the heart we very soon reach the area of "superficial" dulness.

The upper boundary of the heart-dulness is determined by percussion downwards, an inch to the left of the sternum. Normally, as already stated, there should be no modification of lung-resonance due to the heart above the third left cartilage. Extension of dulness upwards indicates effusion into the pericardium or enlargement of the conus arteriosus (infundibulum) of the *right* ventricle, this part of the chamber coming to the *left* of the sternum.

The results of percussion of the heart in a given case according to this method may be shortly noted in the following way:—The left cartilage to which the cardiac *dulness* (in the sense of lung resonance modified by the heart) reaches, is

recorded in Roman numerals III. or II., as the case may be, while below the extension of dulness to the right and left respectively from the middle line is noted in inches in Arabic numerals placed underneath as one would write the results on the patient's chest. Thus $\overset{\text{III}}{3\text{—}7}$ was noted in a case of aortic regurgitation, in the last stage of the disease, and implied enormous enlargement of the left ventricle and great distension of the right auricle, with absence of pericardial effusion, which would have been indicated by extension upwards above the 3rd cartilage. In aortic disease, enlargement of the right side of the heart may be long delayed. The figures $\overset{\text{III}}{1\frac{1}{2}\text{—}4}$ were noted in a case of mitral stenosis and indicated some distension of the right auricle, slight enlargement of the left ventricle, and absence of pericardial effusion. In a case of the same lesion the following results—$\overset{\text{II}}{4\text{—}5}$ indicated great enlargement of the right auricle and of the infundibulum (conus arteriosus) and moderate enlargement of the left ventricle. In a case of pericardial effusion such a dull area as $\overset{\text{II}}{4\text{—}7}$ may be obtained.

When it is remembered that the heart is a contractile organ, constantly undergoing changes in size and shape, the utility of attempting very accurate measurements of it by percussion may

well be doubted. On the other hand, however, it is certain that in most cases an approximate estimation of the size of the heart can be made by means of it. The size of the right auricle enables us to gauge the obstruction in the pulmonary circulation and more or less in the general circulation. When there is enlargement of the right auricle, confirmatory evidence of such obstruction will not usually be wanting—dyspnœa, enlarged tender liver, dropsy, etc. Enlargement of the left ventricle depending, for the most part, upon dilatation of its cavity, is an unmixed evil, unlike hypertrophy of its walls.

In certain chronic cases of cardiac dilatation the area of dulness produced closely simulates that due to pericardial effusion, extension of dulness upwards resulting from dilatation of the infundibulum, extension of the right lower corner from dilatation of the right auricle, and of the left lower corner from dilatation of the left ventricle. When there is no impulse to be felt, the distinction between the two conditions—pericardial effusion and general dilatation of the heart—may be difficult.

AUSCULTATION.

Modifications of the Physiological Heart Sounds. Modifications of the physiological sounds of the

heart in all cases of heart-disease should receive careful attention before the adventitious sounds, that may be present, are even considered. Such modifications may be the only evidence of disease yielded by auscultation. *Adventitious sound or murmur may be entirely absent, throughout a prolonged observation of a heart-case*—one either of mitral stenosis or of simple muscle-failure. Moreover, now and again, even definite modifications of the physiological sounds may be absent in cases of the kind, and the heart sounds cannot be pronounced other than normal.* While it is necessary that the student should know this, he must also know that such cases are altogether exceptional. In hypertrophy of the left ventricle, such as occurs in chronic Bright's disease, when the ventricle is contracting, so to speak, on a heavy burden—with effort—the first sound is often toneless and almost inaudible, while there is no murmur. On the other hand, when the burden of the left ventricle is light—that is the arterial tension low—and contractile vigour fairly preserved, the first sound is apt to be shortened, though its tone is rather enhanced and it comes to resemble the second sound. Such a condition of the heart sounds is met with in fever, and it gives to the sounds some resemblance to the fœtal heart sounds, the first and

* Exception may be taken to this statement as regards the 2nd *S.* in the pulmonary area, but accentuation of this sound may be accounted for by lung conditions.

second sounds being almost indistinguishable. In severe typhus, and rarely in enteric fever, the first sound may cease altogether. As an extreme rarity the second sound may also cease under the circumstances, and the patient might be thought to be dead but for his respiration and the flicker of some artery. A peculiar condition of the heart sounds has been described, in which the first sound is short and almost immediately followed by the second sound: this probably indicates an ominous degree of cardiac muscle-failure. In excited action of the heart, the arterial tension being usually low from vaso-motor influence, the first sound is often loud, and so-called "accentuation" of this sound is a valuable sign of mitral stenosis — its value being the greater the more quietly the heart is acting at the time. *Intensification of the second sound—so-called "accentuation—over the pulmonary valves is commonly present, more or less, in all heart cases.* In dilatation of the arch of the aorta the same condition of the sound is met with in the aortic region.

Reduplication is an important modification of the heart sounds. In the early stages of Bright's disease, while the arterial tension is high and the heart muscle fairly vigorous, reduplication of the first sound is common. The reduplication may be represented phonetically by the syllables "*turrup*" in place of the syllable "lub" or "lup" in use to

represent the normal sound: the reduplicated first sound followed by the second sound may thus be represented by the syllables "turrup-dup" or "turrub-dup." As might be expected, it is at the cardiac apex that this reduplication is best heard. In the later stages of Bright's disease, however, when there is manifest cardiac muscle-failure, reduplication of the second sound is often met with at the apex and over the ventricles. This is the *bruit-de-galop* or galloping sound, which is often accompanied by the palpation sign of a double diastolic shock at the apex (p. 84).

In mitral stenosis, reduplication of the second sound to the left of the sternum below the third cartilage, and in some cases over the ventricles to the apex, is very common, so as to form a valuable minor sign of the disease. An accentuated* first sound, followed by a reduplicated second sound, forms a combination, that, without any murmur, is exceedingly suggestive of the lesion. Besides becoming reduplicated, the second sound in mitral stenosis offers another sign—a negative and minor one—of this lesion, by ceasing to be audible in the mitral area. Thus it happens that either reduplication of the second sound to the left of the sternum, or absence of the second sound from the apex, constitutes evidence in favour of the

* During palpitation, the first sound may actually become audible at some distance from the patient.

existence of mitral stenosis. The first sound followed by a reduplicated second sound may be represented phonetically by the syllables "lub- or lup-ta-ta."

Murmurs:—Unlike the sounds, modifications of which have just been considered, cardiac murmurs have no existence in health: they are prolonged more or less "blowing" sounds bearing no resemblance to the physiological sounds of the heart, which they may accompany or replace. There is one murmur to which the term "blowing" ill applies: it is the auricular-systolic or presystolic murmur, which is further distinguished by being *crescendo* in character and by being brought to an abrupt termination, while at its maximum, by the first sound. The quality of murmurs varies much; some are musical or "noisy" and may rarely be heard at some distance from the patient's body. The quality and loudness of murmurs, in our present state of knowledge, are not sufficiently understood to render their consideration here profitable. Musical and "noisy" murmurs are apt to receive from the student an amount of attention to which they are hardly entitled when regarded from the practical standpoint. An instance of the fact may be given. A patient was admitted to the wards extremely cyanotic and suffering from great dyspnœa. His heart was carefully auscultated, but no trace of murmur of any kind

could be heard. A few days later there was heard at the apex a systolic murmur of musical quality and of such loudness that it could be heard several inches away from the patient's body. When the patient died a few weeks later, the lesion mitral stenosis was found, evidently of long standing, the valves showing no trace of recent change.

When a murmur is heard over the heart:—

(1) Its *rhythm* must be determined. Every murmur coincides with the movement—contraction or expansion—of a chamber of the heart. Thus cardiac murmurs are either ventricular systolic, or ventricular diastolic, or auricular systolic.* When a physiological sound of the heart is present with a murmur the fact should always be noted, and the presence of the sound helps in the determination of the rhythm of the murmur. It must be observed that the murmur, if systolic or diastolic, outlasts the "sound," showing that the latter occupies a portion only of the whole time of systole or diastole. Many mitral systolic murmurs run off from the first sound of the heart, while the presystolic or auricular-systolic murmur runs up to and ends abruptly and at its maximum in the first sound, which is peculiarly modified under the circumstances, as already

* Exception may be taken to this statement concerning certain rare and generally congenital murmurs.

described. The diastolic murmur of aortic incompetence, again, may run off from the second sound, which in cases of aortic dilatation is modified in a somewhat similar way, so that the same term is used to describe it: it is said to be *accentuated.*

(2) The exact spot at which the murmur is heard loudest must be determined in the case of each murmur—its *place of maximum intensity.* It does not follow that this must correspond to one of the four areas that are associated for auscultatory purposes with the four orifices of the heart and their valves (*vide* p. 5).

(3) Lastly, the *direction in which the murmur is conducted* over the surface of the chest must be determined.

In practice the three points are considered together, and it is difficult to isolate each entirely, even in description.

(1) Rhythm of Murmur.

At the base of the heart, over the orifices of the great arteries, murmurs may be ventricular-systolic or -diastolic. Pulmonary diastolic murmurs, over and below the pulmonary area, are rare (p. 250). At the apex, and more or less over the ventricles,

murmurs may be ventricular-systolic or -diastolic or auricular-systolic—so-called presystolic—and the last murmur is usually closely limited to the apex.

During systole the blood is driven from the ventricles into the aorta and pulmonary artery so as to distend these vessels. A systolic murmur over them suggests interference with the current through the orifice and the current spreading out beyond a narrowing of the channel at the orifice. Care has to be taken in the use of the word *obstruction*, often employed with reference to the cause of such murmurs, inasmuch as the so-called obstruction may be only *relative* to the channel beyond. Thus a loud systolic murmur is often heard over a simply dilated aorta, the orifice of which is actually *larger* than that of a normal aorta. The fibrous ring to which the aortic valves are attached yields much less readily than the walls of the vessel beyond it, and so it happens that the stream of blood spreads out as soon as the orifice is passed, and this is the physical condition by which murmurs are commonly produced, so-called *veines fluides* being formed. It is evident that a similar condition will be established if, instead of the channel of the vessel being increased in size, the orifice be diminished. In this latter case *actual obstruction* exists. But a little thickening and stiffening of a cusp, so as to prevent its normal

effacement before the current, or a minute nodule on its margin acts in a similar way as regards the production of murmur, although the obstruction is nominal only. Moreover, in anæmia the blood-change seems to render the current specially productive of murmur.

It follows from the foregoing considerations that the inference to be drawn from the mere presence of a systolic murmur over the aorta is very indefinite. Its significance has indeed to be determined by concomitant circumstances. For instance, a much accentuated second sound following the murmur points to dilatation of the aorta, while the fact of a prolonged harsh murmur, accompanied by a systolic thrill, being present without there being any second sound, more than suggests actual aortic obstruction or stenosis, and the pulse will probably bear out this diagnosis (*vide* p. 161).

Although narrowing (stenosis) of the pulmonary orifice is one of the most common congenital lesions, it is but seldom met with. It is indicated by a loud, harsh systolic murmur in the appropriate area, often accompanied by thrill and less commonly by diastolic murmur. Rare as is actual stenosis of the pulmonary orifice, a systolic murmur in the pulmonary area, on the other hand, is one of the most common of murmurs, and, more-

over, it is the murmur of least significance. Its mode of origin has been much debated. It constitutes the murmur of anæmia *par excellence*, but it is common in most varieties of cardiac muscle-failure. It is common, too, in cases of mitral stenosis (*vide* p. 122). In cases of this last kind the pulmonary artery has often been long exposed to high blood-pressure and tension, and it is a remarkable fact that under such circumstances it becomes subject to an atheromatous process like that of the aorta, when similarly exposed to strain. In anæmia there would seem to be a difficulty in the passage of blood through the lungs—at any rate, it is the right side of the heart that suffers in the first instance. In the latter two cases—mitral stenosis and anæmia—it is possible that the explanation of the systolic murmur in the pulmonary area is that a degree of dilatation is undergone by the channel of the pulmonary artery—in other words, a similar mechanism to that considered with reference to the corresponding aortic murmur. Accentuation of the second sound in the pulmonary area will naturally be present under the circumstances. A change in the blood may still perhaps be allowed a place in the production of murmur. The veins, for instance, certainly become specially soniferous in anæmia. A systolic murmur in the pulmonary area is not

rarely found in persons enjoying good health, when the explanation of its production is not easy. Occasionally some deformity of the chest may suggest an abnormal bend or pressure upon the vessel in some part of its course (p. 73). Too much importance must not be attached to murmurs of the kind, and the general rule must be borne in mind that *a systolic murmur in the pulmonary area is, of all murmurs, of least diagnostic and prognostic value.*

The great majority of basic *diastolic* murmurs are aortic. The presence of the second sound should always be noted, as well as any special character presented by it, such as *accentuation*, which suggests aortic dilatation as the mechanism of the incompetence. A diastolic murmur in the pulmonary artery may, as already noted, accompany congenital stenosis, being due to coincident deformity and incompetence of the valves. Such a murmur is not a very rare occurrence, however, in cases of long-standing mitral stenosis. When first established it usually comes and goes, according to the degree of blood-pressure within the vessel. It is accompanied by dilatation of the pulmonary artery, which, as already stated, under the circumstances may become atheromatous like the aorta. This murmur has been called "the murmur of high pressure in the pulmonary

artery." Septic endocarditis may attack the pulmonary valves.

Coming now to the consideration of the rhythm of murmurs heard loudest at the apex of the heart, and presumably produced at or just beyond the mitral orifice, it is to be noted that besides ventricular systolic and diastolic murmurs such as exist at the base, a murmur may be heard during the systole of the auricle—a murmur which differs from all other murmurs by being *crescendo* and by coming to an abrupt termination with the first sound and when at its maximum intensity.

When a systolic murmur is heard, and has its maximum intensity in the mitral area, we presume that the mitral valves have become incompetent and permit a backward current to take place between them. Having arrived at this conclusion, it is necessary for the observer to ask himself these three questions concerning the state of the mitral orifice and valves :—

(a) *Are the valves sound in structure and incompetent only because they do not receive the support from the contraction of the muscle of the heart that is necessary for the fulfilment of their function?*

(b) *Is there stenosis of the orifice, and is the*

regurgitation taking place through the necessarily deformed valves of a narrowed orifice?

(c) *Is there structural damage of the valve-curtains without stenosis of the orifice?*

With regard to this last question, a difficulty is at once presented by the fact that *it is impossible to separate the effects of structural damage of the valves in the production of their incompetence from those of coincident muscle-failure.* In other words, granted structural damage to the valves, it cannot be affirmed that structural damage is the *sole* cause of their incompetence. The valves may be damaged—structurally changed,—but the essential cause, nevertheless, of their incompetence may be muscle-failure. There are no means at our disposal by which the contributions of these respective factors to the result can be estimated. The valves may be transformed into little more than masses of vegetations in septic endocarditis, but it cannot be doubted that there is in such cases a profound debility of the heart-muscle. Again, in ordinary rheumatic cases in youth, the valve changes may be found to be quite trivial, while the heart is greatly dilated with or without adherent pericardium. In such cases, the change in the valves is altogether inadequate to explain the evidently free regurgitation that occurred during life and the disastrous dilatation of the heart. The *muscle-*

failure factor, it may be presumed, was the essential one. Between these extremes all sorts of blendings of the factors in question are met with.

(a) To return to cardiac muscle-failure as a cause of mitral incompetence, a common feature of such cases is the non-conduction of the murmur to the back. But so many exceptions are met with that the rule is hardly worth formulating. The absence of a history of rheumatism, and chorea, from the history of the patient, is an important consideration in arriving at the conclusion that the valves are sound, and that their incompetence is dependent on muscle-failure only. So much for negative evidence: but positive evidence of a cause of muscle-failure has to be found before the diagnosis can be considered in any sense established.

(b) Any of the minor physical signs of mitral stenosis—such as an accentuated first sound at the apex or a reduplicated second sound to the left of the sternum—helps in resolving this question, while the past history of acute rheumatism or chorea is of the greatest importance. A presystolic murmur is of course at once conclusive of the diagnosis, irrespective of the history. In cases of mitral stenosis, like those of muscle-failure, the murmur of incompetence is in the majority of cases not

conducted to the back, but is there replaced by a pure first sound, but again it must be pointed out that the rule is far from absolute (*vide* p. 122).

(c) Structural damage to the mitral curtains without stenosis of the orifice results from endocarditis, in most cases caused by an attack of rheumatism or chorea in comparatively early life, in a few from septic endocarditis at any age. Attention has been sufficiently called already to the impossibility of separating absolutely the incompetence due to the structural valve damage from that due to coincident muscle-failure of the heart. In the case of septic endocarditis the concomitant symptoms and the general circumstances of the case will usually indicate the nature of the pathological process (p. 294). The lesion of septic endocarditis is usually a much more destructive one than that of rheumatic endocarditis.

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(2) Situation of Maximum Intensity of Murmur.

"A superficial area of half-an-inch will include a portion of all four sets of valves *in situ;* an area of about a quarter-of-an-inch a portion of all except the tricuspid." This often-quoted state-

ment of Dr. Walshe explains the hopelessness of ascertaining the orifice at which a murmur is generated by its being loudest when the stethoscope overlies that particular orifice. Moreover, it is probable that murmurs are not loudest at the precise orifice, but rather beyond the orifice in the direction of the current producing the murmur. The principles on which certain sites are chosen for each orifice vary. What is technically called the "*aortic area*" is *the sternal end of the second right costal cartilage.* Here the arch of the aorta comes up most closely to the surface, and it may well be believed that systolic murmurs indicative of stenosis of the orifice will be readily conducted to this spot. But for murmurs of incompetence the "aortic area" is a much less satisfactory choice, and as a matter of actual clinical experience it is found that, were the absence of a diastolic murmur from this area to be taken as sufficient evidence of the integrity of the aortic valves, many grievous errors of diagnosis would result. For the *isolation* of aortic murmurs, however, the site is well chosen, for it is at some distance from the small area referred to by Dr. Walshe, and all systolic and most diastolic murmurs will be audible at "the sternal extremity of the 2nd right costal cartilage." The actual orifice of the aorta, however, lies deep in the heart, as

is evident from a glance at a transverse section of the heart (Fig. 4). It will be noticed, further, that the infundibulum crosses in front just below the aortic valves. During diastole it can be readily understood how well a murmur, generated by the current proceeding downwards from an aortic leakage, may attain the surface of the chest-wall at the left edge of the sternum below the level of the third cartilages. The sternum readily conducts murmurs that reach it, and between the third cartilages is a spot at which it is common to hear a "double aortic" murmur with great distinctness, while the diastolic portion is well transmitted down to the xiphoid cartilage in most cases. As will be mentioned later, conduction sometimes takes place specially to the left of the sternum and more or less towards the apex.

If the statement that murmurs as a rule tend to be conducted in the direction of the current of blood producing them be correct, the apex of the heart is a well-chosen spot for hearing murmurs due to obstruction of the mitral orifice—presystolic and diastolic—and experience bears out the fact that these murmurs are specially well heard at the apex. In the case of the former the site is even more appropriate than in that of the latter, for the presystolic murmur has a peculiarly limited area of audition, and it is heard

only over the apex in most cases, although the accentuated first sound, which forms almost an integral part of it, may be heard more or less over the heart, while the modified sound in question may be regarded as the last portion of the murmur, and so the idea, that the murmur itself is as widely conducted, is apt to be formed. Rarely the presystolic murmur itself is widely conducted over the heart, and exceptionally it is audible behind in the neighbourhood of the angle of the scapula. The diastolic murmur of mitral stenosis in some cases is sharply limited to the apex, but occasionally it is heard all over the cardiac surface, while reaching the sternum it may be heard over this bone. As there is usually a systolic murmur present as well as the diastolic, the to-and-fro murmur may be indistinguishable from the "double aortic" murmur of similar rhythm. An examination of the pulse and the state of circulation generally is, then, the best mode of distinguishing between the two lesions, and there is seldom difficulty experienced in the process.

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(3) Conduction of Murmurs.

As regards the conduction of murmurs of mitral origin, the presystolic murmur is peculiarly limited to the apex of the heart, and this notwithstanding

the loudness and harshness of the murmur. Occasionally, however, as already stated, the presystolic murmur is heard practically all over the heart, and rarely it is conducted to the back. The accentuation of the first sound, associated with the murmur, is often heard all over the heart, while the murmur is limited to the apex. The diastolic murmur is sometimes, like the presystolic, limited to the apex: more commonly it is audible over the ventricles, above the apex, and rarely it is heard all over the heart, so that when, as is usually the case, there is a systolic murmur at the same time, the to-and-fro combined murmurs resemble those of aortic incompetence, but, of course, the vascular phenomena of the latter lesion are absent, and the diastolic murmur has not usually the soft blowing quality so often possessed by the diastolic aortic murmur.

Though both the presystolic and diastolic mitral murmurs are due to stenosis the currents of blood producing them own respectively a different moving force. In the case of the presystolic murmur the contraction of the left auricle is the force immediately concerned in the production of the current of blood through the orifice. As the cavity of the left auricle diminishes in size it can be imagined that the force of the current gathers strength. Moreover, it may be imagined that the

emptying of the auricle is incomplete at the time the ventricle enters into contraction and abruptly cuts short alike the current from the auricle and the murmur produced by it, the termination of the murmur while at its maximum being marked by the occurrence of the peculiar first sound already so often referred to as characteristic of the lesion. The diastolic murmur of mitral stenosis, on the other hand, is to be associated with the current of blood through the narrowed orifice that is the product of a *vis a tergo* and a *vis a fronte* supplied respectively by the blood-pressure in the lungs, derived, of course, from the right ventricle, and by the diastole of the left ventricle. Unless it is accompanied by a presystolic murmur, which it often is, the first sound need not be accentuated in the case of the diastolic murmur; there is no direct association between the two phenomena as there is in the case of the presystolic murmur. It would seem that the presystolic murmur runs into the first sound because the contraction of the left ventricle abruptly and forcibly stops the current producing the murmur, and that the accentuated sound marks the point of time alike of the contraction of the left ventricle and the stoppage of the current from the auricle into the ventricle, for how can the weak-walled auricle contend for a moment against the massive-walled ventricle?

In another place there will be described recent observations on the condition of the wave of contraction from the venous ostia of the auricle along the wall of auricle and ventricle to the apex, which have shown a tendency to interruption of the wave between the two chambers at what has been called the "bridge" between them. In disease or functional disorder interruption here may become pronounced. Admitting all this, there can be no question that when a presystolic murmur is present it goes on without interruption till it is brought to a close *while at its maximum* by the first sound indicative of the contraction of the ventricle. The presystolic murmur is *crescendo* from first to last; it never shows any indication of slackening of the producing current or any trace of *diminuendo* character, as does the diastolic murmur of mitral stenosis. If the blood-current resulting in the diastolic murmur is produced in the manner stated, it is easy to understand why this murmur should be more or less *diminuendo*. On the other hand, it may be repeated, if the blood-current resulting in the presystolic murmur has as its driving force the contraction of the auricle, its *crescendo* character is explicable by the approximating contractile walls of the chamber gaining power over their contents as they approximate, while the sudden termination of the murmur

at its maximum is explained by the entrance of the ventricle into contraction, with the formation of the first sound of the heart and the immediate arrest of the current of blood from auricle to ventricle.

Aortic obstructive murmurs are conducted upwards in the course of the circulation, aortic-diastolic murmurs downwards and to the left, *i.e.*, towards the apex and over the ventricles.

If the principles on which the apex is chosen as the site of maximum intensity of mitral obstruction murmurs be correct, it may be objected that the same spot cannot be correctly chosen as the site of the maximum intensity of murmurs produced by a blood-current in the opposite direction, namely, backwards to the left auricle. Now this last-named chamber lies essentially far back, and it is only the tip of its appendix that reaches the front. This backward position of the left auricle explains well the frequent audibleness in considerable intensity of mitral regurgitation murmurs at the back, about the region of the angle of the scapula. The free transmission of the mitral regurgitation murmur to the apex of the heart as matter of experience, is probably explained by the physical fact that the mitral curtains project downwards towards the apex, and, as lips projecting in the opposite direction to the regurgitant current are

known to be capable of doing, reflect the murmur, produced by the current, in the opposite direction, that is, in the case of the mitral valves, towards the apex.

It will be evident that the sites chosen as the points of maximum intensity of murmurs produced at the aortic and mitral orifices are not chosen as spots corresponding to the situation of these orifices. In the case of the pulmonary and tricuspid orifice it is otherwise, for these lie superficially, and the stethoscope can be placed directly over the site of the orifice. It has been already noticed that the pulmonary valves lie so near the surface that a shock can be felt over them when they are closed with abnormal force owing to high blood pressure in the pulmonary artery. The site of maximum intensity of tricuspid murmurs is the lower sternum, and the small area of heart uncovered by the left lung.

In a few cases of aortic incompetence, usually of dilatation type, the diastolic murmur may be limited to the manubrium sterni. This is specially apt to be the case as regards a very short murmur, if such it can be called, that immediately precedes the second sound in rare cases of aortic dilatation. This was called the "phw . . . tt" sound by Dr. Walshe, and in some respects it bears a slight resemblance to

the combination of presystolic murmur and accentuated first sound of mitral stenosis, and as in this latter case the observer may be in momentary doubt as to whether he is dealing with a peculiarly accentuated sound only, or with a combination of short murmur and accentuated second sound. "Downwards and towards the left" best describes the usual conduction of the diastolic murmur of aortic incompetence, but it may be heard either all over, or limited to any part of the triangular area thus described: by a line drawn from the "aortic area"—second right costal cartilage where it joins the sternum—along the right border of the bone to the xiphoid cartilage and by lines uniting the extremities of this line with the cardiac apex, which is included. *The whole of this area must be explored before the absence of an aortic diastolic murmur can be safely asserted.* It is not rare for such a murmur to be heard only at the lowest part of the sternum or located to the left of the bone between the third and fifth cartilages. It will be noted that these last mentioned sites are open to objection inasmuch as they are the areas over which tricuspid and pulmonary diastolic murmurs would have their position of maximum intensity, but a tricuspid diastolic murmur is of extreme rarity, and the like pulmonary murmur is little less rare. Aortic incom-

petence generally impresses its special characters on the pulse in greater or less degree.

When the murmur of mitral regurgitation is well conducted to the back it can usually be heard all the way round the side. In the cases already referred to in which the same murmur is inaudible at the back (for the most part cases of mitral stenosis and simple muscle-failure of the heart), and is there replaced by a first sound, the disappearance of the murmur usually takes place behind the mid-axillary line. Occasionally both sound and murmur are together audible at the back. When there is only murmur at the apex, the fact of the first sound becoming audible behind the axillary line points to the regurgitation taking place through a stenosed mitral orifice or between normal valves as a result of muscle-failure, and usually the sound replaces the murmur, the murmur ceasing when the sound becomes audible. Not rarely the 1st sound is present with the murmur at the apex. The conduction or non-conduction of the murmur of mitral regurgitation to the back must not be allowed too much diagnostic weight for, as already stated, there are many exceptions to the general rule. All the facts of the case—history, symptoms and signs—must be fully considered in arriving at the diagnosis.

FREQUENCY OF AUSCULTATORY SIGNS OF MITRAL STENOSIS.

In 1895 the writer undertook a statistical enquiry as to the frequency of the different auscultatory signs of mitral stenosis, taking for the purpose a group of 60 cases, choosing the number 60 in order to make allowance for the occasional omission of the sign in question from the record, so that under no circumstances should he deal with a number of cases below 50. The results were published in the Manchester *Medical Chronicle* for September, 1895, and a short abstract of them perhaps will give the best idea of the relative frequency of the signs in question.

First of all, as regards the *modifications of the physiological heart sounds*. Accentuation of the first sound in the mitral area, *without there being presystolic murmur*, occurred in 13·33 per cent.: presystolic murmur implies the presence of an accentuated sound, and, as will be seen directly, occurred in 53·33 per cent. Thus, taking the cases without and with presystolic murmur, accentuation of the first sound was met with in 66·66 per cent. of the cases.

Reduplication of the second sound somewhere

over the cardiac area, and most commonly in or below the "pulmonary" area to the left of the sternum, occurred almost as frequently: in 65 per cent. of the cases. On the other hand, the second sound was absent from the mitral area only in 11·66 per cent. of the cases.

As regards *murmurs*. Systolic murmur in the mitral area was the most common murmur, occurring in 75 per cent.: the murmur being inaudible at the back in 46·66 per cent., and audible there in 28·33 per cent.

Diastolic murmur, audible at the apex and above the apex, occurred in 66·66 per cent. of the cases: audible above the apex only, in 13·33 per cent. Thus a diastolic murmur audible somewhere over the heart occurred in 80 per cent. of the cases.

Presystolic murmur, again, occurred only in 53·33 per cent. of the cases.

It follows that the pathognomonic presystolic murmur is the rarest, whilst the systolic murmur, which bears only an indirect relationship to stenosis, is the most common murmur, the diastolic apex murmur standing between these as regards frequency: 66·66 per cent.

It is worth noting that a systolic murmur in the "pulmonary" area proved to be the most common murmur of all, having been met with in 83 per cent. of cases.

AUSCULTATION OF VESSELS: ARTERIES AND VEINS.

Apart from the sounds and murmurs heard over aneurysms, which are described in the section on Aneurysm (p. 199), there are sounds and murmurs, audible over arteries and veins of large size which require a short description.

ARTERIAL SOUNDS AND MURMURS.

The femoral artery in Scarpa's triangle is usually chosen for auscultation. When the stethoscope is placed lightly over the femoral artery, *pressure being avoided*, a dull sound, *i.e.*, resembling a cardiac sound, is heard corresponding to the systole of the left ventricle and the diastole of the vessel under the stethoscope. The moment *pressure* is applied with the stethoscope, however, this *sound* becomes replaced, or accompanied, by a systolic *murmur*. The ease with which the *sound* can be obliterated and the *murmur* developed by pressure varies in different cases. Moreover, the *sound* becomes much more of a *tone* and less of the usual dull "thud" under certain circumstances, which probably include a considerable difference between the maximum and minimum blood pressures, and a rapid transition from the one to

the other. Aortic incompetence is the form of heart disease, therefore, that affords the best examples of the modification of the normal arterial systolic *sound* under consideration. In some cases it is easy to obliterate the *sound* and develop the *murmur*: in others difficult. If the *sound* is to be studied, pressure is to be avoided. The *murmur*, indeed, may be regarded as a pressure-phenomenon. In a case of free aortic incompetence when the stethoscope is applied over the femoral artery, *pressure being avoided*, the *sound* in question is heard; and as a rare occurrence in cases of extreme incompetence of the aortic valves, *not one sound* is heard, but *two sounds* are heard, so that there is close resemblance to the physiological sounds of the heart. Even if it were possible for these to be conducted to the femoral artery from the heart, which it is not, in the cases that exemplify the phenomenon, no physiological heart sounds usually exist, for only the " double aortic " murmur of the disease is audible over the heart under the circumstances. The sounds, then, must be locally produced. It is a matter of extreme difficulty to account for the production of the *second sound*. The sign: " double sound in the femoral artery." is commonly called " Traube's sign," after the late Professor Traube, of Berlin, who first described it. It is so rare a sign that few opportunities of hearing

it are afforded even in a large clinical experience of heart cases.

There is another and much more common arterial auscultatory sign met with in cases of aortic incompetence: it has been called Duroziez's sign. Duroziez's sign is altogether different from Traube's, with which it has been unfortunately confused. In Duroziez's sign there is developed *by pressure with the stethoscope a diastolic, as well as a systolic, murmur.* This diastolic murmur is usually less loud than the systolic, and the degree of pressure necessary to develop it varies in different cases. It probably means that there is not only incompetence of the aortic valves, but a high degree of incompetence of the same.

Over the carotids and subclavians the writer has occasionally heard what resembled a reduplicated or "split" sound, such as is described in the text as a "double or reduplicated first sound": "turrup" and the resemblance of this last sign to a short presystolic murmur and first sound is recalled by the arterial phenomenon under consideration. The circumstances under which the sign has been met with do not throw any light on its explanation.

AUSCULTATION OF VEINS.

The jugular and subclavian veins are usually chosen for auscultation, but the femoral vein may be made use of.

Dr. Mackenzie has described "sounds produced by the stretching of the valves in the veins" (p. 178, "The Study of the Pulse," paragraph 147). These are best heard over the "jugular bulb," and may be auricular or ventricular systolic in rhythm.

Murmur audible over veins has long attracted the attention of physicians. It is apt to be specially developed in the condition of anæmia known as chlorosis, that is so common in young women. The *murmur* in question differs from cardiac and arterial murmurs by being *continuous*, though of *varying loudness* and quality. Inasmuch as the flow of blood to the heart varies in amount and rate, under a great variety of circumstances the varying degree of intensity of the murmur in question may be accounted for. Moreover it is practically impossible to prevent the degree of pressure with the stethoscope from varying. The quality of the murmur may be soft and blowing, humming or musical, or the murmur may actually be described as a "roaring noise." Sometimes a fictitious resemblance to a "double murmur" is given by intensification of the murmur during the systole

of the ventricle, usually attributable to the adjacent carotid artery being slightly pressed upon by the stethoscope, and so developing a coincident murmur. There is probably much yet to be learnt about venous murmurs: it is notorious how well-developed they are in some cases: how ill-developed in other cases of anæmia. As a general rule only, to which there are so many exceptions, they are better developed in the relatively benign chlorosis than in pernicious anæmia.

A continuous murmur, though varying in intensity, may sometimes be heard over the spleen and enlarged thyroid. It is usually louder in systole than diastole, so as to prevent some resemblance to a "double" murmur.

PULMONARY INFARCT: PERIPHERAL VENOUS SOURCE.

Although it is perhaps scientifically incorrect to include in a description of Heart Disease the condition of embolism of the pulmonary artery from the detachment of a clot in a peripheral vein, inasmuch as the accident does not imply any cardiac "disease," still the symptoms concern the heart to such a degree that they may well raise the question of the integrity of that organ. The accident is not of very rare occurrence, and it is not by any means necessarily fatal unless, of course, the detached clot is of such a size as to block the trunk of the pulmonary artery. The writer has seen cases in which the source of the embolism was a thrombosed vein resulting from an infectious fever, a vein injured by a fractured bone, and, the most common source of all, a varicose vein. Should such a vein receive an injury and phlebitis be set up, the accident of detachment of a clot is to be apprehended, and absolute rest enjoined for a considerable time. The form of pulmonary embolism that occurs in the puerperal state is

specially apt to be speedily—in fact, immediately—fatal, the trunk of the artery being blocked.

Symptoms.

The patient is seized, unexpectedly, with intense dyspnœa and intra-thoracic distress of indefinite character. Such dyspnœa, if survived, is apt to subside and recur paroxysmally. Any movement of the patient is apt to be followed by a violent access of dyspnœa, in which the patient may be thought to be dying. Physical examination of the heart and lungs is usually negative at this stage. There may be a systolic murmur in the "pulmonary area," but the frequency of such a murmur is great quite apart from pulmonary embolism, and consequently its presence is of comparatively little value *unless the murmur is known to be recent.* The heart's action is usually frequent and may be irregular. Little more than a surmise, however, can be formed as to the nature of the illness, unless a source of venous thrombosis is obvious. In a few days, however, there will usually occur a symptom, that when pronounced is conclusive of the diagnosis, namely, *hæmoptysis.* The actual quantity of blood expectorated is seldom large, but the blood is brought up almost pure and usually dark in colour. Hæmoptysis occurs generally within five days of the sudden seizure, and with

it, or preceding it, physical signs in the lung become developed, either small bubbling sounds or the percussion-dulness and bronchial breath-sound of a large area of consolidation. The pulmonary physical signs, indeed, may closely simulate, and practically be identical with those of pneumonia, but the fever is much less, and at first may be altogether absent or trivial. Pleurisy, with pain in the side and exacerbation of pyrexia, is a common later result, and enhances the resemblance of the illness to pneumonia. The pronounced development of physical signs and especially the amount and pureness of the expectorated blood, may be in striking contrast with the degree and often short duration of pyrexia. In one case which was seen by the writer, and which followed a simple fracture of the leg, the patient woke in the night with hæmoptysis, no sudden or distressing dyspnœic seizure having been experienced. In another case, in which the source of the clot was post-febrile thrombosis of a vein in the leg, the physical signs of consolidation were fully developed, but the pyrexia was much less than is usual in pneumonia, while the expectoration was practically pure blood. In both these cases the physical signs were developed in the lower part of the lungs, as is usual.

INSTRUMENTAL EXAMINATION OF ARTERIES AND VEINS.

While throughout this book a knowledge of anatomy and physiology with regard to the circulatory organs and the circulation is assumed, there are certain views concerning the contractility and mode of action of the heart, that have only recently received general adoption, if, indeed, that word can even now be used. There was at one time a disposition to regard the remarkable rhythmic action of the heart as of nervous origin, the contractile muscle-substance being, as it were, placed in complete subjection to a nervous system, partly local and partly situated in the higher centres of the general nervous system. Even now the multitude of minute nerve-ganglia and their more numerous communicating branches forming a close network, may give us pause, before at once accepting recent views as to the predominance of the muscle-substance itself and the assignment to the nervous system only of the subordinate function of a regulator and controller on behalf of the organism of the individual as a whole. A mere sketch of recent views is as much as it would

be prudent to attempt here. Time will reveal if all of these in detail shall become established, or if they shall undergo in the future very important —it may be essential—alteration.

Anatomically, the fact that the striped muscle-fibres of the heart are branched and have no sarcolemma arrests attention, and physiologically there is the still more remarkable fact of rhythmic stimulus-production, which is known to arise normally at the venous ostia adjacent to the auricle—thus at the commencement of the fœtal tube from which the heart was formed. Having arisen thus in rhythmic fashion, the stimulus is conducted downwards towards the apex of the heart over auricle and ventricle. Its passage would seem to be somewhat less easy at the point uniting these two chambers, and it will be found that under abnormal circumstances there may be here actual arrest of the conduction of the stimulus causing contraction. Although normally the stimulus production takes place at the ostia venosa, artificial and probably pathological stimuli take effect at other parts. "The conduction . . . . takes place within the wall in all directions. Thus it is quite easy to make the heart contract in a direction opposite to the normal" (Wencke-bach, p. 15). To meet recent views, certain properties are ascribed to the muscle-fibres of the

heart, which properties may become altered and modified in disease. Influences are supposed to be at work in the direction of either increasing or diminishing the properties referred to. The four properties assigned to the muscle-fibres of the heart are (1) *stimulus production;* (2) *stimulus conduc-*

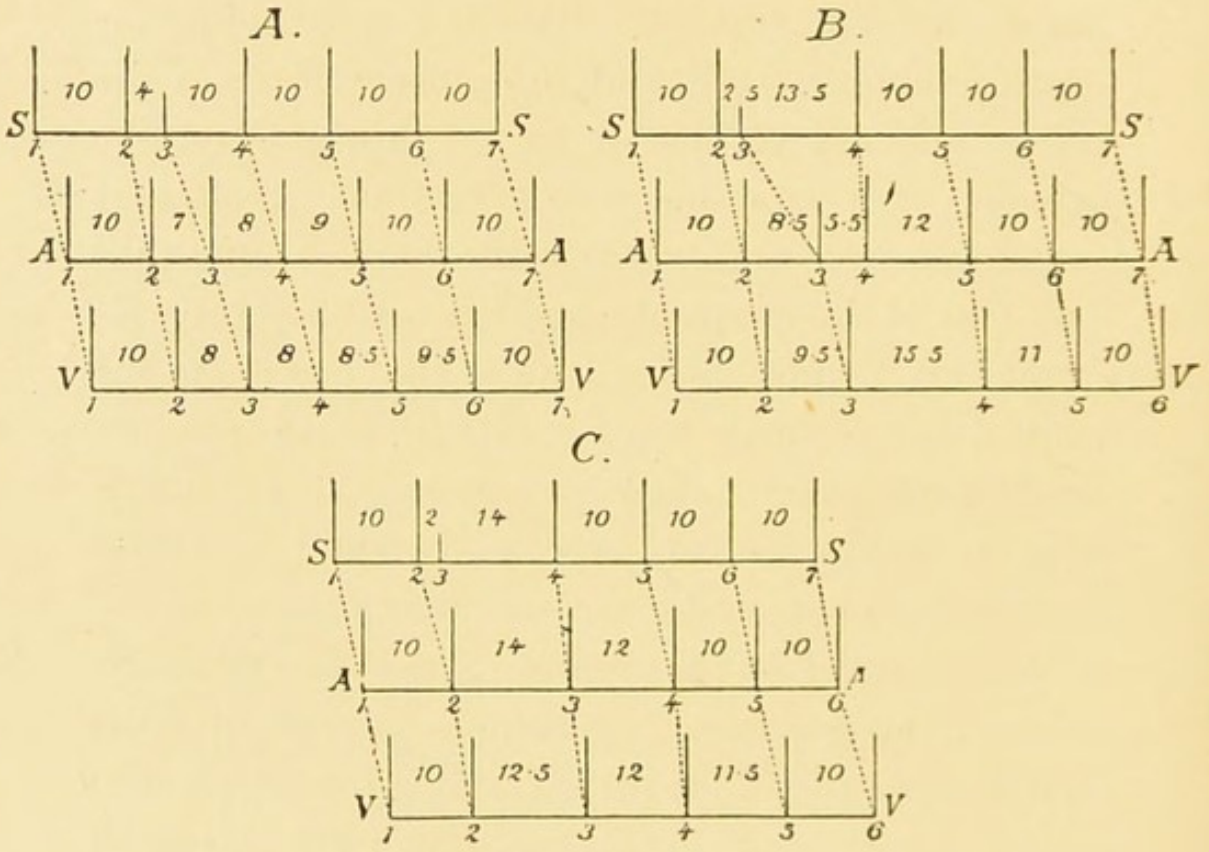


Fig. 6.—After Engelmann.

tion; (3) *excitability of the cardiac muscle-fibres;* and (4) *the contractility of the same.* The following names have been given to the influences modifying each respectively in a positive or negative direction: (1) *Chronotropic;* (2) *dromotropic;* (3)

bathmotropic; and (4) *inotropic.* Thus positive *chronotropic* influences accelerate the rate of the heart-beats and negative influences slow it. *Dromotropic* influences render the conduction of stimulus more difficult or more easy according as they are negative or positive. In this relation the so-called "bridge" between auricle and ventricle has to be remembered. *Bathmotropic* influences modify the excitability of the muscle-fibres of the heart, and are estimated by the lowest stimulus capable of producing a contraction. *Inotropic* influences are concerned with the all-important function of the contractility of the cardiac muscle-fibres or "musculatur" to make use of the convenient German word. It will be shown how the recognition of these properties of the heart-muscle proves useful, especially when irregular cardiac action is under consideration.

With regard to the action of the heart-muscle, it is of the greatest importance to bear in mind that it *either does not respond to artificial stimulus at all, or responds with the maximum power of which it is capable.* The contractions of the heart-muscle are thus, it may be said, *all maximal.*

A no less important consideration is that when once a contraction has taken place the cardiac muscle-fibres for a time pass into a condition in

which they are insusceptible to stimulation and incapable of contraction. This fact explains one of the most common phenomena of irregular pulses. From one cause or another what is called a " premature systole " occurs before the rhythmic period for the next normal systole, and the result is that this latter systole does not take place, inasmuch as when the normal stimulus from the venous ostia reaches the ventricle, it finds the muscle-fibres insusceptible of stimulation and contraction, owing to the recent abnormal systole having left them exhausted and requiring a minute space of time for recovery of function. By the next period, however, at which stimulus comes down from the venous ostia the fibres have not only recovered their contractility, but respond energetically, causing often a pulse-beat of exaggerated size. This last fact is probably in part due to the accumulation of blood that has taken place during the preceding period of diastole. The period during which the fibres remain insusceptible to stimulation is called the "*refractory period.*" As soon as it is over response to stimulation again becomes possible, and the longer the time given for restoration—in other words, the later (within limits, of course) the period of stimulation—the greater the response, because more time has been given for recuperation and the chamber is the more replete.

What is meant by the "law of the conservation of the rhythm of physiological stimulation of the heart" is the regular production of "stimulus-material" and its as regular destruction by normal systole. In the case of a premature systole, for instance, the succeeding normal systole does not occur—is left out—and the heart is at rest till the second normal period arrives. Physiologically, it is known that this compensatory pause (as it has been called) never occurs after a successful stimulus applied to the venous ostia. In the case of abnormal stimulus arising in the auricle again, it is often incomplete, while when the stimulus arises in the ventricle the compensatory pause is complete, and the first spontaneous systole occurs at the moment at which it would have occurred had there been no abnormal stimulation of the heart.

The influence of the nervous system upon the heart would seem to be an indirect one, and exerted upon the four properties of the heart-muscle already referred to: stimulus-production, conductivity, excitability and contractility. Thus chronotropic, dromotropic, bathmotropic and inotropic influences, positive or negative, and in various combinations, may be recognised.

Extra systoles occur more frequently when the patient is lying down than when he is standing up,

because in the latter posture the pulse is usually slower, and greater opportunity is given for their occurrence, inasmuch as the excitable phase is longer than when the patient is standing upright and his pulse accelerated.

The patient is commonly aware of the occurrence of an extra systole, but probably not always in the same way—most patients seem to feel a "stop," as it were, in their cardiac action and also the compensatory exaggerated beat that follows. It has been held that the extra systole may be felt as the chief impulse, as it may be more or less unable to effect the opening of the aortic valves, and so the impulse against the chest-wall is felt with greater intensity. Moreover, the first sound is said to acquire a "booming" character, explained by the contained blood "striking the mitral valves with all the greater force." The "booming" sound has been also associated with the small amount of blood in the ventricle at the time of the extra systole. The apex-impulse, while forcible, is short and unsustained: there is no holding of the apex up against the chest wall, while the ventricle is being emptied.

As Wenckebach asserts: "extra systoles need not be abortive, and may produce an ordinary pulse-beat, while abortive contractions, which cause a very small pulse-beat, or none at all, must not

necessarily be extra-systoles . . . . it is not infrequently very difficult, or indeed impossible, to determine whether one has to deal with extra systoles or early occurring normal contractions" (Snowball's translation, p. 54).

Dromotropic Influence in Practice.

Disturbance of stimulus conduction would seem to be a common cause of irregular action of the heart in man. Each systole, it has been seen,

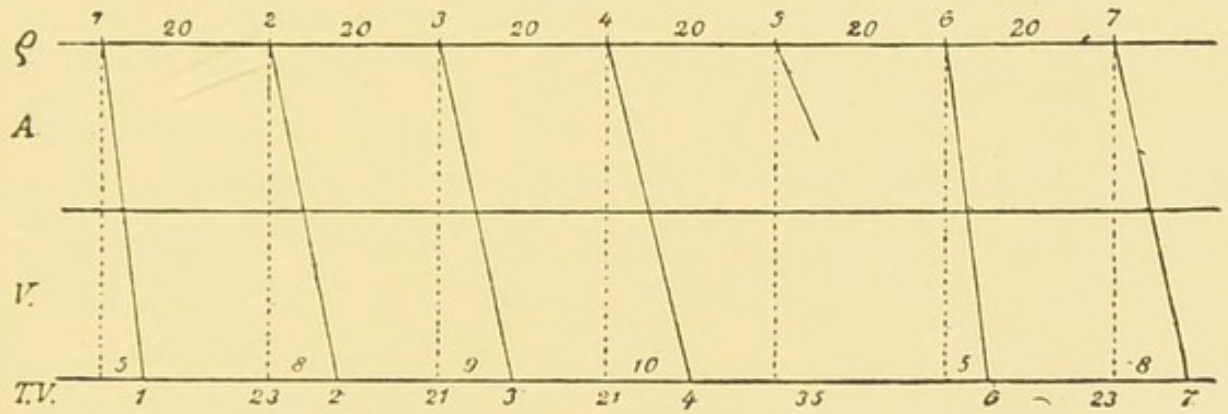


Fig. 7.

weakens the heart-muscle in its various properties of stimulus production, stimulus conduction, excitability and contractility, while each pause gives time for the heart-muscle to recover these properties. Recovery may be said to be incomplete normally when the next systole comes round, a fact of much importance in considering irregular action of the heart. Moreover, strong and numerous contractions reduce these properties

more than few and weak ones. If a longer pause than normal occurs through the missing of a beat, the ventricle has more time to recover than if there had been no such prolongation of the diastolic period. In the diagram (Fig. 7, p. 137) taken from Wenckebach's book it will be noticed that the beginning of the systole takes place at regular intervals, that the wave of contraction reaches the apex five-tenths of a second later in the case of the first contraction, in the second systole 8 points later, and in the third systole only 9 points later, the first systole, after an intermission, damaging the conduction more than the succeeding ones. The fourth systole reaches the apex likewise only 10 points later, but in the case of the fifth systole the stimulus fails altogether to reach the ventricle, having failed to pass the auriculo-ventricular "bridge." During the intermission, however, under the influence of rest, the "bridge" recovers its conducting power, so that the stimulus produces contraction of the ventricle once more after 5 points, instead of 10 points as before the intermission. Between the first and second ventricular-systoles there are 23 tenths of a second: between the second and third, 21 points (12+9): between the third and fourth, again, 20 points (11+9: but between the fourth and sixth systoles, 35 points, the fifth systole being absent: while between the sixth

and seventh systoles, the conduction power of the bridge having been restored during the 35 point period of rest, there is a delay of only 5 points. During all this time the intervals between the offsets of the systoles at the venous ostia have remained the same : at 20 points. The conductivity of the heart-muscle is diminished by increased rate of frequency of contraction, and *vice versâ*, the time for recovery being, of course, less in the case of the frequent pulse.

In Wenckebach's second diagram (Fig. 8.)

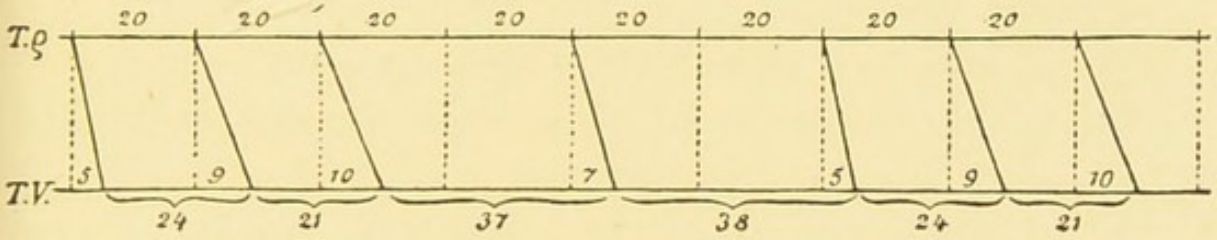


Fig. 8.

it will be noticed that the first intermission only enables conductivity to recover sufficiently, that the delay in the ventricular contraction is represented by 7 points instead of 5. Another period of rest is required before the period of delay is reduced to 5.

Among the most interesting results obtained by Dr. Mackenzie's research is that by which he shows that when conduction is at fault a delay between the auricular- and ventricular-systoles can be demonstrated (Figs. 9—11.

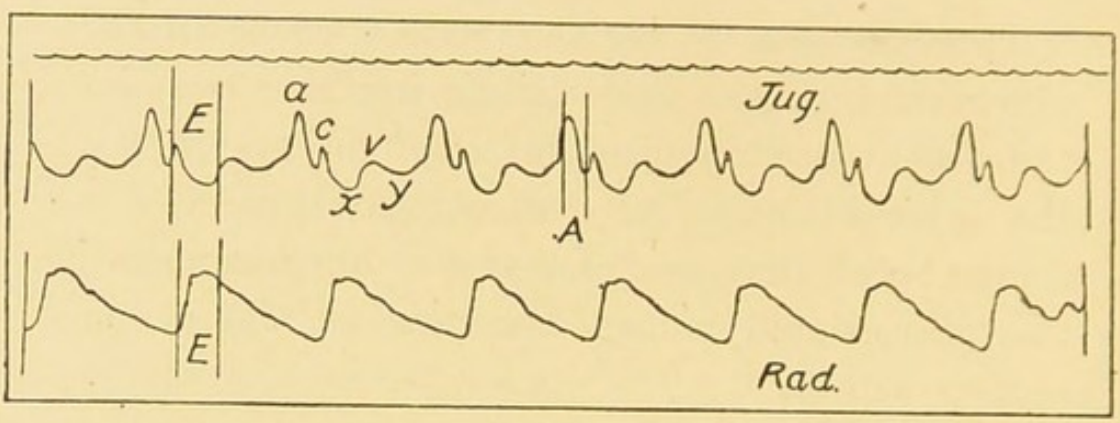


FIG. 9. The space *E* represents the time of the ventricular systole. The wave *a* is due to the right auricle, and the wave *c* to the carotid impact. The space *A* represents the *a*-*c* interval. The time marked in all these tracings equals one-fifth of a second.

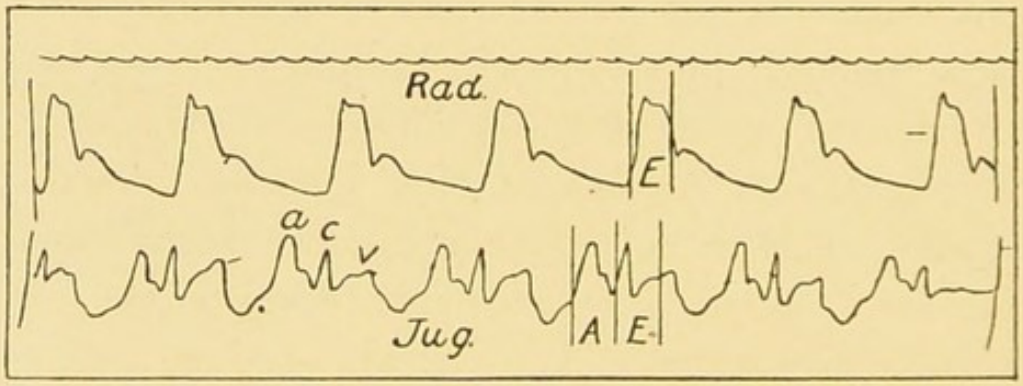


FIG. 10. The *a*-*c* interval (space *A*) is twice the normal period, lasting two-fifths of a second in place of one-fifth as in Fig. IX, indicating a delay in the stimulus passing from auricle to ventricle.

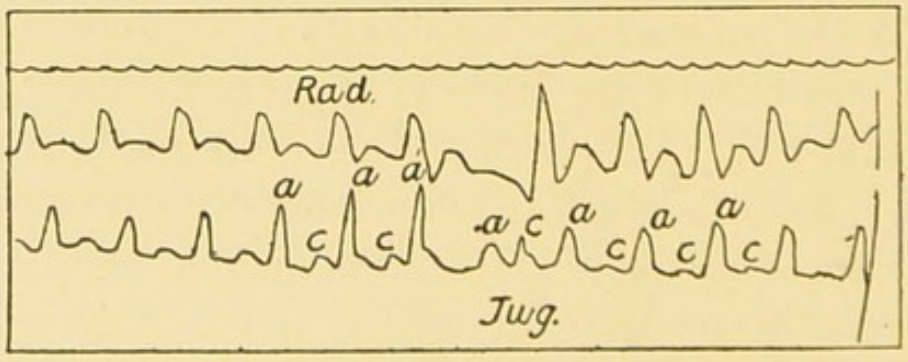


FIG. 11. Shows a gradual lengthening of the *a*-*c* interval till a radial and carotid pulse beat drops out after the auricular wave *a*. The following *a*-*c* interval is shortened.

This may seem at variance with the auscultatory observation that the presystolic murmur *runs into* the accentuated first sound that follows without the intervention of any pause. But it must be remembered that the presystolic murmur usually has disappeared before cases put on their more serious aspect.

Inotropic Influence in Practice.

Disturbance of the important function of contractility has its best example in the well-known, but somewhat uncommon, *pulsus alternans:* this is a frequent pulse, in which there is a regular alternation of larger and smaller beats (Fig. 52). A remarkable feature of this type of irregular cardiac action is its continuance over long periods. Extra systoles never occur with such regular persistence. The *beginning* of the systole in each beat of the alternating pulse occurs *at regular intervals*, but the systole in the case of the larger beats is of greater duration, and in the case of the smaller of less duration, so that the pause that follows the latter is of greater duration than that which follows the former (*i.e.*, the larger beats). In physiological experiments this type of pulse can be produced artificially, and the smaller beats can be made to disappear, but it does not seem that this occurrence has been demonstrated in man.

A variety of pulsus alternans is that in which the smaller beat is delayed: "one high pulse is regularly followed by a low one, and this low beat is separated from the next high one by a shorter pause than that which separated it from the preceding high pulse." This modification of the pulsus alternans can be explained by the supposition that, combined with the affection of contractility (inotropic influence), there is also affection of conductivity (dromotropic influence).

Wenckebach has given a very interesting explanation of the pulsus alternans, which has been already referred to. "Great exhaustion of the heart impairs its contractility: a too high frequency which . . . is able to diminish all the functions of the cardiac-muscle, the conductivity most of all, can also reduce the contractility to a certain extent." The ordinary type of pulsus alternans may, then, be attributed to impairment of contractility. "When the contractility is very much impaired the small short systole weakens the heart less and gives it longer time to recover," according to Wenckebach, and so the development of the pulsus alternans may tend to the preservation of the organism. The other variety characterised by the late occurrence of the smaller beat may be referred to a combined affection of contractility and conductivity.

Bathmotropic and Chronotropic Influences in Practice.

The excitabilty of the heart-muscle is measured by the lowest stimulus that will produce a contraction. It will be remembered that cardiac contractions are all maximal. The excitability of the heart may be increased or diminished (positive and negative *bathmotropic* influence): increase of it shortens the cardiac cycle and augments the frequency, diminution of it lengthens the cardiac cycle and reduces the frequency of the beats. Extraneous stimuli are more likely to affect the heart when it is under positive bathmotropic influence, and extra systoles are more likely to occur.

Powerful negative bathmotropic influence may, again, lead to the missing out of a beat owing to the contraction-wave meeting with a refractory portion of the muscle, the bathmotropic influence acting locally. "The contraction which began at the venous ostia will travel as far as the refractory spot and stop there. The next stimulus, however, will not find the muscle refractory, because it has had a rest, and so will produce a contraction. . . . In this way systoles may fall out periodically, until, finally, the half-frequency is reached." (Wenckebach).

According to Wenckebach, negative bathmotropic influence can be alone assumed to be at work "in cases where the rhythm of the heart remains perfectly normal." If evidence of a diminution of conductivity (negative dromotropic influence) is also present, the droping out of a beat must be attributed to this. When a transitional stage of alternation of the pulse is observed a disturbance of contractility (inotropic influence) is probable. The condition described as "irritable weakness" of the heart is induced by positive bathmotropic influence, whereby the heart becomes exposed "to the action of all kinds of stimuli," which otherwise would have little or no effect. Moreover, "there is a great tendency to extra systoles."

Chronotropic influence is exerted on the heart-muscle, positively or negatively: and the stimulus production is hastened or retarded. It is the cause of "true arhythmia": dromotropic, inotropic and bathmotropic influences (the last acting below the venous ostia) only produce irregularity by interfering with the effect of the regularly recurring stimulus arising at the venous ostia. When there is observed "the falling out of systoles while the original rhythm is preserved (halving, and so on) we must assume that a minimal contraction, perhaps one confined to the cells which

generate the stimulus, has at all events occurred." (Wenckebach).

When there is increase in the production of the motor-stimulus this "reaches the necessary strength again, after a systole, in a shorter time than normally, the cardiac cycle is shortened, the frequency increased." But "increased excitability can have the same effect."

Negative chronotropic influences producing slowing of the pulse are, clinically, almost indistinguishable from negative bathmotropic influences. Moreover, a positive bathmotropic influence may obscure a negative chronotropic one.

THE CARDIOGRAPH.

Tracings of the apex-beat—the apex-beat representing the contraction of the left ventricle show sometimes a slight rise of the trace-line preceding the rise due to the ventricle entering into systole, which represents the contraction of the left auricle, driving home the blood, as it were, and distending the left ventricle. Then comes the more or less abrupt ascent representing the contraction of the left ventricle, a small peak sometimes being formed at the top. Whether there be this peak or not, the curve, of which the line of ascent referred to forms the ascending limb, is flattened and broadened out at the top so as to form a sort

of plateau corresponding to the outflow from the ventricle, during which the apex is held, as it were, against the intercostal wall. Then follows the descending limb of the curve representing the relaxation of the ventricle. Occasionally a slight rise in the trace-line follows, owing to the accumulation of blood in the chamber, till it is abruptly increased by the next auricular or

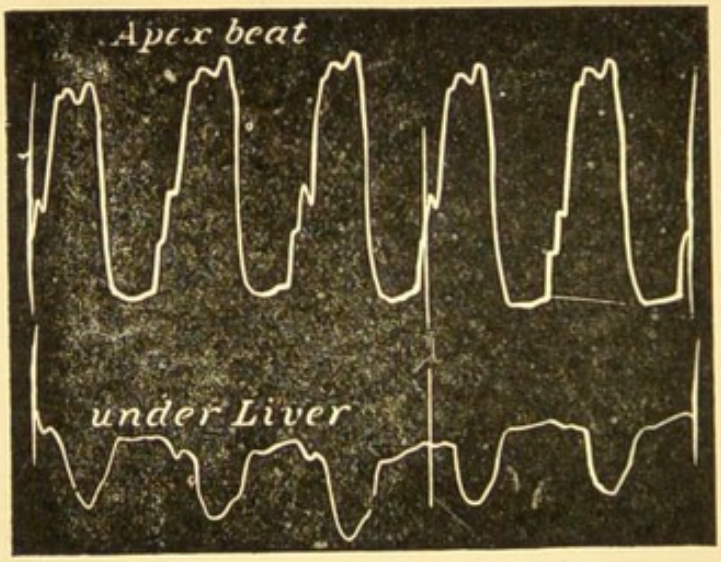


FIG. 12. Simultaneous tracings of the apex beat and of the movement of the liver. When the ventricle empties the liver is drawn up, and this causes the fall in the tracing. (Mackenzie).

ventricular contraction. It has been already explained how the apex-beat is *sui generis*, and to be carefully distinguished from all other impulses of the heart. *There is nothing corresponding to it in the case of the right ventricle.* The impulse that denotes the movements of the right ventricle is of an entirely different kind from that denoting

the systole of the left ventricle—" the apex-beat." When surface movements are detectable due to the systole and diastole of the right ventricle, on the application of the cardiograph, it is found that it is *diastole*, and *not systole*, which is represented by an elevation. When as already stated the right ventricle is engorged and over-distended

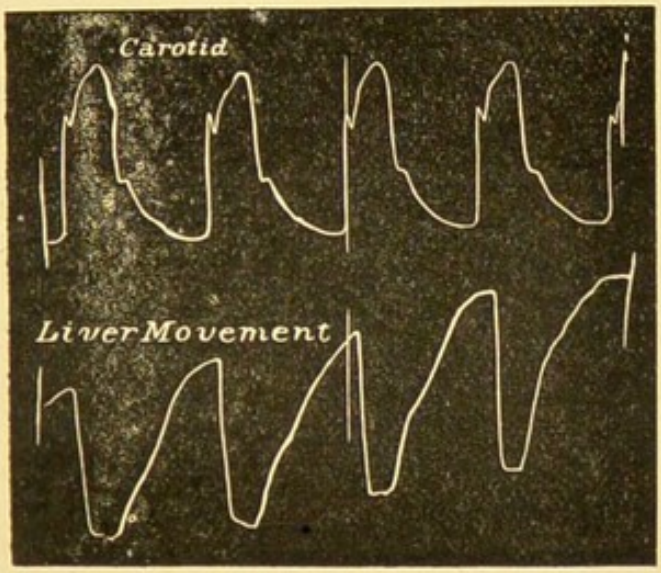


FIG. 13. Simultaneous tracing of the carotid pulse and liver movement. With the appearance of the carotid pulse there is a sudden fall of the liver tracing due to the liver being drawn upwards by the emptying ventricles. (Mackenzie).

the left ventricle tends to get pushed back, as it were, and its impulse, the apex-beat, is interfered with or annulled, so that a simultaneous comparison of the two can seldom be made. Dr. Mackenzie has shown how the systolic protrusion of the apex-beat may become replaced by the depression of the right ventricular-systole, when the right chambers

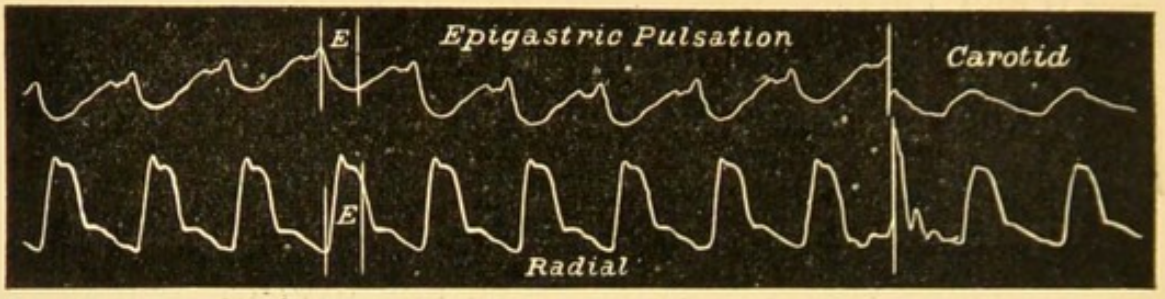


FIG. 14. Simultaneous tracing of the epigastric pulse, due to a dilated right heart, and of the radial pulse. The epigastric pulse shows a retraction during the ventricular systole (*E*), and a protrusion during the filling of the ventricle. (Mackenzie).

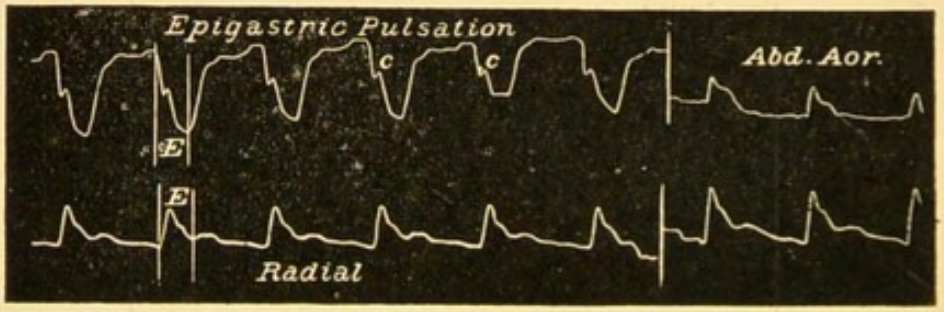


FIG. 15. Shows the same features as Fig. 14, with the exception of the small wave (*c*) occasioned by the shock communicated to the epigastrium by the abdominal aortic pulse. A few beats of the abdominal aorta are also given. (Mackenzie).

become greatly dilated, and the impulse of the right ventricle appears to take the place of the impulse of the left ventricle. Owing to their being communicated through the liver, the epigastric movements of the right ventricle must be carefully distinguished from the true expansile pulsation of the liver, elsewhere described (p. 153). Occasionally the abdominal aorta, or even an aneurysm of the same, produces pulsation in the

epigastrium, but in these cases the impulse or protrusion is systolic in time. It is said that even an enlarged and hypertrophied left ventricle may occasion epigastric pulsation, and in this case the impulse is systolic.

There is no doubt that Dr. Mackenzie is right when he insists upon the observer carefully distinguishing, in palpation, "shock," due to the heart-muscle entering into contraction, from the actual movement that immediately follows it.

THE VENOUS PULSE.

Owing chiefly to the work of Dr. Mackenzie great advances in our knowledge of pulsation in veins have been made during recent years. Long ago the frequency of a double impulse or wave in a cervical vein was recognised, though when a tracing was taken and the two waves represented graphically, their significance was misinterpreted. Moreover, when a single wave was recognised occasionally to take the place of the double wave, such wave was believed to be always ventricular-systolic and the fact overlooked that it might be auricular-systolic. These distinctions, of course, became possible only when the polygraph came to be employed in the investigation. By this instrument simultaneous tracings could be taken of the venous pulse

and the radial pulse, the liver or the apex-beat itself. As regards the double wave referred to and its misinterpretation, it was thought that when (as is usually the case) the waves were unequal in size, the smaller wave represented the contraction of the auricle, the larger the contraction of the ventricle. How impossible it was that this view could be correct will be evident when the following considerations are borne in mind:—Anatomically, there is the vein in the neck communicating with the vena cava, in its turn continuous with the right auricle and ventricle. Between the vein seen in the neck and the right ventricle of the heart, then, there lies the auricle.

For the moment the venous valves may be disregarded; in the presence of dilatation of the veins they seldom remain competent. With the cervical vein, the vena cava, ostia venosa, auricle and ventricle in the mind's eye, let the question be put: The auricle having contracted and sent a backward wave to the cervical vein, when the ventricle enters into systole what will be the state shown by the cervical vein? Will it show a positive wave still larger than that of the auricle, as used to be supposed? No: and for the simple reason that *anatomically* between the ventricle and the cervical vein lies the auricle, and that

physiologically during the first part of ventricular-systole the auricle is in diastole, expanding and producing a *negative wave* in the cervical tracing. How is the frequently present second wave produced? It does indeed represent the ventricular-systole, but *only the last portion of it*, as it were. The first portion of ventricular-systole would have produced its positive wave in the cervical vein but for the interposed expanding auricle, which more than negatives it. *The auricle has to be refilled before it is capable of transmitting to the neck any backward impulse, there may be, from the ventricle.* The greater the venous stasis, the earlier will the auricle be refilled and the greater the amount of the ventricular contraction, that is represented in the cervical vein, and the earlier its period of commencement. The readiness with which the auricles lose their contractility under the influence of over-distension seems to be great, though the diminution of contraction usually takes place gradually, so that the wave, depending upon the systole of the auricle, with its subsequent diastole, becomes less and less, while the wave representing the ventricular-systole, with its subsequent diastole, is augmented, and *occurs earlier.* Thus the auricular wave may disappear, as its cause—the contractile activity of the auricle—ceases, and the chamber becomes a passive reservoir.

These changes will be easily understood by reference to Dr. Mackenzie's diagram, Fig. 16.

Of the same nature as the venous pulse is the liver pulse. They both represent events occurring

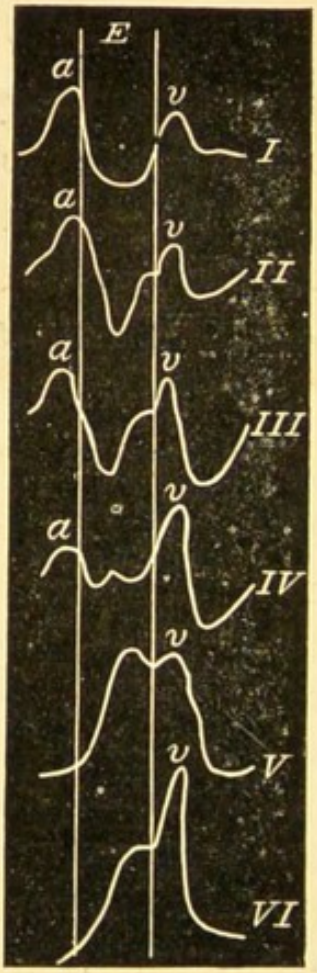


FIG. 16. Semi-diagrammatic representation of the transition of the venous pulse from the auricular type into the ventricular. The space *E* represents the duration of the ventricular outflow through the pulmonary orifice, and the lines enclosing it therefore represent the time of opening and shutting of the pulmonary valves. Coincident with the growth of the ventricular wave there is a diminution and ultimate disappearance of the auricular wave (*a*). (Mackenzie.)

on the *right* side of the heart. So-called "epigastric impulse" is a long-recognised clinical phenomenon, and generally denotes the move-

ments of the right side of the heart — the right ventricle.

"Epigastric impulse," taken to mean the mere impulse of the right ventricle communicated by the liver to the epigastric region, is an entirely different thing from the phenomenon of liver

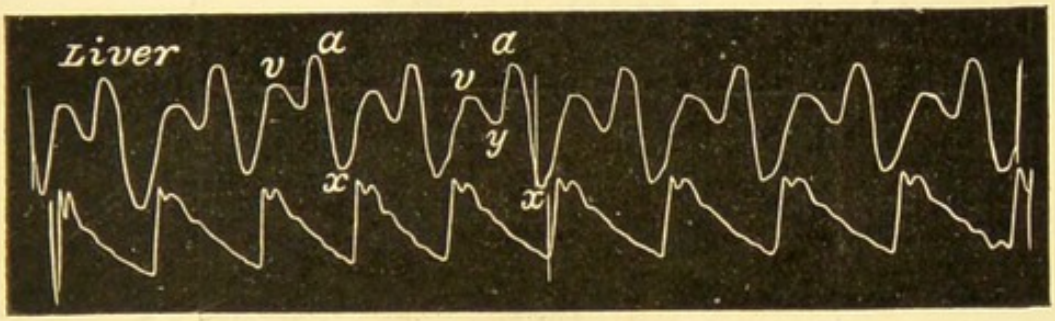


FIG. 17. Simultaneous tracings of liver and radial pulses. Both auricular and ventricular waves represented. (Mackenzie.)

pulsation, which has now to be studied and which is a true expansile pulsation of the liver itself.

True liver pulsation is of much importance as indicative of grave disturbance of the circulation. It may be auricular-systolic* or ventricular-systolic, or its movement may be represented instrumentally like the common cervical venous pulsation, both by an auricular- and a ventricular-systolic wave.

There is one feature, however, met with in the jugular tracing that is not represented in the liver pulse: it is the small wave, or rather interruption of the descending limb of the cervical curve marked c in the diagrams; it is of considerable importance, inasmuch as it represents the impulse of the carotid artery lying by the side of the vein,

* The auricular liver-pulse should suggest tricuspid stenosis because hypertrophy of the right auricle is apt to be pronounced in this lesion.

and accordingly it becomes an useful timing agent. It is not, however, an intrinsic venous phenomenon at all. It is, of course, absent in a liver tracing.

Through the kindness of Dr. Mackenzie it has been possible to illustrate the broad features of venous and liver pulsation by the following illustrations taken from his epoch-making work on the subject: —

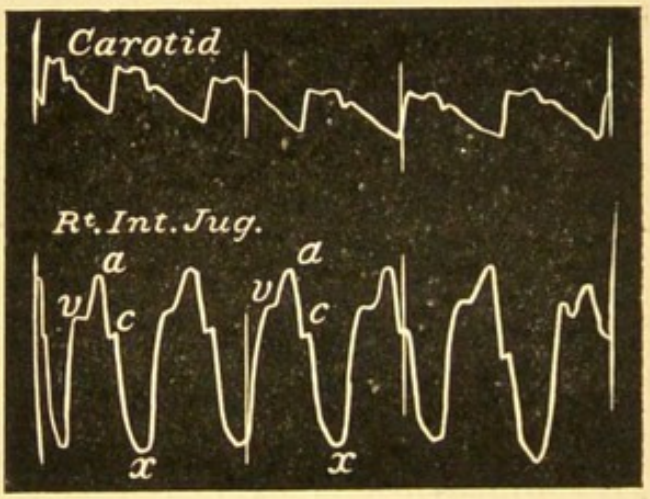


FIG. 18. Simultaneous tracings of the carotid pulse and of the jugular pulse, showing exact synchronism of the carotid pulse with the carotid wave (*c*) in the jugular pulse. (Mackenzie).

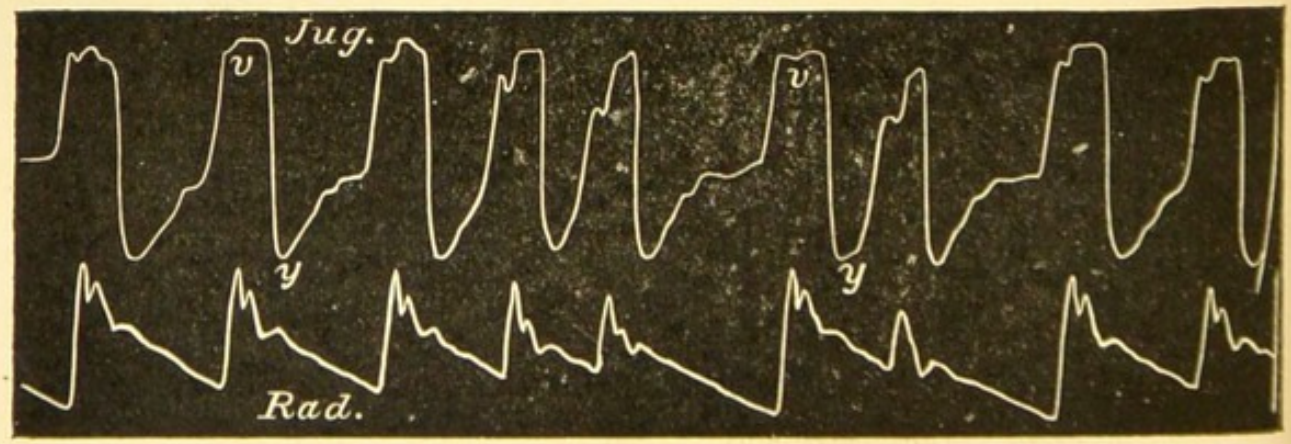


FIG. 19. Simultaneous tracings of jugular and radial pulses: one large wave (*v*) synchronous with and due to the ventricular systole, and one large depression (*y*) synchronous with and due to the ventricular diastole. (Mackenzie.)

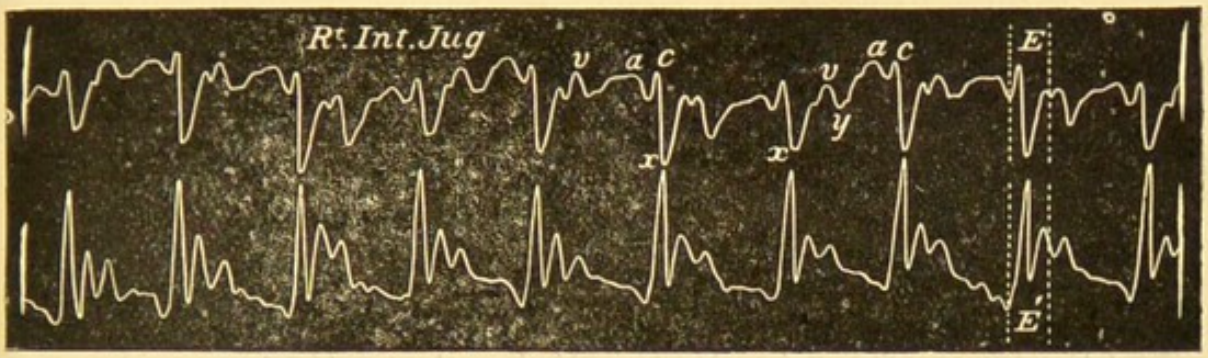


FIG. 20. Simultaneous tracings of the jugular and radial pulses. (*a*) represents the auricular wave ; (*v*) the ventricular ; (*c*) the carotid wave ; (*x*) the depression following the auricular wave ; and (*y*) the depression following the ventricular wave. (Mackenzie).

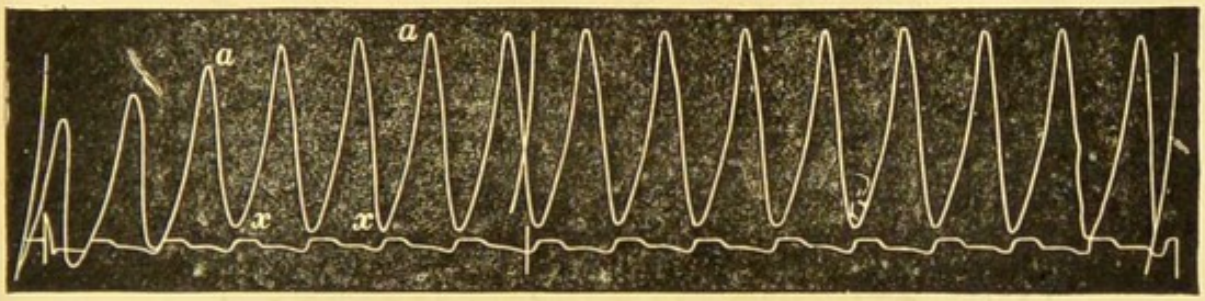


FIG. 21. Simultaneous tracings of the jugular and radial pulses: one large wave due to auricular systole. (Mackenzie).

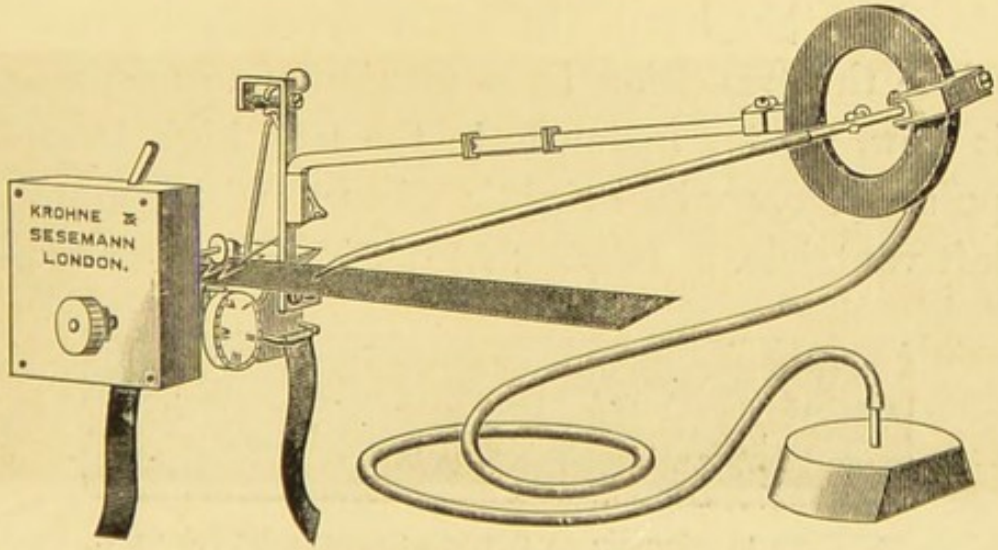


FIG. 22. Mackenzie's Polygraph.

THE PULSE.

What is commonly termed "the pulse" is the difference between the maximum and minimum blood pressure in the radial artery that results from each contraction of the left ventricle. Sometimes this difference is very great and the transition from the one condition to the other is abrupt, as, for instance, in aortic incompetence. (Fig. 23.)

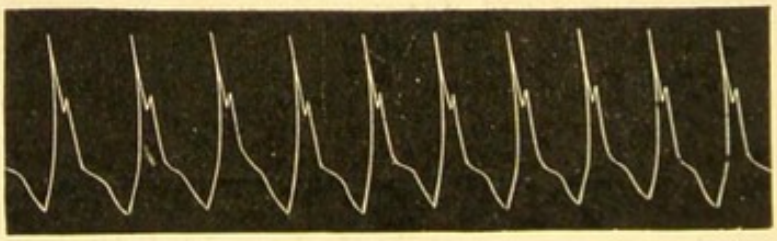


FIG. 23.

On the other hand, the difference may be small, while the transition from maximum to minimum is slow, as, for instance, in the true high tension pulse of chronic Bright's disease with vigorous left ventricle. (Fig. 24.) Sir Wm. Broadbent

FIG. 24. **High tension pulse tracing**: sustained tidal wave. Aortic Notch high above respiratory line.

has well called such a pulse "full between the beats," just as the pulse of aortic incompetence has been called the "pulse of unfilled arteries." The skilled clinicist almost instinctively, besides counting the rate of the pulse, estimates, by pressure with his finger, the force required to obliterate the impulse or, as is said, the "pulse." Moreover, he acquires information as to the sustention of the maximum pressure. The pulse in aortic incompetence may be strong, but it is only momentarily so, and hardly has the maximum been attained, when the pressure collapses, *i.e.*, rapidly assumes its minimum.

In the manœuvre of obliterating the pulse by pressure the observer not only ascertains the force of the impulse derived, of course, from the left ventricle, but when he has applied sufficient pressure to cause obliteration of the impulse, he can roll the emptied vessel between his finger and the radius and so ascertain if there be decided thickening of the arterial wall. It sometimes happens in old people that the arterial coats are the seat of calcareous deposition, and the vessel feels like a row of beads. When such pronounced changes occur in the arterial wall the usefulness of a sphygmogram may be doubted. The radial of old people is often very large, while the thickening of the coats is moderate, and there is no calcareous

deposition: such a "pulse" has been called the "pulse of worn-out arteries." Whenever there is widely-spread atheromatous change, tortuosity of the arteries is common, and when this is found to be pronounced in comparatively young people it suggests long-standing high blood pressure and a tendency to premature degeneration of the arterial system.

Both radial arteries—right and left—should always be examined, and any difference between them noted. Such difference may be due only to

FIG. 25. Unilateral change in pulse in a case of intrathoracic aneurysm. This is an extreme example: a sloping upstroke and blunting of the different waves characterise the earlier stages.

anatomical peculiarity in the arteries, or again, it may be due to local disease of the vessel wall at the origin of the subclavian artery, and the carotid of the same side may be diminished in a similar manner. Aneurysm is always to be suspected when a marked difference between the right and left pulses is met with; but it by no means follows that such exists. Aneurysm does not affect the radial pulse invariably in the same manner. The interposition of an elastic sac simply is far from being the only mechanism of inter-

ference with the radial pulse by aneurysm: sometimes the orifice of the subclavian in the aneurysmal sac is distorted to a mere slit, or there is clot overlying it, or the sac of an aneurysm may compress the subclavian from the outside. A difference between the radials that is often difficult to explain is met with in aortic obstruction and chronic mediastinitis (*vide* Fig. 79.) A radial "pulse" may suddenly become obliterated from an embolism.

In taking a sphygmographic tracing the degree of pressure chosen should always be that which gives the greatest movement of the lever—usually a low degree of pressure in pulses of low tension and a high degree in pulses of high tension. The actual measurement of the pressure recorded on the sphygmograph is unreliable, although it is well to note it.

For purposes of description the following terms are in use in sphygmography: (1) An imaginary line drawn through the inferior extremities of the upstrokes is called *the respiratory line.* (2) The *upstroke* reaches from it to the apex of the curve in the great majority of cases. In the anacrotic pulse, as will be seen later, however, the top of the curve does not represent the top of the upstroke. (3) The peak at the top of the curve or first wave is called the *percussion wave.* In the

anacrotic pulse, however, what may be regarded as the representative of the percussion wave does not form the apex of the curve. (4) The wave following the percussion wave is the *tidal wave.* Its degree of development is of chief importance in estimating the tension of the pulse. In high-tension pulses it is well developed and especially *sustained* or prolonged. On the other hand, in low-tension pulses the tidal wave is ill-developed and may not be discernible. In rare cases of aortic stenosis this wave undergoes peculiar de-development so as to become perceptible to the finger (Fig. 30), so-called bisferiens pulse which may be a unilateral phenomenon. (5) Next comes the *dicrotic wave.* It is best developed in low-tension pulses, and it may become so pronounced as to be perceptible to the finger. Thus, when a pulse gives a double stroke to the finger there are two possibilities to be thought of: (*a*) that the second stroke represents the dicrotic wave, or (*b*) that it represents the tidal wave. The first is a fairly common phenomenon, especially when fever is present; while the second is so rare an explanation of "double-stroke" as always to be most improbable, moreover its cause can easily be discovered by physical examination of the heart.

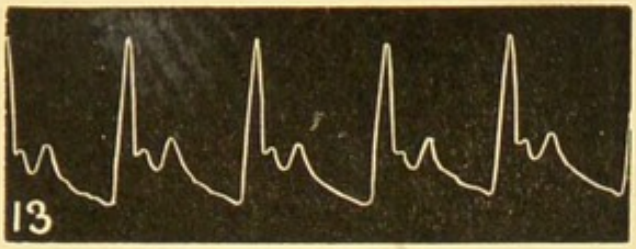


FIG. 26. **Low tension pulse tracing**: large percussion wave, very small tidal wave and large dicrotic wave approaching respiratory line.

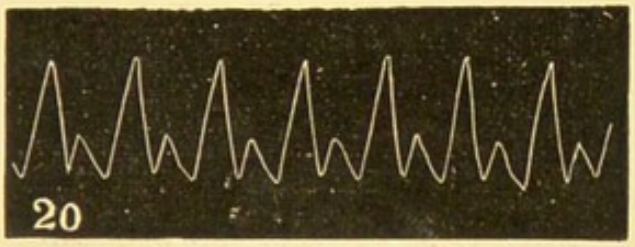


FIG. 27. **Fully dicrotic pulse tracing**: dicrotic notch reaches respiratory line. No tidal wave.

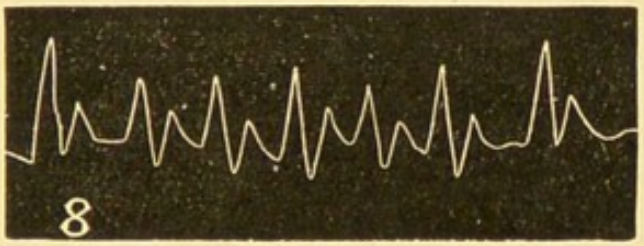


FIG. 28. **Hyper dicrotic pulse tracing**: the aortic notch falls below the respiratory line. No tidal wave.

THE PULSE IN AORTIC DISEASE.

AORTIC OBSTRUCTION.

FIG. 29. **Anacrotic pulse tracing** from case of aortic stenosis: sloping upstroke and the tidal wave forming top of the curve. The dicrotic wave is better marked than in many curves of the kind.

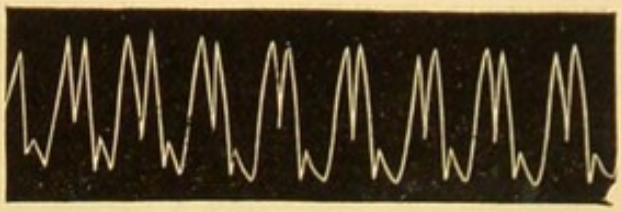


FIG. 30. **Bisferiens pulse tracing** from case of aortic stenosis. The 2nd stroke is formed by a peculiarly modified tidal wave.

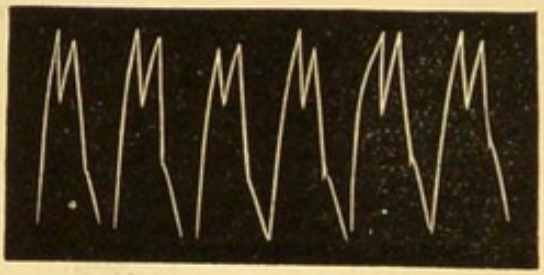


FIG. 31. **Bisferiens pulse tracing** from case of same lesion. A remarkable feature of the bisferiens pulse is that it may be developed on one side only : generally the left.

THE PULSE IN AORTIC DISEASE.
AORTIC INCOMPETENCE.

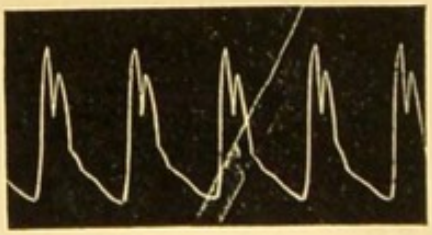


FIG. 32.

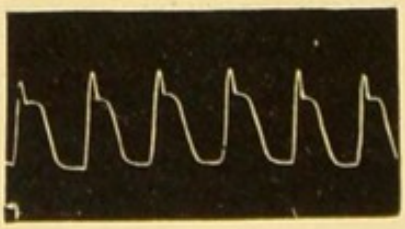


FIG. 33.

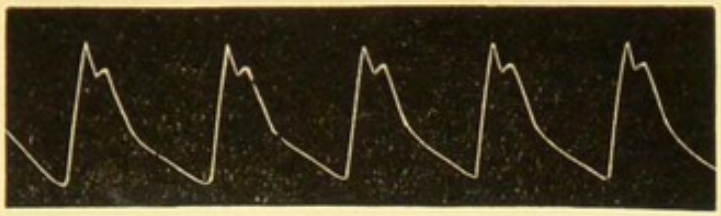


FIG 34.

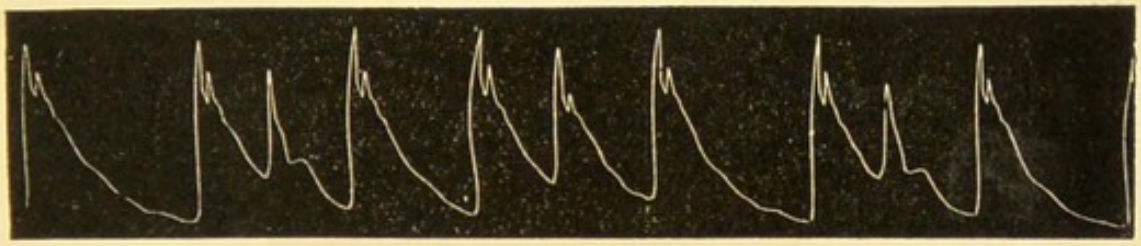


FIG. 35. Tracings from the pulse in cases of aortic incompetence. The percussion wave is sharp and well developed, while the dicrotic wave is deficient. Tracing (Fig. 35) shows irregularity.

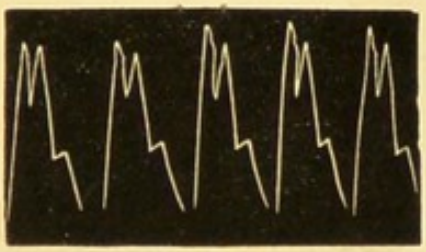


FIG. 36. Tracing from pulse in case of aortic incompetence without stenosis. It would be called by some a "pulsus bisferiens," which it resembles.

FIG. 37. Tracing from pulse in case of aortic dilatation with slight incompetence.

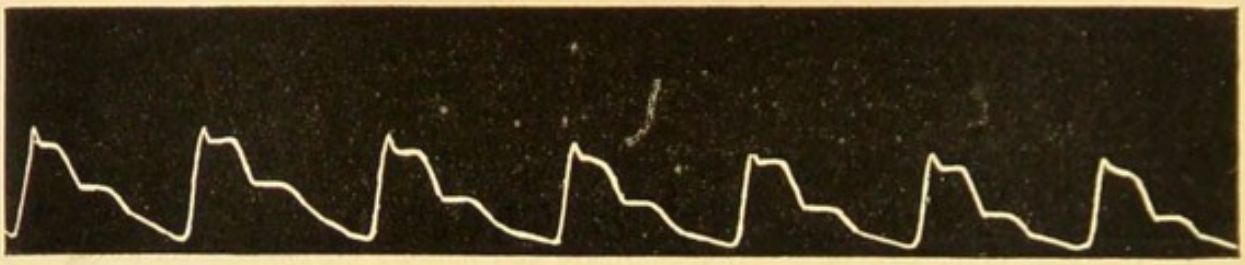
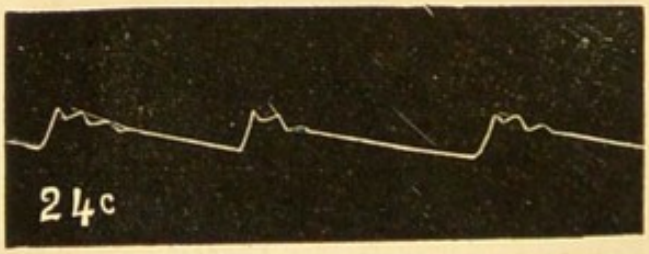


FIG. 38. Tracing from case of aortic dilatation : no incompetence.

THE PULSE IN CASES OF MITRAL STENOSIS.

FIG. 39.



FIGS. 40. High tension regular pulse commonly met with in cases of mitral stenosis (1st stage pulse).

FIG. 41. Irregular pulse in mitral stenosis (2nd stage pulse).

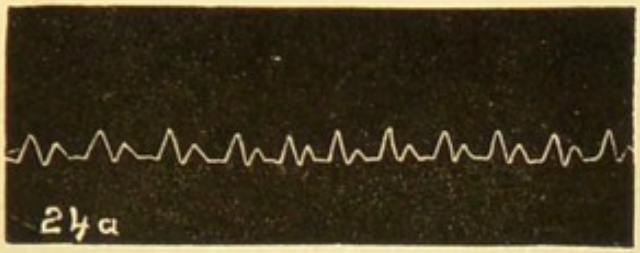


FIG. 42. Regular and low tension pulse in mitral stenosis (3rd stage pulse). The three last tracings were taken from the same patient while under treatment in the Manchester Royal Infirmary and indicate his convalescence (a.b.c.)

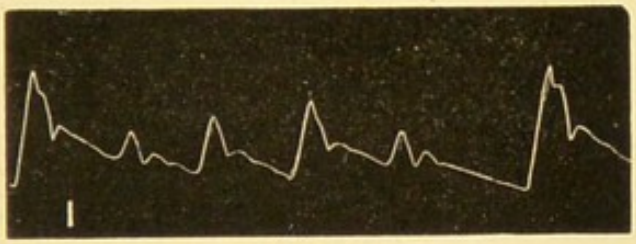


FIG. 43.

FIG. 44. Represent the irregular (2nd stage) pulse of mitral stenosis.

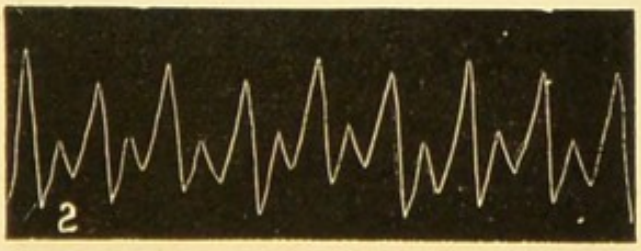


FIG. 45. Hyper-dicrotic pulse-tracing from patient with mitral stenosis, who was at the time feverish from rheumatism, T. 101°. Presystolic murmur was present when tracing was taken.

Pulse in Cases of Mitral Incompetence.

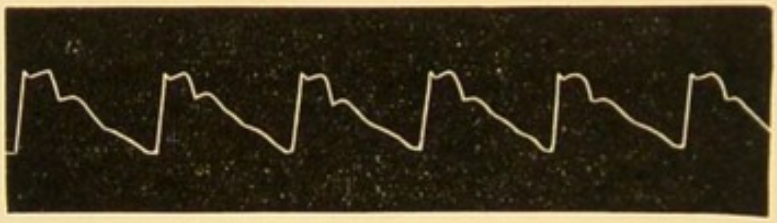


Fig. 46. High tension pulse with mitral incompetence. Case of Bright's Disease. No disease of valves.

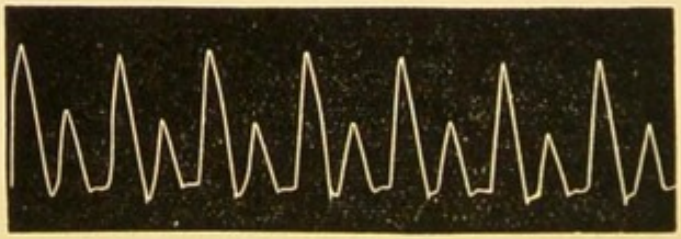


Fig. 47. Low tension pulse in case of septic emdocarditis with mitral incompetence.

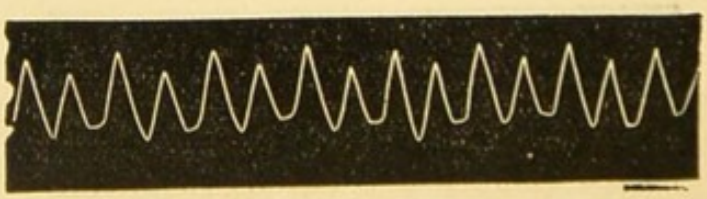


Fig. 48. From same case later: dicrotic wave nearly as high as percussion wave. No tidal wave. Mitral incompetence.

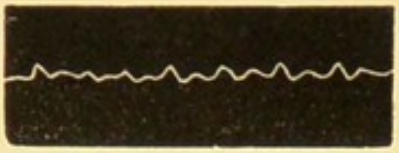


FIG. 49.

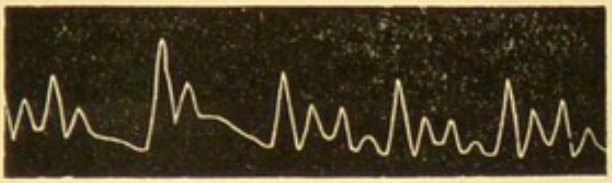


FIG. 50.

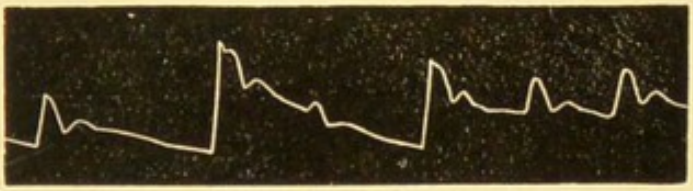


FIG. 51. From case of alcoholic muscle-failure of heart. Gradual recovery of tension during convalescence. Mitral incompetence, but no disease of valves.

(These six tracings show that there is no characteristic pulse of mitral incompetence).

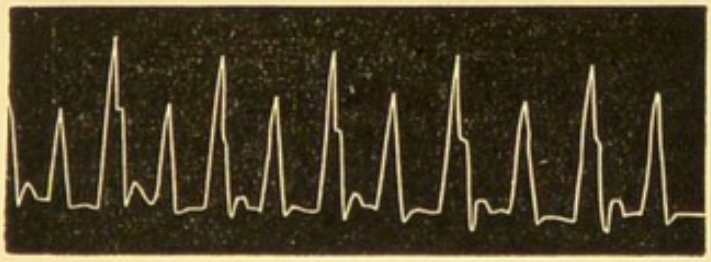


FIG. 52. Alternating pulse: such pulses are usually frequent, and the alternation is persistent over long periods. (Cardiac muscle failure.)

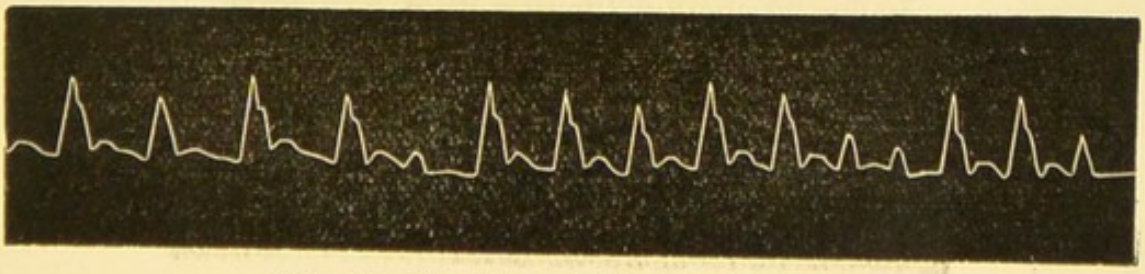


FIG. 53. Irregular pulse in case of pericarditis.

Pulse in Cardiac Muscle-Failure.

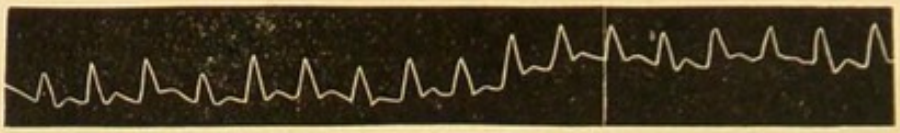


Fig. 54.

Fig. 55.

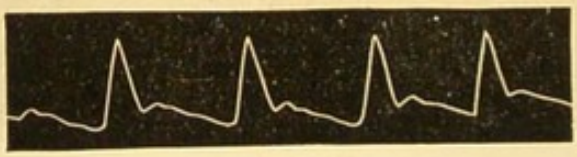


Fig. 56.

Fig. 57.

Fig. 58.

Fig. 59.

Fig. 60.

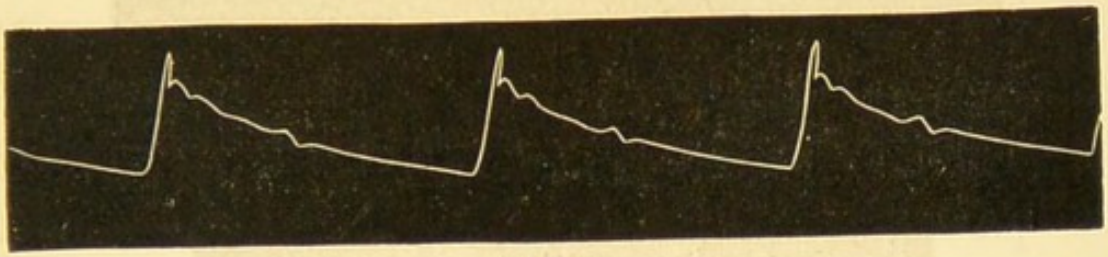


FIG. 61.

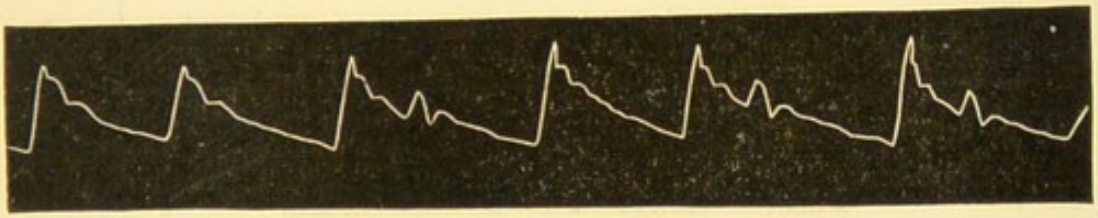


FIG. 62.

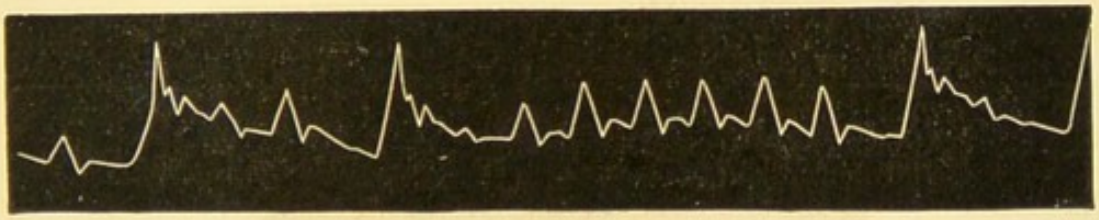


FIG. 63. Show changes in pulse during the course of the cardiac muscle-failure of chronic Bright's Disease ("Granular Kidney" p. m.). Patient was nearly nine years under observation from time to time. When the patient came under observation he had no mitral incompetence, although his arterial tension was then at its lowest (FIG. 54). FIGS. 55. to 60. show the gradual restoration of pulse tension during the patient's first stay in Hospital. The remaining tracings (Figs. 61 to 63.) show the pulse assuming respectively so-called "bigeminal" character, irregularity from "premature systoles," and irregularity of a type that is common in cases of mitral stenosis as well as in cases of cardiac "muscle-failure." In the later stages of his illness mitral incompetence was usually present when the patient came under treatment, but it would soon cease. Only towards the end did it become persistent.

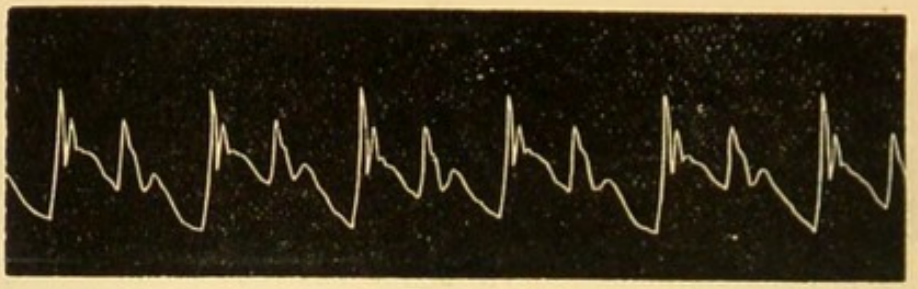


FIG. 64.

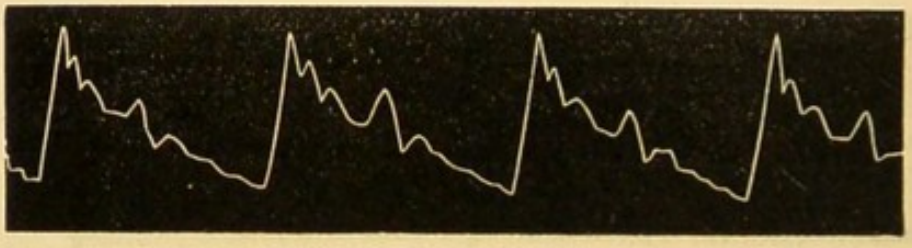


FIG. 65. So-called "bigeminal pulse." Probably there is the regular occurrence of a premature systole after each normal systole, so that the succeeding normal systole does not occur, the heart-muscle not being in a state to respond to the normal stimulus owing to the premature systole having exhausted it. Before the next normal stimulus arrives, however, it has recovered, and a normal contraction results. FIG. 64. represents the bigeminal pulse occurring in a case of aortic incompetence. FIG. 65 was taken from an apparently healthy man (æt. 40), who was subject to the temporary occurrence of such a pulse.

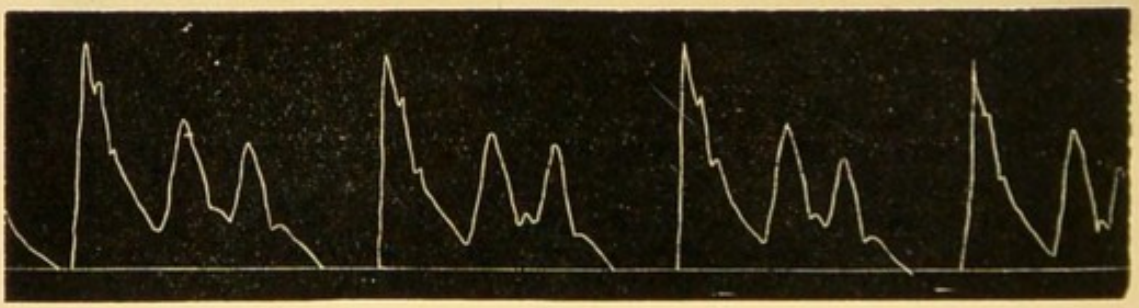


FIG. 66. The so-called "trigeminal" pulse, which is rare.

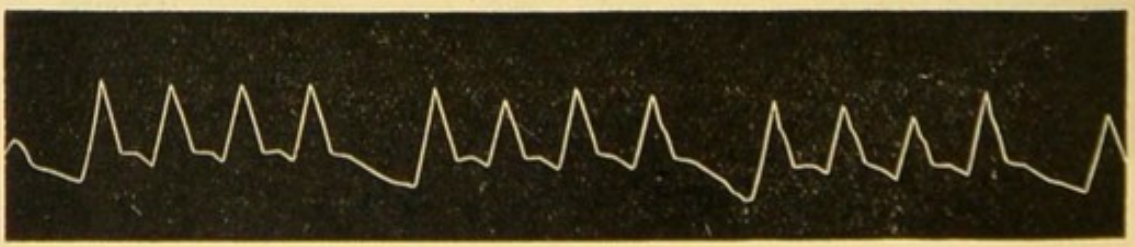


FIG. 67. Groups of four beats separated by intermissions.

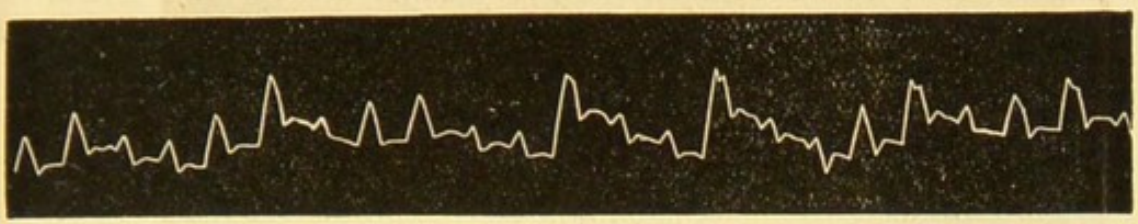


FIG. 68.

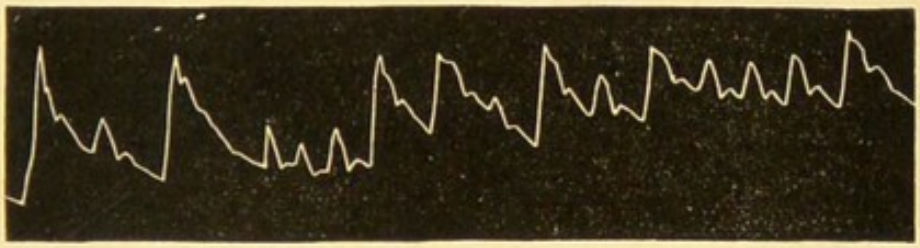


FIG. 69.

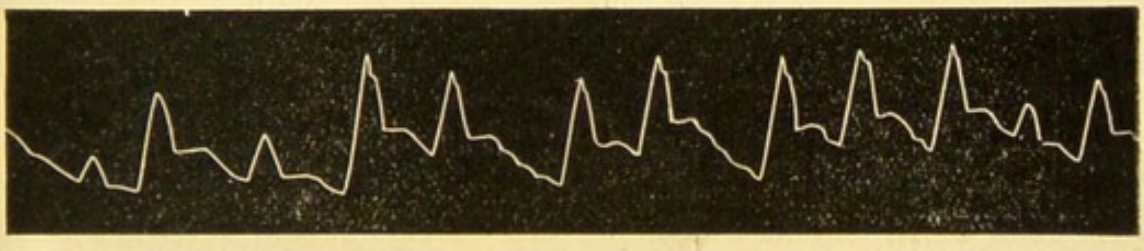


FIG. 70. Irregularity of the pulse. Fig. 68. was taken from the pulse of a gouty patient subject to attacks of irregularity. Usually his pulse was regular and of good tension. Fig. 70 was taken from an *apparently* healthy and active individual whose pulse was always irregular while under observation.

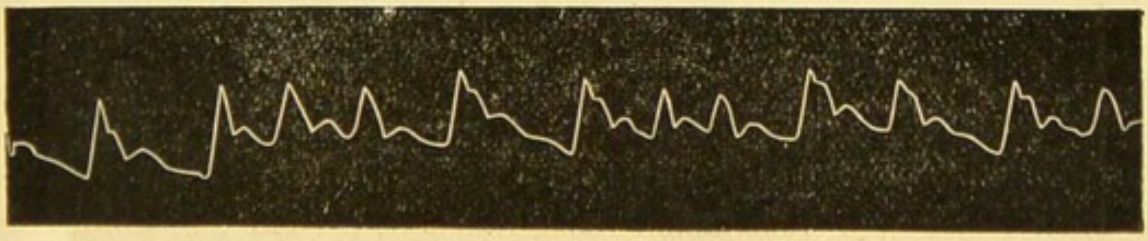


FIG. 71. Another example of irregular pulse in an *apparently* healthy individual.

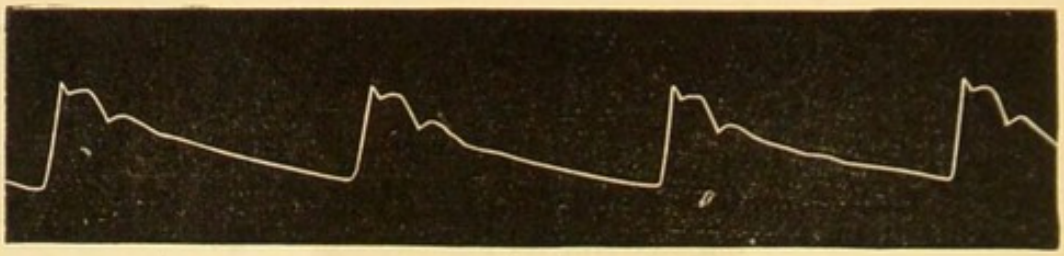


FIG. 72. Pulse in condition known as Bradycardia.

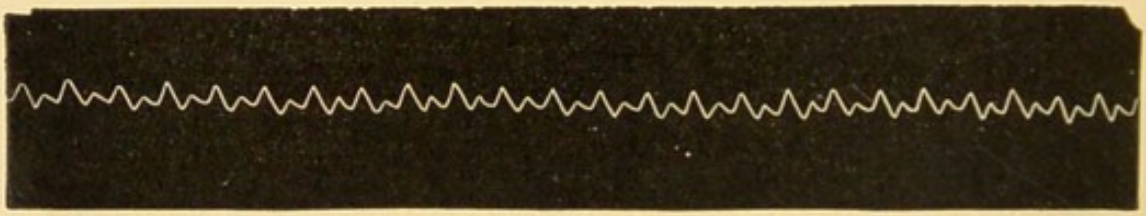


FIG. 73. Pulse in condition known as tachycardia when a frequency of 200 per minute and more may be reached.

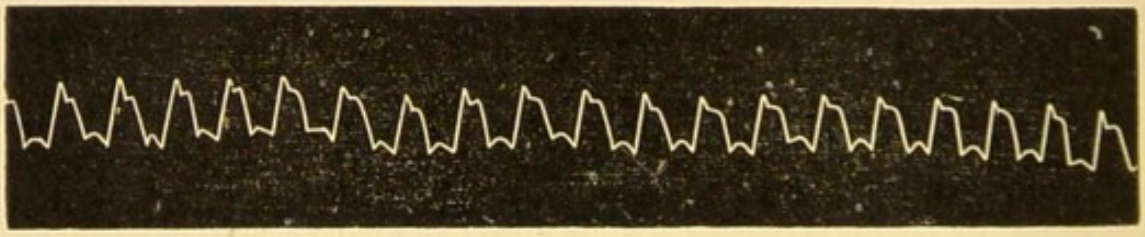


FIG. 74. Pulse in a case of Graves' Disease. Occasionally such a pulse becomes irregular.

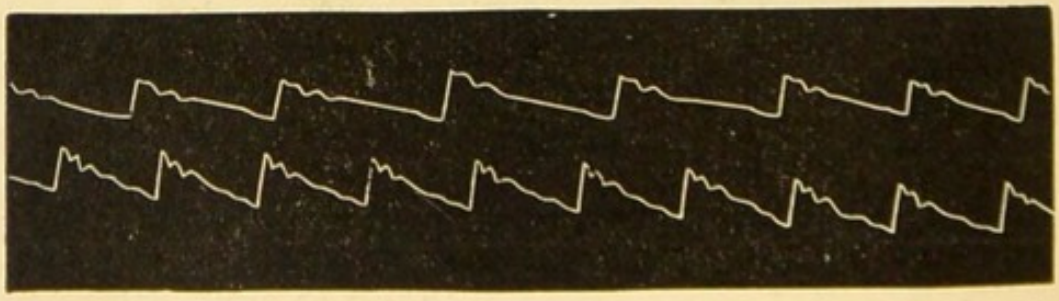


FIG. 75. The pulse in Cheyne-Stokes Respiration, the upper tracing having been taken during the dyspnœal period and the lower during the apnœal.

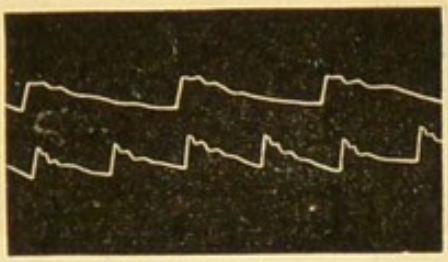


FIG. 76. Same as last tracing.

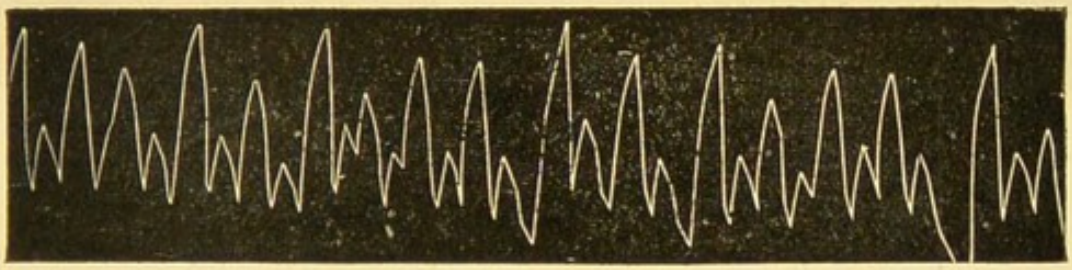


FIG. 77. From a case of great enlargement of the heart with valve incompetence occurring in a young man. The peculiarity of the tracing is that the upstrokes often commence before the completion of the dicrotic wave.

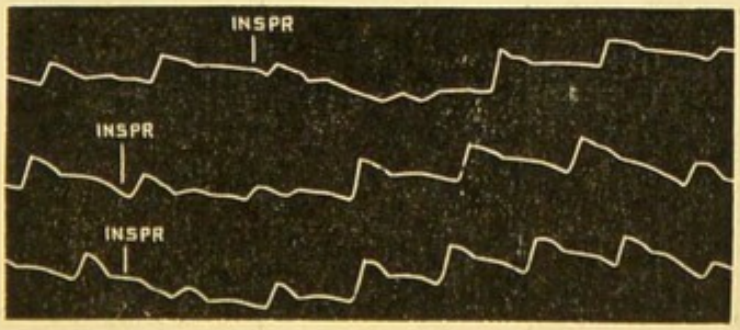


FIG. 78. Pulse in mediastinitis (Dr. Harris).

FIG. 79. From paper by Dr. Harris on "Pulsus Paradoxus" (*Lancet*, 22nd April, 1899), in which he called attention to the fact that this type of pulse may be unilateral.

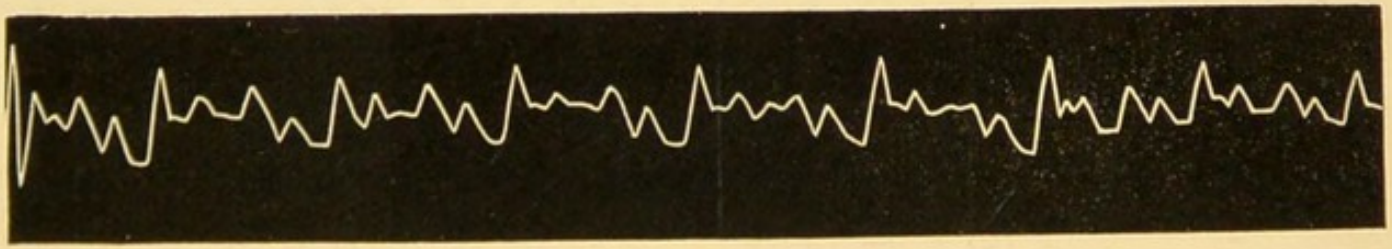


FIG. 80. Pulse in mediastinitis: patient by request breathing deeply and frequently.

ANEURYSM.

The clinical features of aneurysm are of an entirely different kind from those of heart disease. Here only such aneurysms as are combined with heart disease or modify the sounds of the heart, or produce symptoms and signs that may be confused with those of heart disease, will be considered; aneurysms of the thoracic aorta; though certain aneurysms of the abdominal aorta that give rise to epigastric pulsation, will require passing reference. It would be out of place here to enter into a discussion as to the different kinds of aneurysms classified according to their shape, the constitution of their walls, and so forth: lateral saccular aneurysms, fusiform aneurysms, "mixed," "true," and "false" aneurysms, etc. These are matters of pathological rather than clinical interest. The so-called dissecting aneurysm belongs to a different category, and will be specially referred to.

An aneurysm is a *tumour* to all intents and purposes, and its characteristic symptoms are due chiefly to the pressure it exerts upon contiguous structures. Intra-thoracic aneurysm consists, it may

be said, of cystic tumour, the cavity of which is freely in communication with the thoracic aorta, while its internal surface and its whole wall is exposed to the varying pressure of the blood, as it swings from maximum to minimum in the cardiac systole and diastole. By virtue of hydraulic law, the pressure within an aneurysm, in accordance with its size, becomes enormous, while the very variation of the blood pressure within it may be imagined to give it something of the character of a boring or attrition machine: "terebrating." It thus wears away the most resisting and hardest tissue—bone—whether it be sternum, rib, or vertebra.

The *symptoms* of aneurysm, then, are essentially *those that result from pressure upon adjoining parts;* thus they differ altogether from the symptoms of heart disease which are essentially those of failure of the circulation eventuating in general venous stasis. Sir William Broadbent has divided aneurysms of the arch into the two varieties—(1) the aneurysm of physical signs, and (2) the aneurysm of symptoms: "from the predominance of physical signs and symptoms respectively, the former term applying to aneurysms of the ascending aorta and first part of the arch, the latter to aneurysm of the transverse and descending portions of the arch" (p. 374, "Heart Disease," 3rd ed.). While any-

thing worthy of the name of a discussion of the pathology of aneurysm is eschewed, it may be permitted to recall the fact that the essential condition for the formation of an aneurysm is *localized loss of resistance and diminution of elasticity on the part of the aortic wall:* this granted, the blood pressure within the vessel effects the rest. The so-called "dissecting" aneurysm, in which blood is forced between the coats of the vessel, it may be for a very considerable distance, hardly calls for consideration from the clinical standpoint taken up. It does not form a tumour in the ordinary sense, and does not exert pressure on adjoining structures, at least to the production of symptoms. So-called aneurysm of the heart (p. 65), however, has many pathological affinities with the aneurysm under consideration, inasmuch as in it *localized loss of contractility by the ventricular wall* plays the part of loss of resistance and elasticity in the case of the aorta; but clinically it is revealed only by the ordinary indications of heart failure. *Atheromatous and syphilitic aortitis* accomplishes localized loss of elasticity in most cases of thoracic aneurysm. Occasionally there seems to be a congenital local deficiency of structure in the wall, accounting for the development of aneurysm in otherwise healthy young subjects.

Micro-organisms, capable of producing ulcerative endocarditis of the valves of the heart, in a case of the writer's, effected a small patch of loss of elasticity and resistance of the aortic wall with the formation of a small aneurysm just above the aortic valves, though the aneurysm was too small to give rise to any pressure symptoms, and similar cases have been recorded by others. While atheromatous or syphilitic endaortitis lays the foundation of aneurysm in most cases, the effect of *local injury* cannot be entirely ignored. A blow on the upper part of the chest has been experienced not very rarely in cases of aneurysm of the arch. A young man came under the writer's care suffering from abdominal aneurysm, who attributed his disease to a severe blow received on the abdomen, while arterial disease was found post-mortem limited to the abdominal aorta about the site of the aneurysm. Given a weakened portion of the aortic wall, the influence of "strain," as in the making of great and repeated efforts may readily be admitted among the factors at work in the formation of aneurysm. It is no uncommon thing for a slight local dilatation of the aorta to be noticed at a post-mortem examination of a subject dead from other disease and without the formation of any aneurysm, but more important still is the association of actual aneurysmal tumour with such slight

local weakenings and consequent bulgings of the aortic wall. In the ordinary type of aneurysm formation it is the external coat that resists longest, and in the slight bulgings it is the middle coat which suffers destruction chiefly.

Dissecting aneurysm, though of very great pathological interest, is of comparatively little clinical importance, inasmuch as it is seldom long survived, and does not result in the formation of a tumour capable of exerting pressure on adjacent structures. A rent is made in the inner coat and the blood tears up the middle coat and forces its way between the internal and external coats for a variable distance, in some cases actually finding its way again into the proper channel of the vessel. The rent has measured $\frac{3}{4}$ of an inch and 2 ins. in recorded cases, and is often situated near the valves. The degree of violence may not have been extreme. For instance, in one case the lesion seems to have occurred under the influence of the strain implied in pulling the reins of a runaway horse and in retaining the saddle during a jump from a field down into a road at several feet lower level. It was directly after the jump that pain in the chest and a feeling of faintness were experienced, so that presumably the accident occurred at the time of this jump and during the muscular effort necessitated by the drop into the road (*Allbutt's System*,

vol. vi., p. 357). It is curious to note that another case is recorded in which a rent $2\frac{1}{2}$ ins. long resulted from a young, powerfully built and presumably healthy, man leaping "from a wall about 13 feet high into a paved courtyard, alighting on his feet."

THE SYMPTOMS OF ANEURYSM.

Pain. Among the symptoms of intra-thoracic aneurysm pain commonly plays a predominant part, though exceptionally a large aneurysm may be formed painlessly. Much depends on the direction of extension of the aneurysm. Pains complained of across the upper part of the chest, passing occasionally to the shoulders and neck and aggravated by exertion, should always suggest aneurysm as their possible cause. Different conditions, indeed, are at work in the production of pain by intra-thoracic aneurysm, among which the following at least can be identified:—

(1) True *referred pain* of the nature of Angina Pectoris. When the pain assumes the character of true angina the presumption should be that the orifices of the coronary arteries have been caught in the disease of the aortic lining membrane and narrowed. According to Dr. Henry Head, the arch of the aorta should be considered

developmentally and as composed of the following portions:—

(*a*) The part extending from the valves to the level of origin of the innominate artery and representing the bulbus arteriosus, the most headward division of the originally tubular heart.

(*b*) The transverse portion of the arch extending from the innominate artery to the point of entrance of the ductus arteriosus—this part representing the fourth left branchial vessel of the fœtus.

(*c*) The portion beyond the ductus arteriosus.

The ascending portion of the arch *refers* pain to the distribution of the third and fourth cervical and the first, second and third dorsal segments. It is in affections of this portion of the arch that true angina pectoris is so often produced, and in view of the coronary orifices being situated within it, involvement of these orifices is always to be suspected, although disease of this portion of the aorta may possibly occasion referred pain without interference with the coronary vessels. An important area of tenderness is that in

the so-called inferior laryngeal triangle, bounded above by a line drawn from the anterior border of the sterno-mastoid to the level of the crico-thyroid membrane. The posterior side of the triangle runs along the sterno-mastoid muscle to the sterno-clavicular joint, while the remaining third side is formed by the median line. The maximum point of tenderness is situated upon the anterior border of the sterno-mastoid a little below the level of the crico-thyroid membrane. This triangle is seemingly associated with affection of the *transverse portion of the arch*. The descending portion of the arch may refer pain to the distribution of the 5th, 6th and 7th intercostal nerves, which are also the seat of pain referred from the auricle under certain abnormal circumstances (p. 223).

There are two areas of referred pain associated with irritation of the 3rd and 4th cervical segments: the "sterno-mastoid" and "sterno-nuchal" areas (*vide* Fig. 93).

(2) Pain may be due to pressure upon nerves: such as is exemplified when an aneurysm of the descending aorta compresses intercostal nerves. In

this case anæsthesia in the distribution of the affected nerve may finally be produced.

(3) There is the pain resulting from direct pressure of the aneurysm on opposing structures. This type of pain is commonly present, when an aneurysm compresses the bones of the spinal column. It is characterized by its constancy, though it, too, is subject to paroxysmal exacerbation.

(4) Pain, as already stated, has been recorded at the outset of cases of rent in the internal coat of the aorta, and consequent formation of a dissecting aneurysm.

Symptoms due to Pressure on the Air Passages and Lungs or on Laryngeal Nerves. Amongst these, one of the earliest is often a peculiar, dry, noisy ringing *cough*, the characters of which have to be heard to be appreciated. Such a cough is rightly, in most cases, attributed to irritation of the recurrent laryngeal nerve. A similar cough may indicate the early stages of laryngitis. A cough of opposite characters, called by Dr. Wyllie the "Bovine Cough," is occasionally met with in aneurysm, but in the writer's experience such a cough is much more commonly observed in structural disease of the larynx with destruction of the cords or bilateral palsy of the adductors of the cords from other

causes. In it, there is an absence of the "clang" indicative of the forcible closure of the cords, owing to their imperfect approximation.

The laryngeal palsy in thoracic aneurysm, in nearly all cases, is unilateral—left-sided in the great majority of cases, and the right cord can swing over to effect sufficient closure for the production of the sharply explosive element which indicates "with the utmost precision to the ear the first act of a strictly normal cough—the forcible closure of the glottis."

The second type of cough is in essence, on the other hand, "a cough with imperfectly or only partially closed glottis."

Voice. The voice is often preserved in a surprising manner in the presence of paralysis of the left vocal cord, the right cord swinging over to meet its fellow. Still more is this the case, when there is bilateral paralysis or paresis of the abductors of the cords, and it is important in this relation to remember "that in all progressive or organic lesions of the nerve centres or trunks of the motor laryngeal nerves, the abductors of the vocal cords succumb much earlier than the adductors."

There seems to be a considerable amount of evidence in favour of Sir George Johnson's theory that a long continued irritation of the *trunk* of one vagus may, through its afferent fibres, so dis-

turb the nerve centres of the two vagi as to cause either bilateral spasm or bilateral palsy of the laryngeal muscles. As regards spasm, there can be no doubt as to its correctness; as regards paralysis it must be assumed that an inhibitory influence can be exerted on the bilateral centre, and in this relation the law just referred to concerning the earlier failure of the abduction of the vocal cords has to be remembered. Paralysis of the abductors of the cords is indeed a well recognised clinical entity, quite apart from aneurysm, its clinical features being essentially *more or less inspiratory stridor with preservation of the voice*. The writer has observed the condition in locomotor ataxy, and repeatedly as an apparently idiopathic affection.

More than one aneurysm being occasionally met with in the same patient, a subclavian aneurysm on the right side may paralyse the right vocal cord, while an aortic aneurysm paralyses the left.

Direct pressure on air-passages. No less important interference with the air-passages by aneurysm is accomplished by *direct pressure of the aneurysmal sac on the trachea or bronchi*—generally the left bronchus. When a bronchus is compressed, the lung supplied by it is apt to undergo profound changes: it may become collapsed with or without pleural effusion,

or it may become consolidated with exudation often seemingly purulent—at any rate grey—filling its vesicles. Pressure on the trachea produces symptoms that closely resemble those of laryngeal obstruction, and are liable to exacerbation, too, from time to time. The stridor is usually expiratory as well as inspiratory, though more pronounced during inspiration. The voice is essentially preserved, though it is often weak. When a bronchus is compressed, there is interference with the expansion of the corresponding lung and side of the chest—usually the left.

It is most important to note that as the amount of air in the lung tissue is diminished, the resonance usually acquires tympanitic "quality," while the "pitch" rises and the "mass of tone" diminishes, the resonance remaining, however, clear until "complete emptiness" and "dulness" coincide. Under the circumstances—practical collapse of a lung owing to the pressure of an aneurysm on its main bronchus—it will rarely happen that effusion into the pleura is entirely absent, while it is common in the course of an aneurysm, for a large effusion to occur, alone capable of causing collapse of the lung. In Sir William Gairdner's "Clinical Medicine," (Edinburgh, 1862, pages 482 and 483) there will be found illustrations of the lung of a patient with aneurysm, in whom hæmop-

tysis had occurred, though in no great amount. "The left bronchus had its posterior wall deficient for about an inch, and its calibre was almost completely occupied at this point by a firm, grey coagulum of blood, which projected out of the aneurysmal sac. The left lung was almost completely condensed." Sir William later remarked, "Yet this incomplete collapse of the left lung was not accompanied by an empty state of the air vessels. . . . On the contrary, many of the air cells were filled with blood and others with a soft, greyish exudation, being a mixture of fibrine in various proportions with blood and pus."

The writer described in the *Lancet* (April 1893), a case in which a condition of grey hepatization of the lower lobe was combined with later-occurring pleuritic effusion, resulting in collapse of the upper lobe, the original pathological condition being obstruction of the bronchus supplying the lower lobe, by a tumour. There was respiratory "silence" over the lower lobe and bronchial breath-sound over the upper lobe after the occurrence of the effusion. Such a condition could hardly result from aneurysm; but, from an auscultatory point of view, the case is of singular interest as practically proving that bronchial breath-sound is essentially the glottic breath-sound conducted downwards through patent

bronchi; there was silence over the hepatized lower lobe because its main bronchus was obstructed, while the bronchi of the upper lobe remained in free communication with the glottis. When in a case of intra-thoracic aneurysm the whole of the left side is found dull on percussion and "silent," or almost so, on auscultation it must be remembered that the mechanism of this result may be complex —simple collapse, exudation into the vesicles, and effusion into the pleura all possibly contributing to it.

Hæmorrhage. The escape of blood from the sac forms a very important accident of intra-thoracic aneurysm. In many cases the escape takes place into a serous sac and is retained—the pericardium and pleura for instance—or it may occur into a mucous canal, as the trachea, bronchus, or œsophagus and be ejected externally, or the skin itself may give issue to the contents of the sac. In this last case, discolouration of the skin from blood-staining and frequently an inflammatory condition with much tenderness at the impending site of rupture, give warning of the approaching danger.

The importance of the deposition of fibrine within the sac of aneurysms is very great. "The obstacle to a very sudden rupture through a considerable opening is such, that when rupture ultimately takes place toward a mucous

membrane or a cutaneous surface, not only is the opening relatively small, but the flow of blood, impeded by coagula newly formed or of long standing, is also much less profuse than might have been expected, and may even be a mere oozing, or successions of oozings filtered through these obstructions" (Gairdner, *Allbutt's System*, vol. vi., p. 381). On the other hand death is not rarely the result of an appalling hæmorrhage from the œsophagus or trachea. In the former case the patient may die, blanched and convulsed, in a few minutes. When rupture takes place into the pleura the same symptoms may appear, though there is no external hæmorrhage. When, exceptionally, life is prolonged for a few hours after profuse external hæmorrhage, delirium may be marked, apparently the result of the suddenly induced cerebral anæmia. Cutaneous hæmorrhage is perhaps the rarest event. The writer has seen what may be termed a "fountain of blood" rise from the aneurysmal tumour of a recumbent patient, who survived the rupture many days. Dr. Walshe recorded a case in which the patient "must have lived for nearly two months with a gradually increasing extent of his chest-wall and aorta replaced by lint." With regard to hæmoptysis it may occur in all degrees from a "blood-streaked frothy bronchitic sputum" to "a fatal discharge of large quantities

of pure blood." Sir William T. Gairdner has recorded a case in which the patient survived a sudden and large gush of pure blood "renewed an hour or two later, four years and eight months." Tubercle of the lungs is exceptionally associated with intra-thoracic aneurysm of the ordinary type. [The small aneurysms of pulmonary branches in phthisical cavities are not considered here: they belong to an entirely different category and would seem to be formed by defect of the outer coat, which in ordinary aortic aneurysm is longest preserved.]

In one case of intra-thoracic aneurysm seen by the writer the tubercular disease was advanced, and large excavations had been formed. In this relation it must be borne in mind that an inflammatory condition with breaking down of tissue may be associated with the direct encroachment of an aneurysm upon the lung, which condition is non-tubercular. In the tubercular complication, as indeed in that last mentioned, it is conceivable that there may be hæmorrhage, which has its source in the secondary lung-lesions instead of in the aneurysmal sac. Before an aneurysm perforates the trachea or a bronchus an oozing of blood may occur for a very considerable period as already mentioned. In conclusion it may be well to remind the reader of Walshe's statement:—"There

is no conceivable position into which fatal rupture has not occurred."

Affection of Sympathetic Nerves. An aneurysm involving the upper and back part of the arch may compress sympathetic fibres and, paralysing these, produce contraction of the pupil and diminution of the palpebral fissure on the left side. Irritation of the same fibres results in the opposite condition: viz., dilatation of the pupil and enlargement of the palpebral fissure.

Dysphagia, presumably due to the direct pressure of the aneurysmal sac on the gullet, occurs occasionally in an aneurysm of the descending aorta or posterior termination of the arch. Not very rarely, however, when *post-mortem* the gullet seems to have been compromised by the sac, no dysphagia has been experienced or at least complained of by the patient. Moreover, when this symptom is experienced, it is often only transiently so, and patients afterwards omit to mention that it has occurred.

Enlargement of the superficial veins of the thorax, neck, and upper extremities, is believed to be much more common in the cases of solid tumour than in those of aneurysm, but the latter disease is quite as capable of producing the condition as the former. In a patient of the writer's, aged only 22 and suffering from aneurysm of the arch, the front

of the chest was covered with greatly distended veins. The face, neck, arms and upper thorax are apt to become œdematous owing to the pressure of an aneurysm on the superior vena cava. Pressure of an aneurysm of the abdominal aorta on the inferior vena cava may cause interference with the return of blood through that vessel, and so enlarge the superficial abdominal veins and produce œdema of the lower extremities. Albuminuria may result from the renal vein being implicated.

PHYSICAL SIGNS.

Inspection.

Information of the greatest value is to be obtained from a careful Inspection of the chest in cases of intra-thoracic aneurysm. .There may be an obvious local projection from the surface of the chest, and such projection or *tumour* is seen to *pulsate.* It often happens, however, that there is no tumour present, although careful inspection, especially by looking along the surface of the chest in the appropriate area and in different directions, reveals more or less localized *visible pulsation apart from the pulsation of the heart itself.* The site of such tumour or mere impulse varies, of course, with the position of the aneurysm. A very common site is to the right of the upper sternum with a maximum of projection or

impulse in the second intercostal space. A rare site is lower down to the right of the sternum with a maximum in fact under the right nipple, the condition being a pear-shaped aneurysm coming off at a right angle from the ascending portion of the arch. The sternum may be corroded and pulsation be visible over it, or the site of impulse may be to the left of that bone above the third cartilage. In the case of aneurysm of the descending aorta a pulsating area may be visible to the left of the spine behind—rarely to the right—the ribs obviously having suffered more or less absorption.

Tracheal tugging may often be *seen* in aneurysm of the arch; when it is so, the appearance may be regarded as evidence of aneurysm, of the greatest value. Practically, it is pathognomonic, although the same cannot be said of the like palpation sign, minor degrees of downward systolic impulse being *felt* in cases of simple dilatation of the arch, and possibly even without there being any great degree of that condition, under certain circumstances. When an aneurysmal tumour is formed, the colour of the skin is usually normal, but rarely, as already noted, there is discolouration, blood-staining or inflammatory redness, as in a remarkable case recorded by Dr. Ramsay, in which the patient held a bowl to the external rupture of an aneurysm believing it to be, as he said, a "bloody boil."

In considering an aneurysmal impulse, it is always of importance to identify the impulse of the heart itself, so that any aneurysmal impulse there may be, can be at once differentiated. In cases of displaced heart—from pleural effusion, retracted lung, etc.—*the impulse of the heart is absent from its normal site.* It is a vexed question whether the heart in aneurysm undergoes enlargement, apart from induced incompetence of the aortic valves. In the majority of instances, when the heart is enlarged, the aortic valves leak, and the left ventricle takes the chief share in the enlargement. Without aortic incompetence, and even in the case of large aneurysms, the heart may remain of normal size: this much is certain. On the other hand, however, the writer is not prepared to affirm that the heart is always found of normal size in cases of aneurysm in which the valves remain competent, and in which no independent cause of enlargement is obvious. A large aneurysm may, again, displace the heart—generally downwards. The condition in which aneurysmal and cardiac impulses are most readily—indeed, unavoidably—confused, is that in which an aneurysm of the descending thoracic aorta is situated *behind the heart.* In this case, as has already been noted (p. 72), a remarkable precordial impulse results: the whole heart is

seemingly thrust forwards in each systole, and the apex beat, right ventricular impulse, and right auricular pulsation cannot be identified. The greatest impulse, indeed, seems to be over the body of the ventricles. It is important to remember that a similar impulse is met with, when there is a *solid* tumour, in place of an aneurysm, situated between the heart and the spine. This would make it seem that the essential impulse, in the case of aneurysm situated behind the heart is that of the heart itself, rather than that of the aneurysm. In diastole the walls of the ventricles are relaxed, and the heart may be regarded as a thick walled flaccid sac with compartments. In systole the heart becomes a rigid mass of muscle, and, when systole is complete, nothing else, the ventricles being empty up to the supra-papillary space. Post mortem it is not a very rare thing to find the ventricles of the heart in complete systole, and if such a heart be sliced transversely the cavities of the ventricles are found to be obliterated up to the supra-papillary space, and thus a rigid mass of muscle would be interposed at the end of systole between an aneurysm of the descending aorta or solid mediastinal tumour and the anterior chest wall. That the two impulses are blended in the case of aneurysm may be granted, but that they can be distinguished is denied. Obviously the two im-

pulses should not be synchronous, in view of the length of the blood channel between the heart and the aneurysm.

A small, deeply placed aneurysm may cause no visible impulse, though it may nevertheless cause pressure on important structures, for instance on the left recurrent nerve. On the other hand a large aneurysm may give so diffuse an impulse that "*local*" impulse escapes observation from the front, if care be not taken to look *along the surface of the chest* from the sides, downwards and upwards. The *expansile* character of the impulse becomes most pronounced when there is local projection of the sac, the ribs or sternum having given way before the pressure.

The rare condition, in which a collection of fluid in the pleura pulsates with the heart beat, has only to be mentioned. A solid lung may rarely produce a somewhat similar phenomenon.

Palpation.

By palpation the heaving *force* of an aneurysm may be appreciated, as well as the expansile character of the impulse, the latter especially when there is local projection. A thrill—generally systolic, sometimes systolic and diastolic, or almost continuous through systole and diastole—can some-

times be felt over aneurysms. Thrills are represented in auscultation by murmurs, as a rule, and certain questions with reference to diastolic thrill will be discussed under *auscultation*. The impulse of the heart itself should be carefully examined in all cases of aneurysm. Another impulse, as strong as and separate from that of the heart, is presumably the impulse of an aneurysm.

Tracheal Tugging is usually present in aneurysm involving the transverse portion of the arch. The cricoid cartilage should be gently held up with the forefinger and thumb placed on the sides of its lower edge, when the systolic tug downwards will be perceptible.

Aneurysm of the descending aorta may occasion impulse behind, to the left of the spine—possibly to the right of the same.

Percussion.

The dull area in the front of the chest due to aneurysm of the aortic arch naturally varies according to the size and position of the sac. The size of the dull area represents only a small portion of the anterior wall of the sac. "Hence," says Walshe, "practically an intra-thoracic aneurysmal sac is always larger than the results of percussion would indicate." This is obvious.

Making allowance for exceptional conditions, as for instance the aneurysm coming off with a narrow neck at right angles to the first part of the arch referred to p. 193, the dull area due to an aneurysm of the arch involves the upper sternal region, usually extending to the right or left of this in varying proportion, most commonly occupying the second right inter-costal space to some extent.

The whole area of dulness is apt to assume a somewhat *semi-lunar shape with the convexity downwards*. Moreover, the convex lower boundary may or may not be connected to the cardiac dulness by a neck of varying breadth.

This dull area is at once distinguished from a lung dulness by the possession of the following two features: (1) *It crosses the middle line*, and (2) *It fails to reach either costo-acromial angle*. Rarely the aneurysmal dulness is so huge that it runs into the dulness of the heart, the neck of union referred to being obliterated. It is more common for the two dull areas to be entirely separated. Light percussion should only be applied over aneurysms for obvious reasons. Moreover, by increasing the force of the percussion stroke, naturally the extent of the "dulling" influence of the aneurysm is increased, a certain depth of resonant lung being percussed through, as it were, in which case the

resonance, while remaining "clear," becomes "emptier" and "emptier" until the surface of the aneurysm comes into contact with the chest wall and resonance ceases—at least to a slight stroke incapable of giving rise to the phenomenon of "horizontal conduction" of resonance.

While it is very rare for aneurysm of the descending aorta to give rise to dulness to the left of the spine (still rarer to the right), secondary percussion changes over the left lung resulting from aneurysm of the arch are common: pleural effusions and pulmonary consolidations for instance. Moreover, the dulness so produced may be extensive and, indeed, involve the whole of one side of the chest. Such secondary conditions have been already mentioned and explained.

Auscultation.

It is important that the student should be early disabused of the idea that murmur-production is a necessary part of the evidence in favour of aneurysm attainable by physical examination. On the other hand, modifications of the physiological sounds of the heart may afford most valuable evidence just as they do in certain cases of cardiac muscle failure of the heart and mitral stenosis (p. 97). As regards aneurysm of the arch involving the first part, the most valuable modification of the physiological sounds is

accentuation of the aortic second sound. In aneurysm this reaches a degree far in excess of anything observed in simple dilatation of the aortic arch. The impression often conveyed to the mind by the extreme accentuation of the second sound in aneurysm is indeed that the valves are bearing a burden that threatens their integrity. It is important to remember that leakage of the valves is quite compatible with great accentuation of the second sound. In curious contrast with accentuation of the aortic second sound of aneurysm of the arch is practical absence of the first sound and murmur during systole. The observer is aware rather of a "jog" to his head than of an auditory impression. Often, however, there is distinct systolic murmur audible, while diastolic murmur is common enough. In the great majority of cases, diastolic murmur is associated with incompetence of the aortic valves. That a diastolic murmur cannot occur in the absence of incompetence of the aortic valves can hardly be affirmed: the facts of observation seem opposed to such an affirmation, but the question is one of little practical importance. The diagnosis ought never to depend on any single feature, but be based on all the features of the case collectively —symptoms and signs—considered in combination and in the light of the history of the case, and of a knowledge of pathology.

ANGINA PECTORIS.

The great *pain* of heart disease is the clinical entity known under the name of *angina pectoris*, which in the vast majority of cases is associated with obstructed coronary arteries, whether these be obstructed at their orifices or in their course. There is still, however, a small minority of cases in which an indistinguishable pain is experienced in the absence of coronary disease, though in the presence of distinct cardiac lesion, most commonly mitral stenosis There are many varieties of suffering experienced by the subjects of heart disease, moreover, that cannot rightly be called *pain. Of angina pectoris pain is the very essence.* "No one fact in a typical case of angina pectoris is necessarily other than subjective, with the exception of the awful terminal fact of sudden death," says Sir William Gairdner. *The disease,*—the clinical entity—*may be met with in cases in which physical examination altogether fails to discover evidence of disease of the heart.* Nay, the most perfect development of the disease occurs under these circumstances. On the

other hand, in many cases of aortic disease—atheromatous and syphilitic—angina pectoris is found in association with abundant physical evidence of incompetence of the aortic valves. The same process that directly or indirectly rendered the valves incompetent (pp. 46 & 47) has narrowed the coronary orifices.

It is idle to differentiate these latter cases from those that have been called "classical," and yield no evidence of disease on physical examination. Lastly, there is the small minority of cases in which angina pectoris is experienced by the young subjects of mitral stenosis, and in which the coronary arteries have been found normal. (*Lancet*, 21st Nov., 1903, p. 1431, and *Med. Chron.*, March and April, 1905.)

While we insist on *pain* as essential in angina pectoris, there must be admitted into our conception of the disease a suffering associated with, but over and above, the actual *pain*. Such suffering is most difficult of description and even comprehension. It is not a dyspnœa in the ordinary sense, though an element thereof can hardly be denied it. There is a feeling of oppression, a constriction of the chest, or the apparently opposite but really closely related sensation of the chest being expanded from within. "The sense of having a mountain on the chest;" "the front of the chest

seeming to be bulged out in a convex prominence which suddenly terminated at the lower end of the sternum in a sharp and deep depression towards the spine . . . a purely subjective phenomenon;" and "the feeling of the sternum being drawn backwards towards the spine, as well as that of oppresion in breathing, although the action of breathing was attended with no real difficulty," are descriptions recorded by skilled witnesses of their own sensations. None of the three, however, reaches the "inwardness" of that sensation which Gairdner terms "a sense of impending death," and which "Dr. Latham . . . justly elevated to a co-ordinate rank with the pain itself." According to this last author: "Angina pectoris . . . consists essentially of pain in the chest and a sense of impending dissolution" . . . "a sense of dying." A highly intellectual patient used the expression "A sense of dissolution," adding, "not a fear of it." Closely related with this sensation, an idea of which one tries to grasp from the descriptions of sufferers most capable of giving it expression, is what has been called the "unbearableness" of the pain. "If the pain is again as severe as it was before you came, I do not know how I can bear it," said a distinguished patient to his medical attendant. Dr. Walshe probably gives as true an idea of the nature of this suffering associated with,

but over and above, the pain of angina pectoris, as can be reached by a non-sufferer, when he defines it thus: "It is on the part of the patient a profound conviction that life cannot go on unless instantaneous relief be afforded."

The pain usually begins in the sternal region, most commonly at its lower part, and involves more or less of the precordium to the left of the bone, whence it spreads to the left shoulder and down the left arm on its *inner* aspect. It commonly reaches the elbow but may stop half-way. Often the pain goes down the ulnar side of the forearm to the ring and little finger-ends. In many cases the whole hand and all the fingers are implicated. Rarely the pain is *limited to the arm*, but its severity, and especially its development and aggravation by exertion on the part of the patient, should prevent such pain having a local origin assigned to it. The pain may be limited to one part of the limb, as the wrist. Rarely the pain travels backwards to the chest, a circumstance that led Trousseau to liken it to the aura of epilepsy, a resemblance that occurred also to Dr. Walshe. Both arms may be affected in angina pectoris.

Associated with the arm-pain of angina pectoris, and generally following it, is a sensation of numbness of the affected limb. Rarely the sensations described with regard to the chest, as associated

with the pain, are repeated, as it were, in the case of the limb. There may be a sensation of the part being swollen or on the other hand constricted. A sense of motor weakness of the affected arm is common, and may be so pronounced as to cause the patient to raise the affected limb with the sound one, after the manner of a hemiplegic. No actual paralysis occurs, however, and the feeling of weakness soon passes off.

Sometimes, the pain of angina pectoris is referred to the epigastrium—"pit of the stomach." It will be found, however, that the pain is brought on or aggravated *not* by active states of the stomach, but by physical exertion on the part of the patient. Moreover, in some cases, if this epigastric pain be neglected and the exertion persisted in, the precordium and left arm, or both upper extremities, become involved by the pain.

In relation with epigastric pain, it will be convenient to refer to a symptom, that may still further mislead the patient, and even his medical attendant, to thinking that the stomach and not the heart is the *fons et origo* of the suffering. This symptom is the eructation of gas, which is specially apt to occur when the pain is subsiding, so that the relief experienced is attributed to the eructation. An allied symptom very commonly present in angina pectoris is the so-called sensation of *globus*

or *globus hystericus*, owing to its frequency in the hysterical. Feeling of "a lump in the throat" is the familiar lay expression for the sensation, which is too well known to require description. Few cases of angina pectoris have no sensation of the kind. Occasionally young women are the subjects of true and lethal angina pectoris, and the risk of being misled by this, in itself trivial, symptom is great. In one such case the writer was saved from error by the presence of a faint diastolic murmur of aortic incompetence. Very ordinary cases of dyspepsia occasionally complain of *globus* sensation, and its association with hysteria is very far from being an exclusive one.

Dr. Henry Head has called attention to headaches and areas of hyperalgesia associated with irritation of certain spinal segments. Supra-orbital headache is that commonly met with when the "aortic" spinal segments—2nd and 3rd dorsal—are implicated. The affected area often shows some degree of hyperæsthesia or rather hyperalgesia. The areas in question need not be the seat of spontaneous pain, and may have to be sought for by the clinicist. For their discovery Dr. Head recommends pressure with the head of a pin, lightly picking up the skin or pulling up the hair of the scalp. (*Vide* p. 213.)

The whole subject of the distribution and radiation of pain in cardiac disease will be considered

immediately (p. 221). Suffice it now to say that in angina pectoris, such extraordinary radiations of pain as to the leg and testicle seem less difficult of explanation, if the diffusion of irritation in nervous grey matter be supposed to take place in the cerebral cortex rather than in the spinal cord, for in the former close physiological association of parts with distant anatomical distribution, can be more easily understood. Moreover, all pain as far as we know, must mean molecular disturbance taking place in cerebral grey matter—probably that of the cortex: for it is in the brain only that the *ego*—even if we regard it as the sum total of past impressions—can take cognizance of the disturbance. Irritability of the bladder and a profuse flow of urine are rare associated symptoms in angina pectoris. Sickness and vomiting are also rare—the effects of treatment being of course excluded. Delirium, unconsciousness and convulsions are altogether exceptional: they would seem to be due to cerebral anæmia, and the shock of such violent molecular disturbance of the cortex as is implied by an attack. There seems to be ground for saying that "pain can kill," and however rarely, death from simple biliary or renal colic, the calculus being found in the duct, occasionally results.

As regards the attitude of the patient suffering from a seizure of angina, Trousseau wrote:—

"One patient will be motionless on his back, another will incline backwards on the back of his chair, or on his pillows, a third will place himself on all fours resting on his elbows and knees, while a fourth may stoop as much as to bend in two." Usually a fixed attitude is assumed: the movements of the patient are arrested. Occasionally, however, patients—especially female—are restless, and toss about, vociferating loudly and almost continuously. Even in a young woman such symptoms must not be allowed to occasion the case being regarded as one of pseudo-angina. In a case of the kind the presence of a *bruit-de-galop* over the heart prevented the possibility of such an error, and the patient died. The movements of the patient, as just stated, are usually arrested, when the seizure takes place while he is in motion, and the cessation of motion generally brings relief, if not cessation, of pain. Very exceptionally a patient succeeds in "walking-off" the pain, but the attempt, even if successful, is always most dangerous. "I can walk with ease 10 or 15 miles after I have been stopped three or four times at intervals of a hundred yards," was the remark to Dr. Walshe of a patient, who died in a paroxysm of angina pectoris three days later. Then, again, an attack may come on when the patient is at rest, and such an attack may prove fatal. Patients who

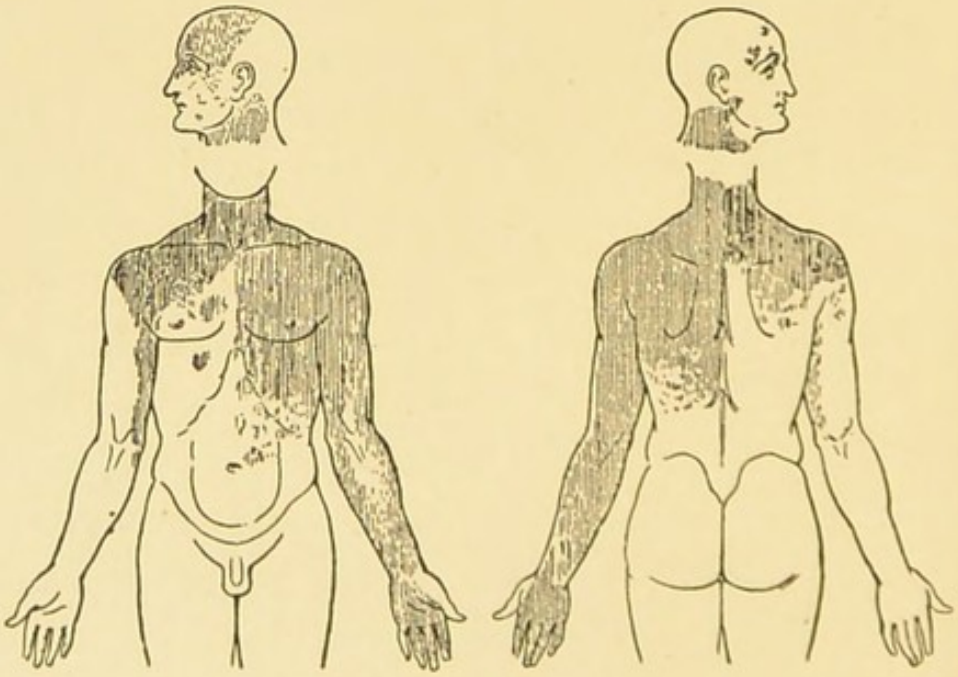
have had angina pectoris are *liable to sudden death* as far as we know *without the experience of pain.*

Closely related to angina are some cases of paroxysmal dyspnœa of sudden and violent nature. Thus a patient of the writer's, who had physical evidence of heart disease in the presence of *bruit-de-galop* and dilatation of the heart, but had never had pain, was seized with sudden breathlessness, and almost immediately died. *Post-mortem* the ordinary lesion of angina pectoris—diseased and obstructed coronary arteries—was found. The patient (at rest in bed) was able to express to the nurse the respiratory suffering, but made no mention of pain. Though their close relationship with angina pectoris is admitted, such seizures cannot be regarded as attacks of angina.

There is a tendency for the paroxysms of the disease to be associated with a rise in the arterial pressure, which has been supposed—in many cases no doubt rightly—to determine the pain. It was the fact of this rise of arterial pressure that led Sir Lauder Brunton to employ nitrite of amyl as a remedy. Such rise of pressure is not, however, met with in all cases, and occasionally the pulse is of low blood pressure both in the paroxysms and between them. There may even, in these cases, be some slight relative rise of blood pressure, that the sphygmogram fails to indicate.

It is important to remember that the pulse varies in different cases: for instance, it may be frequent and of low tension throughout the seizure, or become abnormally infrequent as in a case of Professor Osler's, or irregular or even imperceptible as in Hunter's case ("he found none in either arm"). As Dr. Walshe truly remarks, "in the very extremity of pain the pulse may be perfectly regular and scarcely exceed by half-a-dozen beats per minute the rate normal to the individual." No less true is his statement concerning the pulse in the intervals between the seizures: it "possesses no special character."

FIG. 81. FIG. 82.



To show the widespread tenderness present after a major anginal attack. (Head.)

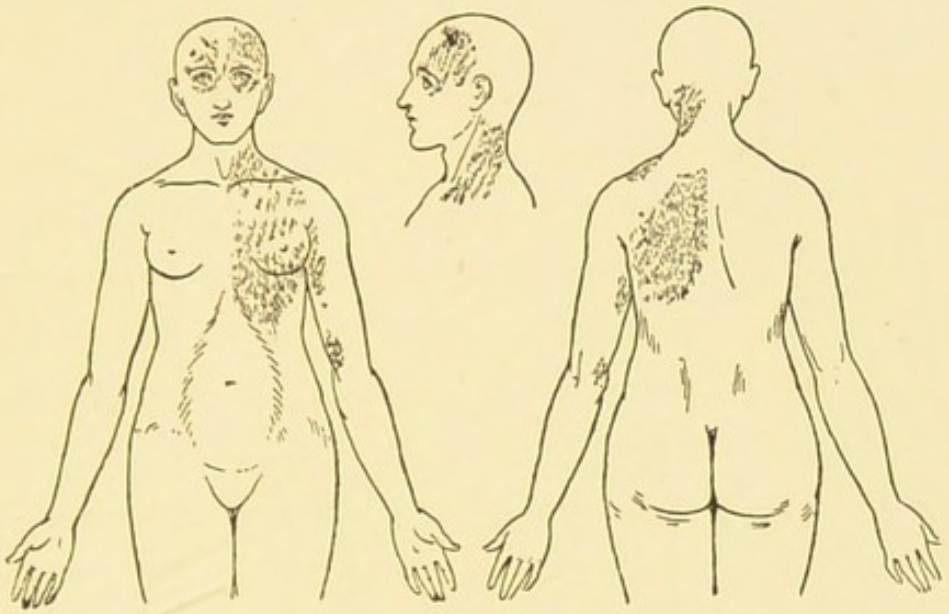


FIG. 83. To show the extent of the superficial tenderness in a Case of angina pectoris apart from an attack. (Head.)

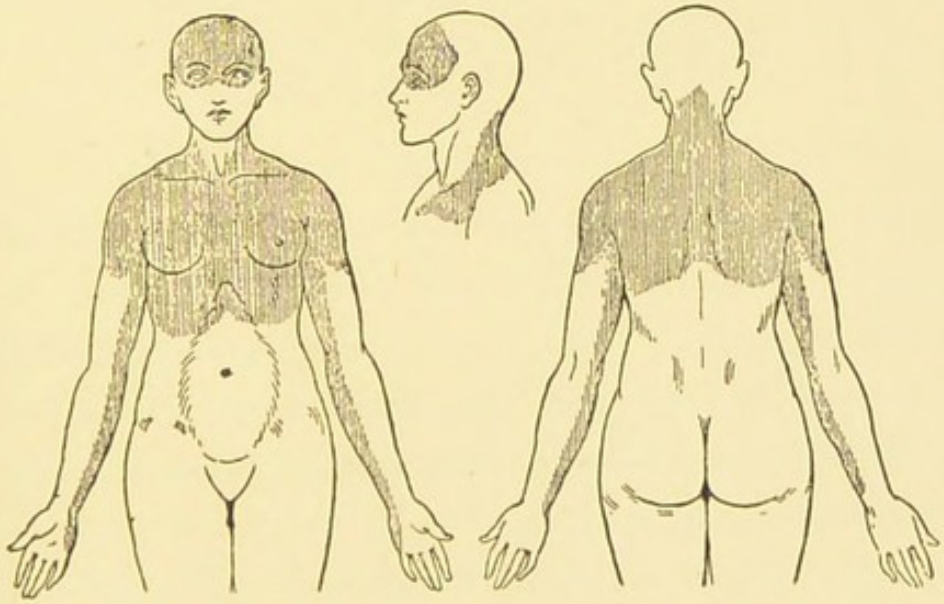


FIG. 84. To show the extent of the superficial tenderness during the time the pain was spreading, and 3 minutes before the quickening of the heart, in an attack of angina pectoris described by Dr. Head.

Fig. 85.

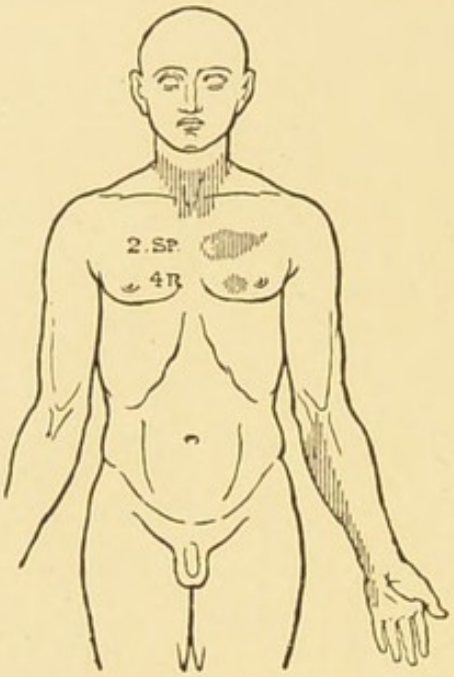
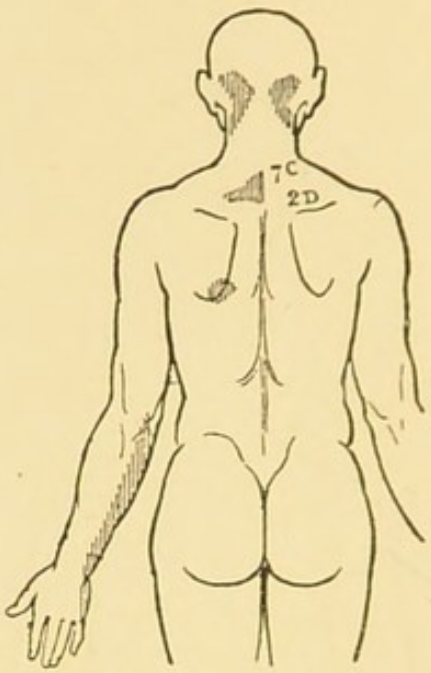


Fig. 86.



To show the areas of cutaneous tenderness during an anginal attack in a man with aortic disease. They represent the whole of the 1st dorsal and 3rd cervical segments together with the maxima of the 4th dorsal.

2 Sp. is placed over the 2nd intercostal space.
4 R is placed over the 4th rib.
7 C at the level of the spine of the 7th cervical vertebra.
2 D at the level of the 2nd dorsal spine. (Head.)

Dr. Head has shown, as already mentioned, that areas of hyperalgesia are usually associated with angina pectoris. They are usually best developed when an attack has passed off and correspond to the sensory distribution of the 3rd and 4th cervical and the 2nd and 3rd dorsal segments, as well as to the 5th, 6th, and 7th dorsal segments lower down. In association with the irritation of spinal segments, certain scalp areas may become hyperalgesic. Such hyperæsthetic areas are met with

in heart cases in general, thus: *occipital* headache or hyperalgesia is associated with irritation in the

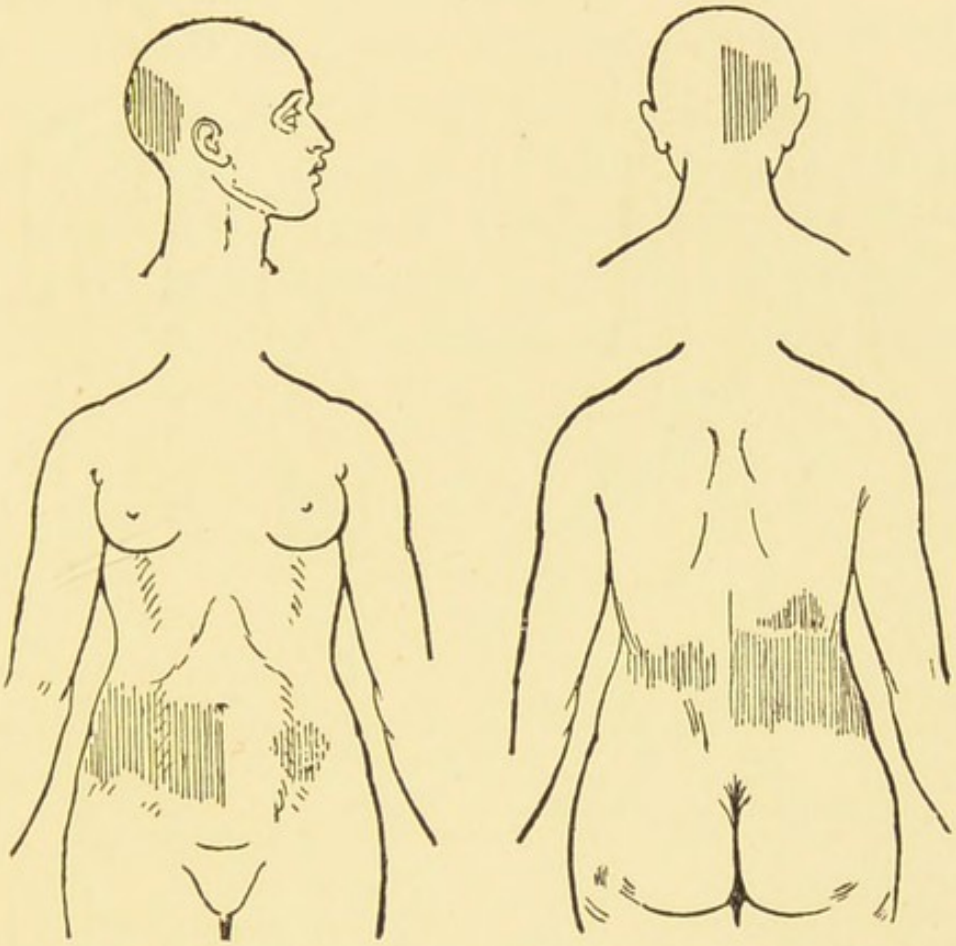


Fig. 87. To show the association between tenderness over the tenth dorsal area on the body and the occipital area on the scalp. (Head.)

10th dorsal segment, the upper border of whose distribution corresponds to the umbilicus. The implication of this area, is, however, usually indirect, and due to the passive congestion of the liver that follows heart-failure. The 9th dorsal segment is associated with the parietal area; the 8th dorsal segment with the vertical area; the 7th

dorsal with the temporal region of the scalp; the 6th segment with the fronto-temporal area; and the 5th segment is probably similarly associated. The 2nd dorsal segment, which is concerned in angina pectoris, is associated with scalp hyperalgesia, or pain in the supra-orbital region. When the 3rd cervical segment is involved, the pain is believed "to shoot from the back through the head to the centre of the forehead."

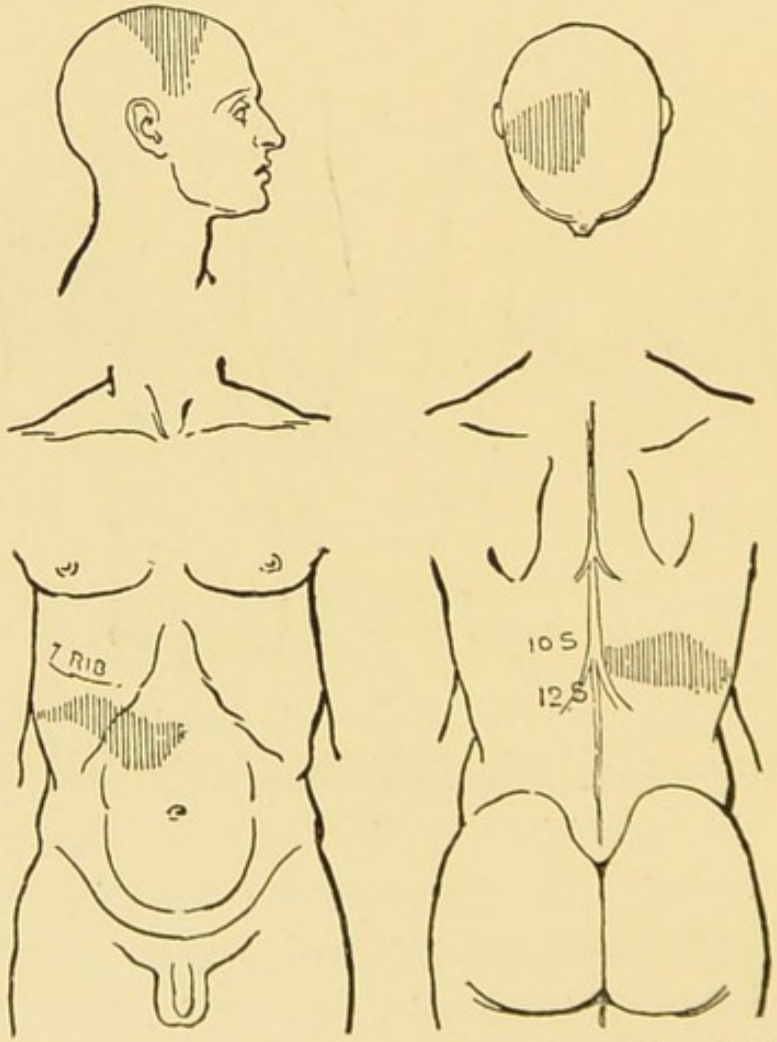


FIG. 88. To show the association between the eighth dorsal area on the body and the vertical area on the scalp. (Head.)

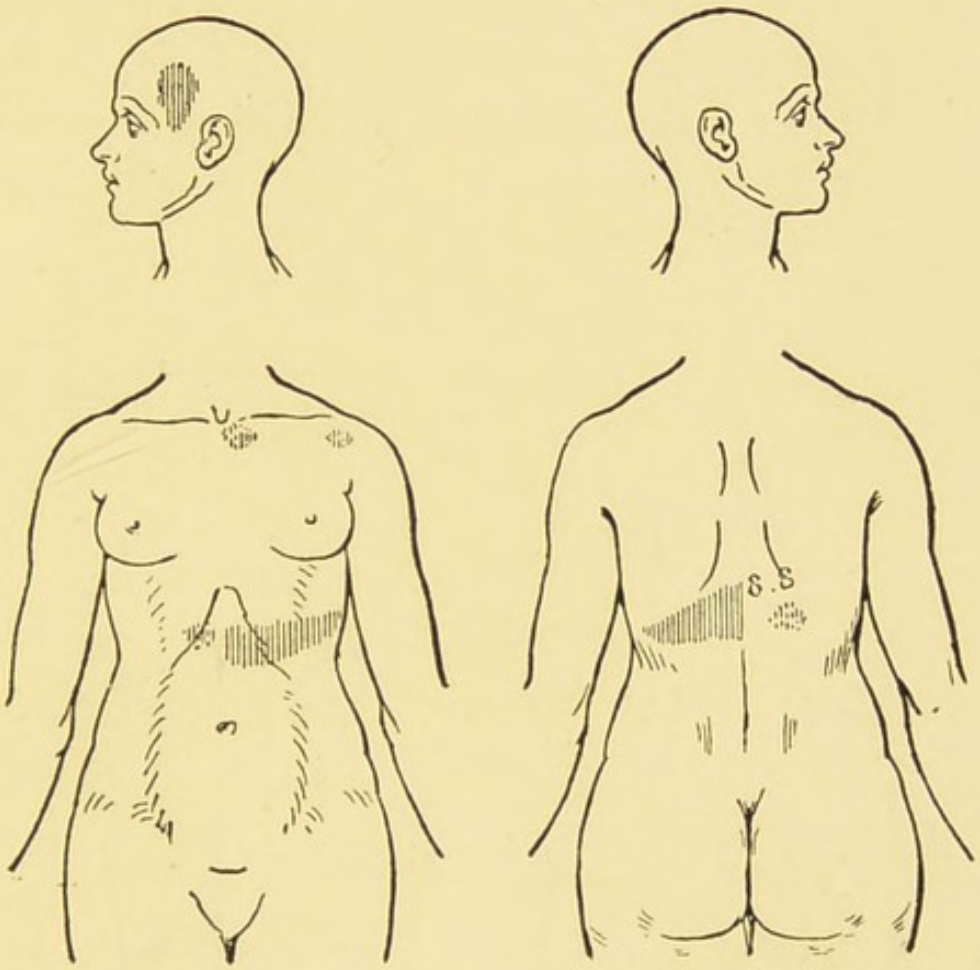


FIG. 89. To show the association between the seventh dorsal area on the body and the temporal area on the scalp. (Head.)

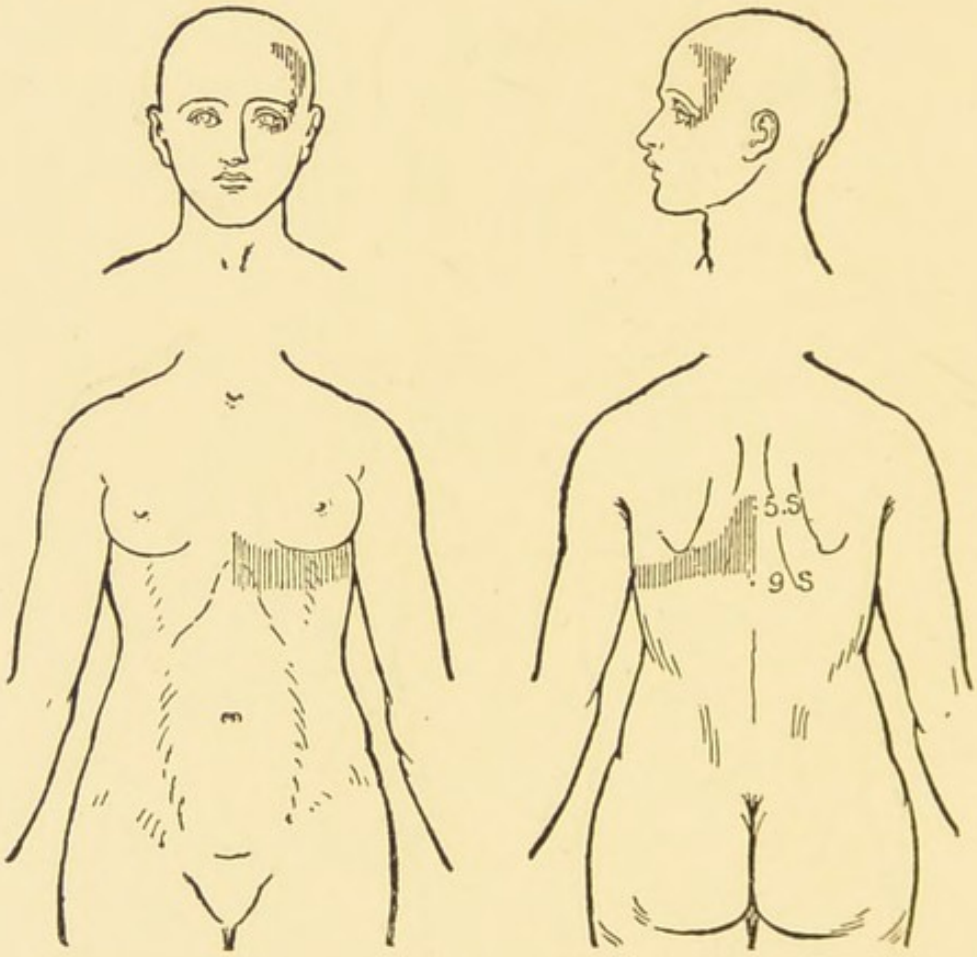


FIG. 90. To show the association between the sixth dorsal area on the body and the fronto-temporal area on the scalp. (Head.)

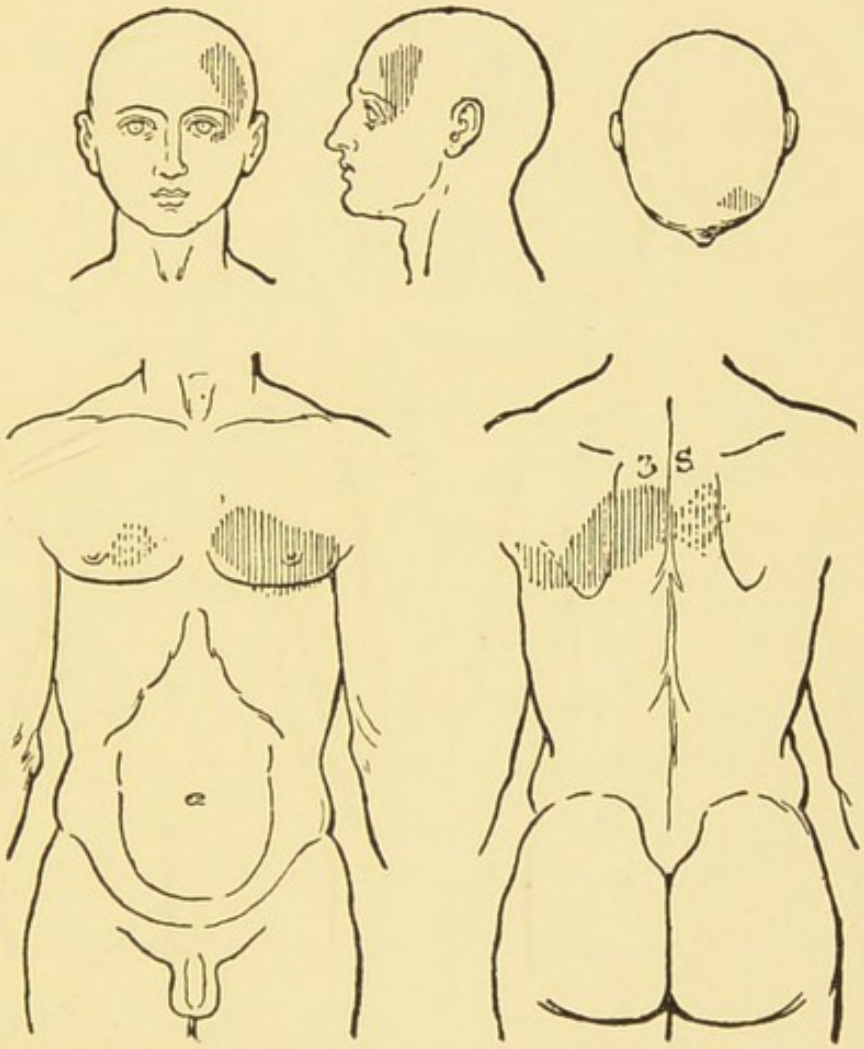


FIG. 91. To show the association between what is taken to be the fifth dorsal area on the body and the fronto-temporal area on the scalp. (Head.)

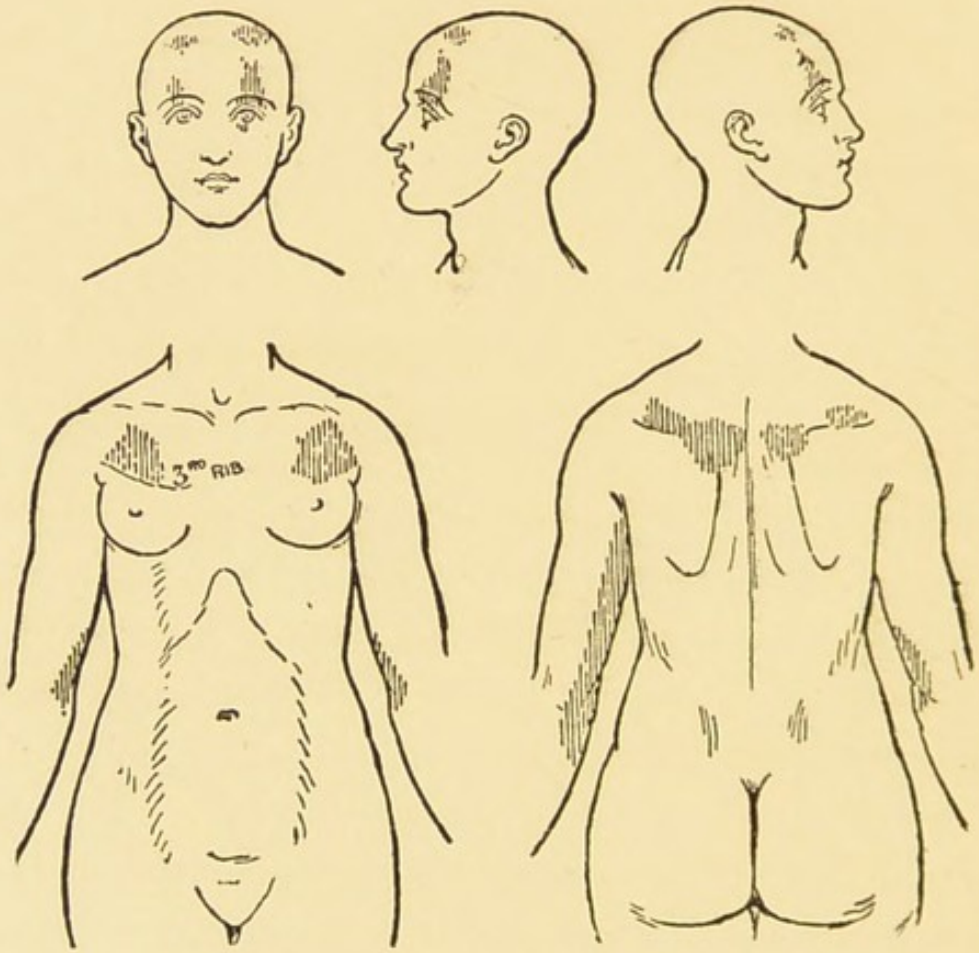


FIG. 92. To show the association between the second dorsal area on the body and the mid-orbital area on the Scalp. (Head.)

Physiological Considerations in Explanation of the Pain of Angina Pectoris.

The work of Drs. Ross, Mackenzie and Head has thrown great light upon the pain of angina pectoris. The "breast pang" of angina was naturally, though probably erroneously, long ago directly referred to the heart, but the radiation of the pain down the left arm, and sometimes, as has been described, down the right too, remained a mystery. The study of development alike in the individual and in the race, of recent years, has produced results of singular value in many ways. It holds the key to the understanding of the remarkable distribution of the pain of angina pectoris. We must go back to a primitive state of the nervous system and consider the segmental departments of which the spinal axis of the nervous system is composed, and the relationship borne to these by the great sympathetic, visceral, or splanchnic nervous system.

"When a viscus is diseased there is local pain which may be regarded as of splanchnic origin (precordial pain in the case of the heart). In addition the irritation is conducted to the portion of the spinal cord, from which the viscus derives its splanchnic nerve, and then spreads in the grey

matter of the posterior horns, when, by the law of eccentric projection, it is referred to the termination of the somatic nerves derived from the segment of the cord—the 2nd and 1st dorsal in the case of the heart. This explains the pain shooting between the shoulders and down the inner side of the arm (2nd dorsal) to the elbow and the ulnar border of the fore-arm and hand and ulnar fingers (1st dorsal)." These are the words of the late Dr. Ross, and the only comment I have to make upon them, is with reference to "the local pain," which Dr. Ross says may be regarded as of splanchnic origin. Good observers have denied to the heart any direct sensibility at all. This is probably going too far. The fact of the referred pain in the case of the heart being in part over, or almost over, the organ itself is a cause of much confusion in arriving at a definite conclusion on the matter.

Speaking of a certain group of mitral stenosis cases, Dr. Head speaks of "palpitation being accompanied by shortness of breath and occasionally by a pain which is said to be situated over the apex of the heart." "This pain does not go through to the back or round the body like a referred pain" . . . "It is apparently a true local pain."

Impressions arising in the heart are supposed to

be carried by sympathetic fibres to those segments of the spinal cord with which they are in association. Such impressions or irritations having arrived at the associated segment of the cord, undergo diffusion in the grey matter and seem to the patient to proceed from the fibres of the somatic spinal system, with which the segment in question is directly connected: so that in the cerebrum, where alone pain can be felt by the *ego*, the pain of angina pectoris is referred to the parts corresponding to the afferent supply of the grey matter into which diffusion has taken place. Similar diffusion may possibly occur in the grey matter of the cerebral cortex. The aura of epilepsy may be constituted by pain referred to a peripheral part.

Concerning spinal cord diffusion the study of development teaches how the upper limb, budding out from the trunk, carries out with it the nerve fibres from the spinal segments concerned, the arm being in the fœtal position of supination. Only the middle fibres, taking the series from above downwards, reach the finger ends. Thus the arm being in supination, the nerve roots that minister to the sensibility of the arm and forearm along their radial border are the *5th cervical*, whose territory extends to the wrist, the *6th cervical* supplying the thumb, while the *7th* supplies most of the palm of the hand and the 2nd, 3rd and 4th

fingers. The little finger and left border of the palm are supplied by the 8th cervical and the ulnar side of the forearm and lower half of the upper arm by the 1st dorsal root, while the 2nd dorsal root supplies the upper half or more of the inner side of the upper arm. These latter distributions are of chief importance in our consideration of angina pectoris, for it is in the areas so supplied, that the pain is so often localised, though it may extend to all the areas described by diffusion in the grey matter of adjoining segments.

The segments, that are specially concerned with pain having its origin in the heart, are given as follows by Dr. Henry Head, and the aortic areas are of special importance in relation with angina pectoris in view of the common association of the disease with aortic changes : —

According to Dr. Head the *transverse portion of the arch* of the aorta refers irritation arising locally to the *inferior laryngeal triangle*, of which the superior angle lies on the anterior border of the sterno-mastoid, at about the level of the pomum Adami. The upper border runs with a slight curve almost directly inwards to reach the middle line at about the crico-thyroid membrane. The posterior border runs along the anterior border of the sterno-mastoid to the sterno-clavicular articulation. It then bends a little outwards,

forming a small patch over the sterno-clavicular joint. The base of the triangle is formed by the median line. The maximum point of tenderness of this area is over the anterior border of the sterno-mastoid, a little below the level of the cricoid cartilage.

The *ascending portion of the arch* of the aorta refers to the area of distribution of the 3rd and 4th cervical and 1st, 2nd and 3rd dorsal spinal segments: that is to the somatic supply thereof.

The areas of the somatic distribution of the 3rd and 4th cervical segments will be seen at a glance in the figure, while their definition in description would be tedious. They are spoken of sometimes as the "sterno-mastoid" and "sterno-nuchal" areas respectively. (Fig. 93, Cerv. IV. and Cerv. III.)

The ventricle refers to the area of distribution of the 2nd, 3rd, 4th and 5th dorsal segments and their somatic supply.

The auricle refers to the distribution of the 5th, 6th, 7th and 8th dorsal segments, *i.e.*, to the somatic supply thereof.

It will be noticed that the auricular segment lies below the ventricular; a fact explained by the bending of the tube that originally represented, from the head downwards, the bulbus aortæ, the ventricles, and the auricles, so that the auricle

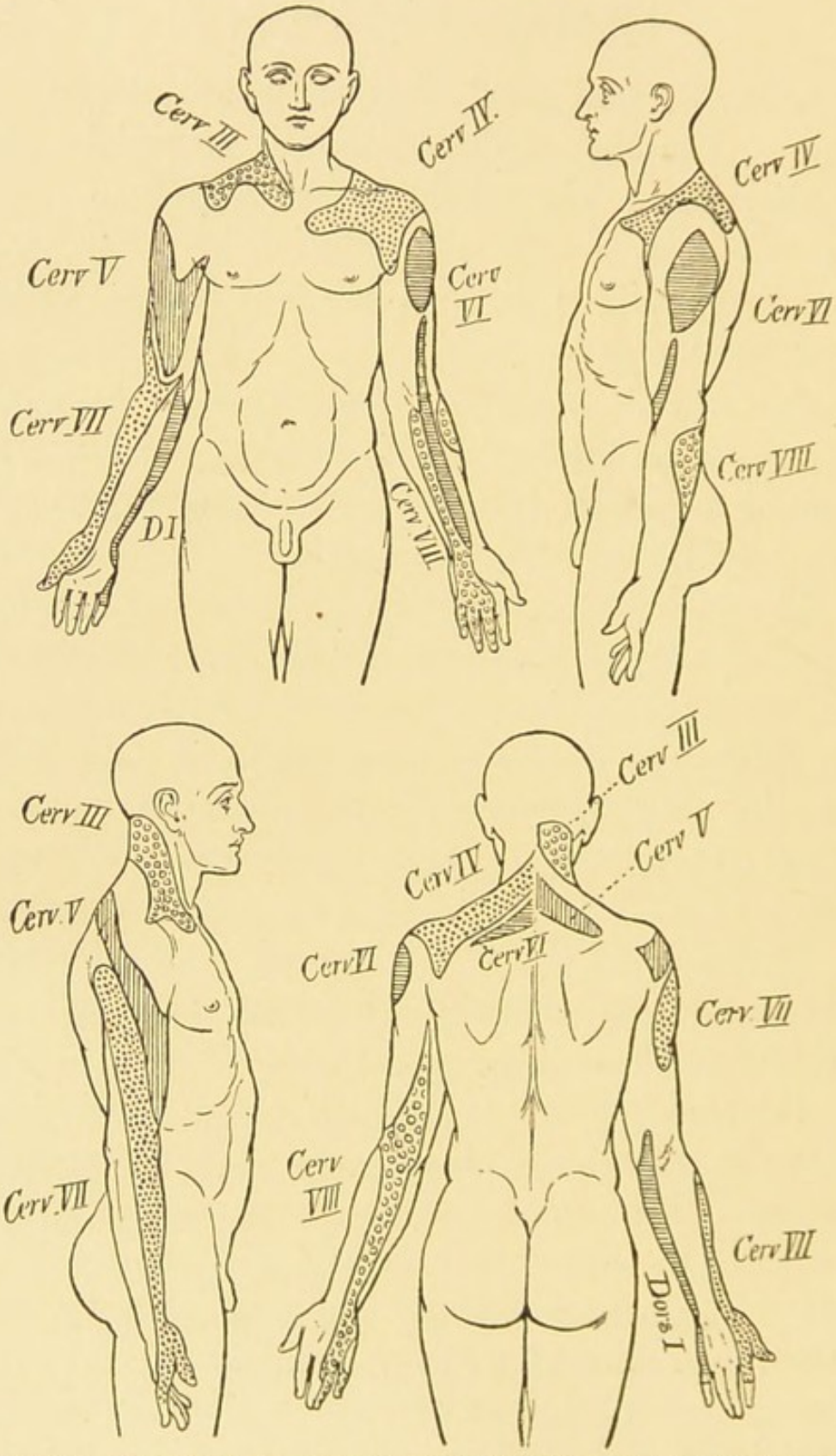


FIG. 93. To show approximately the distribution of the areas from the third cervical to the first dorsal. The amount of overlap can be seen by comparing any area on the right side of the figure with that above or below it on the left side. (Head.)

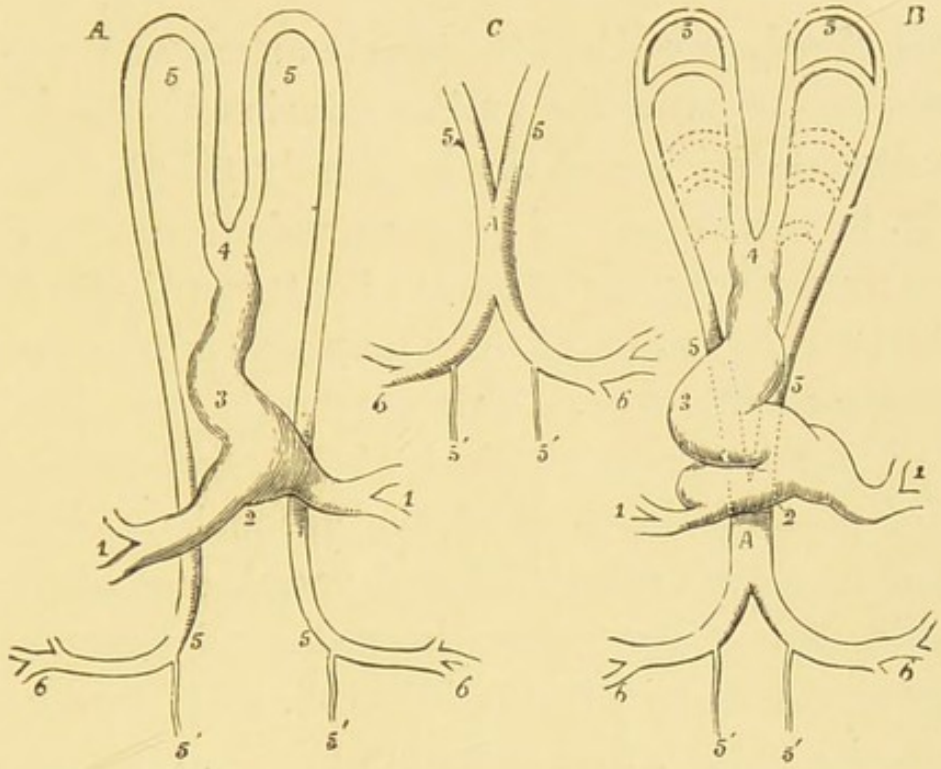


FIG. 94. Diagrammatic outlines of the heart and primitive vessels of the embryo chick as seen from below and enlarged. (A. T.)

A, soon after the first establishment of the circulation; B, C, at a somewhat later period; 1, 1, the veins returning from the vascular area; 2, 3, 4, the heart, now in the form of a notched tube; 5, 5 (upper), the two primitive aortic arches; 5, 5 (lower), the primitive double aorta; A, the single or united aorta; 5^1, 5^1, the continuation of the double aortæ beyond the origin of the large omphalo-mesenteric arteries, 6, 6. The division above 4 is represented as carried rather too far down. (Quain.)

gets above the ventricle (Fig. B). Dr. Head remarks: "The sensory supply of the heart is explained by the fact that the impulses enter the segments of the central nervous system just as if the auricle were still the hindermost portion of the heart."

It is important that the aorta should be considered developmentally as divided into three portions.

The *first* part extends from the valves to the

level of origin of the innominate artery and represents in the adult the bulbus arteriosus—the most headward division of the originally tubular heart (Fig. 94). The *second* part extends from the innominate artery to the point of entry of the ductus arteriosus in the fœtus : one may say the transverse portion of the arch. This part represents in the adult the embryonic 4th branchial vessel. The *third* part of the arch of the aorta, beyond this point, is a true somatic dorsal vessel, and when it is the seat of aneurysm the pain and superficial tenderness lie within the 5th, 6th, and 7th dorsal segmental areas, that is within the distribution of the corresponding intercostal nerves—a distribution that may, however, be influenced by impulses having their origin in the auricles.

Prognosis. In few diseases is care in the formation of a prognosis more requisite than in angina pectoris. John Hunter lived 20 years after his first attack, and his death from a seizure may be regarded as, in a sense, more or less accidental. Dr. Walshe recorded a case of 24 years' duration. On the other hand, Dr. Arnold, of Rugby, died in his first attack. Again the tendency of the subjects of angina pectoris to die suddenly, irrespective of pain, has to be borne in mind in considering the prognosis. The type of angina pectoris associated with mitral stenosis is probably much less dangerous

than the classical type, without valve lesion and even the ordinary evidence of cardiac muscle-failure. On the other hand, in cases of aortic disease, with incompetence, the prognosis is much the same as in cases without valve-lesion: the coronary orifices are probably involved.

In rheumatic young adults with aortic incompetence, angina pectoris is occasionally observed to occur. In such cases one cannot exclude coronary disease, inasmuch as the rheumatic valve-lesion producing incompetence has thrown a strain upon the first part of the aorta, so that premature atheromatous change may have involved the coronary orifices.

COMMON TYPES OF HEART DISEASE.

Having now considered the facts of evidence of cardiac disease in detail, as they are elicited by physical examination, as well as from the statements of the patient and from the observer's general examination, a short description or clinical sketch of different types of heart disease may be entered upon.

The two great *valve-lesions* are mitral stenosis and aortic incompetence, and while they present for the most part a striking contrast in their features, it has to be borne in mind that the two lesions may be met with in the same patient, and that certain of their physical signs may present a curious if superficial resemblance, *e.g.* Flint's auscultatory sign—"pseudo-presystolic murmur" —in aortic incompetence and widely distributed double systolic and diastolic murmur in mitral stenosis.

The patient with mitral stenosis—and she or he is often in adolescence—will usually give the history of an acute rheumatic attack or chorea at least several years before. On the other hand, an absence of such history by no means excludes

the lesion, for, especially in female patients, though occasionally in men too, unmistakable evidence of the lesion is found in the absence of all history of either rheumatism or chorea. Such cases are usually met with before or in middle age.

The typical aspect of the sufferer from mitral stenosis includes vascularity of the face, especially circumscribed flushes on the cheeks. The lips are often livid. The neck shows venous engorgement and often pulsation. The arterial pulse may be quite regular and of good tension, but it is often irregular. Evidence of enlargement of the *right* ventricle is often forthcoming on uncovering the chest—epigastric impulse—while the apex-beat may be deficient or absent, or pronounced and displaced to the left. Thrill is often felt over the apex-beat. The liver is often very large and may truly pulsate, while dropsy may be slight or absent, or, on the other hand, pronounced. There may be no murmur over the heart, or, on the other hand, presystolic, systolic, and diastolic murmurs may all be present, affording an almost constant succession of murmurs. In the absence of murmur, again, the heart sounds may be characteristically modified, or, on the other hand, they may be practically normal. A systolic murmur, preceded or not by the first sound,

is the most common auscultatory sign. The first sound is usually audible alone at the back about the angle of the scapula, the murmur being inaudible behind the mid-axillary line. *On the other hand, the presence of systolic murmur at the back by no means negatives the diagnosis of mitral stenosis.*

The typical sufferer from aortic incompetence, again, is usually a middle-aged man of pale aspect, who denies all history of rheumatism and chorea, but has been exposed to hard work, especially the frequent making of efforts, while he may have lived imprudently, taken drink, had syphilis, inherited or earned gout, or been exposed to plumbism: as well as having dyspnœa on exertion he may be subject to paroxysmal dyspnœa. His carotids pulsate in striking manner, while all exposed arteries are likewise seen to pulsate in exaggerated fashion. The jerky character of his radial pulse is felt to be increased when the limb is raised, while the arterial pulsation can be felt on grasping the forearm with the hand. There is little or no evidence of venous engorgement, the unduly visible apex-beat is increased in area and displaced downwards and to the left. Epigastric pulsation is absent, or such, as there may be, is obviously due to the encroachment of the huge left ventricle. The liver cannot be felt,

and there is no dropsy. A loud double—systolic and diastolic—murmur is audible all over the heart and up to the top of the sternum. The heart *sounds* are inaudible over the precordium, while on pressure with the stethoscope over the femoral artery a systolic and a diastolic murmur are audible.

In both cases, dropsy is probable sooner or later. In the aortic case the liver will ultimately become enlarged, as in the mitral case, and "pulmonary apoplexy," with its hæmorrhagic expectoration is apt to supervene in both cases.

But inasmuch as the coronary arteries have become involved in atheromatous or syphilitic aortitis, a case with aortic incompetence may come before us as one of angina pectoris, or again, inasmuch as the disease of the aortic coats has led to the formation of aneurysm, as a case virtually of aneurysm with the characteristic pressure signs and symptoms of that condition.

It has been pointed out that aortic stenosis is a lesion often borne without serious symptoms for many years, but sooner or later the ordinary indications of cardiac muscle-failure develop—dyspnœa, dropsy, engorged liver, etc. The diagnosis must be carefully made by physical examination, fully detailed elsewhere (p. 103). The slow regular pulse, while cardiac muscle-failure is

absent or incipient, the loud, long, harsh systolic murmur in the aortic area and the deficiency or actual absence of the second sound with or without a usually feeble diastolic murmur are significant.

One or other of the suggestive, if not pathognomonic pulses of the lesion—the bisferiens and anacrotic—may be present and aid in the diagnosis. It will be remembered that the bisferiens pulse may be much better developed on one side or be only recognisable on one side. The great frequency of even a loud systolic murmur in the aortic area without there being any stenosis must be remembered.

Absence of all murmur along with the presence of the ordinary indications of chronic heart failure may be met with in cases of cardiac muscle-failure and in cases of mitral stenosis. The history of the case and any modifications of the physiological sounds present form the best guides to the diagnosis. Thus the history of acute rheumatism many years before, accentuation of the first sound, and reduplication of the second sound would almost certainly indicate mitral stenosis, while in a patient with nocturnal polyuria, albuminuria, and habitual low specific gravity of his urine, a reduplicated second sound over the left ventricle etc., would in all

probability represent the *bruit-de-galop* of cardiac muscle-failure, and the case would presumably be one of chronic Bright's disease with secondary cardiac muscle-failure. In both cases the heart sounds may, however, present no such modifications as have been referred to, and indeed be practically normal.

The occasional coincidence of mitral stenosis and gout, as pointed out by Dr. Sansom, is sufficiently common to make it desirable that the possible combination should be remembered as a not very rare concurrence of pathological processes. The writer has met with the combination sufficiently frequently to convince him of its importance. Under the circumstances the diagnosis may be difficult, as the mitral stenosis may not be revealed by pathognomonic sign. A systolic murmur, for instance, is equally common in mitral stenosis and in the cardiac muscle-failure of gout, while the same may be said of irregularity and a high degree of tension of the pulse.

It is impossible to give a description of a case of mitral incompetence: mitral incompetence occurs under such an immense variety of conditions, in many of which it is far from being the essential feature of the pathological process. Often it is a quite unimportant one. The physical conditions associated with mitral incompetence

have been already described and considered in detail, and the three questions that the clinical observer has to ask himself have been stated (p. 106). Of the rarest condition: structural change in the valve curtains of a nature calculated to render them incompetent independently of any failure on the part of the heart-muscle, septic endocarditis furnishes the best example. Then comes the much more common, but greatly less destructive, rheumatic endocarditis, with which cardiac muscle-failure is more obviously associated in the production of the incompetence of the valves. Even in the case of these forms of endocarditis, it is impossible to deny a muscle-element in the production of the incompetence. The presence of fever has long been known to exercise a peculiarly debilitating influence on the heart-muscle, and septic endocarditis as a rule runs a prolonged and highly febrile course, while the rheumatic toxine probably directly injures the myocardium, just as it often sets up pericarditis.

Mitral incompetence is an exceedingly common concomitant of mitral stenosis. In stenosis the valves are deformed and they are prone to leak. That the regurgitation under the circumstances is of any great importance may well be doubted. In cases of the kind the stenosis is the essential

pathological condition present. Notwithstanding, writers have been fain to attach probably wholly unwarranted importance to such accidental and quite minor incompetence.

Lastly, there is the great class of cases in which mitral incompetence results from debility of the heart-muscle simply. Anæmia, gout and chronic Bright's disease furnish good examples, but the causes that may be at work in the production of debility of the heart-muscle are very numerous indeed, and no good would be achieved by any attempt at their enumeration. Often such causes act in combination—a common combination of the kind is anæmia and hard physical work. The most common type of all is the "senile" heart—bordering on the physiological.

The reader may be again reminded of the occurrence of cases of cardiac muscle-failure without the production of murmur (p. 96).

Sometimes a patient with cardiac muscle-failure has no murmur when upright, but develops one when he lies down.

DIASTOLIC MURMUR AND THRILL WITHOUT ARTERIAL INCOMPETENCE OR AURICULO-VENTRICULAR CONSTRICTION.

Hitherto in this work the object of the writer has been to give an account of our common knowledge of heart disease, as it has been accumulated by the daily experience of those engaged in the treatment of the disease. Beyond such knowledge there is, however, a margin of imperfect knowledge shading on the other side into absolute ignorance. It has been held that the limit of instruction to the student should be that of perfect knowledge, with at most a rare trespass into the imperfectly known. But the student in time becomes the practitioner, and in his practice may happen to meet with cases, about which he has never heard a word from his teachers. To prevent this as far as possible it seems desirable to admit to the student that experience has taught that there are rare occurrences which seem to militate against our common knowledge and belief. It is better he should be warned that such exist, than that his faith in well-established clinical

truths should be temporarily shattered by the encounter of a case of the kind under consideration. Contrary to custom the details of two cases in point will now be given:—

Case I. Mary E.D., æt. 19, was admitted to the M.R.I. on February 11th, 1893. She had had rheumatic fever æt. 11, and had been short of breath since. She had orthopnœa, bronchitis, and a much enlarged, tender liver, but no dropsy, on admission. Her heart was greatly enlarged, and its dulness was represented by the figures $\frac{\text{II.}}{2-6\frac{1}{2}}$. In the mitral area there were heard a systolic and a diastolic murmur, the former being well conducted to the back. In the tricuspid area the first sound was audible followed by a systolic murmur. The second sound was audible. In the aortic area there was a systolic murmur followed by the second sound. In the pulmonary area there was a systolic murmur followed by a greatly accentuated second sound. Later it was noted that there was a double second sound audible at the apex with the murmurs. The diastolic murmur at the apex was often accompanied by thrill. Dropsy began to appear when the patient was in Hospital, and on May 1st it was noted that there was a huge pad of œdema over the lower part of the back but no œdema of the lower extremities. The patient insisted on going home on May 31st, but was re-

admitted to the M.R.I. on June 26th. There was now general dropsy, of which ascites was a prominent part. She died 17th September, 1893.

Post mortem. Pericardium generally adherent. Heart enormously enlarged; all the cavities dilated; tricuspid and mitral orifices admit four fingers each. Mitral valves thickened; chordæ tendineæ hard and short. Vegetations on the auricular surface. Aortic and pulmonary valves apparently competent. Liver "nutmeg." Kidneys venously congested.

Case II. Gerald F., æt. 23, admitted to the Manchester Royal Infirmary 22nd May, 1893. He stated that he had never had rheumatic fever or chorea, but that he had been delicate as long as he could remember, and always more or less short of breath. He had had dropsy eight years before. A week before admission he had had pains in his knees. Palpitation and shortness of breath were the symptoms complained of on admission. The liver was not obviously enlarged, and there was no dropsy. The cardiac dulness was represented thus: $\frac{\text{III.}}{1\frac{3}{4}-5\frac{1}{2}}$. In the mitral area there were audible a systolic murmur, double second sound, and a slight short diastolic murmur. The systolic murmur was audible at the back. In the tricuspid area a systolic murmur and double second sound were audible; in the

aortic area also a systolic murmur and double second sound, and in the pulmonary area the same. On the 2nd June it was noted "there is now a distinct diastolic murmur accompanied by thrill at the apex." The patient went home considerably relieved, on the 8th July. He was readmitted on the 28th February, 1894, but did not survive twenty-four hours.

Post mortem. Pericardial cavity obliterated. Heart enormously enlarged, weighing empty 1 lb. 6 oz. All the cavities extremely distended with dark clot. Tricuspid orifice much dilated, valves normal, pulmonary valves normal and apparently competent. Mitral valves thickened and distinctly indurated; orifice greatly dilated. No vegetations. Aortic valves slightly thickened, but by no means deformed and apparently competent. Aorta itself normal. Liver "nutmeg." Kidneys venously congested.

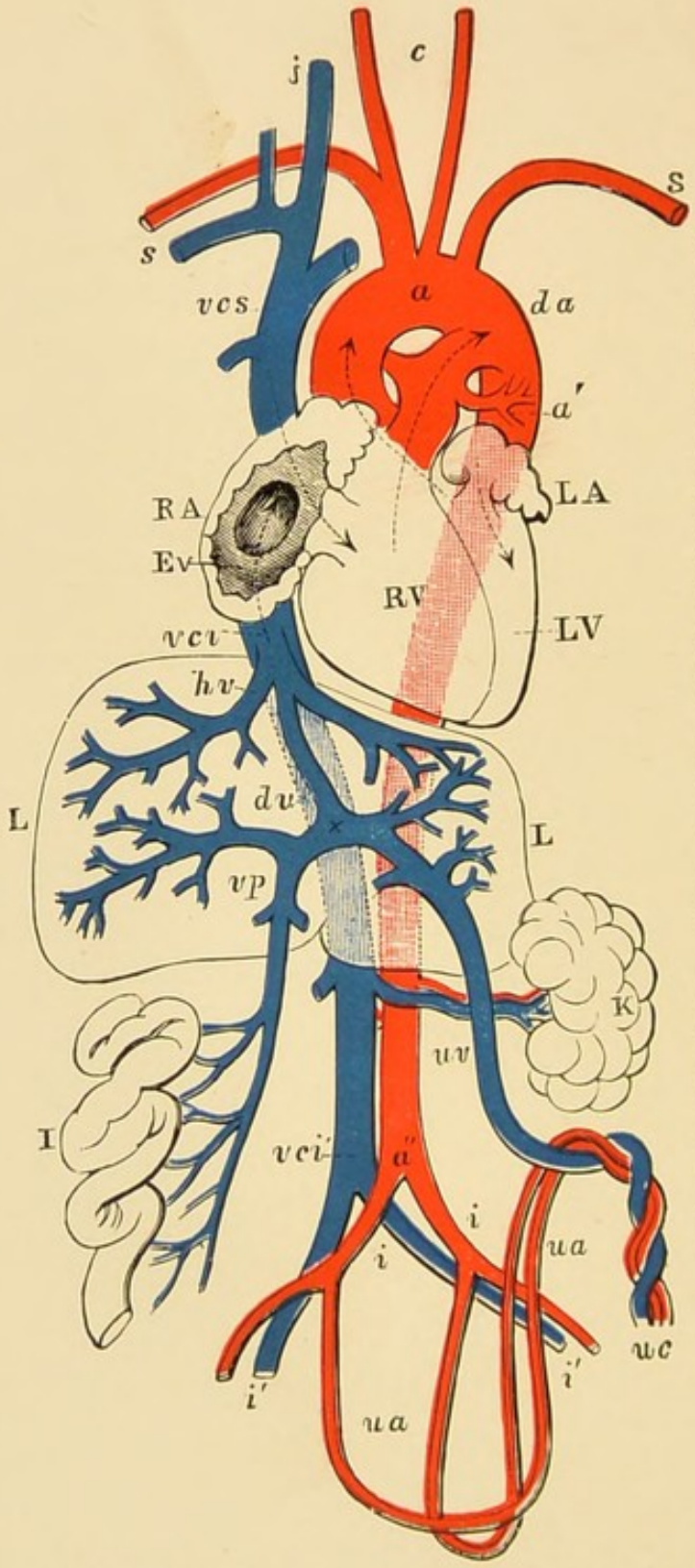
These cases were published in the *Practitioner*, vol. lii., p. 254, in a communication entitled "Diastolic Murmur in Dilatation of the Heart without Arterial Incompetence or Auriculo-ventricular Constriction," in which reference was made to a similar case recorded by Dr. Stokes in 1854.

Both cases were diagnosed during the patients' lifetime as cases of "mitral stenosis," the reasons

for which are too obvious to require mention. Since these cases occurred, the writer has met with others of a similar nature, but the nearest approach to their recognition clinically he has made, has been the remark that the case "*might possibly be* of the anomalous type described in the *Practitioner*." Otherwise the cases were regarded, till the autopsy, as cases of mitral stenosis, and the writer must confess that now he knows no feature by which like cases can be distinguished from cases of stenosis.

One of the patients, observed since the cases were described in the *Practitioner*, had the circumscribed flushes on the cheeks that are so common in mitral stenosis, while the diastolic murmur was accompanied by marked thrill in the mitral area.

The subject of these anomalous cases is further discussed in a paper by the writer, entitled "On a rare combination of physical signs met with at the apex of certain dilated hearts," contained in the *Practitioner* for September, 1896, and another case is recorded in detail.



RA, right auricle of the heart; RV, right ventricle; LA, left auricle; Ev, Eustachian valve: LV, left ventricle; L, liver; K, left kidney; I, portion of small intestine; *a*, arch of the aorta; *a'*, descending thoracic aorta; *a''*, lower end; *vcs*, superior vena cava; *vci*, inferior vena where it joins the right auricle; *vci'*, its lower end; *s*, subclavian vessels; *j*, right jugular vein; *c*, common carotid arteries; four curved dotted arrow lines are carried through the aortic and pulmonary opening, and the auriculo-ventricular orifices; *da*, opposite to the one passing through the pulmonary artery, marks the place of the ductus arteriosus; a similar arrow line is shown passing from the vena cava inferior through the fossa ovalis of the right auricle, and the foramen ovale into the left auricle: *hv*, the hepatic veins; *vp*, vena portæ; *x* to *vci*, the ductus venosus; *uv*, the umbilical vein; *ua*, umbilical arteries; *uc*, umbilical cord cut short; *i i'*, iliac vessels.

FIG. 95. Diagrammatic outline of the organs of circulation in the fœtus of six months. (Allen Thomson.)[1]

1. The red and blue coloration of this diagram represents the fact that the vessels coloured are arteries and veins respectively, and not the quality of blood which they convey.

From "Quain's Anatomy."

CONGENITAL DISEASE OF THE HEART.

THE FŒTAL CIRCULATION.

It may be desirable at the outset to remind the reader of the peculiarities of the fœtal circulation. The purified blood from the placenta is brought back by the umbilical vein. A small part passes through the liver; the greater part, passing through the ductus venosus, reaches the inferior vena cava, and passes to the right auricle. By the Eustachian valve it is there directed through the foramen ovale into the left auricle, and thence into the left ventricle and aorta. Thereby it passes to the head and upper extremities, which thus receive comparatively pure blood, owing to the importance of their development. Returning by the Superior Cava the blood again reaches the right auricle. This time it takes a different direction: passing into the right ventricle, pulmonary artery and ductus arteriosus respectively, it reaches the descending aorta, to be supplied now to the lower part of the body and extremities, finally finding its way back to the placenta by the umbilical or hypogastric arteries.

CONGENITAL DISEASES OF THE HEART.

For the due appreciation of this comparatively small class of heart cases, it is necessary to bear in mind certain facts in the development of the heart and large vessels, commencing with that stage in which the heart is represented by a single tube. "In mammals, the heart appears in the form of two tubes lying in the cervical region, one on either side of the embryo. . . . The two short tubes form the double rudiment of the heart. The situation which they occupy becomes, when the lateral walls fold over to form the foregut, the ventral wall of the pharynx, and the two tubes are thus brought together in the middle line underneath the head-part of the alimentary canal. Here they soon become fused together to form a single median tube" (*Quain's Anatomy*, vol. i., part 1, p. 134). The next step in development that interests the clinical student is the bending of this tube, consisting of its three parts; those three parts named from behind are as follows: (*a*) the union of the two vitelline or omphalomeseraic veins in the posterior part of the heart forms the venous part of the heart; (*b*) the middle part of the tube corresponding to the ventricle and (*c*) the arterial bulb.

The bending in question is of the anterior part of the tube to the right, and of its posterior part to the left, the tube as a whole assuming to some extent the shape of the letter S.

Moreover, the posterior or sino-auricular end of the tube gradually comes to lie behind the ventricular part. The tube is thus divided into the following parts: (1) The part formed by the junction of the principal veins, sinus venosus; (2) the auricular part; (3) the ventricular part, and (4) the aortic bulb (Fig. 96). The sinus venosus consists of two lateral enlargements and a transverse portion. The umbilical, the vitelline veins, and the ducts of Cuvier (formed by the junction of the *primitive jugular* vein from the head and the *cardinal* from the trunk) join the sinus which "is at first in free communication with the common auricular cavity, but the junction presently becomes narrowed." "The sinus now forms a transversely disposed sac lying below and behind the common auricle, with a larger right and a smaller left horn (the latter being tapered off into the left duct of Cuvier)." In this "saccus reuniens" the umbilical and vitelline veins soon open by a common trunk which becomes the upper end of the vena cava inferior. The right horn of the sinus becomes incorporated with the cavity of the auricle. "The transverse part of the sinus and its

left horn are continuous with the left duct of Cuvier, and eventually the transverse part forms the coronary sinus." (*Quain's Anatomy*, p. 141.)

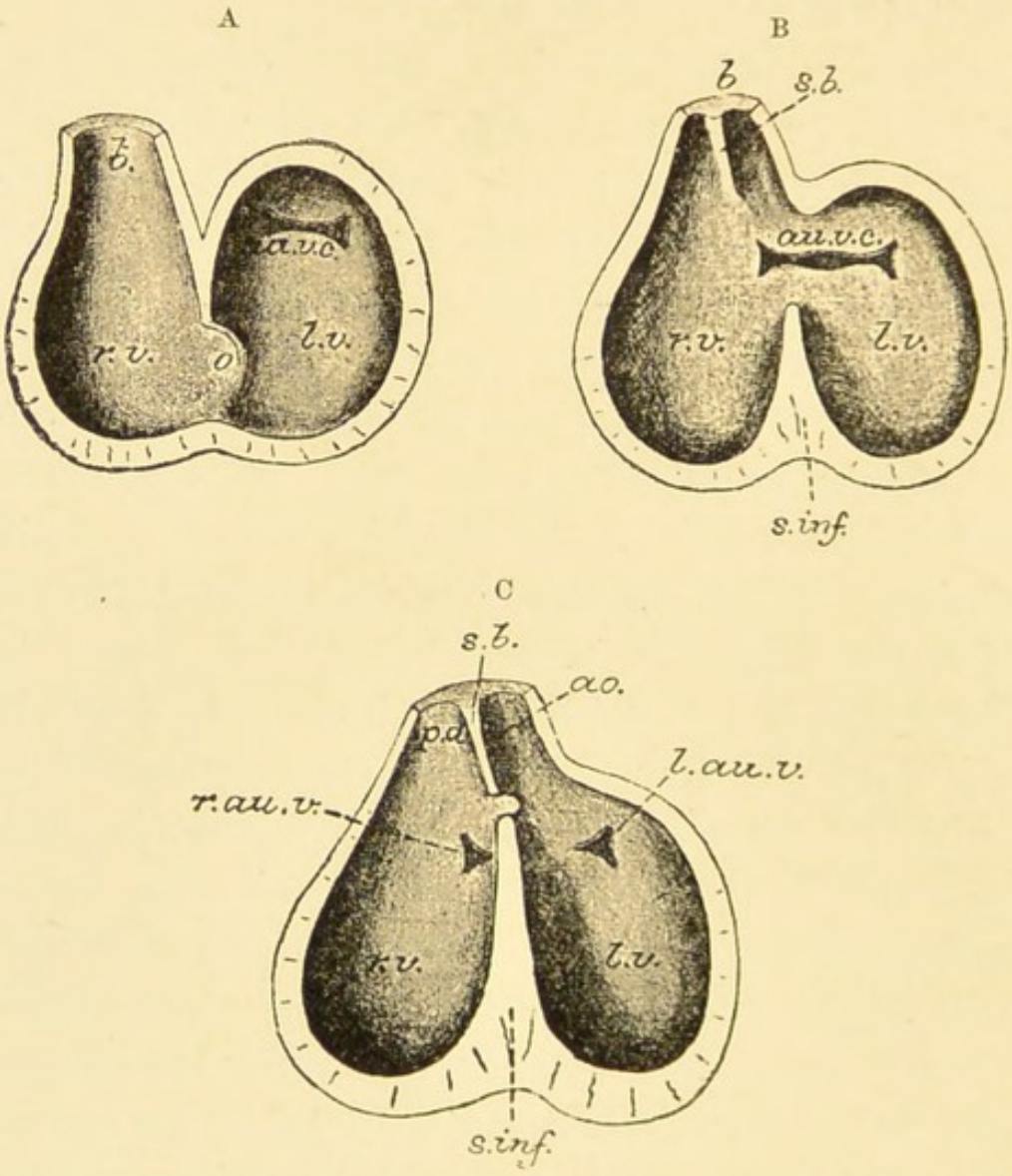


FIG. 96. Diagram to show the formation of the septum of the ventricles and bulb, and the mode of division of the common auriculo-ventricular aperture. (Born.)

au.v.c. (in A and B), auriculo-ventricular aperture, partially divided into two by endocardial cushions; *r.au.v*, *l.au.v*, right and left auriculo-ventricular apertures which have resulted from the division of the common aperture; *r.v*, *l.v*, right and left ventricles; *b*, bulbus aortæ, replaced in C, by *p.a* and *a.o*, pulmonary artery and aorta; *s.b*, septum bulbi; *s.inf*, septum inferius ventriculorum; *o* (in A), orifice between the two ventricles. (Quain.)

The changes that take place in the common ventricle, which eventuate in its division into right and left cavities, will be best studied by reference to Figs. A, B, C, Fig. 96. In A the common ventricle is seen: at its right end it passes into the aortic bulb, while at its left is the single auricular orifice. Figs. B and C show the growth in opposite directions of the septum bulbi and septum inferius ventriculorum, and the displacement downwards and from left to right, and the final separation into two of the auriculo-ventricular orifice. The septum from below grows "upwards and backwards from the antero-inferior part of the tube." The so-called *pars membranacea septi or undefended space* is formed by the lowest part of the septum that divides the original single tube into the aorta and pulmonary artery and grows downwards to meet the septum ascending from below upwards, uniting with the *endocardial cushions*, which divide the common auriculo-ventricular orifice into right and left orifices.

Turning now our attention to the common auricle, we have seen its passage upwards behind the ventricle, the aperture of communication between them being situated on the left side. The aperture forms a canal about which much has been heard recently (Mackenzie, Wenckebach and others). It has been seen how the orifice assumes

a median position, though when the septum advancing from below reaches up to and divides the orifice into two orifices, the right division is situated nearer the septum than the left. The separation is affected by the growth inwards of the so-called "endocardial cushions." The final closure of the septum is affected by the growth downwards of the septum of the bulb, which divides the single canal of the latter into the aorta and pulmonary artery.

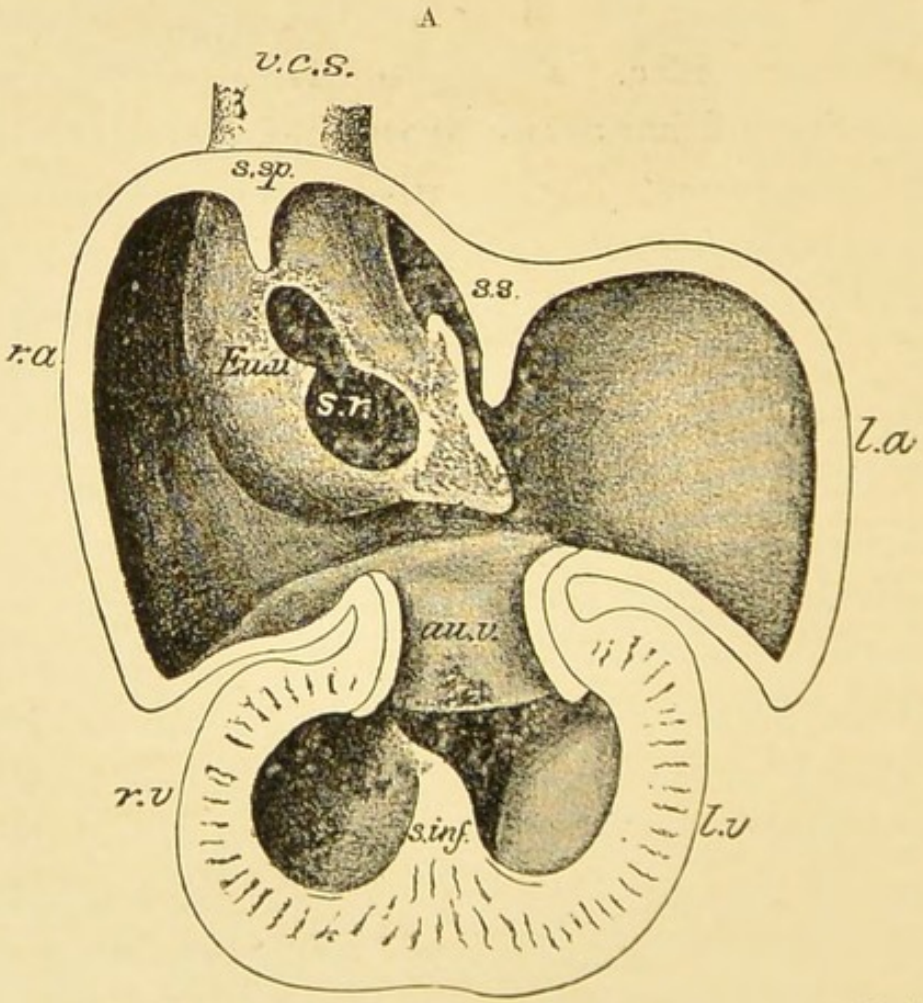


Fig. 97. (Quain.)

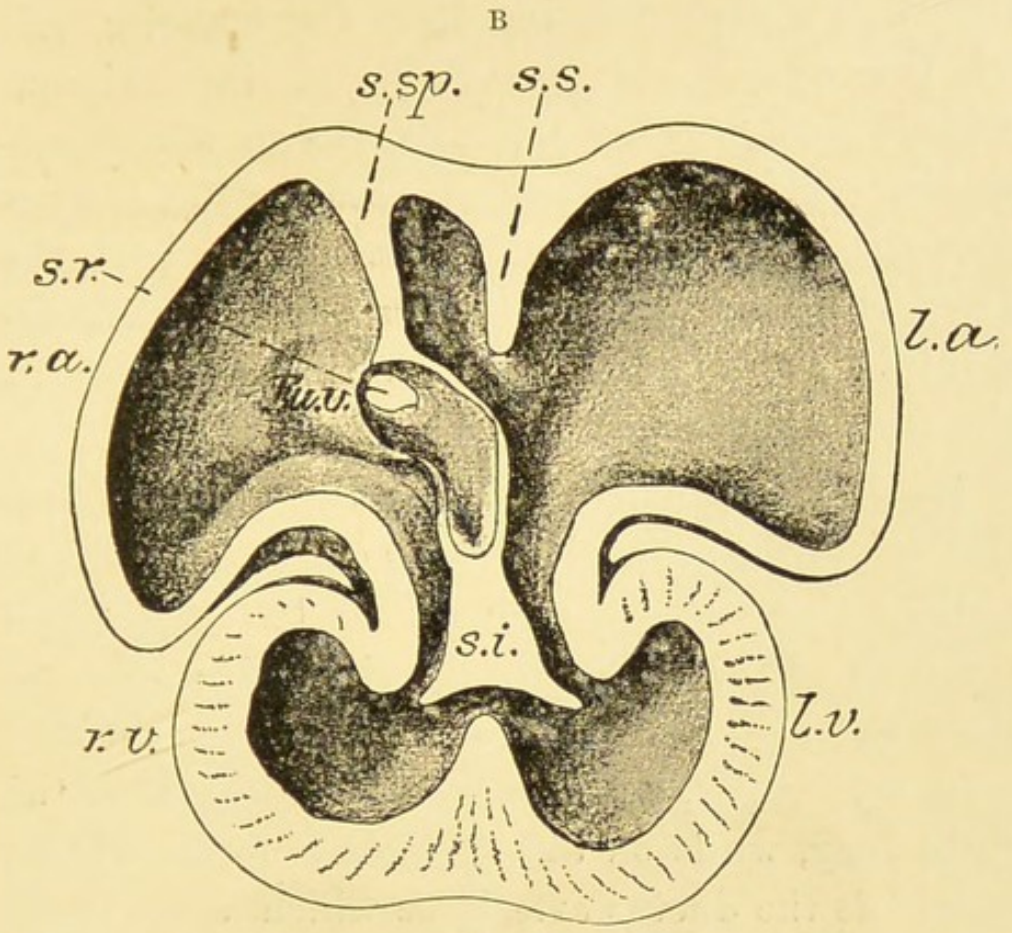


FIG. 98.

Two stages in the formation of the septum intermedium in the heart of the human embryo. (His.)

In A the septum is represented as growing from a triangular area to the left of the sino-auricular orifice; in B it has coalesced with the endocardial cushions, and lies like a stopper in the auricular canal.

r.a, *l.a*, right and left auricle; *r.v*, *l.v*, right and left ventricle; *s.r*, sinus venosus; *Eu.v*, Eustachian valve; *s.sp*, septum spurium; *s.s*, septum superius; *s.inf*, septum inferius; *s.i*, septum intermedium; *v.c.s*, vena cava superior dextra. (Quain.)

The septum between the auricles grows from the upper and back part of the chamber forwards and downwards, this septum uniting with the cushion-like endocardial thickenings of the auriculo-ventricular orifice. Finally the *foramen ovale* forms in this septum, to be ultimately closed by

a second septum starting from the superior wall of the auricle to the right of the original septum and growing like it forwards and downwards. This second septum becomes the *Limbus Vieussenii*, the union of whose margin with the forepart of the valve of the fossa ovalis is instrumental in accomplishing the ultimate closure of the foramen ovale.

In the accompanying figure the ultimate destination of the arterial arches is shown. It will be noticed that the 4th arch on the left side forms the arch of the aorta; on the right side only the right subclavian. The 5th arch on the right side —or rather part of it—forms the branch to the right lung, while on the left side it forms during fœtal life the ductus arteriosus which, after birth, becomes impervious beyond the branch to the left lung, and is known as the *ligamentum arteriosum*. "The upper part of the descending primitive aorta disappears entirely on the right side; that of the left side forms the commencement of the permanent descending aorta."

The condition known as "ectopia cordis" needs only to be mentioned. Three varieties are described: Ectopia cervicalis, pectoralis, and abdominalis. The first is of no practical importance, being met with only in the fœtus. In the second, there is often imperfect closure of the parietes of

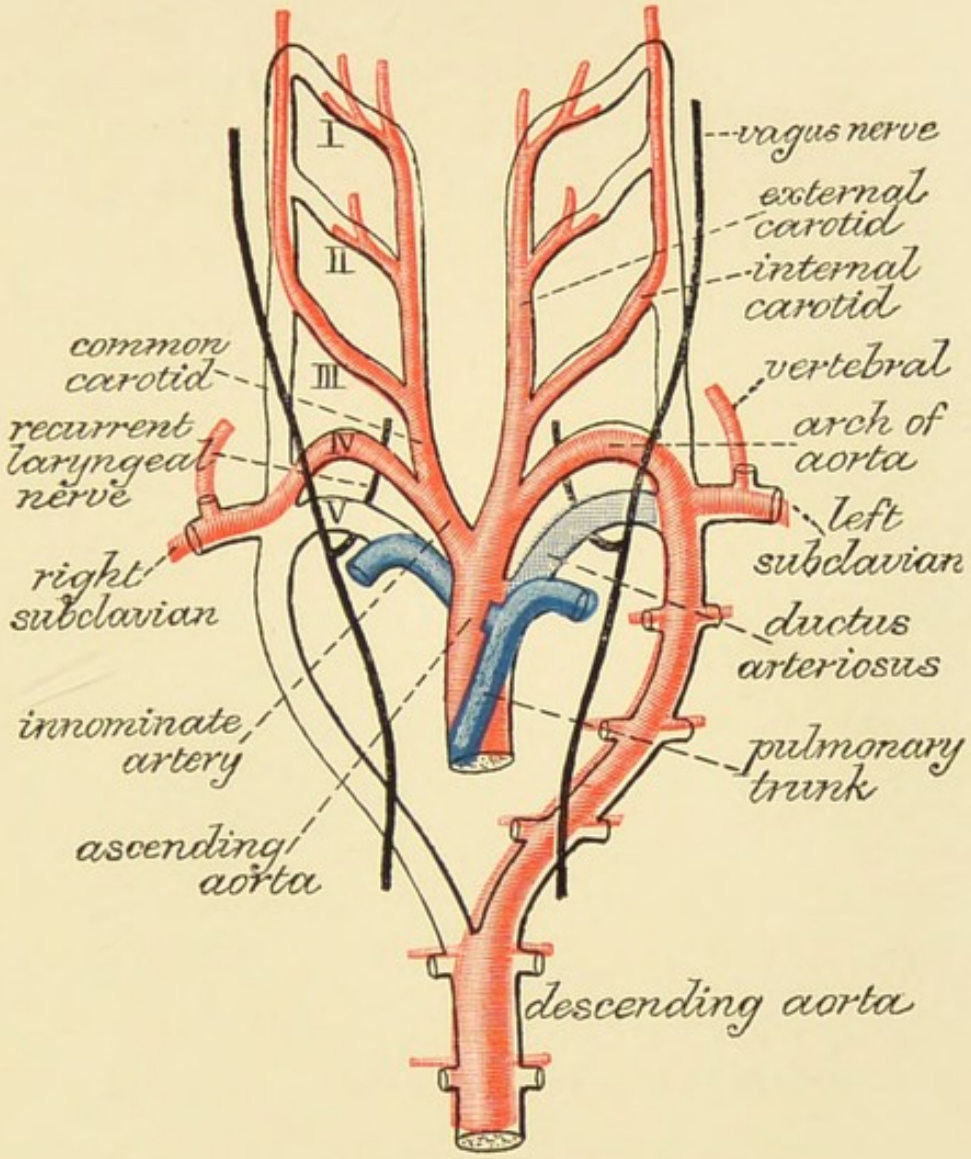


FIG. 99. Diagram to show the destination of the arterial arches in man and mammals. (Modified from Rathke.)

The truncus arteriosus, and the five arterial arches springing from it are represented in outline only, the permanent vessels in colours—those belonging to the aortic system red, to the pulmonary system blue.

From "Quain's Anatomy."

the chest. The third is an extraordinary condition in which the heart lies below the diaphragm. In a subject of such ectopia cordis, who had been a soldier, "the heart was found in the position of the right kidney"!

The heart may be transposed along with other viscera or alone. At the apex of the heart there is rarely an indication of a double apex by the presence of an external groove separating the ventricles. The pericardium may be absent as a great rarity, and a case is on record in which "death was caused by dislocation of the heart during a severe attack of vomiting."

The writer is too well aware of the imperfection of the information given in the above paragraphs. If, however, he has succeeded in raising a desire for more information on the subject in the mind of the student, he will be satisfied.

The varieties of congenital lesions of the heart are numerous, and the lesions are peculiarly apt to be complex. Arrest or irregularity of development and intra-uterine endocarditis are their great source, and both factors are probably often in action together. It is remarkable, moreover, how often rare examples of congenital deformities are brought to light by the abnormal structures having become the seat of septic endocarditis, on the whole a rare disease.

It would be out of place in this work to attempt to consider all the forms of the disease that have been met with often enough to form types. Two forms only will therefore be chosen for description: one because it is by far the most common, and the other because it may be met with in the adult, and is apt to be overlooked or misinterpreted. These two are (1) stenosis of the pulmonary orifice, and (2) coarctation of the aorta at the posterior part of the arch with obliteration of the ductus arteriosus.

Stenosis of the Pulmonary Artery.

This lesion was found by Dr. Peacock to constitute 119 cases out of 181 cases of congenital disease of the heart.

The production of the pulmonary stenosis before the septum is complete, no doubt, tends to interfere with its completion. When the stenosis occurs subsequently to the closure of the ventricular septum, the auricular septum will be found perforated, either by the foramen ovale remaining open or otherwise. The ductus arteriosus, again, remaining unobliterated, may assist in carrying on the pulmonary circulation. When the stenosis is produced subsequently to the closure of the inter-ventricular septum, the tendency is for the right ventricle to atrophy, the

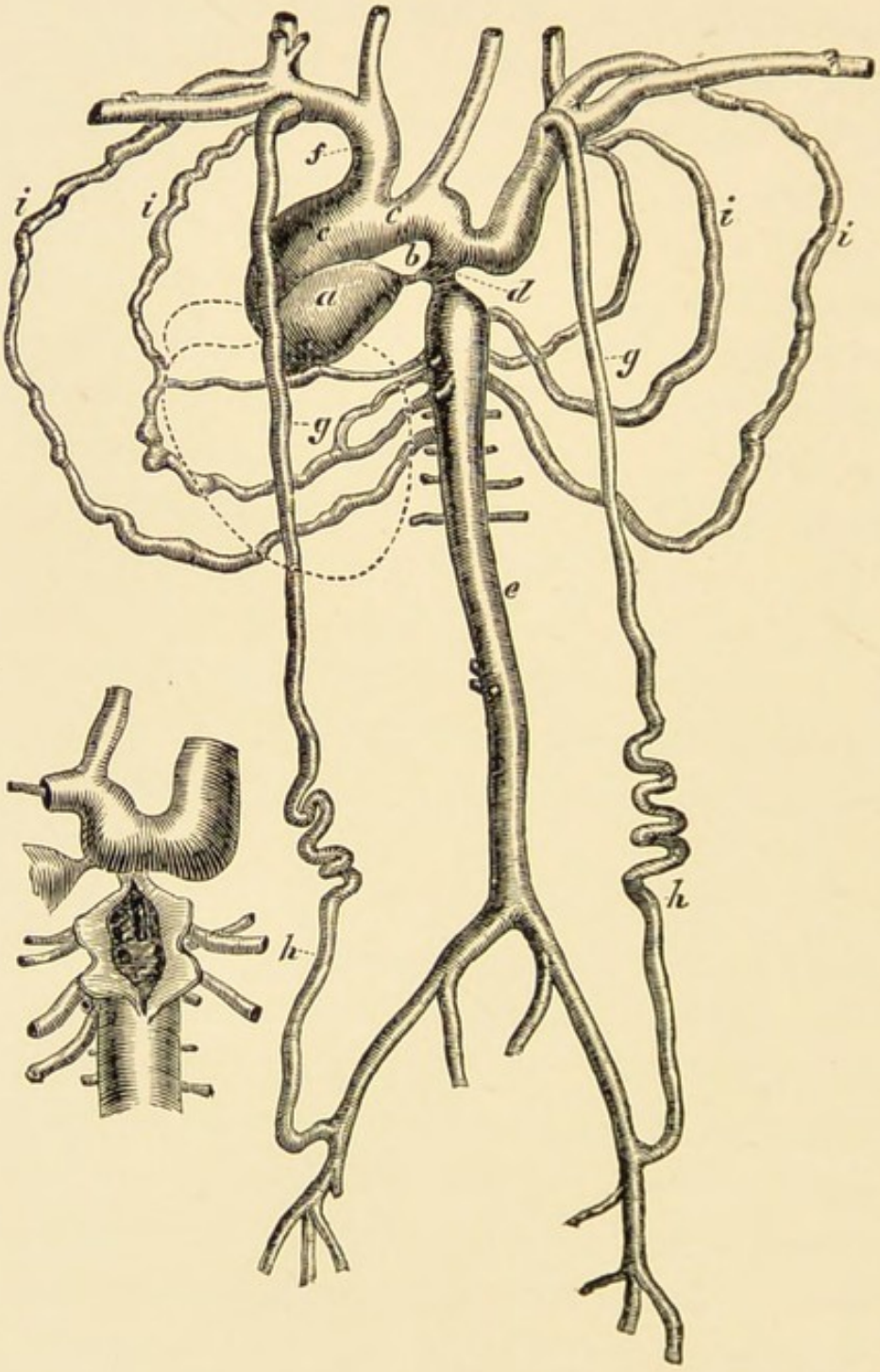


FIG. 100. Coarctation of the aorta: Diagram to illustrate malformation, including extremity of 5th left branchial arch, which forms the ductus arteriosus

From Walshe, "Diseases of the Heart."

foramen ovale remaining patent and the left auricle and ventricle becoming hypertrophied. On the other hand, pulmonary stenosis along with open inter-ventricular septum leads to the hypertrophy of the right ventricle, and the more freely the aorta is in communication with this chamber the greater will be the hypertrophy. Should stenosis of the pulmonary artery proceed to obliteration, the ductus arteriosus remains pervious and conveys blood to the lungs. The bronchial arteries may assist, or special arteries may be formed, arising from the large branches of the arch—innominate, etc., or from the arch itself, or there may be direct communication between the aorta and pulmonary artery.

Coarctation of the Aorta. (Fig. 100.)

The other congenital lesion to be considered—coarctation of the aorta—is formed at the posterior portion of the aortic arch, and involves the extremity of the 5th left branchial arch, which forms the ductus arteriosus. The separation of the arch and descending aorta may be complete, or there may be a connecting cord. The upper intercostal arteries from the 2nd part of the subclavian, communicate with the first aortic intercostals, and become much enlarged and extremely tortuous. The posterior scapular arteries arising from the 3rd part of the subclavian or from the transverse

cervical arteries of the thyroid axes (1st part of the subclavian), likewise anastamose with the upper intercostal arteries. The subscapular arteries also aid in the anastamoses. The internal mammary arteries (1st part of subclavian) anastomose with the aortic intercostal arteries. Diaphragmatic branches of the internal mammary, again, communicate with diaphragmatic branches from the abdominal aorta. Lastly, the superior epigastric branches of the internal mammary, communicating with the deep epigastric arteries from the external iliac, become similarly enlarged and tortuous. Enlarged and tortuous arteries on the back or abdomen should suggest this type of congenital malformation of the aorta, which comes under observation usually in the adult. An allied but rare malformation is obliteration of the aorta just beyond the left subclavian, *i.e.*, between the 4th and 5th branchial arches. In it, the descending aorta receives blood from a patent ductus arteriosus.

There is often question clinically as to mitral stenosis being a congenital condition. Such an origin of the lesion is probably much rarer than is commonly supposed. But malformation may reach such a degree as to occasion obliteration of an auriculo-ventricular orifice, while endocarditic vegetations have been found on fœtal valves. It is rare, however, for mitral stenosis to be met with before the age of 6 or 8 years, though it has been

found at the age of 5 years. Tricuspid stenosis—and it must be remembered that in intra-uterine life, it is the right and not the left side of the heart that is liable to endocarditis—has been met with at the age of 4 months. In another place mention has been made of the fact that when in the adult mitral and tricuspid stenosis in combination are found, the former lesion is always the more advanced. Dr. Carpenter is probably right when in his monograph on "Congenital affections of the Heart" he remarks that "Stenosis of the mitral or tricuspid orifice, particularly the former, is exceedingly rare."

The extraordinary conditions of "Heart with three cavities"—2 auricles with perforate foramen ovale and a ventricle giving rise to an aorta and pulmonary artery in their usual situations, the same with the arteries situated to the left of the ventricle, and again the same with transposed arteries (Carpenter)—and "Heart with two cavities," the ventricle "with a single vessel arising from it supplying both the systemic and pulmonary circulations" need only be mentioned and are of no clinical importance. A remarkable case of a woman aged 39, who had suffered "all her life from cyanosis and palpitation, and died of cardiac failure," and who was found to have *three ventricles*—right, middle and left,—is recorded by Sir Stephen Mackenzie in the *Pathological Society's Transactions*, London, 1879–80.

Symptoms.

The symptoms of the more pronounced forms of congenital disease of the heart are usually recognised soon after birth. *Cyanosis* is usually the first to be recognised. It is specially noticeable in the face, the extremities, and on visible mucous membrane. Efforts, interfering with respiration, exaggerate the lividity in question, so that the crying of the infant is apt to cause it to become "black in the face" in pronounced degree. "Venous stigmata" may be present on the cheeks. *Eclampsia* may be induced or without convulsion the infant may become comatose for several hours, while the cyanosis is intensified. Even *paroxysmal dyspnœa* may occur, there being great temporary increase of the lividity and tumultuous action of the heart. The condition may not be recognised till the child is a year old or older still. The *cyanosis* present has given rise to the old-fashioned name of *morbus cæruleus*. Its cause is less clear than might appear on first consideration, for there may be admixture of blood, as in the case of single ventricle on the one hand, without cyanosis, and cyanosis may be present without admixture, on the other. When there is such lesion as stenosis of the pulmonary artery combined with admixture from

defective septum, the cyanosis is readily explicable. There are probably factors in the production of the discolouration that are not yet sufficiently recognised.

The blood has been shown to contain an abnormal number of corpuscles, as well as an abnormal amount of hæmoglobin; thus in a case of Dr. Gibson's, the blood contained 110 per cent. hæmoglobin, 8,470,000 red corpuscles, and 12,000 white corpuscles per cubic m.m. A similar, though less pronounced change is found in ordinary heart cases of long standing, in which there is a high degree of "venous stasis" and cyanosis. The specific gravity of the blood has been found increased: 1,071—8, but the specific gravity of the blood in the newly-born is normally high.

A marked feature of the cyanosis of congenital heart disease is clubbing of the ends of the fingers and toes, with curved so-called "acorn" nails. This would seem to be only an exaggeration of a symptom of ordinary disease of the heart: clubbed finger ends, for instance, are common enough, though usually in less pronounced form, in cases of the latter, when accompanied by much cyanosis and of long standing.

Among the symptoms of congenital disease of the heart besides those already mentioned are "extreme irritability," gastro-intestinal affections — vomit-

ing and diarrhœa,—jaundice, and hæmoptysis. Patients the subjects of the disease seem apt to die of pulmonary tuberculosis, and are often ill-developed—mentally and bodily. Dropsy is rare in children, but in older subjects the end is brought about—if by the cardiac condition—much as it is in ordinary cases of heart disease, with dropsy, etc., muscle-failure playing the prominent part as in them.

Physical Signs.

The physical signs of congenital disease of the heart, in comparison with the complexity of the anatomical changes, are comparatively simple. In the most common congenital lesion—stenosis of the pulmonary artery—a loud, harsh, systolic murmur, with maximum intensity over the artery in question is usually present, and often accompanied by thrill. Rarely there is a diastolic murmur to the left of the sternum, owing to coincident incompetence of the pulmonary valves. As regards deficiency of the septa: the auricular septum remaining open would seem to be a condition that may not be revealed by any auscultatory or other sign. The writer once met with the condition present in two cases of mitral stenosis, which happened

to prove fatal within a period of a few days, and in which the physical signs had differed in no respect from those of ordinary cases of the uncomplicated mitral lesion. When there is deficiency of the ventricular septum there is often stenosis of the pulmonary artery present, and this latter lesion is much the more likely to have been the cause of murmur present than the patent septum.

The seat of murmur alleged to be due to deficiency of the inter-ventricular septum is, according to Roger and Potain (Broadbent), to be "in the 4th left space, half an inch above the nipple." If any murmur is to be definitely associated with deficient inter-ventricular septum it is probably such a murmur as Sir William Broadbent states he looks upon as diagnostic: "one quite different in character to any that occur in the usual forms of valvular disease. It is harsh and loud, but its great peculiarity is that it never ceases, becoming suddenly louder and higher pitched with the systole, and subsiding into a continuous rumble during diastole."

Dr. Balfour (*Clinical Lecture*, 1898, p. 243) regarded "cases of uncomplicated open ductus arteriosus frequently more or less aneurysmally dilated" as "common enough." An interesting report with post-mortem result was published by the late Dr. Jas. Foulis, *Edin. Med. Journal*, July,

1884, "On a case of patent ductus arteriosus with aneurysm of the pulmonary artery."

The degree of doubt attaching to the significance of the evidence in such cases will, it seems, to the writer, always be great; the signs described being open to a different interpretation.

The systolic murmur audible over congenitally diseased hearts is often very loud and widely conducted over the chest, while associated thrill is common. Dr. Carpenter remarks that "Stenosis of the aorta may originate a bruit heard all over the chest, the maximum intensity being at the apex and not the signs usually recognised as belonging to aortic disease," but some doubt may be experienced in such a case as to the apex murmur being *directly* due to the stenosis. The same author's statement: "From what has been said it will be seen that the conclusions to be drawn from the point of maximum intensity and the directions taken by cardiac bruits in congenital affections of that organ, are very unreliable in themselves, and that great stress must not be laid upon them" is well worthy of note.

Mention has been made of the fœtal heart sounds in the foregoing sections. Students should take the opportunity of becoming acquainted with these in obstetric practice, and noting their peculiarities. Close resemblance between the two sounds so that

they are incapable of being distinguished by the ear is the chief of these. The first sound is like the second, and the intervals between them—1st and 2nd, and 2nd and 1st—tend to be equalised. The so-called *uterine souffle* must also be studied in obstetric practice. The attention implied in the seeking for the fœtal heart sounds, moreover, is excellent auditory practice for the student of ordinary cardiac auscultation.

PERICARDITIS.

Inflammation of the pericardium occurs in varying degrees of intensity and in many different associations. It seldom occurs alone or even as the chief pathological condition. It occurs most commonly in association with acute rheumatism. It has been said that "the number of cases coming under this head is very much larger than that of all other cases put together." When pericarditis, moreover, occurs in the course of acute rheumatism, it seldom represents the only damage wrought by the rheumatic toxine upon the heart: endocarditis will almost certainly co-exist, and in young subjects in whom pericarditis is specially common there may be myocarditis also. This last fact is of great importance in relation with the ultimate dilatation of the heart, that is so apt to result in cases of the kind. At one time it was supposed that an adherent pericardium, the result of pericarditis, exerted a peculiarly pernicious influence upon the heart-muscle or myocardium, but changes of a sub-inflammatory and degenerate type in the myocardium were overlooked. The exudation in

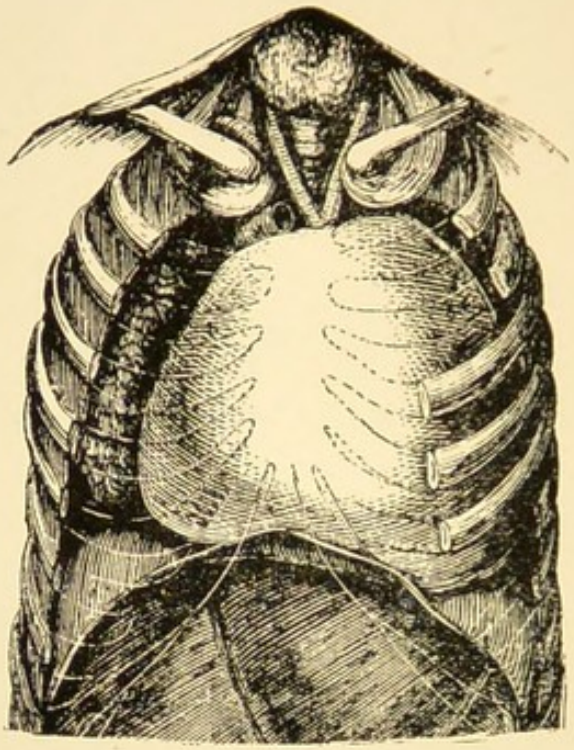


Fig. 101.

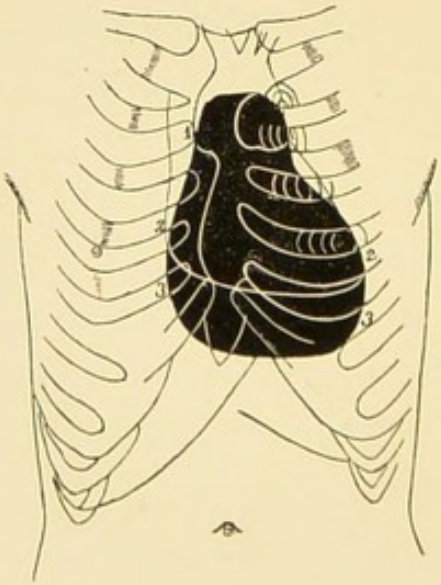


Fig. 102.

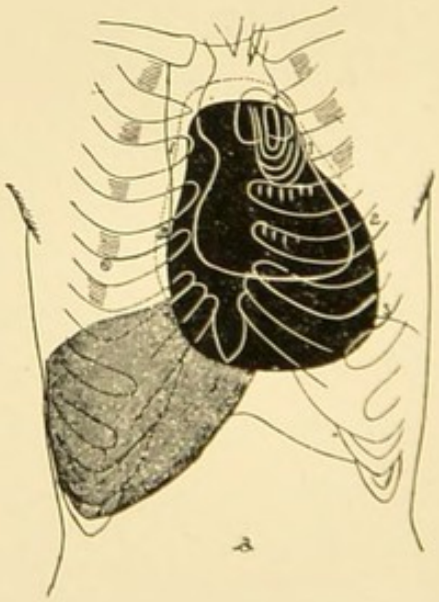


Fig. 103.

rheumatic cases is sero-fibrinous; the changes that occur in the surface of the membrane account for the friction sound that is heard, while more or less copious effusion of serum accounts for the increased area of the dulness that normally represents the heart. When there is no appreciable quantity of effusion the pericarditis is called "dry." Perhaps the next most common cause of pericarditis, is Bright's disease of the kidney. "Granular disease of the kidney" is most frequently complicated with pericarditis. In it, the heart is generally more or less enlarged, and the affection is apt to remain "dry" and without effusion. Friction sound is often extremely well marked, a result to which the hypertrophy of the heart probably contributes.

Septic pericarditis is that associated with pyæmia and various septicæmic conditions. The exudation is apt to be purulent—and presumably purulent from the first—in such cases. Thus a convalescent from typhus was noticed one morning to appear ill. His pulse was found to be extremely irregular, while his "cardiac" dulness had increased in size, and his hitherto normal heart sounds had become indistinct and indeed barely audible; *post-mortem* examination in a few days revealed purulent pericarditis. In the only case of leprosy, that ever came under the writer's observation in the Man-

chester Royal Infirmary, the patient died of pericarditis. Pleuro-pneumonia, generally of the left lung, is not very rarely complicated by pericarditis, or it may be simple pleurisy that seems to spread to the pericardium. Great care must be taken in such a case to make sure that the friction is truly pericarditic and not only *pleuritic of cardiac rhythm* occurring along the anterior edge of the left pleura. Hydatids and abscess of the liver have been known to perforate the pericardium and set up pericarditis.

It is said that a blow over the precordial region has caused pericarditis. Fractured ribs are a more likely cause. Sharp bodies, such as "false teeth, needles, and fishbones," have been known to injure the pericardium from behind, having been swallowed and lodged in the gullet. A gastric ulcer even has led to perforation of the sac.

Pericarditis, if we are to accept friction-sound as a certain indication of the disease, may be a quite trivial ailment and occasion not even discomfort to the patient. Thus a convalescent from bronchitis, with chronic emphysema, was sitting by his bedside in the Infirmary, dressed and ready to go home. Quite casually his heart was auscultated, and, much to the surprise of the writer, a fine grazing friction was heard instead of the usual normal heart-sounds. The indignation of the

patient was great at his discharge being cancelled. He had no pyrexia and no pain, and in a day or two the friction sound had disappeared. The writer met with a similar case when House-physician in the Edinburgh Royal Infirmary. In striking contrast with such cases, are those of purulent pericarditis such as that related as occurring after typhus, in which there was no friction sound to be heard in the presence of a profound toxæmic condition and overwhelming illness. Rheumatism, as the most common cause of pericarditis, is always to be first suspected when the signs of pericarditis are unexpectedly found. Joint-pains are apt to be trivial in the case of children, and, even in the adult, they may have to be enquired for, so slight may they be. A history of previous rheumatism is of some assistance. On the other hand one has to be careful to avoid regarding manifest arthritis as of rheumatic origin, when it is really septicæmic, pericarditis being a complication of both conditions.

Symptoms.

The *posture* assumed by patients suffering from pericarditis varies. Most commonly the patient lies on the back. When there is any great degree of dyspnœa the tendency is towards elevation of

the head and shoulders. The writer remembers a patient with chronic bronchitis and emphysema, who eventually developed pericarditis. His usual posture was orthopnœic, but with the advent of pericarditis and no doubt consequent cardiac failure, he lay flat on his back. On another occasion an old woman was admitted to the infirmary with Chronic Bright's Disease and dropsy. It was noticed that she always not only sat up in bed but bent her body forwards. On auscultation of her heart no cardiac murmur was audible, but on percussion it was evident that she had an enormously distended pericardial sac—apparently a simple dropsy rather than an inflammatory effusion. In a few days the large area of dulness had greatly diminished and the patient no longer retained her peculiar position. Dr. Walshe remarked that "The decumbency is least commonly on the left side": notwithstanding, such decumbency is occasionally assumed, generally when the case has been of some duration. This posture is assumed in order that full play may be given to the right lung, the expansion of the left lung being seriously compromised by the pericardial effusion. If the patient becomes restless, the trunk is moved little, though the arms may be tossed about. Exceptionally there is jactitation of the trunk itself: in delirium usually. An anxious

physiognomy is usually assumed, largely influenced by the degree of dyspnœa present or by pain or indefinite feeling of distress in the chest.

Pain. It has been estimated that pain of one kind or another is present in 70 per cent. of cases of pericarditis (Sibson). It may be "slight in amount or of agonizing severity," the latter exceptionally. Its seat is the precordium most commonly, but it is occasionally experienced in the epigastrium, pressure upon which may aggravate it. Pain is most likely to be present early in the illness. Dr. Sibson long ago noted tenderness of the skin over the precordium, no doubt a "referred" condition. Respiration and movements of the trunk are apt to aggravate the pain. A pain deep in the chest and between the shoulder blades has been found to be increased by swallowing and eructation, or these acts only may determine pain. Dr. Frederick Roberts (*Allbutt's System*) remarks: "In exceptional instances pain of an anginal character shooting up the left side of the neck, to the ear, to the shoulder or down the arm, is associated with acute pericarditis." The writer once met with such a case, the friction sound proving transient and the pericarditis being probably limited in area. Pleurisy is of course a not very rare complication, and apart from it, affection of the diaphragmatic

surface of the pericardium may be supposed to produce characteristic shoulder pain.

Dyspnœa. When the accumulation of fluid has taken place in the pericardium distressing dyspnœa is apt to result, for not only is the action of the heart embarrassed, but the lung-space is seriously encroached upon.

In rheumatic cases, when there is pericarditis, it will seldom happen that this is the only pathological condition in the heart: endocarditis is almost to be inferred, and if there be not actual myocarditis there will almost certainly be debility of the heart muscle under the depressing influence of the contiguous inflammation, whether or not there be effusion in embarrassing amount. Dyspnœa is thus the rule in copious effusions, and as the encroachment is upon the vesicular structure of the lungs, what may be called the "pneumonic type" of respiration predominates, though owing to bronchial catarrh and the presence of secretion in the "bronchial tree," the "laboured type" of respiration may gain the ascendancy. The causes of dyspnœa in pericarditis with copious effusion are thus complex. The condition of the *pulse*, apart from intrinsic cardiac conditions, varies much. It is usually simply accelerated in the early stages, and its tension is low. In malignant (purulent)

cases, it may become very irregular or barely perceptible almost from the outset, as in the typhus case related, but it may be irregular in cases with serous effusion. Rarely the pulse remains normal in rate or is actually retarded. The writer has met with such conditions only in the apyrexial dry pericarditis of "granular kidney." In all cases the pulse should receive the most careful attention of the observer from day to day, inasmuch as its *changes* during the course of the illness may be fraught with significance and with warning.

More or less *pyrexia* is almost invariably present, except in Bright's Disease and in those cases already mentioned in which the only evidence of the disease is a *physical sign* namely *friction-sound*, and in which the physical change undergone by the serous surface may be supposed to have passed little beyond a nutritive change interfering with its perfect lubrication. In chronic Bright's disease and uræmia, it is notorious how little fever there may be in the presence of conditions that usually produce marked pyrexia. On the other hand the decided tendency to *hyperpyrexia* in acute rheumatism must be borne in mind. Such hyperpyrexia may be, and no doubt often is, altogether independent of co-existing pericarditis. Even in a purulent case the writer has found pyrexia barely manifested.

A short, somewhat hacking *cough* is not uncommon, especially when there is effusion, but if there be bronchial catarrh, expectoration is likely to occur. Feebleness of the voice has been noted, but the patient can usually raise his voice with an effort. Such *aphonia* as there may be resembles that occasionally observed in severe spasmodic asthma. There is no reason to suppose that there is any paralysis or paresis of the recurrent laryngeal nerve. *Hiccup*, "distressing and painful," has occasionally occurred, suggesting irritation of the diaphragmatic portion of the sac. *Vomiting* is "sometimes a marked symptom in acute pericarditis." The writer has seldom observed it.

Grave *nervous symptoms* have been associated specially with pericarditis, but it is probable that such nervous symptoms are the result of a general toxæmia, and have no direct relationship with the pericarditis. Among such symptoms are "delirium, coma, temporary insanity, rolling of the head from side to side, and tetaniform symptoms," including *risus sardonicus*. Chorea may, of course, occur in association with rheumatism or alone, usually in a rheumatic subject. In most cases of pericarditis, the mind remains clear. In a rheumatic case the advent of delirium should suggest hyperpyrexia in the first instance, and all the more if joint pains, previously present, diminish or cease.

Occasionally pericarditis with non-purulent effusion runs a protracted course, and in such a case the ordinary indications of disturbance of the circulation — venous stasis — may supervene from cardiac muscle failure, engorgement of the liver and dropsy supervening. In a remarkable case of the kind under the care of the writer, while the dulness due to the pericardial effusion was enormous, there was never any trace of friction-sound, murmur, or bruit-de-galop, the physiological heart-sounds continuing throughout. Post mortem revealed a large pericardial serous effusion with vascular false membranes. Purulent effusion is not necessary, therefore, in order that a pericarditis may be "silent."

Physical Signs.

The diagnosis of pericarditis must rest essentially upon the result of the physical examination.

On Inspection *local bulging* may be noticeable over the distended sac, the ribs being widely separated and raised upwards. Such "fulness" may extend "from the 2nd to the 6th or 7th rib," but to the eye it often seems to involve the whole left side, the movement of which is much interfered with. In children and young subjects this condition may be so pronounced, the effusion being copious, that the accumulation of fluid may be

regarded as pleural rather than pericardial. This will be more intelligible when certain signs observed at the back of the chest in ordinary cases have been considered. Dr. Ashby long ago called attention to the subject.

Over the pericardium slight *local œdema* may be found. This does not necessarily mean that the fluid in the sac is purulent, but it is noticed generally in cases of considerable standing.

Dr. Ewart (*Brit. Med. Journ.*, 1896, vol. i., p. 717) believes that what he calls "*the first rib sign*" is of considerable use in cases of copious effusion. This sign is due to raising of the left clavicle so that the upper edge of the 1st rib can be felt as far as the sternum. "This points to a raising of the clavicle not only in its outer but also in its inner portion, and to a relaxation of the ligament between it and the first rib. The left clavicle is apparently lifted to a higher level than it is possible for the first rib to reach."

Fulness or actual bulging of the epigastrium has been described and is supposed to result from depression of the diaphragm. It must be remembered that the embarrassment of the heart occasioned by copious effusion and the contiguity of the inflamed tissue, may lead to passive congestion of the liver.

Elevation of the apex-beat of the heart has long

had a place among the physical signs of pericardial effusion, and it is comparatively recently only that doubt has been cast upon the sign. The question is as to the impulse seen representing the apex of the left ventricle or a portion of the ventricular wall higher up. "The impulse is not . . . that of the apex of the heart, but rather of its base" (Ewart). In the state of present knowledge it seems impossible to resolve the doubt, but the sign is not of such supreme value that much regret need be experienced if it be lost.

The possible fact of previous enlargement of the heart has to be taken into consideration when the apex-beat is found "somewhat lower than normal," as it is alleged to have been in certain cases of acute pericarditis with effusion.

Diminution or disappearance of visible impulse, as the effusion increases and the heart-muscle becomes debilitated, is only what might be expected to occur.

An "undulatory impulse" is another of the signs formerly believed to be indicative of pericardial effusion, that seems to have "passed."

Palpation.

Palpation is of no great value in the diagnosis of pericarditis, at least taken alone. Considered

along with the results of other physical methods it may prove of greater value; thus *absence of impulse* may be combined with increase of dull area on percussion. It may be used to check the observation of impulses ascertained by inspection, which however may be found hardly appreciable by it. In pericarditis the sign that palpation specially elicits is friction-*fremitus* invariably associated with loud friction *sound*—a much more important sign.

Percussion.

If inspection and palpation have yielded only indifferent evidence of acute pericarditis with effusion, the remaining methods will redeem the value of the physical methods of diagnosis. As fluid accumulates in the pericardial sac the *dull area*, that normally represents the heart, undergoes increase. Extension is usually first noticeable in the upward direction, but very little later lateral extension will be noticeable at the right lower corner to the right of the sternum, and also at the left lower corner. The extension of dulness upwards soon broadens and the whole area assumes the outline of a "bag of fluid spreading out at the base" (Ewart). If the sac becomes extremely distended the pyriform outline of dulness is lost in large degree and becomes rounded. Figs. 101, 102, 103.

Dr. Sansom has called attention to the abrupt transition from fairly full resonance to absolute dulness, that may be met with in pericarditis with effusion. This feature was very well marked in the case related of emphysema complicated with pericarditis in which the usual orthopnœic position of the patient seemed to be negatived by a syncopal condition (p. 264). An important sign of effusion in cases in which the position of the apex-beat can be identified, is the extension of the left border of the dull area some distance to the left of the apex-beat.

Auscultation.

The great auscultatory sign of pericarditis is *friction sound*, which is usually first noticed about the base of the heart, where there is freeest movement. The occurrence of effusion interferes little if at all with the sign, and it is often heard distinctly in the presence of copious effusion. On the other hand it must be remembered that, while in cases of purulent exudation, absence of friction is occasionally observed, like absence of the sign, in what seems *post-mortem* to be ordinary fibrino-serous exudation with abundant false-membrane. may occur. Moreover, in such cases the heart-sounds may hardly depart from the normal, and that throughout the course of an illness of several

weeks' duration. Again, in such cases debility of the heart muscle may be induced to such a degree that the liver becomes engorged and dropsy appears.

It is with reference to cases of the kind, rare though they be on the whole, that it is of great importance to remember the simulation of the dull area of pericardial effusion by the dull area of certain generally dilated hearts. Sir William Gull long ago called attention to the possible confusion between the two very different conditions: pericardial effusion and general dilatation of the heart, in which latter, enlargement of the infundibulum supplies the extension upwards; enlargement of the right auricle, the lateral extension of the right lower corner; and enlargement of the left ventricle, the lateral extension of the left lower corner. Feeble physiological heart-sounds may represent the auscultatory result of examination in both cases.

When friction sound is at all well marked in pericarditis, it annihilates the physiological heart sounds as well as any murmurs there may be, and so the student will be spared the duty of identifying these and describing their characters, in the presence of friction sound. It seems to the writer to be quite useless to attempt a verbal description of pericarditic friction sounds. Suffice it to say—what has been already indicated—that friction sounds, unlike endocardial murmurs, bear no rela-

tion to the physiological heart *sounds*, even when these remain audible, which is exceptional, though of course towards the end of the illness the physiological sounds will have become audible before all friction sound has gone. At the beginning of the illness too, friction sound and physiological sounds or endocardial murmurs may be heard together. Speaking generally friction sound is double—systolic and diastolic—corresponding to the systole and diastole of the ventricle, and there are not the pauses or periods of silence that exist between the physiological heart sounds and precede or follow most endocardial murmurs. With regard to the physiological heart sounds it must always be remembered that they occupy but a portion of the systole and diastole of the ventricles. The idea of presystolic or auricular-systolic friction sound being capable of identification is probably purely fanciful. The murmurs met with in certain cases of free aortic incompetence, in which all physiological sound is obliterated, certainly may closely simulate varieties of grazing friction sound. In the aortic case, abundant other evidence of the valve lesion will always be forthcoming, if the ear cannot alone resolve the doubt, which seldom happens. Of course, aortic incompetence and friction sound may co-exist, but it is in the highest degree improbable that when this rare event occurs, the friction sound will assume characters, which are no less rare.

PNEUMO-PERICARDIUM OR HYDRO- (OR PYO-) PNEUMO-PERICARDITIS.

Cases of the rare condition: the presence of gas within the pericardial sac, have been recorded by Graves, Stokes, Walshe, McDowel and Warburton Begbie. Probably in all clinical cases the sac is placed in communication with a gas-containing viscus. There seems to be no doubt that the condition of hydro-pneumo-pericardium may result from decomposition *post mortem*, and so good an observer as Laennec held that such a condition was not very rare during the closing hours of life. In all cases met with clinically, it may however be inferred that the gas has gained entrance to the sac by perforation of a viscus. In Graves' case there was a hepatic abscess in communication on the one hand with the stomach, and on the other hand with the pericardium. In McDowel's case the communication was with a pulmonary cavity. In Walshe's case, traumatism to the œsophagus and communication of this tube with the pericardium, resulted from a juggler's knife being swallowed, while in Dr. Begbie's case cancerous ulceration of the œsophagus opened the pericardial

sac. In considering a condition so rare, it is important the student should bear in mind that an air-containing cavity in the vicinity of the heart, but without any communication with the pericardial sac, will occasionally—one may say fortunately, rarely—lead to extraordinary acquisition of metallic phenomena by the physiological heart sounds or by quite ordinary abnormal auscultatory signs, for instance "the crepitating and mucous râles of bronchitis and pneumonia, the friction sounds of pleuritis, and finally, the sounds of the heart and the friction signs of pericarditis, may present a distinct metallic character" (Stokes). The most marked instance of the kind met with by the writer occurred in a case of influenza, complicated by pleurisy among other pathological conditions. In it, as Stokes described, "the friction sounds of pleuritis" were the subject of the phenomena, and were audible at a distance from the patient.

To return to pneumo-pericardium: "this condition," as Walshe remarked, "must exist temporarily, be it for ever so few minutes, as the sole result of perforative communication between the pericardial sac and any hollow viscus containing gas; but in this isolated state *it has never been observed, pericarditis having supervened before clinical examination has been made.*"

The signs, to be referred to in diagnosis, imply therefore the presence of *air and fluid*—serum or pus. If the quantity of gas be large, tympanitic resonance may be found over the pericardium on *percussion*, and this be alterable with dulness by changing the posture of the patient, the fluid gravitating to the lowest part and the gas rising.* The influence of "horizontal conduction" of resonance from adjoining viscera over the cardiac region already referred to, must be borne in mind (p. 91). As regards auscultation: the heart sounds with their abnormal characters may be heard at a distance from the patient as mentioned by Stokes. Of the auscultatory phenomena themselves the description in words is exceedingly difficult—practically impossible. Dr. Begbie spoke of an "extraordinary guggling sound which accompanied the heart's action—a sound which cannot . . . . be better described than as a churning splash." Dr. Stokes spoke of "a mixture of various attrition murmurs with a large crepitating and gurgling sound, while to all the phenomena was added a distinct metallic character" (and) "the region of the heart gave a tympanitic *bruit-de-pot fêlé* on percussion." As remarked by Dr. Begbie "These signs will be still more available if the guggling sound has been noticed to succeed a distinct friction sound, and

* It will seldom be safe to make such an investigation.

the tympanitic has replaced a dull percussion note."

Grave as is the condition of hydro-pneumo-pericardium, it is "not necessarily of fatal import." Of course much will depend upon its cause in the individual case.

The student is again reminded of the extreme rarity of hydro- and pyo-pneumo-pericarditis, and of the altogether remarkable character of the auscultatory phenomena to which it gives rise. He must, moreover, remember that a large gas-containing cavity *in the vicinity merely of the heart*, though not in communication with the pericardial sac, may occasion auscultatory phenomena, that may be confused with those of hydro- or pyo-pneumo-pericardium.

ADHERENT PERICARDIUM.

It is probable that various pathological conditions of the heart have been associated together simply because of the presence of an adherent pericardium. The fact of adherent pericardium being not very rarely found *post-mortem* in the absence of obvious pathological condition of the heart itself has been already emphasised. On the other hand, the associated conditions of the heart-muscle are often of supreme importance. Long ago it was pathologically recognised that dilatation of the cavities and universal adhesion of the pericardial surfaces, evidently of long standing, were often associated. In such cases the thickening of the adherent pericardium often indicated a high degree of pathological change in the past. In the consideration of the associated conditions, it has to be borne in mind that whatever debilitates the heart-muscle is likely to take expression in enlargement of the cavities. It is this vital law, that seems to nullify and make of no effect, the actual support to the ventricular walls, that might be supposed to be afforded by the adherent and thickened pericardium. Nay, there are a few exceptional cases

that even give countenance to such a supposition. Dr. Frederick Roberts, in *Allbutt's System*, remarks that "In children the natural growth and development of the organ may be prevented; or it becomes small and atrophied, its walls being grasped and compressed, and its cavities forcibly contracted in size by the dense, thick, tight envelope surrounding them." The explanation of such cases is difficult. The heart of John Hunter was small, and had a thickened pericardium, but he died in a paroxysm of angina pectoris after 24 years of liability to that disease. In another case of angina pectoris, the patient being aged 27, a similar condition of heart was found—that is to say, the heart was small in the presence of old thickening and adhesion of the pericardium. The coronary affection of angina pectoris is not referred to here: attention only is called to the possibility of adherent and thickened pericardium being associated with a small rather than a large heart. While the writer has thought it necessary to call the reader's attention to such cases, he would emphasise the fact that their occurrence is exceptional. In John Hunter's heart, for instance, with its diseased coronary artery, and so impoverished blood-supply, it may be admitted, perhaps, that the pericardium did exert a supporting influence tending to counteract the usual result of cardiac muscle-failure—namely dilatation.

Pericardial adhesion exists in a great variety of forms, in some of which its pathological significance is quite trivial: it may be partial, and such partial adhesions may be drawn out into filaments or bands, bearing witness to the traction to which the adhesions had been subjected. Sibson believed that local adhesions were most common a little above and to the left of the apex, and along the line of the ventricular septum. It is important to remember that pericardial adhesion may be internal, *i.e.*, between its own serous surfaces, or external and with adjoining structures, or both internal and external. Moreover, the degree of thickening varies greatly; in some instances a thickness of half-an-inch is reached. Naturally double adhesions — internal and external — might be expected to embarrass the heart-muscle in greatest degree, and experience would seem to bear out the expectation.

Lastly the pericardium may be much thickened without adhesions of its surfaces.

It has been shown how simple cardiac muscle-failure leads to incompetence of the auriculo-ventricular valves — a condition that ultimately must embarrass cardiac action, even if its immediate effect were to give temporary relief to the heart-muscle. Incompetence of the auriculo-ventricular valves is apt to follow the muscle-failure

associated with thickened and adherent pericardium. On the other hand, in the presence of actual valve lesion, it cannot be doubted that the like condition of pericardium often exerts a harmful influence upon the heart-muscle in its struggle to counteract the effects of the valve lesion.

In the consideration of the effects of valve lesions upon the heart muscle—failure of which is so apt to take expression in dilatation of the cavities—it is well to bear in mind, and especially with regard to rheumatic cases in young people, the possibility of "adherent pericardium," and of its being a factor in the embarrassment of the heart muscle.

Clinical Recognition.

"It is a familiar fact that they (pericardial adhesions and thickenings) are frequently met with at necropsies in various degrees, when they have not been diagnosed during life. It may be acknowledged at once that their diagnosis is often, for obvious reasons, impracticable, or may be matter of great difficulty or mere surmise; not uncommonly, indeed, there is no reason whatever even to suspect their presence." These are the admissions of an able observer (Dr. Fred Roberts in *Allbutt's System*) who, in another place, asserts

that "to teach that the diagnosis of adherent pericardium is impossible, is absolutely wrong and misleading." The writer must, for the present, be placed under this condemnation, but he would not be pessimistic, and still looks for the discovery of some infallible or at least thoroughly reliable sign of the condition. All he contends is that no such sign is as yet forthcoming.

Symptoms.

There is probably no distinctive symptom of adherent pericardium.

Pain has been described as a result—pain of no great severity and of "dragging" character. With such pain there may be precordial oppression, and, it is said, "inability to take a deep breath," . . . . "especially when the external adhesions are extensive." It seems difficult to the writer to attribute such symptoms to the pericardial adhesion: they are symptoms not unknown in cardiac disease apart from pericardial adhesion. As to the other symptoms: they may be relegated to the ordinary category of the symptoms of cardiac muscle failure, to which pathological condition they may indeed be referred. It has been supposed that the right side of the heart is specially crippled in cases of this kind. Dr. John Broadbent remarks: "that when symptoms of right ventricle failure super-

vene in cases in which there is no evidence of left ventricle failure, due to valvular disease or kidney mischief, constant high tension, or other obvious causes, or of lung disease, such as chronic bronchitis, etc., to account for their appearance, the presence of adherent pericardium should be suspected as the cause." But what of the not rare cases of mitral stenosis without distinctive sign?

The writer is loath to enter into any description of signs supposed to indicate pericardial adhesion. When he has succeeded in strongly suspecting the existence of such adhesion during the patient's life, and the suspicion has been justified by the *post mortem* inspection, the success has never depended on the result of physical signs alone—much less on any single one—but upon a wide survey of the case as a whole: its history, its symptoms, and the results of physical examination. At best a "happy guess" was the most he could claim.

Authors commonly think it their duty in text-books to give views that have been promulgated by others, although they themselves have little or no faith in their correctness. The writer of this work does not feel constrained to adopt this system: he does not think it right to burden the student's mind with theories, that he believes have little or no basis in fact. Once, and once only, has

he observed clinically a physical condition on examination of the heart that gave any countenance to the belief that "adherent pericardium" was capable of recognition by physical examination. The case occurred while he was Resident Medical Officer in the Manchester Royal Infirmary. No cardiographic examination was made. There was no proper "apex-beat," but the ventricular portion of the heart, including presumably the apex, fell in with the chest wall during systole, to rebound during diastole. *Post mortem:* Not only were the layers of the pericardium adherent to one another, but the adjoining pleuræ and the external surface of the pericardium were all matted together and to the chest wall.

One feature of the case seemed specially noteworthy, and that was the absence of a true apex-beat. A similar case has never occurred in the writer's practice, though he has been continually on the outlook for it. Rightly or wrongly, the multiple serous inflammation that had given rise to the condition was attributed to syphilis, the patient having been long syphilized, while no more likely cause was in evidence.

Immobility of the apex-beat and of the borders of the left lung have had importance attached to them in the recognition of adherent pericardium—the writer would add—largely from the theoretical

standpoint,* for they are conditions difficult to demonstrate practically.

As regards Friedreich's sign of collapse of the large cervical veins with the ventricular diastole, it seems idle to discuss it, when the polygraph is at work, and may be expected soon to shed fresh light on the sign, if such it be.

So-called *pulsus parodoxus* is another sign that is still *sub-judice*. Dr. Frederick Roberts speaks of it in relation with the diagnosis of adherent pericardium as "by no means trustworthy," and in holding the same belief, the writer is glad to have this recorded opinion of so good a clinical observer.

The condition of pulse termed *pulsus paradoxus* is portrayed in Fig. 79. On examination with the finger the pulse becomes almost or entirely imperceptible during inspiration.

The question of the diagnosis of adherent pericardium will be again considered when chronic mediastinitis is under discussion.

INDURATIVE MEDIASTINO-PERICARDITIS.

The cases of cardiac muscle-failure that come under this designation may be regarded as forming examples of a fairly definite clinical entity, which has been carefully studied in Manchester by

* As this work is passing through the press a case occurs which would make the writer express less scepticism in the matter

Dr. Thomas Harris, whose monograph must be consulted by readers, who desire still further to study the features of the disease.

Besides adherent pericardium in the ordinary sense, there is in cases of the kind "marked increase of fibrous tissue in the mediastinum, not infrequently associated with a caseous affection of the lymphatic glands of the mediastinum, and adhesion of the exterior of the pericardium to surrounding parts" (Harris).

Dr. Harris, in his monograph (*Indurative Mediastino-pericarditis.* London: Smith, Elder & Co., 1895), considers two allied states: namely, (1) pericarditis externa and pericarditis interna, "with very little and sometimes no general mediastinitis," and (2) chronic mediastinitis: "increase of fibrous tissue in the mediastinum without any internal pericardial adhesions."

Indurative mediastino-pericarditis is probably most frequently met with in young subjects, though it is by no means necessarily limited to such.

It occurs more frequently in males than females. Usually the onset is gradual and insidious, but sometimes there has been an acute illness, in which the chest was implicated, and from which the chronic ailing dates. "Tubercle appears," Dr.

Harris remarks, "certainly to be not an infrequent associate of the disease."

For the most part the clinical features, met with, are simply those of cardiac failure. In general aspect, the cases resemble those of mitral stenosis: thus the patient may show congestion of the cheeks, swollen veins in the neck, general cyanosis, and specially congested liver, and perhaps ascites as the prominent, if not only, dropsy. Moreover, in both conditions there may be no cardiac murmur present, or if there be murmur present, it may be only one of mitral and tricuspid incompetence such as is not distinctive of either pathological condition, and may occur in both. It has been supposed that the patient stoops forward specially when he sits up in bed, but this posture is very far from being invariable in mediastino-pericarditis, and it is met with in ordinary heart cases and in some intra-thoracic aneurysms, so that no specific value can be attached to the sign.

The three cardinal symptoms of heart disease: dyspnœa, engorgement of the liver, and dropsy, may all be well represented during the course of the illness, but the last may be met with only late, while it may be earlier represented by ascites only, which no doubt follows, as in cases of mitral stenosis, the special affection of the liver and consequent special disturbance of the portal circula-

tion. In some cases there has been a condition of the peritoneum that has placed in question the origin of the ascites in the liver-congestion, but in this respect "Indurative medio-stinitis" does not differ from mitral stenosis and other valve lesions, in cases of which ascites has been a prominent feature, while there is evidence of inflammatory action in the peritoneum, and especially in the capsules of the liver and spleen. The latter organ is not usually enlarged, following the general rule in cardiac dropsies, but exceptionally it is enlarged.

These may be regarded as the principal features that "Indurative mediastino-pericarditis" possesses in common with ordinary forms of heart disease. In another place (p. 260), the belief has been expressed that adherent pericardium has too often been regarded as the cause of associated cardiac muscle-failure, and it has been pointed out that internal adhesion of the pericardium is not very rarely found *post-mortem* with a practically normal heart. Of course in the disease now under consideration, not only are the adhesions external as well as internal, and the thickening of the tissue concerned pronounced, but from their very nature and associations, they may be supposed to hamper the heart's action to a much greater degree than a simply adherent pericardium, the result of pericarditis interna only.' There seems, besides, no

less reason to apprehend secondary cardiac muscle implication—myocarditis. The writer is not aware that the histology of the myocardium has been worked out with regard to "indurative mediastino-pericarditis." In any case the reader will bear in mind the much more formidable nature of the adhesions of this disease than of those that result from an ordinary and transient pericarditis. While evidence of enlargement of the heart is generally abundantly forthcoming "mediastinal dulness," *i.e.*, a dull area over the upper part of the sternum *crossing the middle line, but reaching neither costo-acromial angle*, is ill-developed or absent in most cases of "indurative mediastino-pericarditis."

In one of Dr. Harris's cases paralysis of the left recurrent laryngeal nerve resulted in paralysis of the left vocal cord, but the case was associated with chronic tubercular (fibroid) phthisis and thickening of the pleura over the upper portions of the left lung.

Two vascular symptoms have been regarded as almost pathognomonic of the disease under consideration, but it is doubtful if they are entitled to have so much diagnostic value attached to them :—

The so-called "*pulsus paradoxus*" consists of extreme diminution or actual disappearance of the radial pulse during inspiration. Dr. J. J. B. Williams (*London Journal of Medicine*) called at-

tention to the sign as long ago as 1850. A similar pulse if perhaps not so perfectly developed as regards its peculiar characters, has been met with apart from "indurative mediastino-pericarditis" in various pathological conditions, including laryngeal obstructions (Brockbank: *British Medical Journal*, June, 1893), dilatation of the heart with mitral incompetence and large pleuritic effusions.

Dr. Harris has called attention (*Lancet*, 22nd April, 1899) to the phenomenon of pulsus paradoxus being limited to or specially developed in the left radial. In the same communication he remarks that: "When a deep inspiration was taken, and the breath held at the end of that act, it will be seen that the pulse in the left radial gradually returned, the successive beats getting larger and larger, until the breath had been held about 25 seconds, when the individual waves were nearly as high as during ordinary breathing."

Schrieber (*Archiv für Experimentelle Pathologie und Pharmacologie*, Leipzig, 1880) maintained that the pulsus paradoxus in mediastino-pericarditis possesses essential differences from the similar pulse met with, under a variety of circumstances, in the following respects: (1) Marked diminution in the volume or complete disappearance of the pulse of all arteries, and not only of that of the radial in the second half or at the end of that act.

(2) The impossibility of replacing the paradoxical pulse by a full pulse by a prolonged and sustained inspiration. (3) The pulsus paradoxus does not require for its development the inspiration to be a deep one. (4) The pulse intermission is accompanied by a regular action of the heart, and the heart shows (5) no weakening of its action during the period whilst an inspiration is being made: (4 and 5 might, however, be modified in cases where there is at the same time endo- or myocarditis). Quoted from Dr. Harris's Monograph, pp. 63 and 64.

The other sign under consideration consists of *the cervical veins filling up and becoming distended during inspiration.* Like the preceding, this sign is not present in all cases of "indurative mediastino-pericarditis." It has been recorded, moreover, in a case of pericarditis without mediastinitis.

SEPTIC OR ULCERATIVE OR MALIGNANT ENDOCARDITIS.

While it is difficult or impossible to give a sketch of the clinical features of non-malignant rheumatic and choreic endocarditis, inasmuch as they are usually chiefly those of the disease with which the endocarditis is associated, septic endocarditis, on the other hand, in many cases presents a grouping of symptoms that forms a definite clinical entity. Irregular forms and special types, no doubt, occur; but this is no more than may be said of enteric and most other specific diseases.

Septic Endocarditis seems to be a peculiarly common disease in the Manchester district. It attacks either the mitral or aortic valves and rarely the endocardium away from the valves, just as it may attack also the lining membrane of the aorta above the valves (p. 178). In the case referred to a small aneurysm resulted from its inroad into the aortic wall; in like manner it may cause aneurysm of the valves of the heart by piercing one endocardial surface, or it may destroy the endocardium in a small area and cause destruction of the myocardium or pierce a valve.

Even the chordæ tendineæ may be the seat of change and destruction wrought by the micro-organisms, to which the disease owes its existence.

It is important to remember that septic endocarditis often attacks valves already damaged by rheumatic endocarditis, as well as valves that are congenitally defective in structure. Under both these circumstances, if they have been previously recognised, the cardiac signs of the disease lose a large proportion of their value.

It is not proposed to enter upon any description of the lesions that result from septic endocarditis, but it is necessary to refer to one of the terms used in the nomenclature of the disease, namely, "ulcerative" (*i.e.*, destructive), and to point out that a "formative" process is often as much, or more, in evidence in the production of abundant vegetations. Vegetations once formed are apt to have deposition of fibrin upon them, which no doubt plays the principal part in the embolic process.

Of the commonly associated conditions of septic endocarditis, there is a large number. As already mentioned, old damage wrought by rheumatism or chorea or congenital malformation is often the seat of attack. Pneumonia is not very rarely associated with the disease. Cerebro-spinal meningitis is a rare associate, as is also gonorrhœa.

The following statements of Professor Dreschfeld are well worthy of remembrance. *Allbutt's System* (Vol. I., p. 630):—

"(1) Infective endocarditis is a disease due to micro-organisms.

(2) This disease is not produced by one specific microbe only; other organisms, separately or together, may give rise to it.

(3) The organisms which most frequently are the cause of the disease belong to the septic and pyogenetic type (streptococci and staphylococci).

(4) Of other organisms, the diplococcus of pneumonia often gives rise to infective endocarditis; the specific organisms of enteric fever, gonorrhœa, diphtheria, tuberculosis do so very rarely: infective endocarditis occurring in the course of any one of these affections or found in valves already the seat of chronic endocarditis or atheroma, is due to septic organisms, and must be looked upon as a mixed infection complicating these diseases.

(5) The organisms more readily attack valves weakened or altered by disease.

(6) Some of the microbes found in infective endocarditis are also found in rheumatic or verrucose endocarditis."

Enough has been said to emphasise the fact that septic endocarditis is commonly an associated

disease. It must not be forgotten, however, that the disease is met with *alone*, or apparently so, and such cases naturally form the most definite examples of the disease.

It is not considered necessary or desirable here to pursue the subject further from the pathological side. The septic associates of the disease are extremely numerous.

Symptoms.

Septic endocarditis may begin either insidiously or abruptly, and its special features are apt to be confused with the features of the disease, it may complicate. The malaise of pyrexia may come on insidiously, or there may be an abrupt commencement with rigor, which may be repeated, usually irregularly, in the course of the illness. The significance of a rigor is a rapid rise of temperature. The same degree of fever or a higher one may be reached without rigor if the temperature rise slowly; on the other hand, a slight rise may produce "chilliness" if it take place rapidly. The degree of febrile malaise varies in different cases. Sometimes one is surprised how well the patient remains. A clear tongue and fair appetite are retained, and the patient complains of being confined to bed. One is reminded of tuberculosis in these circumstances. On the other hand, a few

patients fall rapidly into the so-called "typhoid" condition, and develop dry tongue, quiet delirium, subsultus tendinum, and other features of that toxæmic condition. It is well known that the "typhoid" condition is specially apt to be induced when the kidneys are diseased and "inadequate," but even in the presence of nephritis and albuminuria and hæmaturia patients suffering from septic endocarditis may escape "typhoid" symptoms almost entirely.

The type of fever varies: it is commonly *intermittent*, the temperature reaching normal or becoming subnormal in the morning; on the other hand, it may be *remittent*, the temperature falling in the morning, but failing to reach normal. There may, however, be continuous fever, though usually the fever is more or less irregular, and in this type the "typhoid state" is most apt to be developed. Occasionally in cases of long duration —and the disease has lasted a year and more— prolonged intermissions, lasting 10 or 14 days, occur, the fever ceasing entirely. The disease may, however, be rapidly fatal. Thus in the first case ever observed by the writer (in 1875) a man suffering from "fibroid phthisis" unaccompanied by fever became feverish, while a slight diastolic murmur developed over the aortic area and down the sternum, soon to be followed by a systolic

murmur, which became more distinct as the former declined, and the patient died in a week from the outset of the complication. An elongated clot was found attached to (no doubt to an ulcerated surface of) an aortic valve.

Whether "typhoid" symptoms be developed or not, the spleen is apt to become enlarged so as in most cases to be easily palpable below the left costal arch, and such enlargement of the organ should always be sought for whenever septic endocarditis is suspected. It has been seen (p. 14) that enlargement of the spleen is not an usual feature of ordinary cardiac disease, as is enlargement of the liver. It is true that the spleen may become enlarged, in ordinary heart-disease, from its being the seat of embolism, but in septic endocarditis enlargement of the organ occurs quite independently of infarct, and after the manner of the enlargement observed in Enteric and other specific fevers. Of course, the fact does not exclude the spleen becoming the seat of embolism later. The resemblance of the disease to typhoid may be increased by the occurrence of diarrhœa, and in a case of the writer's an ulcer actually formed in the intestine presumably of embolic origin and caused the death of the patient by perforation. In this case, which was reported by Dr. Williamson in the *Medical Chronicle*, the spleen was very large.

Hæmaturia is a by no means rare feature of septic endocarditis. The quantity of albumen present is usually greater than the mere presence of blood would account for, and renal casts are usually found without difficulty in the deposit. Such hæmaturia must not be confused with that resulting from embolism of the kidney, which is usually transient.

Petechiæ and rashes, that are apparently not specific, may make their appearance. Retinal hæmorrhages may be found, and prove of some diagnostic value. Pains in the joints, as in other forms of septicæmia, may be complained of and are apt to be confused with those of rheumatism. A pronounced degree of anæmia and cachexia is often a feature of cases of septic endocarditis, and its development during the illness may be remarkable. Emaciation is sometimes extreme in cases that run a protracted course.

Such is a rough sketch of the features of what may be called the general illness, and amidst which the striking occurrence of the symptoms of embolism in various organs is apt to take place.

Some of the most striking clinical features of septic endocarditis are due to the process of embolism, which has already been fully considered. Owing to the septic nature of the plug, arteritis may be set up, and the softened wall yielding to

the blood pressure, a small aneurysm may be formed. Among the arteries that have been so plugged are the "posterior tibial, brachial, popliteal, and even abdominal-aorta"; but the left middle cerebral artery is a specially frequently affected one. The aphasia and hemiplegia that result vary in severity. Sometimes these conditions are only transient: at other times the cerebral seizure terminates the case abruptly. A young patient of the writer's had an ordinary embolic hemiplegic and aphasic attack from which, however, recovery was unsatisfactory. During such convalescence as he had from the seizure, he was seized with convulsion and apoplexy, and died in a short time. *Post mortem* his brain was found ploughed up with clot, but the source of the hæmorrhage was not discovered, though the symptoms and history left little doubt as to an embolism-produced aneurysm having ruptured.

Gangrene of an extremity may result from the plugging of a large arterial branch, and such a branch may become the seat of aneurysm in the same way as a cerebral vessel. Such an aneurysm may attain the size of a pigeon's egg or larger.

The *spleen* in septic endocarditis is enlarged quite apart from embolism in most cases; but it

may also become the seat of embolism as in ordinary heart cases.

The mesenteric arteries and their branches are occasionally blocked, and the symptoms have been already considered. Small branches becoming the site of a plug may lead to ulceration of the mucous membrane and possibly to hæmorrhage and even perforation followed by general peritonitis.

The frequency of hæmaturia depending upon nephritic congestion in septic endocarditis must be borne in mind, and the appearance of blood in the urine not too readily assumed to be due to hæmorrhagic infarct. Besides, a so-called "hæmorrhagic diathesis," with petechiæ on the skin and various hæmorrhages, may be associated with septic endocarditis. The petechiæ are possibly embolic in origin. Pulmonary infarct occurs in septic endocarditis as in other heart cases and is apt to be specially developed when the right side of the heart is affected.

TREATMENT OF HEART DISEASE.

NURSING.

One of the great difficulties in the nursing of the patient with advanced heart disease arises from his inability to lie down, and at times, though rarely, even to remain in bed. When dropsy accumulates, again, especially in the case of a large patient, the necessitated immobility of the sufferer is apt to lead to bed sores and sloughing of dropsical parts.

To the bedridden patient, it is often, in heart disease as in fever, a great comfort to have a day and a night bed. The transference may, however, be a difficulty. When only one bed is in use, movements of the patient are, of course, still necessary, and often entail great suffering. With the best intentions, friends and nurses often inflict great suffering on patients in assisting them in such necessary movements. It is hardly possible to handle a heavy patient without causing suffering, and for this reason a mechanical apparatus, by which the patient may raise him-

self largely by his own efforts, is often of great use, though this entails the patient making an effort, which on general grounds is to be strongly deprecated; it is often the best policy to make a compromise and choose this as the lesser evil.

In the Manchester Royal Infirmary most of the beds have a suspended cross-bar, by grasping which the patient can assist his movements. When by any chance a heart sufferer is placed in one of the beds not so provided, the difference observed is most pronounced, and if the patient has had previous experience of the cross-bar in question, he almost invariably begs to be placed in a bed with the apparatus. In private practice a joiner can usually be got to fit up a contrivance answering the same purpose. (Figs. 104 and 105.)

Another useful mechanical aid to the advanced cardiac sufferer is a support to his feet, by means of which he can prevent himself slipping down in the bed, or raise himself if he has slipped down. Any common article, such as the footstool, a pile of large books, etc., can be used for the purpose, provided there is an upright projection at the end of the bed to place it against. The feet of the patient being so supported, he can make use of the suspended cross-bar to advantage in effecting movements in bed with the least possible exertion on his part. Granted that all exertion is injurious,

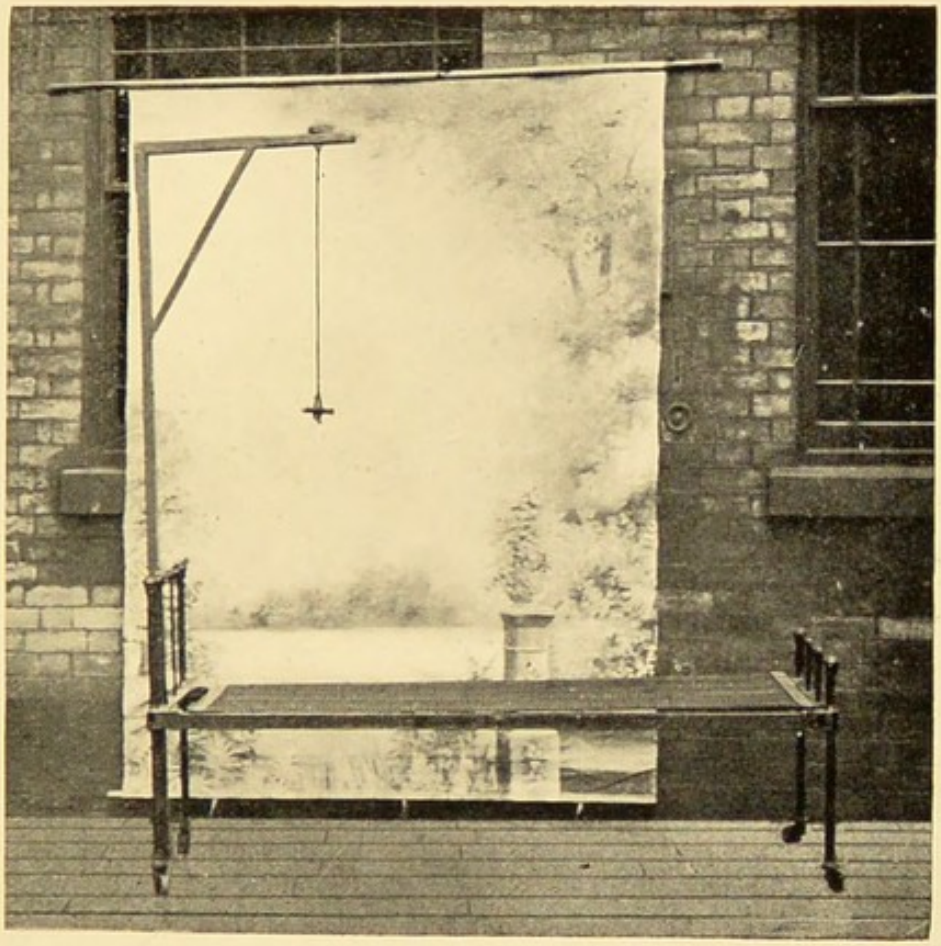


Fig. 104.

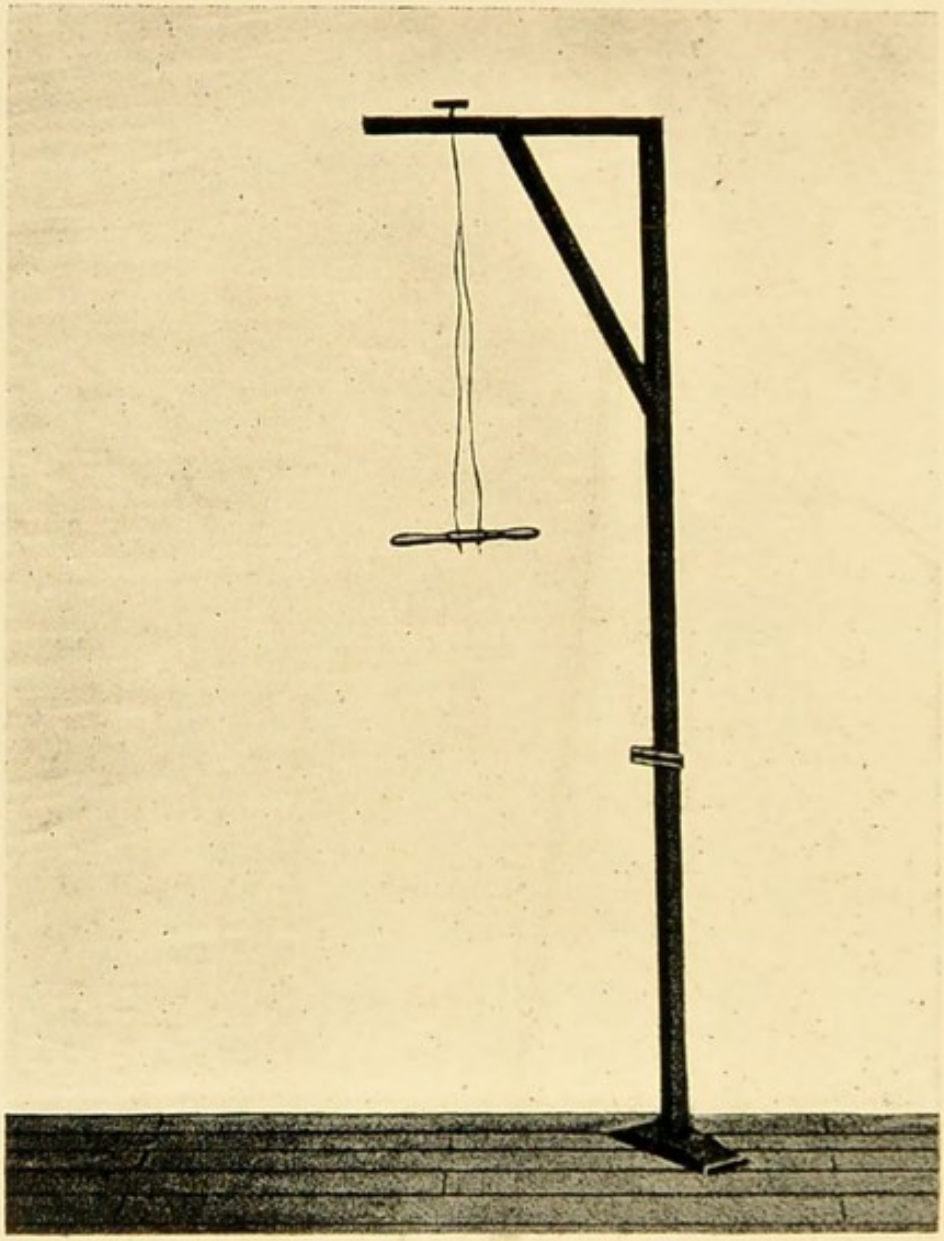


Fig. 105.

there is only to be set against the patient's own efforts, so mechanically aided, his handling by nurses and friends, who, however kindly their intentions, often inflict in the process a considerable degree of suffering. To be the lesser of two evils is all that is claimed for these mechanical arrangements.

DIET.

In the treatment of heart disease regulation of the patient's diet is of supreme importance. The influence of diet upon "wind" in the athletic sense, and upon endurance of prolonged effort, is generally admitted. In the case of two athletes of equal merit striving for victory in a race or other event, the one "trained" and the other not, victory almost certainly will be achieved by the former. The process of "training" may be said to imply regulated exercises and regulated diet, along with every hygienic measure calculated to promote the general health. The result is a condition, of which greatly improved capacity for effort, be it short or prolonged, is the most prominent feature. Another, and, so to speak, passive aspect of the result, is the resistance to injury and power of recovery from injury acquired by the well-trained man; but it hardly concerns the subject of heart disease. As regards the result of training upon the capacity of a healthy individual for effort, there can be no doubt that the heart is chiefly influenced, while the whole "musculatur" of the individual is more or less affected. The

"musculatur" concerned in respiration may be believed to be specially improved by the process, and in this respect to come next to that of the heart itself.

The case of the healthy athlete and that of the patient with chronic heart disease at first sight seem hardly comparable, for the obvious reason that the individual with pronounced heart disease is seldom or never in a condition in which his general "musculatur" can be exercised. No doubt the distinction is very important, but its consideration surely leads to the adoption of such a dietary as will be here recommended, inasmuch as if carbo-hydrates be regarded largely as the fuel of the individual economy, the patient with heart disease has greatly reduced capacity for burning them off by virtue of his inability to exercise his general "musculatur," though his heart-muscle certainly, and his respiratory muscles probably, are, at the same time, exposed to strain—few who have witnessed the "struggle for breath" in a case of severe paroxysmal dyspnœa will be disposed to question the latter statement. But if the capacity of the patient with heart disease to burn off the carbo-hydrates of his food is diminished on account of his inability to take exercise, such diminished capacity will lead to further influence being exerted upon the metabolism of the albu-

minous tissues and especially upon the products of their "tissue-change," or metabolism, for the carbo-hydrates will tend to "eat up," so to speak, the available oxygen that would be better employed in promoting the change and elimination of the products of the metabolism of the proteid tissues.

Another consideration is with reference to the liver. Physiology teaches the importance of this organ both as regards carbo-hydrates and proteid food. The glycogenic functions on the one hand, and on the other the part played by the organ in the preparation of nitrogenous waste for elimination by the kidneys as urea, need only be referred to. Now, in heart disease in all its more severe forms it has been shown that the organ is subjected to "venous stasis" to an extraordinary degree. This condition will almost certainly cripple the functions of the organ, as they are concerned with the dietary in respect of both carbo-hydrates and proteids. It can hardly be doubted, however, that relief will be afforded to the organ in the performance of its functions, if its cells are spared the labour of dealing with excess of carbo-hydrates, and have therefore left to them greater energy to deal with the products of proteid metabolism.

Such are very briefly and imperfectly stated the considerations that tend to support the employ-

ment of the dietary, that will be described later, in heart cases.

There is, however, another side of the question: it concerns the primary digestion of food in the alimentary canal and the customs of modern society as regards meals. The customs of society may be regarded as to a large extent based upon the experiences of the average individual. Sir William Roberts showed that that individual possessed, as a rule, practically an excess of digestive power, and that it was customary to take at meals constituents that had actually a retarding influence upon the digestive processes. The moment these processes are crippled by venous stasis of the organs concerned, however, promotion rather than retardation of them is to be sought. Effectual promotion of the digestive processes will be obtained by a simplification of meals and the organs concerned being left as much as possible free to deal with the food, the digestion of which is their special function. As an illustration, let the ordinary meal of a chop taken with potato or bread or both, and followed by rice pudding, be considered. The digestive changes in the starchy constituents will begin in the mouth by virtue of the saliva and of mastication whereby the action of the saliva is obviously promoted. When, however, the food reaches the stomach, it

is no longer the carbo-hydrate constituents of the meal that are dealt with, but the proteid, and in an acid instead of an alkaline medium. In this acid medium it is generally held that starchy digestion is stayed and does not again proceed till it has passed lower down in the alimentary canal, where an alkaline medium is again provided. If these things be so, the effect of a mixed meal must mean a certain amount of embarrassment in the functions of the stomach—even if this embarrassment be regarded as purely mechanical. With a large proportion of its contents the stomach has no digestive concern. No doubt lower down the alimentary canal the digestion of carbo-hydrates and proteids can go on side by side in the alkaline medium of at least the pancreatic secretion, and possibly of other secretions. In severe illness, as in the fevers, it is possible that this is the chief place of digestion for both constituents of food, but the question is foreign to the present subject. Surely the digestive processes in a patient with heart disease will be greatly promoted if a simplification of meals be made; that is to say, if the patient in question will make a meal of his chop or steak, and take with it neither bread nor potato, nor farinaceous pudding. If he be very weak and his digestive power at a low ebb, the flesh food can be minced, while if a fair

amount of digestive capacity remain and feeling of satiety do not follow the ingestion of meat alone, some green vegetable, well-cooked and best sieved, may be added, which undergoes little chemical change, although it acts as "ballast" to the meal in the stomach and, lower down the alimentary canal, promotes the action of the bowels. Large draughts of water taken with meals presumably interfere with the digestive processes and are to be deprecated—though in certain cases after fasting and a considerable time before a meal, a full draught of water may be beneficial by promoting elimination of the waste products of metabolism.

The capacity for digesting fats seems to vary greatly in individuals. To patients with heart disease, who can digest fat, the writer believes it to be less injurious than carbo-hydrates; but there can be no doubt that any large quantity is undesirable for the cardiac sufferer, though in the case of the patient with his carbo-hydrates cut down to a minimum an increased allowance of fat may be permitted, provided it can be digested and sets up no disturbances in the alimentary canal. In the dietary to be recommended, for instance, cream and butter have a place, although any excess is to be avoided.

In Dr. Pavy's authoritative "Treatise on Food

and Dietetics" (J. and A. Churchill: London, 1875; 2nd ed.), there is related the following experience of a traveller, Sir Francis Head, who, "when crossing the pampas got tired at first with constant galloping and was forced to ride in a carriage after five or six hours on horseback." Sir Francis goes on to relate his experience in the following words:—"But after I had been riding for three or four months and had lived on beef and water, I found myself in a condition which I can only describe by saying that I felt no exertion could kill me. This will explain the immense distances which people in South America are said to ride, which I am confident could only be done on beef and water." In all probability this extraordinary state of the muscular system involves the muscle of the heart—nay, it may be believed that the heart-muscle is principally influenced, for by its capacity for work alone can the "musculatur" of the individual be rendered available. It requires no flight of imagination to picture to oneself the case of an athletic giant, whose aortic valves have become more or less abruptly incompetent. However well developed and sound in nutrition may be his muscles generally, the crippling of his heart renders them of no avail either for temporary effort or sustained exertion. Without "good wind" the largest and best of

muscles do not make the athlete, and "good wind" is essentially dependent on the integrity of the heart. Is there no lesson to be learnt from the experience of Sir Francis Head in the treatment of heart disease? The incapacity for exertion on the part of the patient, whenever his heart is gravely damaged, may be said to interfere with the perfect metabolism, that, on the contrary, a man in the open air and exercising most of his muscles, may be expected to have. But if this be true in relation with proteid food, it is no less true in relation with carbo-hydrate food, which is essentially fuel-food. But supposing that abundance of the latter food is given to the bedridden cardiac sufferer, can he avail himself of it to the full? Moreover, under the circumstances, are the toxic waste products of proteid metabolism likely to undergo such complete change as will render them innocuous when the carbo-hydrates stand in the way of their oxygenation. To the writer the simple statement of a man unprejudiced by theory is worth much. Moreover, the system of dietary referred to by Sir Francis Head is essentially that of the prize-fighters and other athletes of last century. Let the reader disregard the modern system of "records" and read the description of the athletic "events" of the past century, largely like Sir Francis Head's riding achievements "done

on beef and water," and the writer believes that the comparison will not altogether be in favour of the present system. It has been found in the Prussian Army and elsewhere that sugar is a very valuable food for the athlete about to engage in severe exercise, as no doubt it is: it is fuel to his system. But how is the poor cardiac sufferer, incapacitated for all exertion, to make like use of such fuel? Theory, however, in such matters must give way before the results of experience, and the writer for the last eight years has been treating his heart cases on such a diet as he is going to recommend with most excellent results, and it gives him mental pain to look back upon the days when he gave little attention to the matter of diet and let his patients with heart disease unwittingly aggravate their sufferings by a largely carbo-hydrate diet, which gave them distressing flatulence, and took the place of food of infinitely greater value as far as their heart-muscle was concerned.

The question of albuminuria in relation with a largely albuminous diet may give concern to some. The frequency of albuminuria in cases of heart disease is notorious—few cases, indeed, run their course without its occurrence sooner or later. On the other hand, there can be no doubt that in a proportion of cases the state of mere "venous

stasis" passes into that of a true nephritis with epithelial changes of inflammatory nature. Lastly, there is the important class of cases in which the heart failure is secondary to chronic Bright's disease, especially "granular kidneys." The apprehension of a largely proteid diet setting up nephritis seems to be based on theoretical conditions entirely. The writer has seen no case that gave the slightest countenance to such apprehension. On the other hand, that gross feeders in general are liable to albuminuria and glycosuria there seems to be only too good reason to believe; but the latter condition is significant. If proteids were taken in excess, carbo-hydrates were taken at the same time in as great, if not greater, excess. For the subject of actual chronic Bright's disease, whose heart-muscle is manifestly failing, it is difficult to determine the best line of diet. One hesitates to order the ordinary cardiac sufferer's largely proteid diet: all the writer will say on this subject is that he believes such a diet is better borne than might be expected from theoretical considerations. An absolutely milk and fruit diet, on the other hand, suits admirably, but patients weary of it, and few will submit to it for a lengthened period. To make a compromise under the circumstances seems to be the best policy to adopt: to let the patient have a *spare* mixed diet,

though there is no reason why the stomach should not be relieved by a simplification of meals. In these cases the maintenance of regular action of the bowels is very important, and to aid in that result green vegetables may be given freely with the flesh-food.

Dietary for Patients with Chronic Heart Disease.

What may be considered to be the most suitable diet for the ordinary cardiac sufferer is indicated below:—

Breakfast. China tea, or (if preferred) coffee, and cream; toast, buttered cold, the bread cut in thin slices and toasted slowly so as to be crisp, and fruit—bananas, etc. Sugar is better dispensed with: it may be replaced by "saxin," if the patient insists on sweetening his tea or coffee.

Luncheon. Mid-day meal: any kind of plainly-cooked flesh-food, with thoroughly cooked green vegetables, which are often best passed through a sieve. If desirable these will "carry" a large quantity of butter, which should be of good quality. If something of the nature of "pudding" be insisted on, custard or junket (curds and whey) may be permitted, and even a small quantity of cream therewith.

Afternoon tea. At 4 or 5 o'clock a cup of China tea and cream without sugar. Nothing to be eaten.

Dinner. Evening meal: a *little* clear soup *(consommé)*, which promotes the flow of gastric juice. Any kind of flesh-food. Thus fish and joint may be partaken of, provided the quantities of each be small. Green vegetables, as before. If only one kind of flesh-food has been taken, custard or junket may be allowed, though not recommended.

Certain concessions may be made to individual patients: thus an egg may be allowed with breakfast, or the *fat* of bacon provided it causes no indigestion; fruit may be allowed after luncheon or dinner (though carbo-hydrate, it seems to interfere with proteid digestion much less than, say, a rice pudding).

Potatoes, bread and farinaceous puddings are to be absolutely forbidden with luncheon and dinner, and little fluid is to be drunk with these meals. Half-a-glass of old whiskey or brandy in half a small tumblerful of water may be allowed with these meals, and in certain cases nearly a glass (best taken in warm water) of such spirit at bedtime. On no account is anything to be eaten with this last allowance.

The genuine idiosyncrasies of patients must be respected. Merely alleged inability to take this

or that kind of flesh can usually be demonstrated to the satisfaction of the patient to have no real foundation on experience. Some patients can easily digest articles that are difficult of digestion by most people: for instance, pork and toasted cheese, and there is no reason why they should be interdicted under the circumstances.

An important matter is the dieting of the poorer class of private patients, for such a dietary, as has been described, is an expensive one. As regards proteid, from the economic point of view, fish and cheese are specially valuable, and, as Dr. Hutchison remarks, "the despised herring and bloater 'offer the largest amount of nutriment for a given sum of any animal food' (Smith), and two salt herrings contain as much animal proteid as need enter into the daily dietary of an ordinary man." Cheese is another valuable source of proteid for the poor, but individuals vary much in their power of digesting it. A pound of Cheddar cheese represents the total casein and most of the fat in a gallon of milk. Cheese may be regarded as made up of one-third water, one-third nitrogenous matter and one-third fat, while moderately lean beef contains 73 per cent. of water. Beef thus contains less than half as much nourishment as the same weight of cheese. Again, one pound of cheese yields 2,000 calories of energy, which is

more than three times the amount yielded by a pound of moderately lean beef; but "a pound of cheese can be obtained at about one-sixth of the cost of three pounds of beef, which is its nutritive equivalent." (Hutchison.) The casein of cheese forms soluble salts with alkalies, as bicarbonate of potassium. "As much of the bicarbonate as will lie on a threepenny piece is sufficient to dissolve a quarter of a pound of cheese, if the latter be first grated or chopped up into fragments." (Hutchison.) "By the addition of milk and eggs a very savoury and exceedingly nutritious pudding . . . can be prepared at a very small cost." It has been well remarked that "if cheese is ever to take the place that it ought to have, as a cheap and convenient form of proteid food, some such method must be employed, for it is the difficulty with which cheese is digested that renders it an impossible food to many persons." It is in the stomach chiefly that the digestion of cheese gives trouble, for lower down the alimentary canal it is dealt with as easily as meat, while its absorption is no less easy.

In his admirable book, "Food and Dietetics" already so often quoted from, Dr. Hutchison remarks:—"It is as a cheap source of proteid . . . . that buttermilk is chiefly deserving of notice." Moreover, "its casein is present

in a finely flocculent form." As regards the fatty constituents of food—and it must be remembered that when carbo-hydrates are cut down to a minimum, the importance of fat is enhanced—margarine is a cheap and valuable substitute for butter. It is made from the fat of the ox by melting and slowly cooling so that the stearine solidifies first, while the palmitine and olein so separated are removed by pressure and churned up with a little milk, constituting margarine which contains 82·7 per cent. of fat, or practically the same amount as butter. Margarine differs from butter in containing ·25 per cent. of butyrin and fatty acids soluble in water in place of 7·5 per cent. Inasmuch as the irritating butyric acid is apt to be formed from butyrin by decomposition, this is by no means to the disadvantage of margarine. The comparative absence of casein from margarine is another point in its favour, diminishing the risk of decomposition.

As regards carbo-hydrate food, it is cheap enough, a fact that explains to a great extent the large excess of this kind of food wont to be given to the poorer sufferers from cardiac disease, while there is a corresponding diminution of albuminous food. The important part played by bronchial catarrh in heart disease has to be remembered in this last relation, for there is reason to believe that

carbo-hydrates promote the catarrhal process, while fats exert a contrary influence, and the value of cod-liver oil to the phthisical may be related to this fact. Bacon fat and "dripping" are again cheap and valuable forms of fat.

It will often happen in the course of heart disease that ordinary meals cannot be taken, and liquid food—milk, beef-tea, chicken broth, white of egg and water, liquid custard, etc.—has to be given in small quantities frequently. Even such liquid nourishment may be rejected, and koumiss or peptonized milk may then be tried, which failing, abstinence from all food by the mouth for eight or twelve hours is often the best line of treatment to adopt, a mustard poultice being sometimes applied to the epigastrium during the fast. It is less exhausting to a patient to take no food than to swallow food only to be immediately rejected. Small pieces of ice, or small quantities of warm water, may be allowed during the fast. Even larger quantities of warm water may be allowed if grateful to the patient.

In koumiss there are the products of a double fermentation: the sugar being partly changed into lactic acid, while a small portion of it is converted into alcohol and carbonic acid is given off. The casein, again, is precipitated in fine flocculi. The impossibility of large "curds" being formed

in the stomach such as may result from the ingestion of ordinary milk is an important advantage of koumiss and peptonized milk. The writer has seen enormous masses of "curd" vomited by patients with heart disease, or found the same lying in the stomach *post-mortem*—a condition of things that must have been highly detrimental to the patient's chances of recovery.

BLEEDING.

Although the treatment of heart disease, as of many other diseases, by bleeding has almost ceased to be practised, there are a few circumstances in which it may be usefully employed. First of all, venesection—a vein in the arm being usually chosen—may possibly save the life of a patient whose right auricle is distended and paralysed, and whose right ventricle is threatening asystole, the pulmonary circulation being profoundly obstructed. Great engorgement of the veins and intense lividity of the face, lips and tongue, are the great indications for phlebotomy.

Not only is the engorged liver of heart disease often exquisitely tender, but it may be the seat of spontaneous pain and extreme discomfort. When this is the case, a few leeches applied over the right hypochondrium may effect much relief.

"Dry-cupping," again, for which large cups should be used, is often very useful in œdema of the lungs and "suffocative bronchitis," such as is sometimes witnessed in cases of mitral stenosis, even before the advent of dropsy. Dry-cupping, and even "wet-cupping," may also be of use over the kidneys when these organs threaten to pass from a state of venous stasis to one of nephritis.

TREATMENT BY DRUGS.

Among the drugs that have proved useful in the treatment of heart disease digitalis is pre-eminent. Its active principles are chiefly glucosides, and have been named digitoxin, digitophyllin, digitalin and digitalein. Another glucoside—digitonin—has "the irritant action of a saponin and, like a saponin, suspends insoluble bodies in water" (Cushny). It is this last body that accounts for the frothiness of the infusion as well as for the comprehension of some of its most powerful ingredients. The infusion should always be fresh, and in such condition it is perhaps the best preparation of digitalis. It is, however, liable to vary in potency, "for the amount and character of the active constituents seem to vary not only in different seasons and in plants grown in different soils, but in different parts of the same plant." Moreover, it is of great importance that the crude drug from which preparations are made should not have been kept too long. Owing to such considerations, the writer has for many years used Nati-

velle's granules of "Digitaline Crystallisée" (digitoxin?), each granule containing the $\frac{1}{240}$ of a grain of the active principle. These granules he has always found to be very active and accurate in dosage, but their cumulative effect is pronounced, and he has widely separated the doses, seldom giving more than one granule in eight hours and usually one in twelve hours. Moreover, the irritant effects of drugs of the digitalis class are well developed in the granules, and sickness and vomiting are apt to result from their use if continued over too long a period or from their too frequent administration. For instance, early in his clinical experience, the writer gave up the use of the granules because of the frequency with which they produced epigastric pain and vomiting, when given three times daily, *i.e.*, three times in twelve hours during the day—none being given during the night. Dr. Mackenzie has attributed vomiting almost entirely to the action of digitalis upon the muscle-fibres of the stomach; but it is difficult to acquit the drug of an irritant effect on the mucous membrane. Moreover, drugs of the same class have an unquestionable irritant action on the gastro-intestinal mucous membrane, as, for instance, when squill acts as an emetic and euonymin as a purgative. The medulla oblongata would seem to be early excited by digitalis, and

not only, in consequence, may vomiting be an early symptom of poisoning, but the inhibitory action exerted by the drug upon the heart itself is exercised through the medullary centres and pneumogastric nerves.

When the writer was a student and junior hospital resident the action of digitalis was regarded as tending to throw the heart into tetanic spasm, the diastole becoming imperfect, the systole too prolonged—a view his clinical and pathological experience by no means confirmed. This is indeed its action on the heart of the frog. On the other hand, exceptionally in frogs, it is found that the heart comes to standstill in diastole. Two influences exerted upon the mammalian heart by the drug must be distinguished—(1) one just referred to exerted through the medulla and pneumogastric nerves tending to slow the heart and increase its diastole, so that more blood is sent forwards with each systole; (2) the other upon the heart-muscle itself, by which its "tone is exaggerated." In this latter respect, the effect resembles that upon the frog's heart. It is held that "if the heart is weak and dilated, digitalis and its allies tend to lessen this dilatation—that is, the relaxation of the ventricle during the diastole is less than before the administration of the drug. If, however, the heart is normal or does not dilate much during

diastole, digitalis increases the relaxation."—(Cushny: p. 411).

When an irregularly contracting heart becomes steadier under the influence of drugs of the digitalis class—as occasionally happens—the effect has been ascribed to an inhibitory action lessening the irritability of the heart-muscle; but this is doubtful.

In the so-called second stage of the effects of digitalis, there is infrequency of cardiac action, but, the diastole being carried to a higher point, more blood is expelled with each systole. The conduction of stimulus from auricle to ventricle may be blocked, while the irritability of the ventricular cardiac muscle being increased, systoles may start in it. The systoles of the auricle become weaker, and may temporarily cease. In medical practice this stage is seldom observed, and when it does occur, the administration of digitalis should be at once stopped or diminished. It follows that the third stage of the action of digitalis should never be observed clinically. This third stage is constituted by such an increase of the irritability of the heart-muscle, that the inhibitory influence of the vagus loses control. The exaggerated irritability of the heart-muscle leads not only to acceleration of the heart, but to irregularity of action of the auricles and ventricles. Finally, the

ventricle ceases in diastole, though the terminal paralysis is believed to be due to "poisons formed by its own activity," rather than to the direct action of the digitalis.

The influence exerted by drugs of the digitalis class upon the arterioles varies: digitoxin contracts the arterioles of the body generally, while strophanthin, convallamarin and digitalin act specially upon the arterioles of the splanchnic distribution, the arterioles of the limbs dilating, probably under the increased pressure resulting from the vascular contraction in the splanchnic area and increased vigour on the part of the heart-muscle. The arterioles of the heart itself are believed to respond to digitoxin by contraction, whilst they are little or not at all affected by strophanthus.

The essential difference between the action of strophanthus and that of digitalis would seem to be the less degree of contraction of the arterioles produced by the former, and this difference concerns non-splanchnic and peripheral areas chiefly. The action of scilla maritima is of the same nature as that of digitalis and strophanthus. Its tendency to produce vomiting in large doses is probably associated with its action as an expectorant in medicinal doses. This tendency, though better developed in the case of scilla, is not entirely

absent in other members of what has been called the Digitalis Group, which includes convallaria majalis (lily of the valley), apocynum cannabinum, and adonis vernalis (pheasants' eye), &c. The tendency of digitalis and strophanthus to produce sickness, for instance, is only too well known in clinical practice. The writer has found digitalis and strophanthus so useful that he has seldom employed convallaria and apocynum except in cases in which digitalis and strophanthus have both failed. Under the circumstances a successful result—diuresis—is more likely to be obtained from drugs that act specially on the kidneys, such as caffeine, theobromine, and theocine (theophilline), provided the latter organs remain structurally sound. With reference to the Digitalis Group, it is interesting to find a member of it that is usually regarded simply as a purgative, namely, euonymin (extractum euonymi siccum). In the choice of a purgative in heart cases, the fact of the drug possessing an action in the direction of the heart similar to digitalis, may be worth remembering.

Diuretics.

It sometimes happens that when in advanced heart disease with dropsy no response to treatment can be obtained by the use of drugs of the digitalis class, whose principal action is upon the heart-muscle, a diuresis at least may be set up and great relief afforded to the patient by the employment of drugs whose principal action is upon the kidneys. The fact was much impressed upon the writer's attention many years ago by a case of aortic incompetence which had failed to respond in the smallest degree to the most trustworthy drugs of the digitalis class. There was a great amount of dropsy present and things looked exceedingly gloomy as regards prognosis, but the remarkable absence of albuminuria under the circumstances suggested the use of a drug exerting its influence specially upon the kidneys—diuretine. A profuse diuresis ensued, and to the marvel of all concerned, the patient was able to walk out of the hospital convalescent for the time being at all events.

Three drugs that may be so employed to act upon the kidneys are caffeine, theobromine and theophylline (theocine), of which the last is the most powerful. Both theobromine and theocine may produce sickness, if given too freely. The writer has obtained the best results from "diure-

tine," a double salt of sodium-theobromine and salicylate of sodium. Another drug is agurin, a double salt of sodium theobromine, and acetate of sodium.

The caffeine series are purin derivatives, and related to the xanthine bodies of the urine, caffeine itself being trimethyl xanthine. The drugs in question, have some action on the circulation and in the usual ways: *i.e.*, acting on the nervous centre in the medulla and on the heart-muscle itself, the increase of blood pressure that ensues resulting in part from stimulation of the vaso-motor centre, and in part from stimulation of the heart-muscle itself. Theobromine has little or none of the former action, and is alleged to dilate the coronary arteries when experimentally perfused through the heart. On the other hand, chloral may be used with caffeine to remove the vaso-motor stimulation produced by the latter drug, and it is said that the resulting diuresis is thereby enhanced. A stimulating influence, moreover, is exerted on the respiratory centre by the drugs under consideration. Caffeine loses methyl groups in passing through the body, becoming dimethyl and monomethyl xanthine. The caffeine series of drugs may certainly be given with greater confidence when there is reason to believe that the kidneys are structurally sound, but it is open to question if they should be interdicted in the presence of actual Bright's disease.

Vaso-Dilators.

The introduction of this class of therapeutic agents into use in the treatment of heart disease is due to Sir Lauder Brunton, who found that he could relieve the paroxysm of angina pectoris by the inhalation of nitrite of amyl, and that the relief afforded was preceded or accompanied by relaxation of the arterioles, spasm of which he had previously found to be associated with the paroxysm. Knowledge of the rise of arterial pressure in the paroxysm suggested the use of the remedy. Some of the drugs used for the purpose of vaso-dilatation are organic nitrates instead of nitrites. Nitro-glycerine (trinitrate of glyceryl) and erythrol tetranitrate furnish examples, but it is probable that these drugs are broken up by alkalies into a mixture of nitrates and nitrites, the latter proving the active constituents.

The essential action for which these drugs are employed is dilatation of the arterioles, which seems to be produced by the direct action of the compounds upon the contractile fibres of the arterioles. After inhalation of nitrite of amyl the head and neck are specially flushed. The pulse

is accelerated, presumably due to depression of inhibitory influence, and to the fall in blood pressure. After a time the acceleration of the pulse gives place to slowing, attributed to "a direct depressing action on the muscle." In the frog it is said that the heart is usually slowed from the beginning. Respiration is accelerated and inspiration rendered deeper by the inhalations of amyl nitrite, though the breath may be held at the beginning of the inhalation owing to the local action of the drug on the nasal mucous membrane: a respiratory reflex action. The kidneys are litle influenced by the nitrites, though the action of these remedies may prove useful in combination with that of digitalis and its allies by removing local arteriole spasm, and so promoting diuresis. "Owing to nitrite of amyl changing the hæmoglobin to methæmoglobin and nitric-oxide-hæmoglobin compounds" the blood may assume a dark-chocolate colour under its use. "In man usually very little of the hæmoglobin is thus transformed" (Cushny); and the effect would seem to be so small in therapeutic application that it may be disregarded. Nitrite of amyl acts much less efficiently when swallowed than when inhaled, owing to decomposition taking place under the action of the gastric juice.

Nitro-glycerine is specially apt to produce head-

ache, which may also result from inhalation of nitrite of amyl. Nitro-glycerine, moreover, keeps up its action for a much longer period, but in this respect is inferior to erythrol tetranitrate. Nitrite of sodium is also useful for maintaining depression of arterial tension, being given every four or six hours for the purpose in doses of 1 or 2 grains.

The essential use of the vaso-dilators is to diminish high arterial blood pressure, and the success of their application will to a large extent depend upon the skill with which discrimination of favourable conditions for their action is made. The nitrites are too often used in modern practice without such discrimination being exercised, and under such circumstances it is surprising that these drugs have maintained their reputation as well as they have done. The time-honoured spiritus ætheris nitrosi is too commonly administered as if it acted as a nitrite, which it does in little more than in theory, for when mixed with water, it rapidly loses such nitrite action as it possesses when freshly prepared. Liquor ethyl nitritis is, on the other hand, an efficient preparation.

TREATMENT OF PAROXYSMAL DYSPNŒA.

While the treatment of chronic dyspnœa resolves itself essentially into the treatment of the heart, the paroxysms of dyspnœa, which are apt to arise especially in certain varieties of heart disease, call for immediate treatment. Most of such paroxysms are associated with a rise in arterial pressure due to a widely-distributed contraction of the arterioles, and in their treatment vaso-dilators generally prove of great service. When vaso-dilators fail and the bronchial tubes are not blocked with secretion, a hypodermic injection of morphine and atropine is the only remedy that can be trusted to relieve. When, however, there is much blocking of the bronchial tubes by secretion or in Bright's disease, morphine becomes highly dangerous, and if given at all must be given in infinitesimal doses and guarded by atropine or strychnine. When there is evidence of the lungs being engorged and the bronchial tubes loaded with secretion dry-cupping over the chest is indicated. It may be said that in severe dyspnœa, the less physical evidence there is of obstruction in the bronchial tubes and lungs the more is morphine indicated, and *vice versâ*.

In cases of paroxysmal dyspnœa the inhalation of oxygen is often useful, the degree of cyanosis being the best indication for its use. In a grave case of heart disease it is always well to have a cylinder of oxygen available in case of need.

MANAGEMENT OF THE BOWELS.

In the practical treatment of heart disease few details require greater discrimination than those pertaining to the management of the bowels. Physiology teaches the immense importance of the splanchnic area and its unquestionable influence upon the systemic circulation. A man may bleed, as it were, into his splanchnic vessels until syncope is induced. This fact alone may give us pause in the use of the drugs now under consideration. Judicious therapeutics imply not only a knowledge of the physiological action of drugs, but also of the special state of the circulation in the patient to be treated. Can it be wise to treat alike by the application of purgatives, the patient with granular kidneys and incipient failure of a still vigorous heart, whose sphygmogram indicates a high degree of arterial pressure, and the patient with advanced muscle-failure of his heart whose sphygmogram shows a small pulse, of lowest tension and pronounced dicrotism? In the former case purgation is clearly indicated: in the latter case in all probability only harm will result from purgation, and

it may be actually dangerous. The indiscriminate use of purgatives in heart-cases is less common now than it used to be, but the writer cannot be as sanguine in the matter as a recent author, who remarks, with reference to the attempt to remove dropsy by purgation, "that this measure is comparatively seldom used now." It is the duty of the physician, in the first instance, to ascertain the circulatory condition of the patient: this having been done, he may look for guidance to the pharmacologist as to the means to be employed, if it be decided that purgation is requisite.

A very important consideration as regards the use of purgatives is the exertion on the part of the patients that frequent action of the bowels implies. Again, the degree of arterial tension present has to be considered—and careful examination with the finger suffices to recognise the condition present in this respect—for the patient with high tension is not likely to suffer from the exertion of having his bowels moved in anything like the same degree as the patient with low arterial pressure, so that the rule just considered is still applicable. Another question that arises is the frequency with which purgatives should be administered. It is often possible, by the use of mild laxatives and by diet to keep the bowels free without actual purgation. Even when

this is the case, the patient with high arterial pressure often derives benefit from a sharp short purgation, while the patient with low pressure is only more quickly exhausted by the maintenance of relaxed bowels, and severe purgation only hurries him to his end.

The physician has decided that the case before him is one that requires purgation: the choice of a drug has to be made, and in the same case running a prolonged course it is probably beneficial not always to use the same agent.

One of the most commonly employed drugs to produce purgation is calomel, which is credited with special antiseptic influence on the intestinal contents, and (with less reason probably) with special power in the direction of lowering the systemic arterial tension. Though out of place in this section, mention may be made of the diuretic effects attributed to this drug in doses of 3 grains given 3 times daily in cardiac dropsy. The experience of the writer as regards this method of treatment has not been such as to cause him to recommend it, as may be gathered indeed from his omission of the drug when considering diuretics. Theoretically, saline purgatives are specially indicated in heart cases, as their effects are largely due to their physical rather than their vital action: they act by osmosis resulting

in a flow of fluid from the blood-vessels into the alimentary canal. Moreover, their action is likely to be specially pronounced when the tissues are waterlogged by dropsy. Of the saline purgatives it has been said that their "characteristic effect is not irritation, but retarded absorption." "Increased peristalsis is due . . . . to the large amount of fluid contents" (Cushny).

In order that the salines may act efficiently as purgatives they should not be given in too large a quantity of fluid, and they may often be combined with a vegetable purgative that causes increased peristalsis, *e.g.*, pulv. jalapæ co. pulv. rhei co. The saline, however, acts more rapidly than the vegetable preparation, so that the latter may be taken in the evening, and followed in the morning by the former. The saline purgatives, if given in too large a quantity of fluid, tend to pass off by the kidneys, and thus act as diuretics instead of purgatives.

From the foregoing considerations it is evident that the saline purgatives have a large field of usefulness in the treatment of heart disease.

Of the vegetable purgatives in common use the most active, croton oil, is occasionally necessary in the treatment of heart disease in persons possessing in unusual degree *dura ilea*. The irritant action on the intestinal mucous membrane and increased

peristalsis by which the vegetable aperients effect their therapeutic result are exemplified by this drug, especially the former, so that in certain lower animals enteritis without purgation can be induced. Castor oil, again, is frequently employed in heart disease for its mild action on the bowels.

So-called anthracine purgatives used in the treatment of heart disease are rhubarb, senna, and aloes. The writer has found 20 grains of compound rhubarb powder suspended in an ounce of peppermint water and taken three times daily (or half that dose) very useful in the cardiac muscle-failure of gouty patients and in that met with in the subjects of chronic Bright's disease. It probably acts, to a large extent, by keeping the bowels free and also as a stomachic. Senna is a common constituent of the "black draught" of hospitals, in which it is usually combined with sulphate of magnesium. Aloes may be combined with a small dose of blue pill and extract of hyoscyamus as an useful occasional purgative in chronic heart cases in the early stage. With regard to the necessity of the presence of bile in the intestines for the action of rhubarb there seems to be a measure of doubt, but it is only seldom that catarrh of the bile ducts in heart disease actually stops the flow of bile into the intestines.

The resinous class of purgatives are represented

chiefly by jalap, than which, in the form of compound jalap powder, there is perhaps hardly a more frequently employed purgative in heart disease. Scammony, elaterium, podophyllin, colocynth, and gamboge are other members of this class of purgatives. Mention has already been made of euonymin in the description of the digitalis class of cardiac stimulants. Although this fact hardly constitutes a reason for preference of this drug, one grain of euonymin with three grains of iridin and one grain of extract of hyoscyamus constitutes an excellent so-called cholagogue purgative in heart-cases. In the exceptional presence of complete obstruction of the bile duct from catarrh, these resinous purgatives are contra-indicated, inasmuch as the presence of bile in the intestines is probably necessary for their activity.

THE "NAUHEIM" TREATMENT OF HEART DISEASE.

In the present day this fashionable treatment of heart disease, real or fancied, cannot be passed over in silence. The treatment presents two aspects: (1) the influence of bathing in waters of varying temperature, and possessed of peculiar ingredients—salines and free carbonic acid; and (2) the influence of exercises of the muscles of the body, the exercises being accomplished in various ways and bringing into play a large or small proportion of the "musculatur" of the patient. The prevailing opinion seems to be that the action of the potent waters is largely a cutaneous reflex one exerted upon the heart and peripheral vessels. At Nauheim great facilities are afforded for modifying the properties of the waters made use of. Thus ordinary brine baths, thermal brine baths, effervescing baths and current thermal brine and current effervescing baths, besides shower and hip baths, are available. The natural warmth and the amount of saline ingredients, including salts of calcium and iron, as well as the quantity of carbonic acid, peculiar to the waters of the

different springs, are capable of arrangement in the bathing houses. In a so-called "course" of baths it is customary to alter from time to time the duration and frequency of the baths, the amount of saline and carbonic acid content and the temperature of the water. As regards reduction of temperature, it is noteworthy that the stimulation of the skin by the carbonic acid gas enables a colder temperature to be borne without discomfort than otherwise would be the case.

The effects claimed for the Nauheim bath treatment are, stated shortly, slowing of the pulse and dilatation of peripheral vessels, with consequent relief afforded to the heart-muscle.

The treatment under consideration has been imitated with varying success in different health resorts, and even at the patient's home, the saline and gaseous contents of the water being artificially supplied.

The writer believes that no useful purpose would be served by his entering into detail concerning the application of the baths in the Nauheim treatment. Such treatment is naturally best carried out by those who make it their business. He cannot, however, forego an expression of his regret that the advocates of the Nauheim treatment should at its comparatively recent resuscitation have sought evidence in its favour from the results

of percussion of the heart, a method of physical examination that is peculiarly open to fallacy. All the more does he regret this in view of the fact that ample evidence of "betterness" will always be forthcoming in the improvement of the patient's "wind" when the state of the heart has undergone real amelioration.

The second part of the Nauheim treatment consists of various muscle exercises. That there are limits to the treatment of heart disease by rest has long been admitted. In 1854 Dr. Stokes expressed his belief that in certain cases of cardiac-muscle failure the patient must "pursue a system of graduated muscular exercise," and he remarked that "it will often happen that after perseverance in this system the patient will be enabled to take an amount of exercise with pleasure and advantage which at first was totally impossible, owing to the difficulty of breathing which followed exertion." Again he remarks: "The symptoms of debility of the heart are often removable by a regulated course of gymnastics or by pedestrian exercise, even in in mountainous countries." ("The Diseases of the Heart and the Aorta," Dublin, 1854, p. 357.) . . . We may often observe in such persons the occurrence of what is commonly known as 'getting the second wind.'" These quotations sufficiently show that Dr. Stokes fully recognised that

"graduated muscular exercise" might have its place in the treatment of heart disease, and that in the emphatic declaration of absolute rest as an essential part of the treatment of heart disease without discrimination there may be risk of exemplifying the "falsehood of extremes."

In Ziemssen's "Handbook of General Therapeutics" Oertel clearly recognised the part that may be played by careful pedestrian exercise (hill-climbing) in the treatment of heart disease. Later the Brothers August and Theodor Schott developed the modern system of Nauheim treatment, in which "graduated muscular exercise" is combined with bath treatment. Zander proposed to give the requisite exercise by ingenious instruments. The details of the Schott treatment by bathing and resisted movements, in which latter the resistance is supplied systematically by a skilled attendant, must be studied in special works dealing with the subject.

In order that this system of treatment may prove of benefit, discrimination of suitable cases is all important. The writer would emphasise the expression used by Dr. Stokes—"debility of the heart"—for the type of case that was in his mind's eye. Most stout people may be said so to suffer, though such individuals are hardly entitled to be placed in the category of the

subjects of " morbus cordis." In days not so long gone by the fact that anyone was ailing at the heart was matter of conversation in bated breath among his friends, if matter of conversation at all. Now a course of treatment for " debility of the heart" has become a fashion of the day, fit to form the subject of polite conversation and to be regarded by the recipient with some slight egotistic exultation. At least he is in the fashion, even in his ailing.

TREATMENT OF ANGINA PECTORIS.

The treatment of angina pectoris must be materially different from that of ordinary chronic cardiac-muscle failure. Dr. Mackenzie tells us that "angina pectoris can occur when the excitability, the conductivity, and the power to produce a rhythmical stimulus are unimpaired" (*Brit. Med. Journ.*, 7th October, 1905), and he concludes that "we are forced to the conclusion that angina pectoris is an evidence of the impairment of . . . contractility." There is much in the history of the disease to recommend this view, but at the same time it is remarkable that the failure of so important a function should be survived for so long a period as 20 years, as for instance in the case of John Hunter, whose heart was found *post mortem* to be small and covered with thickened pericardium. In the case of Dr. Arnold, who died in his first attack of angina, "the walls of the right ventricle . . . were in some parts not much thicker than the aorta, the cavity large. The walls of the left ventricle, too, were much thinner and softer than natural." "There was but one coronary artery, and, considering the size of the heart, it

appeared to be of small dimensions." Supposing such failure of contractility to exist, vaso-motor spasm and rise of blood pressure may be expected to abruptly throw a great strain upon the heart muscle, whose contractility is at fault—and this too, even though the patient should never have what is entitled to be called a "high-tension" pulse. As a matter of clinical experience it is known that relief can be afforded in a majority of cases of angina pectoris by the administration of vaso-dilators. The nitrites are the most efficient remedies of the kind at the present day, but previous to their employment it was well known that alcohol was a most useful remedy, no doubt largely because of its action as a vaso-dilator. It is usual to begin the vaso-dilator treatment of the paroxysm of angina by the inhalation of nitrite of amyl. The patient is given a box of small glass phials or capsules of the remedy, each wrapped up in linen. When a paroxysm is impending the patient takes a capsule and crushes it between his finger and thumb. He then holds the broken capsule to his nostrils for inhalation of its contents. If relief be obtained, remedies of the vaso-dilator class may be prescribed for internal administration. Nitro-glycerin, erythrol-tetra-nitrate, nitrite of sodium, etc., with the object of anticipating and preventing the paroxysm. If attacks occur in the night a

vaso-dilator may be given at bedtime, just as it may be for nocturnal paroxysms of dyspnœa, another and probably allied condition that is apt to be associated with vaso-motor spasm. Sooner or later it is usually found that the activity of vaso-dilators in affording relief, diminishes with use, and their influence may finally cease. When this happens the hypodermic injection of morphine and atropine must be had recourse to, a minute dose being employed in the first instance for two reasons:—(1) that the susceptibility to morphine varies in different individuals, while the patient may have "granular kidneys," and no opportunity have been afforded for examining the urine, and (2) almost certainly the dose will have to be increased in subsequent paroxysms, while it is most desirable to use *the smallest efficient dose*. The morphine should always be *guarded* with atropine as a respiratory stimulant. Much secretion in the bronchial tubes is probably a greater contra-indication, as regards the use of morphine, than Bright's disease itself.

As a regular mixture, 4 or 5 minims of Liq. arsenici hydrochlorici and Liq. strychninæ in ℥ss of water may be given diluted after meals three times daily. In some few cases this mixture seems to be really useful. Unfortunately it often fails, and generally so in severe cases.

Diet is a very important matter to the sufferer from angina pectoris. Indigestion is apt to exert a harmful influence on the malady, and flatulence may possibly determine a paroxysm. It is all important to feed the patient on the kind of food that will improve the nutrition of his heart muscle —the contractility of which there is reason to believe is impaired—and to prevent the evolution of gas. Both objects can be carried out by such a dietary as has already been sketched out (p. 316). The patient should *rest*, in the fullest sense of the word, for some time after meals. Moreover he must be cautioned about the risk of any exertion —running up even a short stair or hurrying for however short a distance to catch a train, walking against a strong wind, &c.—and of going out into a cold atmosphere from a warm one, as out of a warm room into the open-air on a cold day. Confidence will be given him by the knowledge of his having a box of amyl capsules or nitro-glycerine lozenges in his pocket—even the carrying of the brandy or whisky flask may be legitimate, provided it is used and not abused. The spirits should always be diluted for immediate use.

A quiet and regular life should be led, and the patient *must* learn the lesson: neither to worry nor to hurry. Engagement in congenial occupation is, however, conducive to long survival, as

John Hunter's history exemplifies. Nay, his death in the end may with much probability be attributed to an accident—a legitimate and practically unavoidable emotion: anger.

The peculiar form of angina pectoris that is rarely associated with mitral stenosis seems to be much less lethal than the angina pectoris of coronary disease—if we may so call it,—and greater reluctance must be felt before adopting treatment by morphine in these latter cases, which are met with usually in young people, for "facilis descensus Averni."

In the extremely rare event of failure to afford relief by morphine injections, a few whiffs of chloroform may be given, which greatly promote the action of the morphine.

TREATMENT OF THORACIC ANEURYSM.

The treatment of thoracic aneurysm differs essentially in its object from that of ordinary heart disease, even although by virtue of incompetence of the valves, aortic disease of the heart be actually present. A preliminary prolonged rest in bed is a necessary part of the treatment of aneurysm—a rest of five or six weeks' duration at least, at the end of which time activity may be *very slowly* resumed. The writer deprecates deprivation to any considerable extent of liquid in the diet, which, however, should be spare and nutritious. Iodide of potassium is the only drug he is disposed to recommend. There can be no doubt that patients with aneurysm have their symptoms greatly relieved by rest in bed and full doses of iodide of potassium, though it may be difficult to apportion the result to the rest and the drug. It used to be the writer's custom to give 20 grains of iodide of potassium three times daily till lately, when because of the suspicion that these large doses contributed to the frequently present rapid pulse, he gave the drug in doses seldom exceeding 10 grains and often less. He feels uncertain that

the results of recent cases have been as good as those of former cases treated with the larger doses, but it is difficult to form a precise opinion in the matter. The writer's disposition is certainly to return to the larger doses. As regards the actual cure of the disease by the drug, a sanguine opinion cannot be given.

In the terrible condition of pressure on the trachea by aneurysm, paroxysmal attacks of dyspnœa are apt to arise without obvious cause. In these, hypodermic injection of morphine and atropine often afford extraordinary relief most difficult of explanation. At the same time, if there be much secretion in the bronchial tubes, the treatment is not free from risk, and the patient must not be brought too much under the influence of morphine. Very small doses should be given at first and cautiously increased according to circumstances.

Naturally, it is of the greatest importance that the condition of direct pressure by an aneurysm should not be confused with the effects of indirect obstruction produced at the glottis by interference with the innervation of the larynx. The two conditions in practice are not always so easily distinguished as in theory they might seem capable of being. For instance, in the case of direct pressure the voice is often weak and the

inspiratory stridor is greater than the expiratory.

When an aneurysm of the descending aorta causes incessant pain by pressure on the bodies of the vertebra and full doses of iodide of potassium fail to relieve, recourse must be had to morphine and atropine hypodermic injections without delay, and the dose given must be efficient and repeated according to circumstances.

TREATMENT OF PULMONARY EMBOLISM.

(p. 36.)

For the distressing dyspnœa that attends this accident the inhalation of oxygen and the hypodermic injection of morphine and atropine are the most useful means of treatment. The most absolute rest must be insisted on. The patient must on no account be moved for physical examination of his lungs. If there be hæmoptysis the diagnosis is secure enough.

TREATMENT OF SEPTIC ENDOCARDITIS.

With regard to the treatment of septic endocarditis, there is little to be said. The only field of therapeutics in which we are likely to meet with any success is by the inoculation of bacterial vaccines or possibly by the administration of antitoxines. The former, though yet on its trial,

seems the more likely to be successful, inasmuch as it attacks the specific micro-organism responsible for the disease in the particular case and the dosage of vaccine can be accurately governed by the determination of the opsonic index. (A. R.)

TREATMENT OF PERICARDITIS.

Speaking generally, the treatment of pericarditis should be "expectant," to use that somewhat sarcastic expression. It is at least better that the treatment should be such, than that it should add to the sufferings of the patient, while evidence of its efficacy is extremely doubtful, even from the optimistic point of view. The application of blisters is specially referred to here. It goes without saying, that if blistering is deprecated in the treatment of pericarditis, which at least can be recognised by physical examination, it is infinitely more to be deprecated in the treatment of endocarditis or supposed endocarditis for, that a systolic murmur is the result of endocarditis, is seldom a matter of certainty. If the writer has felt himself compelled to protest against the treatment of the direct rheumatic affections of the heart by blistering, he would protest even more vigorously against the blistering of rheumatically affected joints. In the pericarditis of Bright's disease nothing could be worse than treatment by blistering by the

common means—cantharides—with the risk of suppression of urine entailed.

Only when the amount of effusion is so great as to threaten life is the operation of paracentesis pericardii to be entertained, unless the effusion has been found purulent by exploratory puncture. Dr. Sibson, than whom there has been no greater authority in all matters concerning medical anatomy, recommended that the trochar should be "inserted into the distended pericardium at a point just above the upper edge of the sixth cartilage at the lowest part of its curve, more than an inch within the mammary line; and that the instrument should penetrate gently inwards with a direction slightly downwards, so that it may advance into the collection of fluid below the level of the heart." He states that " by this proceeding the collected fluid will be alone penetrated, and the heart will be quite untouched." Unfortunately, these directions refer only to the " case in which the heart has been previously healthy, and is of the natural size," in which case " its lower border is elevated above the level of the 5th space, when the effusion into the pericardium is at its height. When the heart is enlarged and " to be felt heating in the 5th or even 6th space at the time of the acme of the effusion," Dr. Sibson recommended the selection of a point " at the space between the left edge of the ensiform

cartilage and the right border of the 7th cartilage in the epigastric region." He stated that "the lower border of the fully-distended pericardium is usually a little above, and sometimes even below, the lower end of the ensiform cartilage," and drew the conclusion that the "pericardium may therefore be safely punctured through a point corresponding to the middle or the lower portion of that cartilage." He is speaking, of course, of the sac greatly distended with fluid. ("A System of Medicine," edited by J. Russell Reynolds, M.D., F.R.S., vol. iv., p. 436–7.)

When the patient can be given an anæsthetic perhaps the best plan is to make an incision with aseptic precautions down to the pericardium, which can then be punctured by trochar and canula, the serum being allowed slowly to run off. Should the fluid be purulent the pericardial sac itself must be incised and a drainage tube inserted. Recovery has taken place after the latter procedure.

The writer has resorted to surgical interference only under special circumstances, and when the effusion has seemed to be threatening the life of the patient. The great majority of cases of pericarditis do not require operation, which should never be adopted without very careful consideration of all the circumstances of the case and of the necessity of each individual case.

MECHANICAL REMOVAL OF SEROUS EFFUSIONS.

From time to time in many cases, and towards the end in most cases, of heart disease, the removal of fluid by mechanical means becomes necessary. This is specially likely to happen with regard to ascites and hydro-thorax. Less commonly, Southey's trochars and tubes may be used for the purpose of removing fluid from the cellular tissue. Curious differences in the freedom with which the serum flows through these tubes are observed, suggesting differences in its composition: in some cases an admirable result is obtained, while in others the fluid soon ceases to flow and no benefit to the patient is achieved. In all surgical procedures, however trivial they may seem, the greatest aseptic precautions must be taken, and especially is this the case when œdematous cellular tissue is subjected to acupuncture simply.

VOLUME OF THE BLOOD

APPENDIX.

THE VOLUME OF THE BLOOD IN RELATION TO HEART DISEASE.

By J. Lorrain Smith, M.A., M.D.

In the investigation of the close relation which exists between disease of the heart and anæmia, or other morbid conditions of the blood, too little attention has been paid to the quantitative variations in the volume of fluid contained in the vessels. The histology of the red and white cells has been carefully studied, and a considerable amount of important work has been done on the chemical and physical character of the plasma. In contrast to this, however, the problems connected with the volume of the blood have been so little dealt with that their importance can scarcely be said to be realised. Yet it requires no argument to prove that variation in blood volume is a change of primary importance in anæmia. The routine examination of the percentage of corpuscles or hæmoglobin in a cubic millimetre of blood is only at best a partial observation, and to complete the investigation we must ascertain the number of cubic millimetres of blood

present in the blood-vessels at the time of the examination. This elementary consideration is not clearly enough expounded in text-books on hæmatology. Again it is not difficult to show that increase in the volume of the blood, when it occurs, must throw a strain on the heart, and must add to the embarrassment of the circulation which characterises heart disease. It is even worthy of inquiry whether or not cases arise where an illness, which at the beginning of its course was essentially a condition of plethora, has in the later stages added the morbid effects of cardiac disease as well, owing to the dilatation of the heart which has occurred.

The discussion of plethora as a possible condition of the circulation is of very old standing, and its existence was formerly assumed by many pathologists. Cohnheim and others, however, arguing from their failure to produce plethora in the normal animal by injecting its vessels with excessive fluid, came to the conclusion that a true simple plethora could not under any circumstances be more than a transitory condition. (Lectures on General Pathology: chap. vii.)

What these experiments proved, however, was that when fluid is injected into the vessels of the normal animal, it is rapidly excreted by the kidney, or otherwise, and this process continues

until only the normal quantity of fluid remains in the vessels. In other words, the kidneys, etc., act as regulators of the volume of the blood. It was apparently not clear to Cohnheim that it was impossible to attach to this series of experiments any argument as to the change which may occur when, from any cause, the regulation of the volume of the blood breaks down. The wide acceptance of Cohnheim's conclusion was in no small measure due to the fact that there was no reliable and applicable method of measuring the volume of blood actually in the vessels at a given time. It is, further, necessary to make observations on human beings, in whom the varieties of anæmia and dropsy have been carefully studied. Only by such an investigation could we obtain data regarding the possibility or otherwise of plethora.

The carbon monoxide method of estimating the volume of the blood in the living subject makes it possible to carry out the investigation which is required. This method has been applied to the study of the volume of the blood in man and animals in the normal condition. It has also been applied, to some extent, to the investigation of anæmia and blood disease, and its extension to the corresponding and closely related problems of heart disease is obvious. In view of the interest

in the study of heart disease of the whole investigation, Dr. Graham Steell has asked me to write a short account of the method, with a view to indicating the kind of problems which its application may be expected to solve.

The principle on which the method depends may be put as follows:—Since the colour of hæmoglobin is definitely altered by union with carbon monoxide, it is possible by measuring the change in the colour of the blood caused by inspiring a known quantity of the gas, to calculate how much hæmoglobin there is in the body, and from this to deduce the volume of the blood. The subject of the experiment breathes, let us say, 100 c.c. of the gas into his blood. A sample of the blood when tested shows by the change in its colour that 20 per cent. of the hæmoglobin has united with CO. We know then that in the body of this subject there is hæmoglobin sufficient to contain, when saturated, 500 c.c. of CO. The next point to determine is the quantity of blood in which this amount of hæmoglobin is distributed. The result is obtained by the hæmoglobinometer, which tells us the percentage of hæmoglobin in a given volume of blood. The standard scale of the hæmoglobinometer is obtained from observation of the colour of the blood of healthy young men, and "100 per cent." of the

scale is merely an average from the results. It is possible, however, by direct analysis to find the value of "100 per cent. hæmoglobin" in terms of capacity for CO, and this is actually 18·5 c.c. per 100 c.c. of blood. By this means we reduce our observations on the total amount, and on the percentage of hæmoglobin in the blood to the same terms, viz., capacity for CO. It is then a very simple calculation to find the volume of the blood. Assuming that in this case the subject's blood contains 100 per cent. hæmoglobin, the capacity for CO being therefore 18·5 c.c. per cent., the calculation would run as follows:—

$$\frac{500}{18{\cdot}5} \times 100 = 2700.$$

the volume of the blood in c.c.

Full details of the method and apparatus as they were originally described by Dr. J. S. Haldane and myself are to be found in the *Journal of Physiology*, vol. xxx., p. 331.

The method is easily applicable to the normal subject. The person must breathe a quantity of carbon monoxide gas sufficient to saturate the hæmoglobin 10—15 per cent. The effects of this are inappreciable by the individual breathing it. The first experiments we carried out on ourselves, and on the men and women students in the Medical School. Naturally, with patients, when

from weakness of the heart or from poverty of the blood there is any embarrassment of the respiration, the method can only be used with caution. Even anæmic patients, however, suffer very little disturbance, and the whole observation does not last more than 15 minutes.

This method has, as I have said, been applied in pathology chiefly to a study of the blood. I found it possible to carry out an investigation on the blood in chlorosis and in pernicious anæmia (*Trans. Path. Soc. of London*, vol. 51, p. 311). Chlorosis is a condition which, though persistent, is eminently curable, and rarely, apart from complications, has grave consequences. It often comes on rapidly, and it occurs early in the patient's life. It often disappears also with great rapidity. By the CO method it is demonstrated that the total amount of hæmoglobin in the circulation of the chlorotic subject is approximately normal. The essential change in the disease is therefore an increase in the volume of plasma. If the total amount of hæmoglobin remains normal, and yet a cubic millimetre of the blood has, let us say, only 50 per cent. of the normal amount of hæmoglobin, there must of necessity be double the normal number of cubic millimetres of plasma. In contrast to this, in pernicious anæmia there was found marked deficiency in the total amount of

hæmoglobin. The volume of plasma, on the other hand, varied irregularly in this disease. Sometimes it was subnormal, and at other times it was greatly increased. It was found, however, as in cases of chlorosis, that a reduction in the excessive volume of fluid in the circulation was at once followed by a great alleviation of the symptoms of the disease.

The results of this direct investigation clearly lead us to a conclusion opposed to that of Cohnheim regarding the impossibility of plethora in the circulation, and this conclusion is of the utmost importance in relation to the problems of heart disease, as we shall see. In the first place, the plethora which occurs in anæmia explains in a very natural way the cardiac symptoms which are so often found to accompany the disease. The blood is poor in quality and its respiratory value is greatly diminished. Hence there is dyspnœa on exertion. To compensate for these defects quickened action of the heart is called for; but the excessive volume of blood which has to be propelled through the vessels, and which constantly tends to overfill the heart, leads to a certain amount of dilatation of the left ventricle. In the next place, the excessive plasma is a direct cause of the œdema which accompanies anæmia. In chlorotic patients there is œdema of the feet,

and the tissue spaces throughout the body generally are well filled with lymph. As the patient's blood improves the œdema disappears.

As regards the bearing of these results on the problems of heart disease, it has, in the first place, been shown that in anæmia a condition of persistent plethora becomes established; secondly, the accumulation of fluid in the blood-vessels explains certain of the respiratory and cardiac symptoms which are associated with anæmia, and which closely resemble those of heart disease. Further, a direct connection has been made out between plethora and general œdema.

The occurrence of œdema and dropsy is one of the three cardinal symptoms of heart disease, and it would *à priori* seem remarkable that a plethora of the lymphatic system should occur without any accumulation of excessive plasma in the blood-vessels. One would rather expect to find that in heart disease failure in the action of the heart had led to passive overfilling of the circulation, and that as a secondary result of this, and only after this had been established, fluid began to accumulate in the lymphatic spaces also.

It was possible, however, to put the explanation on a sound basis by the investigation of an example of failing circulation. The opportunity was

given in a case of chronic pericarditis, with cyanosis and ascites, where plethora and œdema resulted from embarrassed circulation. (*Trans. Path. Society of London*, vol 53, p. 136.)

The cause of the embarrassment of the circulation in this case was adherent pericardium, and the sac of fibrous tissue which had arisen from the union of the two layers was of an unyielding character and would greatly hinder the movements of the heart. At the same time, as the patient was a boy, 12 years old, his heart must have been growing, and this also would be impeded by the thickened pericardium. Hence there was a two-fold cause of embarrassment of the circulation. The patient was under the care of Dr. McKisack, in the Royal Victoria Hospital, Belfast, and when he came to the Hospital, on November 15th, he had enlarged liver and spleen, and dyspnœa, but there was no sign of ascites. In spite of the fact that he was kept in bed, ascites developed after a month, and in six weeks he had hydrothorax as well. The ascites was clearly marked by the middle of December, and along with this it was observed that his weight had increased 11 lbs. During the same time the hæmoglobin fell from 114 per cent. to 89 per cent., and the volume of his blood increased from 2,557 c.c. to 3,355 c.c. The increase in the volume of the blood, the fall

in the percentage of hæmoglobin in the blood, the sudden and marked rise in body weight, are all explained by the fact that a plethora of fluid in the circulation, and in the lymphatic spaces, had resulted from failure of the heart.

Grawitz has pointed out that the first sign of failure of compensation in the heart is a diminution in the number of the corpuscles in the blood, and it is clear that in this case a plethora inside the vessels had occurred before there was any sign of ascites. At the date of the first examination, the blood was only slightly under double the normal volume, and the total amount of hæmoglobin was more than double the normal. This large mass of blood was rich in hæmoglobin (114 per cent.), and the corpuscles numbered 6·34 millions per cubic millimetre. The blood, while it was concentrated, was also greatly increased in volume.

This case is itself sufficient to show that in the interpretation of the phenomena of an embarrassed circulation, it is necessary to have before us measurements of the volume of the blood. Here the mere observations of the percentage of hæmoglobin alone were more or less futile. The highest percentage was 114 per cent., and the lowest 89 per cent. Neither of these figures in themselves are in any way remarkable, and by

themselves give us no indication of the profound changes that had occurred. The case shows that here at least dropsy was preceded and accompanied by an increase in the volume of plasma in the blood. Finally, it introduces the element of compensation by hæmogenesis. We are accustomed to regard increase in the muscular power of the heart as the only form of compensation for embarrassment of the circulation, but here it is shown that the increase in the volume of the blood was in a measure compensated for by the increase in the total amount of hæmoglobin. By this means the respiratory value of each c.c. of blood propelled through the lungs was about the same as that of normal blood, but the weakened heart had to circulate an abnormally large volume of blood, and hence there was cyanosis.

The results from the investigation of this case help us to appreciate to some extent the type of problem in the study of heart disease which the carbon-monoxide method will enable us to solve.

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