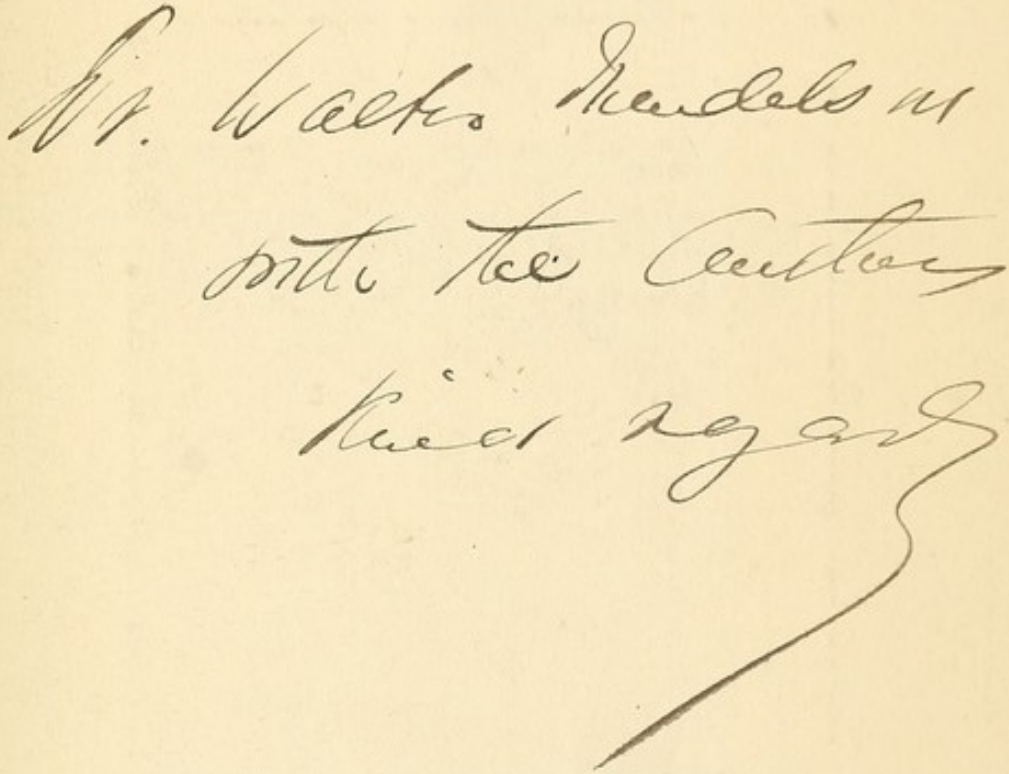
Diseases of the heart and aorta / by Thomas E. Satterthwaite.



Diseases of the Heart and Aorta

BY

THOMAS E. SATTERTHWAITE, M. D.

PROFESSOR OF MEDICINE IN THE NEW YORK POST-GRADUATE MEDICAL SCHOOL; CONSULTING PHYSICIAN TO THE POST-GRADUATE, ORTHOPÆDIC AND BABIES' HOSPITALS; PRESIDENT OF THE MEDICAL ASSOCIATION OF THE GREATER CITY OF NEW YORK

"Progre is the Law of Life"—BROWNING

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PREFACE.

The author of this volume has tried to place before the general practitioner a brief, simple, but practical, presentment of cardiac and aortic affections, chiefly from the standpoint of his personal experience.

Unusual opportunities for seeing the clinical and pathological aspects of these diseases have furnished him with the material illustrating the relation of clinical signs to post mortem appearances. Over a hundred cases from his individual records are given at some length.

But the book is not an encyclopædia. Little space is given to the Anatomy or Surgery of the Heart or Aorta, to Congenital Anomalies or Tumors, or to Parasites of the Heart. And there are obvious omissions of subjects that are sometimes treated at length in books on Heart Disease. For example, only brief mention is made of the Sphygmograph, because it is thought to mislead rather than help at the bed-side or in the consulting room. The practical value of the Sphygmanometer, however, is recognized. Nor do theories or methods of treatment that have little, or merely a historical value, receive much notice. At the same time it is believed that the exceptional quantity and character of the data made use of, will give proper emphasis to the newer views expressed. From this point of view, the comparative values of diagnostic signs, and the relative frequency and gravity of valvular diseases are subjects that may enlist special interest. A good deal of space is given to modern methods of treatment.

The volume is based on a series of articles that originally appeared in our Medical periodicals, but have been revised, while new chapters have been written. The majority of the cuts are original.

With all its failings, for which the author must be responsible, it is hoped that the general practitioner will find it a useful addition to his store of handy-books, helping him to manage his heart cases more intelligently, and therefore with more satisfaction to himself and his patients.

March, 1905.

"That writer does the most, who gives his readers the *most* knowledge, and takes from them the *least* time."—*Colton.*

DEDICATED
TO
I. B. S.

Chapter I.

THE DIAGNOSIS OF HEART DISEASES.

Success in the diagnosis of heart diseases calls for method, thoroughness and a proper consideration of objective and subjective symptoms. But method is of the first importance, and in fact is absolutely essential. Both positive and negative conditions should be recorded in a systematic manner. For this purpose a blank such as is given on page 8 should be used. It is comprehensive enough for ordinary clinical use, and if filled, will tell its own story. Then it should be put aside for subsequent reference.

After noting the *date* and the patient's *name* and *age* it may be necessary to determine the *height*, *weight* and even the *measurements* of the body or limbs. Such data are very important, as for example, in the management of the fat heart, which is closely related to general obesity, where scales and measurements are essential aids in diagnosis and treatment. But measurements are hardly less useful when wasting diseases, such as tuberculosis and carcinoma, complicate the cardiac trouble. The measurement chart on page 9 is adapted for these cases.

First in order among physical signs is the *pulse*. It may be *frequent* or *infrequent*, according to the number of pulsations per minute, the normal being placed at 72, or from 70 to 75 for an adult male. However, *quick* and *frequent* are not synonymous, for the word quick means merely that each expansion of the vessel is brief, in point of time. The word *large*, applied to the pulse, implies that the vessel is well distended during cardiac systole. On the other hand, in the *small pulse*, the systolic distention is less than normal. The pulse usually gives important indications in heart disease. Oppolzer, it is said, would make a diagnosis of aortic insufficiency before his hospital class, after merely placing a hand on the posterior tibial. For he held that the trip-hammer rhythm is pathognomonic.[1]. In arterio-sclerosis a diagnosis may be made from the resistance felt in the walls

[1] It probably is, excepting in aneurism and anaemia.

EXAMINATION BLANK.

Name	Date	Age	Weight
Pulse	Resp.	Height	Temp.
Palpitation	Head Symptoms		
Pain Apex Præcordium Liver Elsewhere	Other Subjective Symptoms		
Phys. Appearance Impulse	Bloodvessels Thrill		
Aortic Direct Aortic Regurg. Mitral Direct Mitral Regurg.	Pulmonary Direct Pulmonary Regurg. Tricuspid Direct Tricuspid Regurg.		
Rhythm { Pulse Cardiac			
Liver	Spleen		
Lungs	Digestion		
Urine			
Notes			
Treatment { Medicines Exercises Diet Baths			

Measurement Chart.

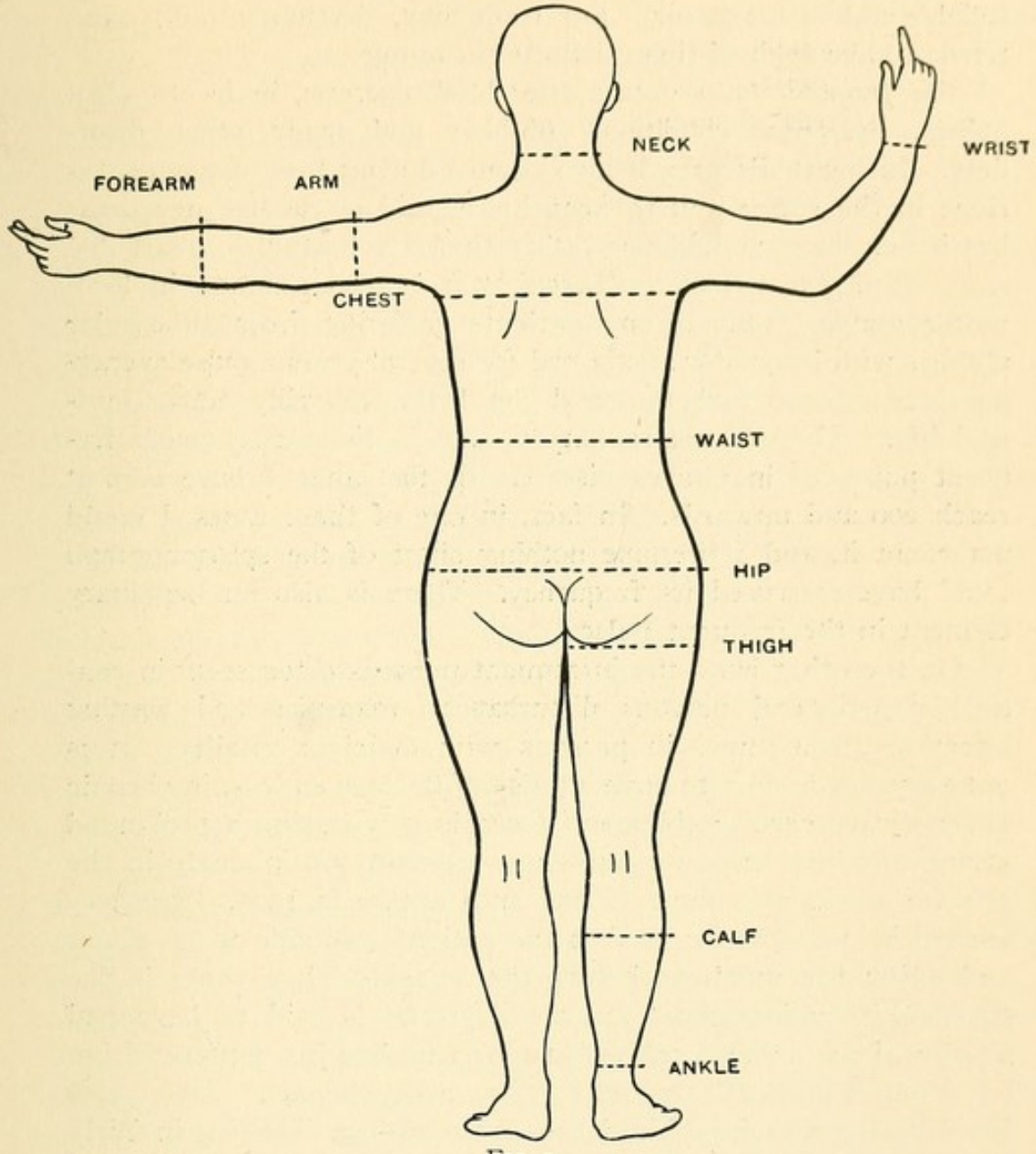

Fig. 1.

Name.....Age.....Height.....

Date	Neck	Arm	Chest	Forearm	Wrist	Waist	Hip	Thigh	Calf	Ankle	Date	Weight

of the artery. Indeed, next to auscultation, the pulse is our most reliable aid in diagnosis. For frequency, rhythm, rapidity and tension have each of them definite meanings.

The *frequent pulse* occurs after brisk exercise, in fevers, alcoholism, hysterical conditions, phthisis and many other disorders. In heart diseases it also occurs during loss of compensation; in the coffee and tobacco heart, and in cardiac neuroses; but it may be a coincidence rather than a symptom in heart disease. It is *paroxysmal* or *chronic*. It is also common in locomotor ataxia. One of my patients suffering from tubercular phthisis with locomotor ataxia had for several years a pulse averaging between 120 and 130, and yet led a tolerably active business life. The greatest rapidity is seen in the paroxysmally frequent pulse, as in Graves disease. In the latter I have seen it reach 200 and upwards. In fact, in one of these cases, I could not count it, and I presume nothing short of the sphygmograph could have measured its frequency. There is also an hereditary element in the frequent pulse.

On the other hand the infrequent pulse is often seen in central or peripheral nervous disturbances, toxæmias and wasting affections; sometimes in persons with deficient vitality. It is not very uncommon to have a pulse of 60 or even less, in chronic degenerative cardiac changes. Occasionally during a prolonged attack of heart failure, a pulse may remain continuously in the 30's for weeks at a time. I had such a case in 1900. Recovery ensued, at least so far as that the patient was able to go about and enjoy life much as before the attack. Then there is the *physiologically infrequent pulse*. Napoleon is said to have had a pulse of 40. I saw a patient in 1901 who had just suffered from an apoplectiform attack. His pulse, also, was 40. Two years later when I examined him it was from 36—40. He was in fairly good condition. There has been no change since then, so far as I know. A patient I saw in 1903 with Dr. Swasey of New Britain, Connecticut, has had a pulse of about 28 for the past year (1904), and at last accounts was attending to an active business, and had recently returned from a trip to Europe.

In these cases it is important, however, to distinguish between the infrequent pulse due to infrequent cardiac action, or mere failure of the blood waves to reach the wrist. In a lady recently under my care, where the heart contractions averaged 80, only 30 pulse beats were felt at the wrist, when she first came under my observa-

tion. She had been under a severe nervous strain, but made a successful recovery.

Some of the more important varieties of pulse are worthy of mention.

The *alternating pulse* is indicated by a fulness or diminution of every alternate beat. In the *intermittent pulse* some of the heart waves fail to reach the wrist, as was the character of the pulse in the case just given. In the *deficient pulse* the *"missed beats"* are due to failure of the heart to contract. In the *bigeminal* and *trigeminal* pulse the second and third beat respectively fails to reach the wrist.

The *arhythmic* pulse is one of the signs of heart failure. When there are three even or similar heart beats, the pulse is said to have the *triple* or *gallop rhythm,* similar to the rat-tat-tat of a galloping horse. Sometimes as in *delirium cordis,* which is apt to occur in profound heart failure, the pulse is wholly irregular in force and frequency, and as regards the intervals between beats; and yet as we all know, recovery is possible. This matter will be further considered in connection with heart sounds. It is always well to note the rate of *respiration* in heart diseases, because it is often hurried or embarrassed. In the *Cheyne-Stokes* variety the patient stops breathing. Then shallow respirations begin slowly, increasing in depth and rapidity, until they reach a certain point, after which they decline, getting slower and more superficial until they finally stop. This cycle is then repeated and occupies from a half a minute to two minutes. In acute heart diseases and in complications, the *temperature* also gives important information. As *palpitation* is one of the cardinal symptoms of heart disease no examination is complete without a record of its existence or non-existence. *Head symptoms* should also be noted. They are apt to be marked in aortic disease. *Pain* should also not be overlooked. Pain at the apex is common in endocarditis. The subjective pain of angina in the præcordial region in distinctive. Præcordial pain on pressure may be due to pericarditis. Pain over the ensiform is also one of the signs of pericarditis. Pain over the liver is common in loss of compensation, owing to the congestion of the organ. Pain in the left arm and shoulder is also common in heart disease and arteriosclerosis. It is important to determine whether there is an *impulse.* This is apt to be wanting in myocardial affections. Failure of the impulse is a warning that heart failure may suddenly

supervene. A *thrill* indicates obstruction at a valve or in a vessel. It is one of the most important signs in mitral stenosis.

In listening to the heart, one should make note of what is heard at each valve, both during systole and diastole. The failure to discover the more uncommon heart lesions, has been largely due to neglect of this precaution. According to the present scheme, the character of the direct and indirect sounds at each of the four valves should be recorded.

In cases of irregular pulse the *cardiac rhythm* should be noted as distinguished from the *pulse-rhythm*.

In chronic affections, the *liver, spleen* and *lungs* are pretty sure to be engorged with blood at some time or other, and the engorgement may become permanent. In the lungs, chronic interstitial thickening and embolic deposits are also to be found, with bronchorrhea, if not chronic bronchitis. The *digestion,* too, should be considered. It is apt to be disordered in chronic heart diseases.

The last few years have seen important modifications in our views of the normal position and shape of the heart. Before the X-ray was used in clinical medicine, the teachings of Luschka and Spalteholtz (1900) were successively accepted as guides for students of topographical anatomy, because their drawings from frozen sections were thought to represent living conditions. Now, however, it is realized that these represent only the relations of the organ to its environment, as seen in death.

Besides better mehods of manual percussion, with *skiagraphy, fluoroscopy*[2] or *fluorography* have so combined to confirm the inaccuracies of the anatomists that corrected diagrams of the heart with its relations to the other thoracic and abdominal organs have become necessary (Figs. 3 and 4). This fact has been recognized in some of the more recent text-books.

Until a comparatively recent date, various plans were in use for mapping out the heart. Some examiners were able to outline it fairly well by percussion, while others relied on dulness and flatness. Believers in the latter plan make the areas of cardiac dulness and flatness fall within two triangles, the larger including the smaller; the larger triangle having for its vertical side a line let fall perpendicularly from the episternal notch; for its base, a horizontal line drawn to meet it from the upper border of the sixth left costal cartilage at its

[2] First brought to the attention of the profession by Dr. H. Campbell Thompson, of the Middlesex Hospital, in *The Lancet,* of Oct. 10, 1896, and Dec. 12, 1896.

junction with the rib. The hypothenuse connecting their extremities is drawn from the vertical line at the level of the upper border of the second left costal cartilage to the point usually occupied by the heart's apex. The lesser triangle is formed of the same vertical and same base line; but the hypothenuse, leaving the vertical line at the level of the third left costal cartilage, passes downward, paralleling the other hypothenuse. The areas of dulness and flatness are presumed to be found within the larger triangle, flatness within the smaller. This conception is manifestly erroneous. Besides, flatness is no proper guide for the dimensions of the heart. The flat areas change with every breath, as the to-and-fro movements of the lungs alternately diminish and enlarge them. Further, the heart neither corresponds to angles, nor has them during life. Another class of practitioners does not attempt to delimit the heart, but simply indicates by vertical lines the extreme right border and extreme left border and the apex. In women and in stout people there is, of course, manifest difficulty in all these matters, and yet fluorography shows that in a male of ordinary build, the heart can be mapped out in its entire contour up to the origin of the great vessels with sufficient accuracy for practical purposes. For, by the X-ray, the whole inferior border of the heart is brought plainly into view; indeed, as the diaphragm descends it leaves a vacuum between itself and the heart. This line cannot be delimited, however, by any kind of percussion. But inasmuch as X-ray work is not often available in the office or at the bedside, it is well to know that in ordinary practice enough of the cardiac borders can be determined by percussion for practical purposes, and in the following way:

With a dermatographic pencil[3] draw on the skin a horizontal line from nipple to nipple, defining each by a circle (Fig. 2); next draw a vertical line from the episternal notch to the point of the ensiform appendix. Then trace by percussion the right and left borders of the heart. This can be done nearly to the inter-mammillary line. Designate the apex by an X. Connect the two lines at the apex by continuing the curved lines on the same arcs of circle, as have been already drawn above the horizontal line, and the contour of the heart will be indicated with sufficient accuracy. Except in stout people, or women with flabby breasts, the nipples are reliable landmarks.

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[3] Faber's is not so good as the *Express wax crayon* used in marking express packages.

A tracing made with these crayon on a sheet of French *vegetable fibre paper* laid on the skin may be kept for subsequent reference.

By these two simple lines the relation of the heart is shown to the middle line of the body, and the dilatation of the several chambers of the heart brought into contrast. Fig. 2 is the *cardiogram* of a patient who was under my care in 1891. It shows clearly the contractions that took place between March 1st and April 12th and how a record of them was kept. Of course, this was an unusual case, the patient being a neurotic subject, with mitral regurgitation and sub-acute dilatation of the heart.

With the aid of these recent discoveries, the position and contour of the heart may be outlined as follows: The heart's dulness commences in the second right intercostal space, at the edge of the sternum, just above the 3d rib. Curving outward to the right it follows the line of a segment of a circle from this point to the apex. The line crosses the cartilages of the 3d, 4th and 5th ribs, reaching the sternum at the fifth right costo-sternal junction; but the line is never more distant from the sternum than the breadth of a rib, and the most distant point is on the 4th rib. (Fig. 3).

On the left side the line of dulness commences in the second interspace, just below the 2d costal cartilage, and, curving to the left so as to form a segment of a circle, joins the curve of the right side, at the apex, in the 5th space. This curved line crosses the third costal cartilage at about the breadth of two ribs inside the chondro-costal junction, and the 4th rib a little more than the breadth of a rib from the margin of the nipple, crossing the cartilage of the 5th rib about two ribs' breadth inside its costal articulation. The apex should be the breadth of 1½ to 2 ribs inside the inner line of the nipple.

The heart in life is ovoid in shape, its right and left borders comprising regular arcs of circles which meet at the apex, forming, of course, not an acute angle, but a rounded point.

In determining the contour and position of the heart, feel for the apex beat. In a doubtful case (as in myocardial disease) identify the point at which the heart sounds are best heard by the stethoscope. If the sounds are still obscure let the patient walk briskly around the room, a few times, so that the organ will act with more energy. In spare people there should be little difficulty in mapping out the outline of the heart on the right side by percussion, at least from the 2d right interspace as far as the 4th right interspace, or possibly the 5th right costal cartilage, and certainly to the inter-mammillary line (see Fig. 3).

However, it is not essential to map out the entire contour by percussion, for given the apex and the arc of a circle

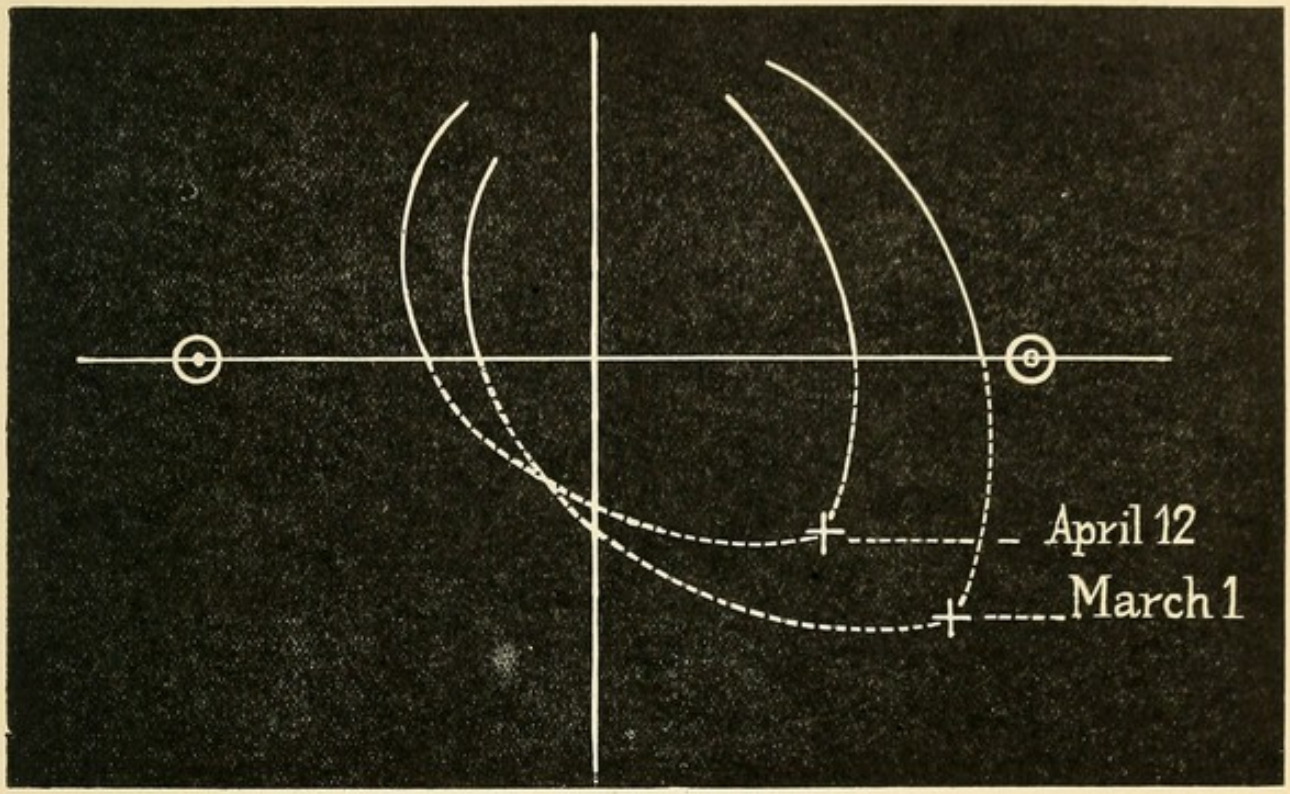

FIG. 2.
Cardiogram.

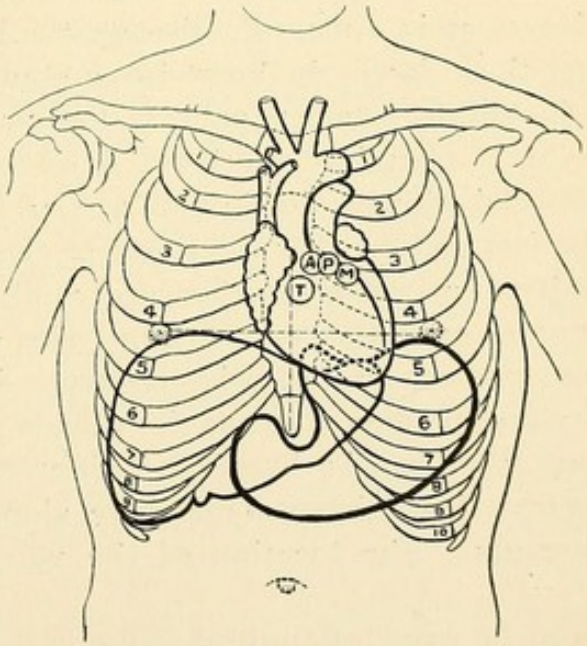

FIG. 3.
Relation of the heart to the viscera.

between the 2d right interspace and 5th rib, the remainder of the arc can be established by simply extending its known portion to the apex. (Fig. 2.)

Percussion can not delimit the contour between the 5th right sterno-costal junction and the apex, because of the interposition of the liver and stomach. However, this is not very material, for the reasons just given. On the left side it is easier to define the heart's margin, because the greater resonance of the lungs makes the percussion note (owing to its greater distance from the sternum) contrast more sharply on the left than on the right side. Usually we can delimit the heart as far as the 4th rib, sometimes as far as the inter-mammillary line (Fig. 3), occasionally a trifle lower. But on either side we have determined the line of curve, and we have but to continue both and they will intersect at the apex. It is true, as Gerhardt says, (*Lehrb. d. Ausc. and Perc. Tuebingen,* 1890) that the heart is capable of *displacement* to one side or the other from 1¾ to 2¾ inches; but this displacement is exceptional, or due to changes in posture. In the case of one of my patients (Case XLVIII) with spinal curvature, the apex was displaced beyond the nipple by the curvature, and subsequently brought 2⅝ inches inwards, by corrective treatment. Emphysema crowds the heart downwards; tumors and effusions displace it laterally; dilated abdominal viscera push it upwards. Sometimes, though rarely, when there is a general descent of the viscera as in Glénard's disease, the heart falls. But notwithstanding these facts, the heart has a standard position in the chest, and in healthy average patients the contour is such as has been described. Certainly the heart is so fixed that it does not ascend or descend with the diaphragm, as Gerhardt has claimed.

Owing to the fact that the four valves are so close to one another that the extremity of a Bowles' stethoscope can be made to cover all of them, more or less, at one time, the ear cannot distinguish between the various sounds distinctly, if placed immediately over any one of them. But as the sounds are conveyed by the blood current, they can be heard and differentiated with considerable accuracy, if we listen somewhere along the course of the several currents. The location of the valves is shown in Fig. 3.

As seen from the front, the mitral valve is behind the 3d left interspace, the breadth of a rib from the edge of the sternum. The aortic valve lies behind the left margin of the sternum, adjoining

the 3d interspace. The pulmonary lies between the two, on about the level of the aorta.

The tricuspid lies behind the sternum, a little to the left of the median line, and opposite the junction of the 4th left costal cartilage with the sternum. All of the valves are somewhat to the left of the median line. These statements as to the shape and position of the heart and the location of the valves, vary from some that have been given; but they are the results of careful personal study.

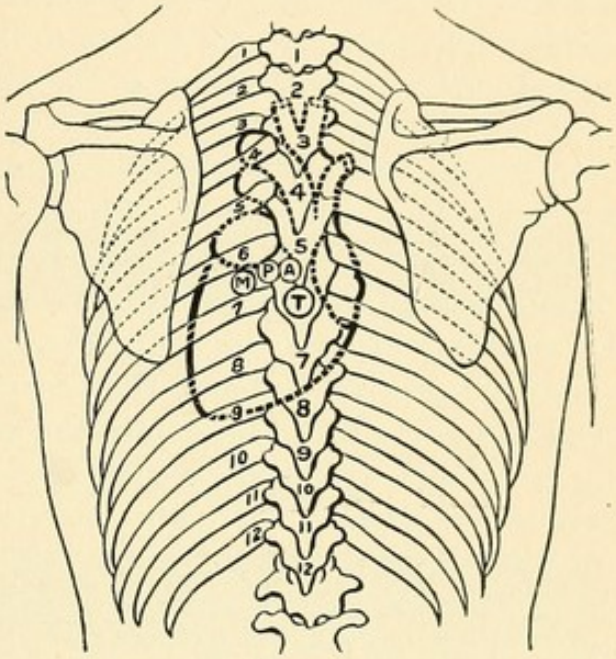

FIG. 4.
The heart as seen from behind.

In this connection the following note from Holden's Anatomy[4] is apropos. It says, "Anatomists differ much in the descriptions they give of the relations of the valves to the thoracic walls, in fact, no two agree in all the details." And yet it is proper to say here, that my statements agree pretty closely with those given in Holden, which are that the left auriculo-ventricular valve is opposite the 3d left intercostal space and about one inch to the left of the sternum. The pulmonary valve lies immediately behind the junction of the 3d left costal cartilage with the sternum; the aortic valve is on a level with the upper border of the 3d left intercostal space, just at the left of the middle line of the sternum. The position of the aortic and pulmonary valves as given by Holden I regard as too high, but the difference in our views is not, after all, a very material one.

[4] Edition of 1901, p. 189.

Seen from behind, the upper level of the heart corresponds with the center of the 4th dorsal vertebra, and the lower margin of the 5th rib, on the left side; the upper margin of the 6th rib on the right side. The apex is opposite the 8th left interspace, about midway between the spines of the vertebræ and the free border of the ribs. The mitral valve is opposite the 6th interspace, close to the left margin of the 6th dorsal vertebra. The aortic lies to the left of the median line, opposite the point where the 5th dorsal spine overlaps the 6th; the pulmonary lies between them; the tricuspid covers the median line, though slightly more to the left than the right, and is opposite the root of the spine of the 6th dorsal vertebra (Fig. 4).

In considering heart murmurs the physiological action of the heart must be taken into consideration. The movement of the blood is caused by the contraction of the auricles, ventricles and vessels. The blood enters the auricles by the veins, and then is expelled by the auricles through the auriculo-ventricular openings or valves into the ventricles; when the ventricles are filled, they contract and force this blood back into the vessels, the left ventricle driving a column of blood through the aorta into the greater or systemic circulation; the right ventricle driving another column of blood into the lesser or pulmonary circulation. Then follows a contraction of the great vessels, the aorta and pulmonary artery. In health the action of the ventricles in closing is attended with a sound or tone, due to three principal causes: 1. The closure of the auricular-ventricular orifices. 2. The muscular action of the ventricles. 3. The vibration of blood in the ventricles. Both auricles and great vessels contract during the filling of the ventricles (diastole) and hence any sound produced during this period is called *diastolic,* but they are not synchronous, the vessels contracting at the beginning of diastole, and the auricles at the end. A systolic sound is produced during the contraction of the ventricles (systole), and the word *presystolic* is accepted as indicating a sound produced at the end of diastole, or during the contraction of the auricles; or in fact, any sound not produced during the time for the contraction of the aorta. The second sound is chiefly due to the closure of the aortic and pulmonary valves, and the vibration of blood in the aortic and pulmonary arteries. Valves, muscular action, chordæ tendinæ, vessels and the vibration of the blood, produce heart sounds.

Now, supposing the time occupied by these actions were divided into eighths, one-eight would be occupied by the contrac-

tion of the large vessels and auricles, three-eighths by the contraction of the ventricles; the remaining four-eighths or one-half, by the filling of the auricles and ventricles.

The following diagram illustrates the rhythm of the heart under normal conditions:

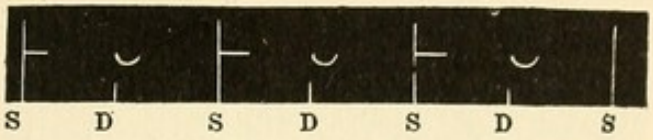

Fig. 5. Vierordt.

When we listen at the apex or at the ensiform cartilage the first sound will be more accentuated, as in Fig. 6.

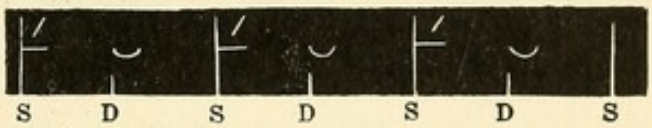

Fig. 6. Vierordt.

On the other hand, if we listen at the base, the second sound will be most accentuated, as in Fig. 7.

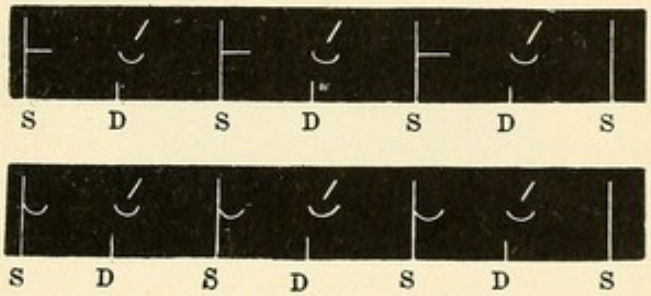

Fig. 7. Vierordt.

In health the sounds are modified, as to intensity, by the elasticity of the chest, thickness of superimposed tissue, especially by fat, and in women by the breasts, and in all by age. Sharp accentuation of the second sound is, if continuous, a very sure sign of hypertrophy of the corresponding ventricle. And it is particularly important as related to the second pulmonary sound, which, if accentuated, means dilatation and hypertrophy of the right ventricle. In arterio-sclerosis of the aorta, the second aortic sound may be slightly resonant or bell-like. And yet when there is heart failure the accentuation of the second sound fails. But all heart sounds are more or less faint in heart failure, pericardial affec-

tions and emphysema. In valvular affections the heart sounds are replaced by murmurs.

The *tick-tack* rhythm is known as the *embryocardial* or *pendulum* rhythm, and is abnormal in the fœtus.

The heart sounds may be *doubled* or *trebled*, as is shown in the following diagram (Fig. 8):

Fig. 8. Vierordt.

Sometimes there is a triple rhythm as in Fig. 9.

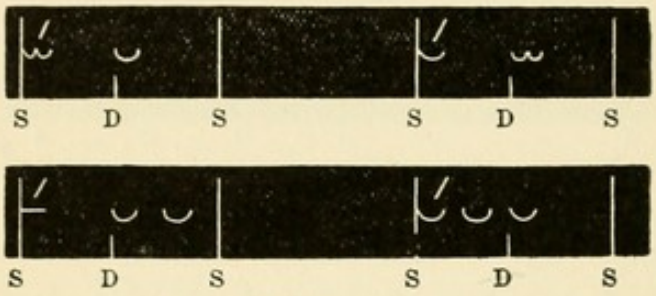

Fig. 9. Vierordt.

I heard a triple rhythm as in Fig. 10

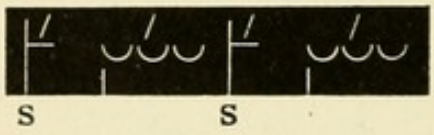

Fig. 10.

with Dr. Dudley in 1901, when the patient was in a very weak state after laparotomy, and when there was a short beat followed by a long one, in the radial pulse.

Or we may have a *gallop rhythm*, as shown by the following diagrams, but this is not always a pathological condieitn.

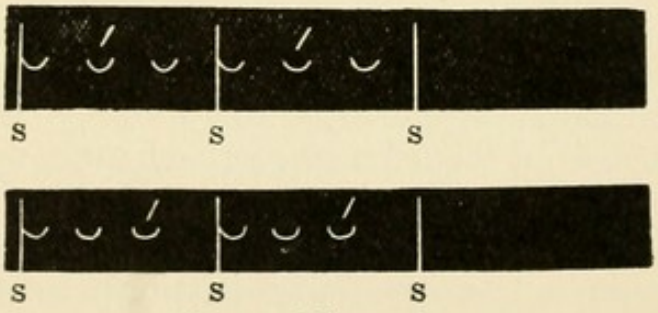

Fig. 11. Vierordt.

Potain claims that some *division of the sounds* occurred in 20% of persons he examined. In 99 of his cases the first sound was divided in 61 instances; the second sound in 30; in 8 both were divided. He thinks division is caused by respiration; that the division of the first sound is associated with the end of expiration and the beginning of inspiration; division of the second with the end of inspiration and the beginning of expiration.

A murmur either supplants a tone (sound) or co-exists with it. Sometimes murmurs replace both tones. Occasionally murmurs are so loud that they can be heard at a distance from the patient. Others can only be heard with the greatest difficulty. In fact, very slight murmurs may only be elicited by movements of the body; or if the patient runs around the room.

Patients should, if practicable, be examined both in the recumbent and upright positions. Sometimes, murmurs cannot be heard in the upright position, and only if the patient lies on the right or left side. Mitral lesions are often best heard when the patient is recumbent.

Murmurs are divided into:

1. *Valvular* and
2. *Accidental.*

Under the latter are included those that are disconnected with valves, papillary muscles, and chordæ tendinæ. They are due to altered conditions of nutrition in the heart muscles, or alterations in the quality or constitutents of the blood, and are found exclusively during systole, and at any single valve; often at all valves together; usually with slighter murmurs in the vessels of the neck, of a light blowing character. But most important, from a diagnostic point of view, is that all the consequences of valve defects are absent in these accidental cases. The exciting causes are chiefly anæmia, or some form of blood deficiency, fever, cancer, consumption, or pressure of neighboring organs on the heart. In pernicious anæmia the murmurs are very loud.

Any kind of diminution in heart pressure also causes accidental murmurs. Less often they are caused by transitory disturbances of the functions of the myocardium, or the papillary muscles, causing temporary insufficiency of the valves. Other accidental sounds may be caused by the act of respiration.

Corresponding to the four valves of the heart there are normally four *tones* or *sounds,* and as each valve may leak, we have four additional sounds, making eight in all.

A *murmur* is the name given to an adventitious sound heard in connection with the heart sound, when there is valvular endocarditis or valve distortion. Murmurs vary in *pitch, quality, duration* and *intensity*. They are charactertistic of valvular diseases, but may be absent, and are not always heard (5 per cent.)[5]. Endocardial murmurs must be carefully distinguished from pericardial and respiratory sounds. Respiratory sounds are limited to inspiration, so that in order to eliminate them, the patient should hold his breath. An endocardial murmur is synchronous with systole or diastole, and is deep seated. A pericardial murmur is heard over any part of the heart, but is best heard at a distance from the valves or apex. It is a sound that is very near the ear. Pressure by the stethoscope increases pericardial friction sounds, but does not affect the endocardial.

None of the valve sounds are best heard immediately over the valves, because the sound is conducted in all cases to the surface, along the line of the flowing blood current; but the pulmonary, being nearest the surface, is heard best at points just above or below the valve, and so the tricuspid; while the aortic and mitral, for similar reasons, are best heard at points remote from the valves.

We find, accordingly, that the pulmonary obstructive murmur is best heard at the junction of the 2d left interspace with the sternum,

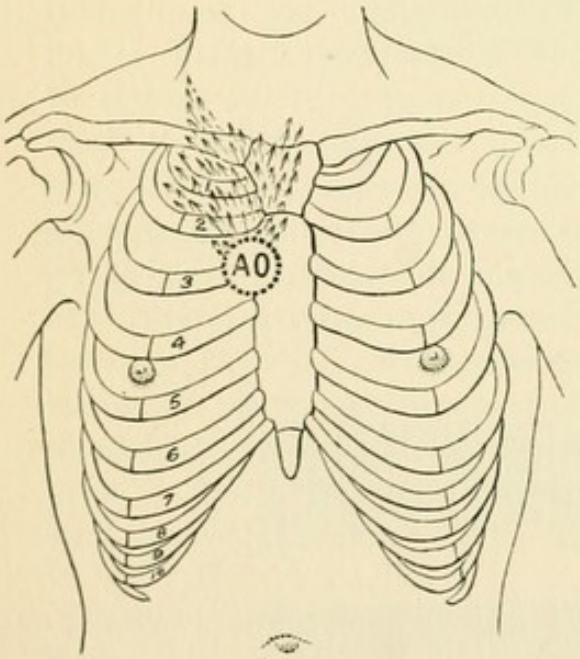

Fig. 12.

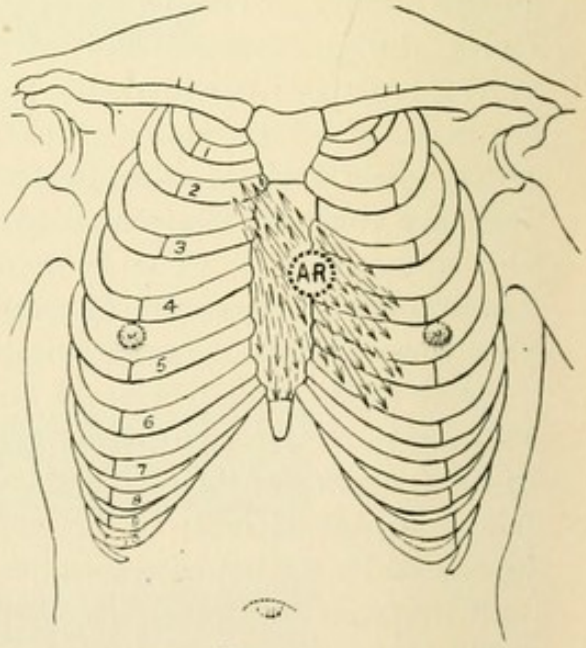

Fig. 13.

[5] According to my figures.

or above its actual position, and is conducted upwards. (Fig. 15.) The pulmonary regurgitant murmur is heard a little below this point and is conducted downwards; but as the pulmonary valve is the most superficial of all, the sounds are always near the ear. (Fig.

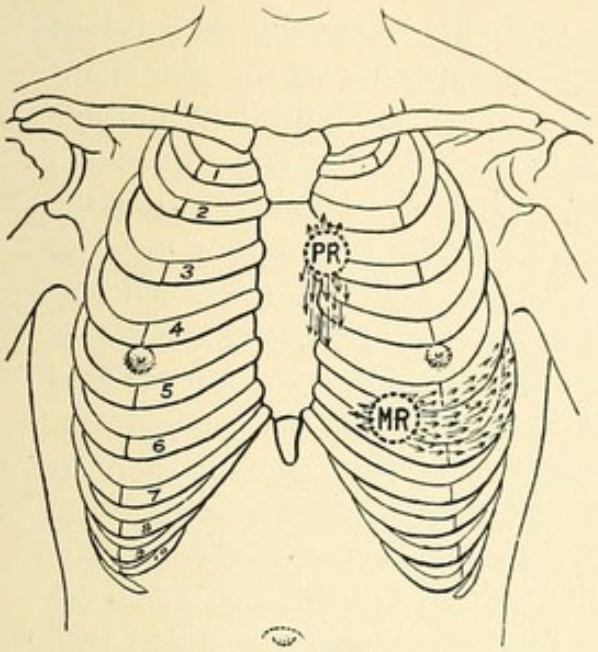

Fig. 14.

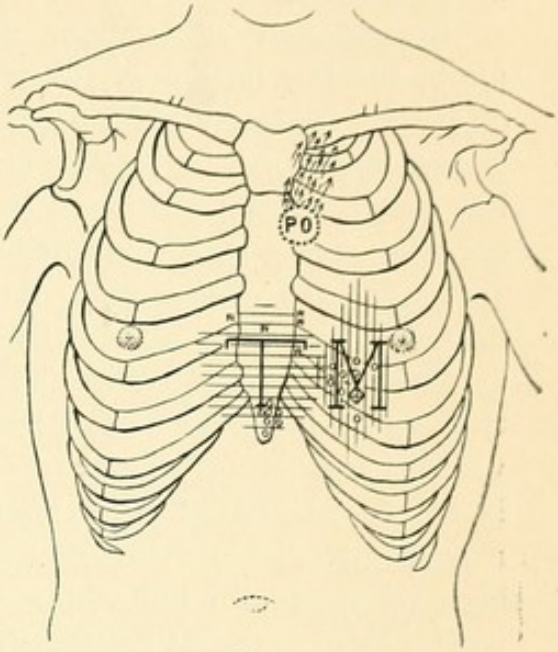

Fig. 15.

14.) The aortic obstructive murmur (Fig. 12) is best heard over a somewhat larger area than the pulmonary and at the junction of the 3d right cartilage with the sternum, and the sounds are conducted upwards chiefly. The aortic regurgitant murmur is also best heard somewhat below the normal position of the valve and at the junction of the 4th left costal-cartilage with the sternum. This murmur is conducted chiefly towards the ensiform cartilage, nipple and apex, but to a slight extent also in the reverse direction (Fig. 13). The mitral obstructive murmur is heard best at a still more remote distance, viz., over a limited area above the apex, usually about midway between the ensiform appendix and the nipple. The area of mitral stenosis, however, is not always well defined. Occasionally, but still rarely, the murmur of mitral obstruction may be heard over any part of the shaded area marked M (Fig. 15).

The mitral regurgitant murmur (Fig. 14) is heard at the point farthest from the valve of all, viz., over the apex. But the murmur is conveyed to the left, not uncommonly to the axilla, and sometimes to the scapula, a matter not hard to understand by aid of Fig. 4; for not only in dilated hearts may the bor-

der of the heart reach to the scapula, but the blood current, from the position of the mitral in the back of the thoracic cavity, and the direction of its current must necessarily cause the murmur to be carried to the back. The *mitral area* is indicated by the large letter M (Fig. 15).

In tricuspid obstruction the murmur is usually heard best along the left border of the ensiform appendix at the points indicated by the small letters *o o* (Fig. 15). The regurgitant murmur is best heard over the sternum, mostly at the junction of the 5th left costal cartilage with the sternum, but occasionally also on the opposite side. It is indicated by the small letters R R R (Fig. 15). The *tricuspid area,* however, is a large one, and indefinite. It is indicated by the shaded area over which is the large letter T (Fig. 15). But it must be remembered that while the base of the heart is tolerably fixed by the great vessels and structures that compose its root, the heart itself is apt to be dilated and hypertrophied both in endocardial and myocardial diseases, so that the relation of the sounds to the bony landmarks of the thorax will vary correspondingly. Accordingly, the meaning of these sounds must be read in connection with other objective and subjective phenomena, in order to be properly appreciated.

No examination of the heart should be made without at the same time an examination of the *lungs, liver* and *spleen.* Patients are often seen by the consultant where tuberculosis of the lungs is the main disease, and the cardiac a secondary affair; most of the symptoms of the latter depending on the lung disease. On the other hand, spitting of blood, which is common in some forms of cardiac disease, may be wrongly attributed to a non-cardiac lung disease.

The liver should be examined for *size, position* and *tenderness.* In cardiac diseases it is apt in the later stages to extend below the free borders of the ribs, and may even reach the umbilicus. It may be tender to the touch. Sometimes, especially in very advanced cases, a *hob-nail* surface can be felt. In one of my cases of enlarged liver the left lobe pushed up the heart so that the apex was above the nipple.

The spleen is also apt to be enlarged when the liver is. It is often the seat of embolism, and as in case No. V, may have suppurative infarcts.

It is needless to say that the *urine* should always be examined.

Attention to the *digestion* is also imperative in the management of cardiac diseases, especially in persons of middle life and beyond. Indigestion alone is not an infrequent cause of death.

In anæmia there is the *venous hum*, or *"bruit de diable."* This is a *continuous murmur* heard over the internal jugular veins at the root of the neck, but it is not pathognomomic of anæmia, as in 50% of the cases where it has been observed there has been none. It is often of a musical quality, and usually more distinct on the right side. The intensity of the murmur is increased by the upright position; by turning the head away from the side which is being auscultated; and by a deep inspiration. It is also modified by the pressure of the stethoscope. The cause appears to be some alteration in the calibre of the vein,[6] due to compression or adhesions attaching it to surrounding parts; or it may be due to a diminished flow of blood. It has been claimed in some of these cases that the vein is "pouched," which would account for the murmurs. The venous hum is not heard in all cases of anæmia, and the intensity of the murmur is not proportionate to the alteration in the quantity or quality of the blood. In anæmia a bruit is also heard over other veins. A continuous murmur, described as being like the wind "blowing through the rigging of a ship under bare poles," is also sometimes heard on either side of the xiphoid cartilage, and it is thought to indicate constriction of the inferior vena cava. In anæmia there is sometimes a systolic murmur in the pulmonic area. It is most distinctly heard in the 2nd left intercostal space, close to the sternum, and is the most constant of the murmurs associated with anæmia. This is said to be due to dilatation of the right ventricle, owing to malnutrition, without change in the pulmonic orifice, so that it is relatively narrowed, and therefore capable of producing a murmur, as in organic stenosis. The same theory holds good if the heart is diminished in size, as it often is in anæmia. For in such case the orifice would be relatively dilated and equally capable of producing a murmur. There are other theories, such, for example, as that the murmur is due to pressure on the pulmonary artery by a distended left auricle. It has also been claimed that the murmur is due to dilatation of the conus arteriosus and pulmonary artery, and that this condition is to be found at the post-mortem examination of patients who have had this functional murmur during life. This view seems at the present time to be

[6] Colbert, *Dis. of the Heart*, London, 1901, p. 72.

reasonable, but it requires substantiation. The *mitral systolic murmur* heard in some cases of anæmia, at the apex, and carried to the left, even to the angle of the scapula, is due to regurgitation from relaxation of the muscular fibres about the mitral orifice, so that the valve leaflets do not come into proper apposition. This is either due to malnutrition of the myocardium, to the musculi papillares, or to simple dilatation of the left ventricle.

The *tricuspid systolic* murmur in anæmia is heard in the tricuspid area. It is due to dilatation of the right ventricle. If the anæmia is slight, the murmur may be absent.

The *aortic systolic murmur* in anæmia is heard in the aortic area, and is thought to be due to relative narowing of the aortic orifice from dilatation of the left ventricle. In these so-called *hæmic murmurs* the position and direction of the murmurs correspond with those of the organic variety; but they are usually soft, and are not conducted so far as in the latter variety.

Exocardial sounds may be mistaken for cardiac murmurs. Of these there are several varieties. In health there is no sound produced by the heart pressing against the pericardium, but when either surface is roughened by disease, a friction sound is produced. It is usually best heard over the right ventricle and the base of the heart. The sound is limited to this particular area, and is like that of two bodies rubbing against one another. It is described as "grating, creaking," etc., and is intensified by pressure with the stethoscope, deep inspiration, or changes in posture. It also changes its position from time to time. The sounds correspond to the contraction and relaxation of the ventricles.

A *pleural friction sound* may also be produced by the movements of the heart, but it ceases when the breath is held.

Chapter II.

ENDO-CARDIOPATHIES.[1]

Endocardial diseases are usually, though not always, localized about the valves, and may or may not be inflammatory. Consequently, the word endocarditis,[2] sometimes used as synonomous with endocardial diseases, is not sufficiently comprehensive. A better term is endocardiopathies, which adequately includes all endocardial affections.

Though endo-cardiopathies were alluded to as early as 1684 by Thomas Willis in his "Practice of Physick," and attracted the attention of Merkel, Senac and John Hunter in the century following, physicians gave them little attention, until exploited by Corvisart in 1808. During the remainder of the century, however, they were studied from almost every available point of view by such men as Kreysig, Andral, Corrigan, Bouillaud, Virchow, Walshe and Stokes, American physicians contributing in later years valuable material.

Affections of the endocardium constitute about one-half of the total of cardiac diseases, and as the inflammatory differ materially from the non-inflammatory in etiology, prognosis, and treatment so we may classify them on this basis; or, on the other hand, may divide them into the *primary* (*i. e.*, inflammatory), under which fall the vegetative, infiltrative, ulcerative, stenotic or sclerotic varieties, as distinguished from the *secondary* (*i. e.*, non-inflammatory), where the changes are secondary to the former, and are such as are caused by the mechanical stretching of the muscular or fibrous tissues.

Some have adopted the plan of classifying them on the basis of their alleged causes. Thus Litten (*Phila. Med. Jour.*, May 5th, 1900) has distinguished a rheumatic, scarlatinal, typhoid and pneumonic endocarditis, etc., but lately we have discovered that endocarditis follows, and appears to be caused by, a number of diseases of less moment, such as coryza, diseases of the skin, and gastro-intestinal disorders. A more simple method is to separate endocardiopathies into the *acute*, *sub-acute* and *chronic*.

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[1] Originally published in the *Virginia Med. Semi-Monthly*, April 26, 1901.
[2] Introduced by Bouillaud in 1841.

The coincident relations of the various *micro-organisms* to the endocardial inflammations have been frequently noted, and cultures have produced endocarditis in the rabbit. But the variety of these organisms is confusing. In 1886 Weichselbaum distinguished two varieties, the staphylococcus pyogenes aureus, and the streptococcus. Netter found the diplococcus of Frænkel; others have found the bacterium coli; others the gonococcus of Neisser, etc. But Michaels (*Phil. Med. Jour.*, May 5th, 1900), opened a number of rheumatic joints and found no micro-organisms. Still, notwithstanding this divergence of opinion there is reason to believe that some forms of endocarditis are of microbic origin.

Acute rheumatism is generally taken to be the chief cause of endocarditis. In fully 40 per cent. of my cases there was an antecedent history of rheumatism, and as many as one-third of the cases of acute rheumatism I have found were followed by endocarditis. These are conservative figures in the light of statistics that follow.

And yet, figures do not always have much significance when rheumatism is concerned, because the word, both with the laity and physicians, is loosely applied. However, Latham found that in 136 cases of acute rheumatism the valves were affected in seventy-four, or 54 per cent., Gibson in 184 out of 325 cases, or 56 per cent. (*Gibson's Diseases of the Heart*, 1898, p. 397).

In this connection it is interesting to observe that chorea has a close genetic relation with acute articular rheumatism and that Fagge found few fatal cases of chorea without organic valvular changes similar to those of rheumatic endocarditis.

There is also a manifest relation between the age of a patient and the initial attack of rheumatic endocarditis. For example, when infants or children have an acute attack of rheumatism, they are liable to endocarditis, perhaps in 70 per cent.; and yet acute rheumatism is rare in infancy and young life, though comparatively common in the decenniums between 30 and 50. After this period it rarely develops.

There is no rule as to the date at which endocarditis appears in acute articular rheumatism. It may occur at any time during the attack, or may precede it. After rheumatism, pneumonia follows as one of the most frequent causes of endocarditis. The toxin of the pneumococcus, however, administered to rabbits by Carnot and Fournier (*Arch. de Med. Exp.*, XII, p. 357,

Schmidt's Jahrbuch), was followed by acute inflammation of the heart muscles, intestinal hæmorrhages, degeneration and fragmentation of the voluntary muscles, while the valves were not affected.

Gibson, on the other hand (*Edinb. Med. Jour.*, Nov., 1900), has reported a case of diplococcal infection resulting in pleuro pneumonia. At the autopsy the aortic segments were seen to be ulcerated, and in a thrombus adherent to the aortic valves there were found diplococci, leucocytes and fibrin. Endocarditis is quite often found in tuberculosis, but the cause of the disease has been usually attributed in these cases to a streptococcus, staphylococcus, Frænkel's pneumococcus, or Friedlander's capsule bacillus.

Endocarditis is also frequently associated with surgical diseases, such as osteomyelitis, erysipelas, dysentery, pyæmia and septicæmia, puerperal fevers, and furunculosis; and the staphylococcus pyogenes aureus has been most frequently found associated with the lesions of these affections. In erysipelas, an associated endocarditis has been traced to a streptococcus, and with it endocarditis has been produced experimentally by a number of workers. In gonorrhœal endocarditis Leyden found the gonococcus of Neisser in the valvular deposits. They had the distinct biscuit form, and were colored satisfactorily by Gram's method.

In scarlatina, endocarditis was seen to develop by Trousseau[3] and others. It may occur at any time during the disease or with its sequelæ. But a characteristic micro-organism has not been found, as yet, in this form. Influenza affects the heart in many ways, but chiefly attacks the muscular substance, through the poisonous influence of the toxins; though endocarditis has been attributed to influenza. Endocarditis occurs occasionally in variola, but if the primary disease is severe, the cardiac affection is (as often happens in endocarditis) associated with other affections, such as a myopathy or pericarditis. Endocarditis occurs occasionally in measles, but syphilis rarely attacks the valves, though the myocardium is occasionally involved. While, as already stated, many different sorts of micro-organisms have been found in the ulcerated valves, the etiological relation they hold to the diseased condition is still doubtful. This problem may eventually be settled by determining whether or not the micro-

[3] Trousseau, *Clin. Med.*, vol. 2, 1869, p. 188.

organisms are to be found in the *initial* lesions of the valves.

There is a close relation between endocarditis and some forms of Bright's disease. Inasmuch, however, as, according to my investigations,[4] lithæmia is a causal factor of Bright's disease in from 50 per cent. to 75 per cent., the close relation between rheumatism and endocarditis is plainly shown. But Bright's disease is apt to be a late phenomenon in endocarditis.

Endocarditis is most frequent after ten and before forty, but there is a manifest relation between the age of the patient and the seat of the disease. In the fœtus, the right side of the heart, doing the most work, is most frequently affected; in extra-uterine life it is the left heart, for similar reasons. Accordingly, age and the character of work to be done must be considered in estimating the liability to endocarditis. In extra-uterine life, disease affects the mitral or aortic valves by preference, next the pulmonary and tricuspid valves, but these latter in a comparatively small number of instances. There is little difference in the tendency to endocarditis between men and women, though it is generally held that mitral disease is more common in women and aortic in men.

The beginning of an endocarditis is marked by an invasion of the substance of the valves by toxins, micro-organisms and inflammatory exudates, while on their surfaces the shining endothelium becomes opaque and gives birth to minute rounded flesh colored papillary bodies, which are one or two millimeters in height when first seen, and situated near the free edges of the valves. On the mitral they develop on the auricular surfaces at a distance of 2-3 millimeters from the free edge, while on the aortic they form on the ventricular surfaces. They are at the point of maximum contact. To these excrescences are attached particles of fibrin from the blood, and these, together with ulcerated portions of the valves, may be carried into the general circulation and cause embolism. There are all degrees of infiltration in these valves, with or without ulceration, and the disease may extend and involve the myocardium. But wherever ulceration takes place, there is at the same time a sclerotic change coincident with it, so that destructive and constructive processes go hand in hand, nature attempting to repair as disease destroys.

Pathologically, the changes consist, first, in a thickening of the small vessels, with hyaline degeneration and perhaps partial sclerosis of the smaller arteries, followed by a small-celled infiltra-

[4] *N. Y. Med. Rec.*, March 7, 1889.

tion and proliferation of connective tissue, with eventual destruction of muscle cells. The process may be so extensive that the greater part of a valve is destroyed or even converted into an aneurismal sac. I have seen an example of the latter accident.

In the chronic forms, there is often a deposit of the salts of lime in the valves, or along their attached margins, and the process extends down over and into the chordæ tendinæ, contracting and stiffening them, while the papillary muscles are also apt to undergo fatty and calcareous degeneration. Such stiffened portions of the endocardium occasionally rupture.

Endocarditis develops insidiously, as a rule. It may not be discovered unless looked for. Years often pass before it is recognized. Much depends on the situation. If confined to the walls of the heart it seldom shows any signs. I have seen a few such cases.

It is interesting to know exactly what is found in valvular diseases at autopsies, and the tables of Sperling prepared from the Records of the Berlin Pathological Institute between 1868-'70, are the best I have met with. They may be compared with my own tables of a smaller number of cases arranged on the same plan.

SPERLING'S TABLES.[5]

300 *Cases of Endocarditis.* 1868-70.

268 cases	= 89 per cent.	left side of heart.
3 cases	= 1 per cent.	right side of heart.
29 cases	= 10 per cent.	both sides of heart.
300	100	

Affections of One Valve Only.

200 Cases = 66.7 per cent.

Mitral valve only	157 cases	= 78.5 per cent.
Aortic valve only	40 cases	= 20. per cent.
Tricuspid valve only	3 cases	= 1.5 per cent.
Pulmonary valve only	0 cases	= 0.0 per cent.
	200	100.0 per cent.

Combined Valvular Lesions.

100 Cases = 33.3 per cent.

Mitral and aortic	71 cases	= 71 per cent.
Mitral and tricuspid	9 cases	= 9 per cent.
Mitral and pulmonary	2 cases	= 2 per cent.
Aortic and pulmonary	1 case	= 1 per cent.
Aortic and tricuspid	0 case	= 0 per cent.
Mitral, aortic and tricuspid	16 cases	= 16 per cent.
Mitral, aortic and pulmonary	0 case	= 0 per cent.
Tricuspid, pulmonary and mitral	0 case	= 0 per cent.
Tricuspid, pulmonary and aortic	0 case	= 0 per cent.
All four valves	1 case	= 1 per cent.
	100	100 per cent.

Gibson's Dis. of the Heart and Aorta, 1898, p. 413.

Embolism.

84 Cases = 28 per cent.

76 with left side disease—8 with right side disease.
Kidney, 57.
Spleen, 39.
Brain, 15.
Digestive organs, 5.
Skin, 4.

AUTHOR'S TABLES.

65 *Cases of Endocarditis*, 1872-1888.

56 cases = 86 per cent. left side alone affected.
0 cases = 0 per cent. right side alone affected.
9 cases = 14 per cent. both sides affected.

65 — 100 per cent.

Affections of One Valve Only.

18 Cases = 27 per cent.

Mitral valve only	6 cases	= 33.33	per cent.
Aortic valve only	12 cases	= 66.67	per cent.
(Including diseases of ascending portion of arch)			
Tricuspid valve	0 case	= 0.0	per cent.
Pulmonary valve	0 case	= 0.0	per cent.
	18	100.0	per cent.

Combined Valvular Lesions.

44 Cases = 67 per cent.

Mitral and aortic	31 cases	= 70.5	per cent.
Mitral and tricuspid	2 cases	= 4.5	per cent.
Mitral and pulmonary	0 case	= 0.0	per cent.
Aortic and pulmonary	0 case	= 0.0	per cent.
Aortic and tricuspid	2 cases	= 4.5	per cent.
Mitral, aortic and tricuspid	4 cases	= 9.4	per cent.
Mitral, aortic and pulmonary	1 case	= 2.2	per cent.
Tricuspid, pulmonary and mitral	1 case	= 2.2	per cent.
Tricuspid, pulmonary and aortic	0 case	= 0.0	per cent.
All four valves	1 case	= 2.2	per-cent.
Pulmonary and tricuspid	2 cases	= 4.5	per cent.
	44	100	per cent.

Embolism.

11 Cases = 16 per cent.

All occurred in connection with left side disease and as follows:

Kidney, 6 times.

Spleen, 4 times.

Brain, once.

Liver, twice.

It will be noticed that there is a general agreement between the two tables, except as to the comparative frequency of aortic and mitral diseases. Probably in my tables a larger number of cases would have altered this relation.

If, however, in my 65 cases we throw out the affections of the first part of the aorta, the incidence upon the valves stands as follows:

.. Aortic insufficiency	49	times
2. Aortic obstruction	39	"
3. Mitral insufficiency	38	"
4. Mitral obstructions	33	"
5. Tricuspid insufficiency	8	"
6. Tricuspid obstruction	4	"
7. Pulmonary insufficiency	4	"
	175	

But as single valve lesions were rare, the total foots up 175, an average of from two to three valve lesions in each case. The following is the order of frequency, as recorded in my office cases:

Aortic disease	56 per cent.
Mitral disease	35 per cent.
Tricuspid disease	6 per cent.
Pulmonary disease	3 per cent.
	100 per cent.

On the other hand, in 50 cases from my clinic, as taken by myself and assistants (not verified by post-mortems), the incidence was put down as follows:

1. Mitral insufficiency.
2. Aortic insufficiency.
3. Aortic obstruction.
4. Mitral obstruction.
5. Tricuspid insufficiency.
6. Pulmonary insufficiency.

This is not very unlike the order of Walshe (*Diseases of the Heart*, London, 1873, p. 105), which is—

1. Mitral insufficiency.
2. Aortic stenosis.
3. Aortic insufficiency.
4. Mitral stenosis.
5. Tricuspid regurgitation.
6. Pulmonary incompetency.
7. Tricuspid stenosis.

Dr. George S. Middleton,[7] of Glasgow, puts the order of frequency from his dispensary cases (unsupported by post-mortems) as—

1. Mitral insufficiency.
2. Mitral stenosis.
3. Aortic incompetency.
4. Aortic stenosis.
5. Tricuspid disease
6. Pulmonary disease.

[7] *Lancet*, Oct. 26, 1889.

And yet I should prefer not to take any purely clinical evidence as a basis of statistics, for the following reasons:

In my 65 cases with clinical histories and post-mortems, while endocardial disease was recognized by those who had charge of the patients in 95 per cent., 37 cases of aortic disease were only noted in 23, or 62 per cent.; while in 31 cases of mitral disease, it was detected in only 19, or 61 per cent. In other words, there was a positive failure to locate in 39 per cent. of actual lesions. This revelation of the results of actual experience in hospitals, where the physicians were among the best we have had, shows how futile it is to base conclusions on clinical evidence only. And yet up to this time it has been the main stay of clinicians. On the other hand, it is expecting too much to require a physician to differentiate every valvular lesion at the bedside, or in the consulting room. As the best clinicians often fail to recognize them now, so they will continue to do for all time. The reasons are threefold. In many instances they give no sign, or if they do, attendant circumstances prevent them from being appreciated. I have even heard a distinguished diagnostician say that a diagnosis of a specific valvular disease made at a first examination had little value. The truth is that in well-established forms of organic valvular disease a specific diagnosis can usually be made correctly at a single examination; while in less pronounced cases several examinations may be necessary.

As Stokes said, in 1855, "The difficulties of special diagnosis are still infinitely greater than many might be led to expect." But of course we shall gradually overcome some of these difficulties, as we frame better rules for diagnosis.

On the other hand, the diagnosis of endocardial disease, on the post-mortem table, is comparatively easy, and rarely liable to misinterpretation, though clinicians do not all take this view. The chief difficulty lies in determining whether or not valves are sufficient. However, the ordinary water test is, I think, satisfactory, if applied by an experienced pathologist; and the latter can also determine whether the valve affected has been the seat of inflammation, or has been dilated or distorted by muscular action, etc.; in other words, whether the endocardial disease is primary or secondary.

The symptoms of an acute benign endocarditis are variable and inconstant, and may escape detection. On the other hand, it may be announced by unmistakable signs. A patient is seized

with intense præcordial pain, dyspnœa, arrhythmia or rapid pulse, perhaps with some fever or even cyanosis, and the ear applied to the chest detects a rough, loud or harsh murmur. Occasionally, the suspicion that the patient is having an acute exacerbation of the chronic disease leads us to apply the ear. More rarely a sudden strain ruptures a valve that has been previously softened by infiltration, or made brittle by atheroma or senile changes. Such an event is usually announced by a *musical murmur*. An acute attack will be more readily detected by keeping in mind the various affections that appear to cause endocarditis. In the acute septic form there are irregular chills and sweats, with fever. Other signs have already been noted as belonging to the benign form, to which should be added pretty uniform tenderness and enlargement of the spleen, with sometimes similar conditions of the liver and kidneys. The urine should be dark colored; *i. e.*, bloody, if a kidney develops an infarct.

According to my hospital tables, as I have said, endocarditis has recognizable murmurs in 95 per cent. of the cases, and the three most prominent signs, following the auscultatory, are dyspnœa in about 50 per cent., palpitation in about 25 per cent., and præcordial pain in about 10 per cent. Other less constant symptoms are cough, weak or irregular action of the heart, dizziness, epigastric pulsation, orthopnœa, cyanosis, delirium and œdema. In only 5 per cent. there were no characteristic signs during life. But as I have said, it is one thing to be able to distinguish endocarditis, or in fact any endocardiopathy, inflammatory or not, and quite another to locate the precise lesion accurately.

Of the endocardial *murmurs* there are two kinds. First, the *organic;* second, the *functional*. The former are heard when there is a mechanical hindrance to the flow of the blood from ulceration, sclerosis or rupture of the valve. The functional murmurs are caused by *relative—i.e.*, muscular—insufficiency, which occurs when the orifice is dilated so that the valve margins do not come together accurately; or, when from degeneration or weakness of the papillary muscles, the valves are not held in place; also, when as in anæmia, especially in convalescence from long continued illness, there is an alteration in the composition or amount of the blood. An irregular pulse with præcordial pain and dyspnœa, or even a systolic murmur at the apex, does not necessarily indicate that the murmur is due to organic disease; but a systolic

murmur at the base is likely to be functional, if it is limited to the left side of the sternum, and there is no thrill.

Organic murmurs during the development of an endocarditis are usually harsh and loud. The French talk about the sawing murmurs (*bruit du scie*), the rasping murmur (*bruit du rape*), the musical murmur (*bruit d'oboe*), the bellows murmur (*bruit du souffle*). These are usually organic; functional murmurs are low, soft and *always* systolic. But a single examination may not suffice to distinguish between the two. The organic murmur will be more apt to continue; while the functional will disappear under tonic treatment or rest. The point of greatest intensity of a murmur is somewhere in the course of the blood current beyond the obstruction, and is usually due to the breaking up of the current. Just as in the stream of water flowing through a narrow orifice, it is not at the point of greatest obstruction where the noise is loudest, but where the water expands beyond the obstruction, and is broken up into diverse currents. When a blood current passing over a rough surface, or through a narrow passage, can be felt, the sensation is called a *"thrill,"* sometimes a *"purring thrill."* (*fremissement cataire*), because it is like the thrill felt by the hand pressing on a purring cat.

In endocarditis we do not always need to be alarmed if the pulse is frequent or infrequent. Neither condition should be treated as a disease. A pulse of 50 or 60 may be characteristic of the man, and so a pulse of 100. It is not at all a rare thing to find a patient with an average pulse of 60; but it is uncommon to find a pulse of 100 or more. We should first inquire if these abnormal rates of the pulse are not individual or family characteristics. I have known the most serious mistakes to be made in such cases. A man with a pulse of 50 to 60, or even 120, may not realize that there is anything peculiar about the action of his heart, and may be quite as able to do his daily work as the next man. And yet physicians are quite apt to treat these conditions, by trying to bring the rate to the recognized average of seventy-two. In such cases, drugs should be the last remedies resorted to. The frequent pulse sometimes follows surgical operations, or injuries to the thorax or neck.

The rhythm is usually affected in endo-cardiopathies, both in the acute and chronic forms, and always in broken compensation. If the pulse is large, it generally indicates cardiac hypertrophy; the hard pulse, rolling under the fingers, means arterio-sclerosis,

the feeble pulse is found in the fatty heart, the soft pulse in anæmia and in fevers. Pulsation of the jugulars suggests tricuspid regurgitation; the capillary pulse aortic regurgitation. The pulse may be unequal—that is, more easily felt in one radial than in the other—but this peculiarity may be congenital or due to arteriosclerosis, or other causes.

The *sphygmograph* is a pretty instrument, but it is less used than formerly, because it is apt to mislead. It is of value in clinical experimentation, but its uses at the bedside are few. Clinicians are using it less and less in this country. It should not be relied upon for differential diagnoses in valvular diseases.

In endocarditis there are, not infrequently, attacks of tumultuous action, with distressing palpitation, the impact extending over a considerable area. In the intervals between these attacks, the action of the heart may be quite regular, the apex beat inappreciable to the finger, and a "thrill," which was distinctly felt, may disappear.

Auscultation yields the most important information. Supposing a valve, say the mitral, is obstructed to a considerable extent, so that it cannot close perfectly, the blood will necessarily leak back into the left auricle, during the contraction of the left ventricle. In such case the first sound, which is due chiefly to the closure of the mitral valve and to muscular action, is replaced by a murmur caused by the leaking or regurgitant blood passing through the obstructed opening, and this sound is best heard between the apex and the axilla or spine of the scapula, where it is conveyed, in accordance with the rules governing the conduction of sound. If the new deposits in the valve are soft and smooth, the murmur is soft; if very rough or irregular, it is loud or harsh. This is provided the heart's action is strong; if it is weak, there may be no appreciable murmur. Sometimes a harsh murmur suddenly disappears, while the action of the heart continues the same. Some portion of the obstruction has then been swept away. There are not the hard and fast areas in which to hear the several murmurs, as laid down in some books, and there is quite a little difference of opinion as to the locality of these areas among teachers of physical diagnosis. The truth is that the point of maximum intensity for determining mitral regurgitation is between the apex and the axilla or scapula; but in mitral stenosis the point of maximum intensity extends from the apex upwards and downwards perhaps as much as an inch or more, and a less distance

to the right. In aortic obstruction the obstructive murmur is heard best over the right 3d costo-sternal junction, or at the junction of the second right interspace with the sternum, or even as far over as the corresponding space on the left side; while the aortic regurgitant may be well heard along a broad area spreading like a fan from the aortic area to the apex, or even to the ensiform appendix. As the point of maximum intensity for the tricuspid is located at the junction of the left fifth interspace with the sternum, it is not far from the mitral area, and its murmurs may be conveyed to that area; hence it may be difficult to make a diagnosis between these two lesions. Pulmonary lesions are so rare that they are curiosities; most of them are due to congenital malformations of the heart. In general, the murmurs indicative of the greatest danger are the diastolic.

Percussion is at first negative, but, as endocarditis progresses, the contour of the heart gets larger and more ovoid. This enlargement is the most important sign of organic heart disease, because it is unequivocal.

The heart swings like a pendulum in the cavity of the chest, suspended by its great vessels; so that the apex is carried well outside the nipple in some cases, especially in lateral curvature, where the spinal concavity is generally to the left. It may also be displaced to the right by fluid in the chest, and by lying on the right side. Still, as we only examine in the upright or recumbent positions, it is relatively fixed, and we find the apex in the fifth space, the left border of the heart the breadth of a rib inside the nipple, and about twice that distance below it. The right auricle is about the only part of the heart outside the right border of the sternum. Two-fifths of the heart lies to the left of the median line.

Angina pectoris is not uncommon in endo-cardiopathies. Both forms, which are best classified as the *mild* and the *severe*, are usually brought on by mental or moral excitement, indigestion, over-exertion, and a number of minor causes, especially those that influence the special senses. They are always, in my experience, capable of being controlled by suitable remedies, though drugs may prove ineffectual when rest, massage, electricity, baths or a change of scene will succeed.

Endocarditis gives rise to various symptoms in other organs, for there may be hyperæmia of the lungs, embarrassed respiration, engorgement of the kidney and chylopoëtic tract, and even general dropsy.

A distinct picture is produced in these cases by *embolism,* where particles detached from the diseased endocardium, or clots formed about the valves, in the auricular appendages, or about the papillary muscles, are carried to distant organs. These accidents may cause few symptoms, and yet may involve the brain, causing alarming results, and even *sudden death.* But if the collateral circulation is rapidly established, little or no functional disturbance will be produced. As terminal arteries, however, are found in the brain, lungs, spleen, kidneys and heart, the occlusion of large vessels in these organs is apt to be followed by severe symptoms, such as chills, vomiting, pain and hæmorrhage. Benign emboli may cause only arrest of function, but the malignant or septic will certainly produce abscesses that in turn will furnish foci for others. *Embolism of the brain* occurs most frequently along the line of the branches of the left carotid, the trunk of which lies directly in the course of the circulation. The embolic masses find their way through this carotid to a branch of the Sylvian artery, and if there is occlusion of a large branch, loss of consciousness, hemiplegia and aphasia usually follow.

In young or middle life, embolism is the rule; in advanced life, *apoplexy. Embolism of the lungs* has characteristic features. If a vessel of any considerable size gets plugged there is apt to be pain, vomiting, cough, dyspnœa, hæmorrhage, and expectoration of frothy mucus; perhaps cyanosis, suffocation and syncope. *Embolism of the liver* may be ushered in with chills, pain, swelling, tenderness and icterus. *Embolism of the spleen* also shows itself with a chill, fever, and severe pain in the organ, which should be enlarged and tender to the touch. *Embolism of the kidneys* similarly may be ushered in by chills, fever, pain and albuminous, perhaps bloody urine. *Embolism of the mesenteric arteries* is revealed by colicky pain in the abdomen, diarrhœa and discharges of black blood. *Embolism of the retina* is sometimes seen with the ophthalmoscope. If septic, it causes inflammation and destruction of the globe. *Embolism of the skin* may cause purpura or gangrene.

In fact, embolism arrests the function of the part where the infarct is lodged, and if septic, produces destruction of tissue.

So long as compensation is imperfect, there are also other complications. For whenever the heart begins to labor, congestion of the veins and capillaries of the lungs immediately results, and then the bronchial mucous membrane, alveoli and passages become swollen and œdematous; with eventual desquamation of epithelium, and transudation of mucus, serum and blood. Embarrassment of

respiration ensues, and it is heightened by the increased efforts of the lungs to aerate the abnormal quantity of blood in the pulmonary vessels. Such a condition may be only temporary, constituting pulmonary œdema; but if it become chronic, the character of the lung tissue is changed, for the continued venous engorgement is followed by deposits of pigment matter. This leads to what is known as *brown induration of the lungs,* and even rupture of pulmonary vessels; indeed, *pulmonary hæmorrhage* is not uncommon in chronic heart diseases.

The liver also becomes enlarged from a similar cause, and *pigmented.* In fact, there is a congestion of the entire chylopoëtic system, which continues so long as the heart is embarrassed. As soon as there is congestion of the venous system, it is shown by a bluish color of the skin or visible mucous membranes.

Thrombosis may also occur, and cases have been described by Welch, A. A. Smith, and MacGregor. (See *Amer. Medicine,* May 25, 1901, and Brannan, *Med. Rec.,* Feb. 22, 1902.)

For a similar reason the kidneys become swollen, and later tough and firm. The urine is diminished, but the specific gravity is increased; it may contain blood, and usually a little albumin; sometimes a little sugar, varying from ½ to 2½ per cent. Oedema, due to prolonged distention of the veins of the peripheral system, hepatic or renal implication, deserves attentive consideration. But all cases of œdema about the ankles, hands or face need not alarm us. They may occur from lack of exercise or anæmia, or temporary compensatory failure, and will disappear under appropriate treatment. Accumulations of fluid, however, in the abdominal cavity, or œdema ascending gradually from the ankles to the trunk, are very serious matters, pointing to a fatal issue at an early date.

If the valvular disease is at all serious, dilatation and hypertrophy of the right or left ventricle will supervene. Hypertrophy is essentially a *compensatory* change, enabling the heart to do the work required of it, notwithstanding the valvular disease, and to re-establish the proper balance between the arterial and venous systems.

In aortic disease compensation produces the *long heart,* due to dilatation and hypertrophy of the left ventricle. On the other hand, in pulmonary stenosis, which is rare, of course, the right ventricle dilates and hypertrophies. As soon as the former cardiac balance has been restored by the means just described, we say that compensation has been established, for then the consequences of the valvular disease have been overcome, for the time at least. While,

however, *compensation* is being established the patient is short-winded, cannot walk any distance, has præcordial pain owing to the dilatation, perhaps fainting fits, and feels physically exhausted at the end of the day. But after compensation has been established all these symptoms disappear, and he feels as well as most persons, except if called on for some extra exertion, or upset by some emotional disturbance. Enlargement of the liver helps to detect lack of compensation. It indicates dilatation of the right ventricle and right auricle as well. If there is obstruction in the pulmonary circulation, the right auricle is pretty certain to be dilated.

But it is a mistake to think that compensation calls only for dilatation and hypertrophy of particular chambers of the heart, corresponding to certain valves. If the valvular lesions are at all serious, both ventricles and both auricles are eventually more or less dilated and hypertrophied, owing to the close relation they hold to each other.

Chapter III.

ACUTE ENDOCARDITIS: BENIGN AND MALIGNANT.[1]

For convenience sake acute endocarditis may be said to have two prominent types, the benign or non-suppurative, and the malignant or suppurative. The latter variety has also been known as the mycotic, infective, etc., but as both contain micro-organisms in the endocardial deposits, and are associated with systemic infections, these terms are inappropriate. Nor has the word ulcerative, as applied to endocarditis, any distinct value, because all forms tend to produce ulceration.

The acute benign form is by far the most frequent. In an analysis of forty-eight cases of endocarditis, verified by post-mortems, some years ago[2] I found that it was rarely produced by an injury, but more often resulted from systemic poisoning, such as rheumatism, scarlatina, measles, etc., though in about half my cases I did not discover the cause. In the light of our present knowledge, however, we may attribute it also to a large number of minor ailments, including gastro-intestinal affections, tonsillitis, etc., which are now known to immediately precede the attack. I saw such an instance in 1901 at the New York Orthopedic Hospital, with Dr. Hibbs, in an otherwise healthy boy of seven, who entered the hospital to be operated on for congenital dislocation of the hip. After the operation, from which there was at first no unfavorable reaction, he developed an obstinate diarrhœa, in the course of which there was a sharp attack of endocarditis. In this case the gastro-intestinal tract appears to have been the source of the infection. I am inclined to think that various systemic poisons, many of them still unknown to us, produce the disease. But it is an incident of systemic affections, rather than a separate entity. In most cases endocarditis selects those portions of the endocardium which are at or about the valves. The first gross evidences of disease consist in the formation of minute reddish excrescences near the free valve margins. In the mitral leaflets they are on the auricular surfaces, in the aortic leaflets on the ventricular surfaces.

Soon after these soft excrescences appear they are covered

[1] Published originally in the *Medical Times*, May, 1901.
[2] *N. Y. Medical Record*, Feb. 27, 1886.

with layers of fibrin, hence the term *diphtheritic*. Two processes are now possible, either a healing, with thickening and puckering of the leaflets, or a degeneration that leaves broken surfaces or ulcers. And yet, when this degenerative process goes on there is about it a regenerative process by which Nature attempts to heal the part and restore its function. At all times micro-organisms, in greater or less number, are found in the fibrin or granular débris of the ulcerating surfaces. The degenerating matter, or the fibrin attached to it, may produce infarcts in various parts of the body.

In extra-uterine life the left side of the heart is most frequently attacked; in congenital disease the right.

Patients seldom suffer much from the simpler forms of endocarditis in their early stages. They often complain of some præcordial pain, though symptoms such as this may be due to an associated pericarditis, the irregular action of a hypertrophied heart, or pulmonary complications. In a large number of cases there is a noteworthy frequency of the pulse (a rate of 120 to 140 is not uncommon at first). Dyspnœa, orthopnœa and palpitation also are of pretty regular occurrence. But it is the accidents and complications that are most dangerous to life. In a disease which in nine out of ten cases lasts for years (sometimes twenty and thirty, and even more) it is natural that these accidents should at times occur; and we find them in the shape of infarcts in the kidneys, lungs, spleen or brain, etc., in one-third of the patients.

The danger therefore increases as the disease becomes chronic. For in the great majority of cases patients survive the acute stage, though embolism may occur, even at that time, as we have seen.

Often the acute or benign form eludes observation. However, we should be on the lookout for it in acute rheumatism, especially when the temperature rises to 100° or 102° F.; and also in pneumonia and scarlet fever. It is less frequent in typhoid, erysipelas, bronchitis and gastro-intestinal affections.

The diagnosis is beset with some difficulties, or otherwise it would be recognized more frequently. The signs include palpitation, dyspnœa, præcordial pain, insomnia, often rapid and irregular pulse, headache, and anxiety. In the first attack there may be no enlargement of the heart, and therefore no dislocation of the apex. In adults we have to deal most often with acute exacerbations of a chronic malady, or the recrudescence of a latent endocarditis. In the majority of cases the disease is either in the mitral or aortic valve. One of the main difficulties encountered, however, in making a

diagnosis, is that the acute attack is apt to be engrafted on a chronic, or at least a latent, condition; or, the lesion may be masked by a pericarditis.

In acute endocarditis the first requisite is rest in bed. As far as possible we should avoid drugging; and stimulation by alcoholics may also be harmful. But the diet should be carefully regulated. Starchy and saccharine substances should be prohibited. For children, milk is the best food, and it is also good for adults, if it agrees with them. It may be diluted with one-third to one-half lime-water in children; with Seltzer and Vichy in adults. The patient should also be kept in a quiet room and free from all annoyances. As soon as practicable, efforts should be made to combat the systemic disorder. If it is rheumatism, the salicylates should be given with caution, in 5 to 10 grain doses, every two to four hours, with an alcoholic or diffusible stimulant; but not in sufficient quantity to produce vertigo, ringing in the ears, or constitutional symptoms. A little palpitation may be let alone. If, however, the heart acts tumultuously, a cold compress may be put to the præcordial region, and bromides, such as the bromide of sodium or monobromate of camphor, given. The latter is an excellent remedy, in 2 grain doses. Two or three grains of Dover's powder every two hours is also excellent. Aconite, in 1 minim doses every two hours, is soothing if there is fever. Phenacetine may also be given to adults, in 3 to 5 grain doses, with a stimulant at bedtime, if the effects of the drug can be watched. Digitalis should not be given, or, if at all, with great caution. It is a dangerous remedy in these cases. In intense arrhythmia strophanthus may, however, be tried cautiously, because it is soothing; but it is apt to be unreliable and, like digitalis, it should be given in emergencies only, and for a very short time. The milder remedies should be tried first.

The following two cases illustrate the benign type.

Case I. Acute Benign Aortic Endocarditis Engrafted on the Chronic Form.—E. B., twenty-two, colored, was admitted to hospital December 11, 1880. Three weeks previously he had been taken with præcordial distress, palpitation and dyspnœa, slight cough with white sputum, orthopnœa; at times nausea and vomiting.

On physical examination the heart was found hypertrophied, with apex to left of nipple. Wavy movement of the epigastrium. Expiration prolonged, and high-pitched behind. Murmur at apex with first sound. Pulsation of jugulars. The murmur,

supposed to be mitral regurgitant, was carried to the left, but was better heard above the scapula than below it. The patient died of pneumonia. At the autopsy an abnormal amount of fluid was found in the pericardium, and some in the pleural cavities. Both lungs were solidified at their bases, and pigmented. Nutmeg liver. Kidneys and spleen congested. The heart was hypertrophied. Pulmonary opening dilated. Right heart dilated. Mitral valve normal, but aortic valve the seat of fresh vegetations, and posterior cusps thickened and inflamed.

This case was an acute benign aortic endocarditis with hypertrophied heart. The murmur at the apex, supposed to occur with the first sound, heard best behind and above the scapula, proved to be one of those instances where the murmur of aortic regurgitation is conveyed to the apex from the aortic. And it opens the question whether the regurgitant murmur was conveyed to the apex by the sternum, the solidified lung or the heart walls. It also illustrates the importance in these doubtful cases of noting that in the aortic regurgitant the murmur *may* be conveyed above the scapula; and further, that the second sound may be mistaken for the first. The pulmonary, though dilated, appears to have been sufficient.

Case II. Acute Benign Mitral Obstruction; Pericarditis.—C. W., twenty-one, single, was admitted to hospital January 25, 1884. He believed himself in good health up to five weeks before admission, when he had an attack of acute articular rheumatism.

On physical examination a systolic murmur was heard at the apex. Liver enlarged; albumin and casts in the urine; œdema of legs. These symptoms improved, but at the end of four weeks there was an increased œdema, which reached to his scrotum. About this time a "purring thrill" was noticed at the apex and a double murmur at both apex and base. The patient died a few days later, in coma. At the autopsy the pericardial sac was found to contain a large amount of serous fluid, with fibrin in flakes. Both visceral and parietal layers of the pericardium were covered with a fibroplastic material. On the mitral valves were villous granulations sufficient to produce obstruction. The other valves were normal. Lungs congested and œdematous.

This case illustrates how an associated pericarditis may obscure an endocarditis. The "purring thrill," however, pointed to mitral obstruction.

Septic or *suppurative endocarditis* is a comparatively rare disease. In a series of forty-eight cases of endocarditis I found it in

only three, or about 6 per cent. And from a review of others of my cases to date, I think it is even less common than these figures would imply. In fact, it is an unusual form of purulent infection. The lesions in the valves may be vegetative or ulcerative, but always suppurative; so that the emboli are necessarily septic, eventually producing metastatic abscesses.

So rare is the disease that it often escapes notice, but there are certain signs which should attract our attention. They are those of anæmia, embolism and metastatic abscesses, in conjunction with the other subjective or objective phenomena of endocarditis and sepsis.

If, in a case of puerperal fever, pneumonia, suppuration from bones or joints, gonorrhœa, or any form of infection, or in traumatism, signs of endocarditis develop, we should suspect the septic or suppurative form, especially if there are chills and fever (even though periodic) with an enlarged and tender spleen, and bloody urine.

In suspected malignant endocarditis the blood should always be examined. Sometimes a pyogenic coccus can be cultivated, indicating that there is a septicæmia; or the gonococcocus may point to the cause of the disease, just as the pneumococcus suggests the lung, and the colon bacillus the intestines as the gates of infection. Usually with the anæmia there is marked leuocytosis, together with destruction of red blood cells, which may be reduced to 1,000,000. (Ewing.)

If blood examinations in a suspected case fail to react for typhoid, malaria or tuberculosis, a diagnosis of malignant endocarditis may be made by exclusion. (Cabot.)

In a case of gonorrhœal endocarditis, however, reported by Stein (*Wien. Klin. Woch.*, Nov. 22, 1900), Weichselbaum found streptococci, the inference being that the gonococci simply made the soil favorable for other bacteria, and that the case was one of mixed infection. I am inclined to think that there are several microorganisms concerned in the production of this acute infection.

The diagnosis is manifestlyy difficult, and yet is not impossible, if all the phenomena are taken into consideration. The signs include præcordial pain, dyspnœa, headache, insomnia, irregular chills, sweats and fever, anxiety, rapid and irregular pulse, and the physical signs of endocarditis, with septic or pyæmic manifestations; in short, the signs of endocarditis, with a tendency to metastatic abscesses. Inasmuch, however, as in most cases the acute form of the disease is engrafted on an old one, we should also inquire as to

whether there have been previous signs of endocarditis. Therefore, we should expect to find some dilatation of the heart, with a dislocation of the apex outwards or downwards, or both. The organic murmurs of septic endocarditis are, so far as my experience goes, confined to the aortic and mitral valves.

The disease is usually fatal in a few days, but cases have been reported where it has lasted for months. Recoveries must be very rare. In my opinion most of the cures reported have been in cases where there has been embolism, with chills and sweats, but the emboli have been benign. Personally I have never known a case of recovery in malignant endocarditis. I have, however, known recovery to follow gonorrhœal rheumatism with metastatic deposits, and I have also seen one recovery in pure pyæmia. Septic endocarditis should not, therefore, be altogether hopeless.

The following are illustrative cases:

Case III. Acute Malignant Endocarditis; Caries of the Carpal Bones.—A. C., colored, aged forty-six; was admitted to hospital November 5, 1878. He was first taken sick on October 25th, but had never before been seriously ill. At first he had pain in the left knee, and about the same time there were chilly sensations, followed by headache, fever and backache. He took to his bed, and remained there until removed to hospital. On admission, there was found to be pain on the left side (spleen?), and in his left leg. His wrist also was swollen, tender and intensely painful. No fever, but great prostration. On November 28th, after showing no signs of improvement, he passed blood per rectum. Probably there had been an evening rise of temperature for some time. It remained between 101° and 102° until December 3d. Towards the last it rose to 104°, pulse to 116 and respiration to 46. At the post-mortem examination no cardiac hypertrophy was observed, but at the aortic orifice, beneath one of the cusps, was a verrucose growth the size of a small chestnut, while the valve was ruptured. On removing the brain, pus was found in the meshes of the pia mater. At the wrist (left?) the first row of carpal bones was found necrotic or carious. Duration of the disease, six weeks. In this case the evidence pointed to necrosis, or caries, of the carpal bones as the source of the acute endocarditis, though the evidence connecting it is not as satisfactory as one might wish, because at the time no special attention was directed to the endocarditis.

Case IV. Lobar Pneumonia; Malignant Endocarditis.—The history of this case was sent me from St. Francis Hos-

pital. A patient of alcoholic habits, aged forty-one, was admitted in November of 1882, with a lobar pneumonia. There was high temperature, but no cardiac murmurs were detected. Other and apparently more important conditions obscured the cardiac disease. At post-mortem examination the aortic cusps were found extensively diseased, each segment exhibiting vegetations, while one carried a growth the size of a hickory-nut, and was ruptured and ulcerated. An abscess was also found in the course of the coronary artery, and a sinus led from it to the fungating mass in the aortic valve. The spleen contained an infarct. Duration of the illness, about five weeks. The malignant endocarditis was probably closely related in etiology to the pneumonia.

Case V. Malignant Endocarditis; Suppurative Infarcts.—A gentleman of this city was taken sick with an attack of fever that confined him to his room for four or five days. He then felt better and went downstairs, but soon returned and took to his bed. During the first two weeks of his illness the temperature ranged from 98° to 100° F., his pulse from 140 to 150, sometimes reaching 160. During the last two weeks of his life it had a wider range (120 to 150). At an early period the diagnosis of obstructive endocarditis was made out. The respiration was never embarrassed, except when the patient sat up. There was never any impairment of motion or sensibility, nor did he experience any pain, except on one occasion when his physician attempted to turn him over on his left side. He then cried out suddenly, "You have killed me," and placed his hand over the region of the spleen, groaning with pain. He recovered from this attack, but died subsequently with suppression of urine.

The post-mortem examination revealed a stenosis of the aortic, which would hardly admit the passage of the first joint of my little finger. The surfaces of the cusps were marked by calcareous concretions and vegetations. The heart, and especially the left ventricle, was dilated and hypertrophied. The kidneys presented the usual appearances seen in the large variety of chronic diffuse nephritis, and also contained both recent and old infarcts. The lungs also had infarcts. The *spleen* and the meninges, however, showed the most important lesions. The first-mentioned organ measured about nine inches in length, and was the seat of numerous infarcts of various ages, some red, others brown and others yellow, while one had been the point of origin for an abscess from which a pint to a pint and a half of dirty, grumous, offensive matter was discharged.

Emboli were also found in the meshes of the pia, with attendant suppuration. This acute attack was plainly engrafted on an old rheumatic endocarditis, but why the emboli were suppurative was not determined.

Chapter IV.

MITRAL INSUFFICIENCY.[1]

Mitral insufficiency, regurgitation or incompetency is a comparatively common valvular affection, and the least serious of any, so long as it is uncomplicated. But it is seldom the only valvular lesion. From my tables it appears that in 86 per cent. it was associated with aortic, pulmonary or tricuspid disease, the combination with aortic being the most common. The most frequent cause of insufficiency is endocarditis, which is also most frequently caused by lithæmia. Under the influence of this and other constitutional vices vegetations form along the borders of the leaflets, which thicken and then retract, while the tendinous cords and papillary muscles also become infiltrated and, contracting, hold the leaflets back. Another cause of inorganic insufficiency is the *rupture* of a *leaflet*. All of these phenomena I have seen. Finally, the orifice may be involved in a new growth, or atheroma may prevent closure. Usually, however, the latter infiltrates a leaflet without interfering with its closure.

Of the inorganic or relative form there are many varieties, and it may be a temporary or permanent condition. One of the most common causes is violent physical exercise, such as young men are subjected to, in training for athletic sports. In one of the physical culture schools of this city I have been told by the manager, who is also a physician, that most of the prominent athletes under his tuition are affected with mitral regurgitant murmurs during their training. Or an aneurysm of the arch causing the large heart so common in aortic disease is pretty apt to entail some relative, that is, inorganic, dilatation; or in plainer language, stretching of the mitral orifice. This is because the whole left heart must dilate and the tendinous cords and papillary muscles stretch (the leaflets usually failing to enlarge in size, so as to fit the enlarged orifice).[2]

In a somewhat similar way the fatty heart may dilate, the leaflets failing to enlarge proportionately. Now, it is quite apparent that this form of insufficiency is capable of remedy, provided the condition governing it is removed.

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[1] Published originally in the *N. Y. Med. Journal*, Feb. 12, 1902.

[2] In some instances the valve leaflets do actually enlarge to compensate for the enlarged valvular openings.

Probably it is quite common as a temporary affair—for example, after a set at tennis, a boat race or a running match, in recovery from fevers, or after an infection or in cardiac neuroses. At autopsies we are not likely to see very many of these accidents, because they do not cause death. On the other hand, we not infrequently find at autopsies an artificial mitral insufficiency where post-mortem softening has set in. This condition is less often seen now than formerly, owing to the system of post-mortem refrigeration that is at present in vogue.

In mitral insufficiency there is such an imperfect closure of the mitral leaflets that, during systolic contraction of the left ventricle, more or less blood leaks back into the left auricle, already partly filled with blood coming from the lungs. Necessarily the left auricle dilates and then hypertrophies, because it has more blood to be driven into the left ventricle. And inasmuch as the left ventricle has to use more force in order to supply the aorta with its proper quantum of blood, it also hypertrophies after dilating. But overfilling of the left auricle dams the blood back on the lungs and offers such resistance to the column coming from the right ventricle that this also hypertrophies and gives way, causing dilatation of the right auricle, whenever the tricuspid yields.

The most characteristic sign of mitral insufficiency is a systolic murmur between the apex and the axilla or scapula, due to the leakage of the mitral during the contraction of the left ventricle. This murmur will vary in quality; it is usually rough and loud, rarely musical. Inorganic murmurs are softer and have more of a blowing character. Regurgitant murmurs are also intensified by slight exertion.

Accentuation of the second sound over the pulmonary artery is another sign which is due to the sudden closure of the pulmonary leaflets, caused by the strong resistance ahead of them in the auricle.

The right ventricle gets to be hypertrophied rather than the left when compensation is accomplished, so that we look for greatly increased transverse dulness; for the left border of the heart may extend from an inch or two to the right of the sternum as far as to and beyond the left nipple.

In the early development of mitral insufficiency, of the organic form, the compensatory symptoms usually go hand in hand with the lesion, so that although a systolic murmur is present, the affection may not be appreciated by the patient or those about him. Yet there will result some embarrassment of the pulmonary circulation,

which will be shown by a little shortness of breath on exertion, perhaps by an increase in the pulse rate, with more or less irregularity.

There may also be spitting of blood, for at this period the pulmonary vessels are all dilated and cause some bronchorrhœa. When compensation is fully established it is at first through the hypertrophy of the left ventricle, but eventually and chiefly by the right, and then the pulse will be slow, full and regular. Compensation may last a long while, with patients who are fortunate enough to combine both a knowledge of their condition and the ability to control adverse incidents. In my records I have not a single instance of uncomplicated mitral insufficiency in which death was attributable to it; though it may certainly cause death when complicated with some other valvular trouble.

With laboring men and those exposed to unusual vicissitudes there will necessarily be lapses, the breakdown at the end coming earlier; and yet during all this time the systolic murmur may continue to be loud, while its quality, whether rasping, filing, blowing, etc., or even musical, will depend upon the physical character of the orifice and adjacent parts.

In my opinion, however, organic insufficiency passes over eventually into stenosis or obstruction.

Acute relative insufficiency is a temporary condition that will mend with rest and systemic treatment; while the forms that result from muscular weakness, as from the poison of diphtheria, the continued fevers and infections generally, from faulty innervation or fatty degeneration, will improve synchronously with improvement in the conditions producing them.

And yet in the mitral insufficiency of day laborers, and in adherent pericardium, there is less likelihood of compensation, because the conditions that cause it are apt to be permanent. In uncomplicated cases the prognosis is good, but complications are to be expected.

Ruptures or lapses of compensation are ushered in by relaxation of the ventricular walls, causing venous congestion, first of the pulmonary system and then later of the systemic. At the end the systolic murmur grows faint and may even become inaudible, owing to the deficient force of the left ventricle. The second pulmonary sound also will become progressively weaker, owing to the yielding of the right ventricle.

Venous pulsation, or certainly a wavy motion in the veins of the neck, indicates giving way of the right ventricle. The surface becomes livid, there is palpitation, with a weak and intermittent pulse,

and the cardiac impulse grows faint or disappears. The liver may be swollen and tender; the urine scanty and albuminous. About this time dropsy may be expected, perhaps delirium cordis, and death is a natural sequence. Or death may result from asystole, though uræmia or pulmonary hæmorrhage may close the chapter.

Up to the period of breaking compensation the three cardinal signs are: 1. A systolic murmur between the apex and the axilla. 2. Accentuation of the second pulmonary sound. 3. Increased transverse dulness of the heart. In and after breaking compensation the diagnosis must be based on the previous history, because the abnormal transverse dulness may be the only one of the three cardinal physical signs left from which to construct a diagnosis.

If obstruction coexists, as it does in from 70 to 80 per cent. of the cases, we must expect a systolic thrill at or near the apex in from 15 to 60 per cent., and a presystolic murmur in at least from 10 to 30 per cent. In children or young people there may be a bulging of the præcordia.

Mitral insufficiency is, as I have said, comparatively easy to diagnosticate in uncomplicated cases, as the following instance will show:

Case VI. Mitral and Tricuspid Insufficiency.—L. A., a cabinet-maker, born in France, seventy years old, was admitted to hospital January 3, 1881. Eight days previously he was taken with shortness of breath, wheezing, slight cough and spitting of blood. Soon his legs began to swell. On examination fluid was found in the pleural cavity. Patient cyanotic; heart sounds indistinct. A few days later, a mitral systolic murmur was made out, with increased heart dulness and diffuse heart beat. A cardiac murmur, loudest over the ensiform cartilage, was attributed to tricuspid regurgitation. Later some lung consolidation was discovered. At the post-mortem examination the aortic and pulmonary valves were found normal, while the right cusp of the mitral was thick and contracted and bound up. Left auricle greatly dilated. Mitral admitted the tips of seven fingers. Valves of tricuspid thickened and restricted in movement; weight of heart, 23 ounces. Both lungs contained red infarcts. Right chest full of serum; left chest nearly full. This case was examined by several of our best clinicians, the result being an absolutely correct diagnosis in essential particulars, the chief point of interest to us being that both the mitral and tricuspid lesions were noted.

But though mitral regurgitation is comparatively easy to diag-

nosticate, it is unsafe to pin our faith too exclusively on auscultatory murmurs, as indicative of organic disease. For occasionally functional murmurs are produced at the apex, by the patient's posture or other causes. These murmurs, however, are not apt to be accompanied by an accentuated secondary pulmonary sound. They may also be due to impoverished blood or muscular vibration. Owing to the extreme rarity of a tricuspid insufficiency (according to my records about 4 per cent.), it is apt to be disregarded in practice. It may, however, be combined with mitral insufficiency, as in Case VI. It is, however, difficult of diagnosis, not only because the centre of the tricuspid area is tolerably close to the mitral, but the two areas overlap to some extent. In uncomplicated cases of tricuspid insufficiency, however, there is no accentuation of the second pulmonary sound; the systolic murmur is not conveyed to the left as much as it is to the right; there is venous pulsation of the veins in the neck, and a dilated right heart.

In instances where there are merely fresh vegetations on the valves sufficient to cause very slight insufficiency no murmur may exist. After all, mitral insufficiency is comparatively easy to determine, because the valve is to the left and behind, and as the murmur follows the regurgitant current, which flows more or less backward as it goes upward, it is carried somewhat towards the angle of the left scapula. In my experience the diagnosis of pure mitral insufficiency is made in three quarters of the cases.

The treatment is considered in Chapter XIX.

Chapter V.

MITRAL OBSTRUCTION.[1]

Our conceptions of mitral obstruction originated with Corvisart, Napoleon's able physician, and the teacher of Laennec, who tells us that in 1819 Corvisart first called attention to the "purring thrill" of mitral obstruction, which he described as a "particular sensation, which in some cases is perceptible to the hand when this is applied to the region of the heart," and is a "sign of the ossification of the valves, and particularly of the mitral valve. Indeed, this phenomenon is observed in almost every case in which there is some contraction of the auricle of the heart." Leaving out of consideration Laennec's erroneous views about "ossification," it is enough to note that he clearly associated the discovery of the "purring thrill" with his distinguished teacher.

He is also equally emphatic in attributing the discovery of the *diastolic bruit* of mitral obstruction to his contemporary, Bertin, but the description of it is all his own. Alluding to one of his personal cases, he uses these words:[2] "The contraction of the auricle, which was extremely prolonged, was performed with a dull but strong sound, precisely resembling that produced by filing wood. This was accompanied by a vibration sensible to the ear, and is evidently the same as is felt by the hand. At the close of the contraction one could distinguish by a louder sound, accompanied by an impulse and perfectly synchronous with the pulse, the contraction of the ventricle, which was three-fourths shorter." Three cases of this kind, he tells us, had been described by Bertin, and were verified by autopsies.

Twenty-four years later Fauvel first called attention to the subvariety of diastolic bruit now known as the *presystolic,* which he defined as a "loud, rasping bruit" preceding the first sound or murmurmur and ending with it. In English circles W. T. Gairdner,[3] of Edinburgh, is usually credited with defining it, which he did under the name *"auricular systolic bruit."* According to Gairdner, it was a murmur preceding the first sound, running sharply up to it, then coming abruptly to a stop.

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[1] Originally published in the *N. Y. Med. Journal,* May 10, 1902.
[2] *Mediate auscultation,* London, 1846, pp. 555 and 617.
[3] *Clin. Med.,* 1862, p. 598.

Obstruction at the left auriclo-ventricular orifice, which for convenience sake rather than because it is the best term, has been called mitral stenosis, is not a very uncommon lesion. Walshe[4] puts it fourth in his list, which is as follows:

1. Mitral insufficiency.
2. Aortic stenosis (obstruction).
3. Aortic insufficiency.
4. Mitral stenosis (obstruction).
5. Tricuspid insufficiency.
6. Pulmonary insufficiency.
7. Pulmonary stenosis (obstruction).

It also occupies the same position in one of my lists (sixty-five valvular cases) verified by autopsies, which is as follows:

1. Aortic insufficiency, 49 times.
2. Aortic stenosis (obstruction), 39 times.
3. Mitral insufficiency, 38 times.
4. Mitral stenosis (obstruction), 33 times.
5. Tricuspid insufficiency, 8 times.
6. Tricuspid stenosis (obstruction), 4 times.
7. Pulmonary insufficiency, 4 times.

One hundred and seventy-five valve lesions in 65 autopsies.

And in fifty cases taken from my clinic by myself or assistants (not verified, of course, by autopsies), the incidence as to mitral obstruction was the same, being:

1. Mitral insufficiency.
2. Aortic insufficiency.
3. Aortic obstruction (stenosis).
4. Mitral obstruction (stenosis).
5. Tricuspid insufficiency.
6. Pulmonary insufficiency.[5]

It may be laid down as a fact that mitral obstruction in general implies regurgitation, though the mitral regurgitant murmur is apt to be absent in the later stages of obstruction. In the sixty-five cases alluded to they coexisted in 45 or 70 per cent. In fact, advanced obstruction seems to be almost impossible without regurgitation; certainly in stenosis, where the orifice or leaflets or their attachments are rigid.

On the other hand, regurgitation not infrequently occurs without obstruction, as for example, in the early stages of organic in-

[4] *Diseases of the Heart*, London, 1873, p. 105.
[5] Endocardiopathies, *Virginia Med. Semi-monthly*, April 26, 1901.

sufficiency and in all cases of the relative variety. The relation of obstruction to regurgitation is sometimes close, according to my figures, and yet it is variable. There is clearly a sequential relation between the two, regurgitation being as a rule preliminary to obstruction, and subsequently its associate. So far from the standpoint of pathological anatomy.

Clinically, there is also a close relation between the two, but the interest of the physician turns chiefly on the determination of which is the predominating disease. On this point hangs the prognosis, for mitral regurgitation has a comparatively favorable outlook, while mitral obstruction seriously modifies the expectation of life.

Well marked mitral obstruction is, however, somewhat rare. In seventy-one instances, complete as to clinical histories and autopsies, taken by myself from French, English and American authors (not including my own cases), advanced obstruction occurred in only thirty-eight, or about 53 per cent. In twenty-nine of my personal cases the severe ones were nine, or only 31 per cent. The larger ratio in the foreign cases is, perhaps, because rheumatic affections are more common abroad, especially in Great Britain, than in this country. So that, on the whole, the severe cases average somewhat less than one-half the total. But it is in this class that we encounter the greatest difficulty in diagnosis, because the diastolic murmurs are apt to be faint. How great this difficulty has been may be judged from the records of the Massachusetts General Hospital, where, as late as 1900, in forty-eight cases of mitral obstruction, as proved by autopsies, but twenty-three, or 45 per cent., were recognized during life. (Cabot, *Phys. Diag.*, New York, 1900, p. 163.)

Another reason for failure in diagnosis (other than the one given) is, as was intimated at the outset, that the data on which to base rules for diagnosis have been defective. An instance is well shown by Fagge,[6] who tells us that previous to 1871 there were but twenty-eight cases on record where presystolic murmurs had been found, on post-mortem examination, to have been associated with mitral obstruction. And yet this sub-variety of the diastolic murmur had been known for upwards of thirty years, in fact since 1843.

Speaking broadly, there is a wide difference between the general effect on the heart and system at large, between chronic obstruction and chronic insufficiency. In the former systolic contrac-

[6] *Guy's Hospital Reports*, 31, Ser. 3, 16, 1871.

tion of the left ventricle fails to force a requisite quantity of blood into the aorta, because part of it escapes through the leak in the mitral valve. To overcome this difficulty the left ventricle hypertrophies and remains hypertrophied as long as the leak is considerable; but as chronic insufficiency passes over into chronic stenosis, so the hypertrophied left ventricle synchronously finds its task more easy until at length, if the patient survive, the orifice is so reduced as to represent nothing but a chink or cleft, and the leak is no longer considerable. But now the blood is very greatly delayed in its passage from the left auricle, and an undue quantity is detained there. Dilatation and hypertrophy of the left auricle naturally ensue, for greater force must be used by the auricle to expel the blood during each diastolic interval.

Assuming, then, that chronic insufficiency of the mitral precedes chronic obstruction, this change in the walls of the left auricle is the second of the series. The third is the arrest in the hypertrophy of the left ventricle, or even an atrophy, due to the fact that the insufficiency has been succeeded by stenosis (obstruction).

The accumulation of blood in the left auricle, and the consequent backing of the venous blood into the lungs, produce in turn hypertrophy of the right ventricle, because more work is thrown on it. If, however, this ventricle finds itself incompetent for the task and dilates, the tricuspid valves will also stretch and hypertrophy, and dilatation of the right auricle will follow. The vicious circle is then complete.

At and beyond a certain grade of obstruction, therefore, the insufficiency is relieved. Unfortunately a substitution of the one for the other does not improve the expectation of life. But it emphasizes the importance of combatting the systemic disease at an early period by treatment directed to preventing continued infiltration of the valves and their attachments, because there appears to be no controlling influence in nature to counteract it. In this connection I believe I may say that the profession is hardly willing to seriously consider Balfour's suggestion of opening the heart and cutting the constriction. Even supposing this procedure were practicable, it could not arrest the constitutional process which produced the stenosis.

The following cases, taken from my records, illustrate some of the points I have made.

Case VII. Aortic and Mitral Disease.— ———, aged thirty-one. Admitted to hospital October 23, 1885. The patient com-

plained simply of dyspnœa and rheumatic pains. On examination the heart's action was found to be rapid and irregular. There was a systolic murmur at the apex conveyed to the left. Death took place after fourteen hours' stay in the hospital. At the autopsy the heart was found to weigh 18 oz. The mitral was dilated and the tendinous chords and margins of the valves were covered with vegetations. Mitral orifice dilated so as to admit three fingers (two fingers being the ordinary size of the opening). Aortic valves thickened and the seat of vegetations, but sufficient. Pericardial sac contained 4 oz. of serum. Left ventricle hypertrophied and dilated. Right ventricle not hypertrophied. Lungs œdematous. Kidneys of the large, white variety. Infarctions of spleen.

Now, such a case as this I hold to be in a stage preliminary to obstruction (stenosis), though as a matter of fact the orifice was really dilated. But given a mass of vegetations about any valve, let them continue to develop, and the patient will surely have stenosis if the disease continues and he lives long enough. In this case the aortic disease appears to have been insignificant as compared with the mitral, the aortic valves being classed as sufficient; so that the hypertrophy of the left ventricle may be fairly charged to the mitral insufficiency.

On the other hand, the following case, No. VIII, may be regarded as one of tolerably advanced obstruction in which the regurgitant element was supplanted by the obstructive. We observe that the heart in this case was small as compared to No. VII. There was no hypertrophy of the left ventricle, but the dilatation of both the right auricle and right ventricle indicated that the anatomical changes had shifted over from the left to the right side, as was to be expected in this particular case of valvular lesion.

Case VIII. Mitral Obstruction (Stenosis) and Regurgitation.—————, aged thirty-eight, teacher, was admitted to hospital Nov. 19, 1891. About a month before admission she was taken with dyspnœa, weakness, dyspepsia and epigastric pain. On a first examination, a bruit preceding the first sound was heard at the apex, and it was propagated to the middle of the axilla, so it was thought. At any rate, a correct diagnosis of mitral regurgitation and stenosis was finally made. About a week after admission she was taken with spitting of blood and râles, and cogwheel respiration was heard at the base of the right lung. On November 29th the patient's mind became affected. Pulse moderately strong, but irregular.

Delirium ensued and then death. At the post-mortem examination 20 oz. of fluid were found in the peritoneal cavity. There were hæmorrhagic infarctions in both lungs. Weight of heart 8 oz. Right ventricle dilated. Tendinous chords contracted. Mitral did not admit a single finger. No hypertrophy of left ventricle. Aortic normal. Pulmonary valve a little thickened and dilated. Right auricle dilated. Nutmeg liver. This case is a good example of the changes that may be found in the heart and system at large, in the middle period of mitral obstruction, after compensation has been established.

It is astonishing how small the orifice may become. I have occasionally seen it so reduced that it would not admit the little finger.

During the first period where there is hypertrophy of the left ventricle, the apex is carried out to or perhaps beyond, the nipple, but as this ventricle contracts the apex recedes within the nipple line, usually remaining within it during the course of the disease, unless there is some complication, such as the common one, aortic disease.

When in a subsequent stage the right ventricle is enlarged and begins to labor, it thumps against the wall of the chest and its increased breadth is recognized by percussion. It may extend as much as two inches beyond the right sternal border. But with all these changes the heart is seldom much enlarged, for in uncomplicated cases it will average in weight from 15 to 18 oz. only.

In Dyce Duckworth's cases (*St. Bartholomew's Hospital Reports,* 13, 263), 264 in number (though in many the ante-mortem diagnosis was not verified by autopsies), the average age was thirty-five. In my first series of 100 cases I found the limit of age seventeen and seventy-eight, but 68 per cent. of them died under forty, and at the average age of thirty-five. In the second series the average age was thirty-three. Sansom (*Albutt's System,* p. 908, Vol. 6) found the average age of death 32.7 years.

There has been a disposition among English writers, particularly, to state that the female sex is most often affected. In Broadbent's fifty-three autopsies, according to Sansom, there were thirty-eight females to fifteen males. In a series of seventy cases, however, I have found that the preponderance of females was not noteworthy (thirty-six to thirty-four), and Sansom's seventeen autopsies have given the proportion of only ten females to seven males.

The value of the pulse by itself as a diagnostic factor is small. Walshe, already quoted, speaks of it as regular. James Andrew (*St. Bartholomew's Hospital Reports,* 13, 1877) makes it small, rapid and irregular. As a matter of fact, it will be irregular until compensation is well established, but regular during well-sustained compensation, and irregular again with failing compensation. But in all stages it is apt to be small and feeble at the last; becoming intermittent with irregular rhythm, which is often in marked contrast to the prevailing strong or heaving cardiac impulse.

The characteristic presystolic murmur associated in our minds with mitral obstruction has various qualities. It is sometimes spoken of as "rumbling" or "rolling," but in my experience it is more often loud and "rasping," or "sawing," though it may have other qualities and be faint or barely audible. It is contemporaneous with the thrill, if there be any, and indicates the passage of blood from the auricle into the left ventricle in the diastolic interval. The murmur may occupy the whole of this interval or any part of it. In fact, there may be a double diastolic murmur. Though, properly speaking, all diastolic murmurs are presystolic, the term presystolic, by common acceptance, is limited to those at the extreme end of diastole. This murmur may come or go. When the patient is weak or the orifice small, it may be impossible to hear any murmur. Again, other murmurs may mask it. In twenty-seven of my own cases, proved by autopsies, I found the presystolic murmur, which presumably existed

heard	in 4	=	15 per cent.
not heard	in 19	=	70 per cent.
falsely interpreted	in 4	=	15 per cent.
	27	=	100 per cent.

In Fagge's series of forty-seven cases a presystolic murmur was noted in only seven, or 15 per cent., which tallies with my experience. On the other hand, Samways, in 156 cases as proved by post-mortems at St. Bartholomew's Hospital (*British Med. Jour.,* 1898, i, p. 36), found a presystolic murmur audible in about 60 per cent., and in Hayden's fifteen autopsies it was noted in twelve, or 80 per cent. However, in the latter's total of eighty-one cases sixty-six were not confirmed by autopsies. In eight of Fagge's twenty-eight cases there was a double murmur, diastolic in character. In other words, there was a diastolic murmur in 28 per cent. apart from the presystolic, which was 15 per cent., so that the total

of his murmunrs in the diastolic interval was 43 per cent. In a series of twenty cases I found

a simple mitral presystolic murmur in	3 =	15 per cent.	
simple systolic in	4 =	20 per cent.	
presystolic and systolic in	5 =	25 per cent.	
no murmurs in	8 =	40 per cent.	
	20 =	100 per cent.	

From my figures, therefore, it appears that the simple presystolic murmur was noted in only 15 per cent., the systolic in 20 per cent., the double murmur in 25 per cent., or a total of diastolic murmurs equaling 40 per cent. My figures agree, therefore, pretty nearly, with those of Fagge.

In one of my twenty-seven cases, the presystolic murmur, it was stated, was carried to the left of the nipple, in another to the axilla; in two it was heard as high as the second rib; usually is was about at the apex. Probably in the case where it was said to have been carried to the axilla it was confounded with a systolic murmur. In one instance it was confounded with a Flint murmur carried down from the aorta to the apex, and in this particular case the aortic leaflets were so distorted as to make the explanation of Sir Walter Foster apply, namely, that the sound was caused by the aortic stream impinging on a stiff mitral leaflet. But there may be no murmur, as I have indicated, and as the following case shows:

Case IX. Mitral Obstruction, Anæmia, etc.— ———, aged thirty-two, domestic, was admitted to hospital Jan. 22, 1877. She was the mother of eight children. About a week before admission her urine became scanty, and there was a swelling of her abdomen. She had orthopnœa, anasarca, uræmia and cyanosis. Pulse weak and intermittent, heart sounds faint and irregular No murmurs. On March 31st she was discharged improved, but was readmitted on May 22d with a return of symptoms. She died two days later, of erysipelas. At the post-mortem examination the right ventricle and auricle were found dilated and thin. Stenosis (obstruction) of mitral with extensive calcareous deposits. Aortic thickened, but not rough. Pulmonary and tricuspid normal. Weight of heart, 19 oz. Lungs œdematous. Kidneys granular and pigmented.

The absence of murmurs when examination was made is comprehensible, when we consider her weak condition, and that she was in the final stages of mitral obstruction.

To distinguish between the mitral obstructive and the aortic regurgitant, feel the carotid pulse and not the radial, which is later

than the carotid; then carry your stethoscope by successive steps from the base to the apex, and you are certain to determine which is the first and which the second sound.

It is important also to note if there is a peculiar quality, pitch or duration or intensity in either murmur. If there is it will help to distinguish the one from the other.

W. S. Thayer (*American Journal of the Medical Sciences,* November 19, 1901, p. 538), however, from a study of 74 cases of aortic insufficiency, where the lesion was determined by post-mortem examinations, has concluded that "in uncomplicated cases of aortic insufficiency a rumbling, echoing, presystolic or mid-diastolic murmur, limited to the region of the apex," occurs in fully half these cases (*Medical Record,* January 18, 1902), from which it would appear that the value of the presystolic or diastolic murmur as a sign of mitral obstruction is not so great as has been held.

There are other signs of aortic disease, such as the "long heart," Corrigan pulse, and dilated aorta, that assist in the diagnosis of aortic insufficiency, while the aortic direct murmur is carried upward into the vessels of the neck.

From my two series of cases I gather that a purring thrill exists in from 10 to 35 per cent. Samways, in 196 cases, found the thrill in less than 33 per cent. It is usually noted in the fourth left space, sometimes in the fifth or sixth, again in the fifth, sixth and seventh. The thrill in this situation probably always denotes stenosis of the mitral or implication of some other valve, usually the aortic. So far as the mitral is concerned, it means that the opening is small. The thrill continues through the period of effectual compensation; indeed, a strong thrill means good compensation, and loss of thrill heartweakness. The accentuation of the second sound over the pulmonary is a very important sign. It indicates that the left auricle is over-filled, the extreme back pressure from the blood in the auricle against the pulmonary valve causing the accentuation of the second pulmonary sound. Epigastric pulsation is a late symptom. It is usually associated with a large, tender and pulsating liver, and is an unfavorable sign, indicating that compensation is failing and the right side of the heart becoming involved, so that dropsical effusions are not off.

Embolism is a special feature of mitral obstruction, as Cases X and XI show.

Case X. Mitral Obstruction; Embolic Pneumonia.— ———, aged twenty-eight, widow, was admitted to hospital Dec. 20, 1877. She had suffered from cough for five years, and seven years from

heart trouble. Eight years previously had an attack of inflammatory rheumatism. The patient was found on admission to be anæmic and much emaciated. Breathing short and rapid, cough with frothy expectorations, pulse 120, temperature 101.2° F. On examination, she was found to be in the third stage of phthisis. Heart's action weak, but not out of proportion to her general debility. No organic lesion discovered. Patient developed uræmia suddenly and died Dec. 22d. At the autopsy the heart was found to weigh 14 oz. and was stated to be normal, except as to the mitral, which had a button-hole opening three-quarters of an inch long, in its longest diameter. The lungs were the seat of embolic pneumonia. Liver atrophic and nutmeg. Cause of death, embolic pneumonia.

But cerebral embolism may also cause death, as in the following case:

Case XI. Mitral Obstruction; Cerebral Embolism.————, aged fifty, clerk, was admitted to hospital Dec. 12, 1883. He had suffered from many attacks of inflammatory rheumatism. In 1882 there appeared symptoms of slight cerebral embolism as shown by aphonia, which lasted two days. On entering the hospital he was found to have dyspnœa and abdominal pain with consolidation at the apex of the right lung. There were also murmurs at the apex of the heart with the first sound, and at the base with the second sound. Albumin and granular casts. Pulse strong and regular, later becoming weak and irregular, during an intercurrent attack of rheumatism with uræmia and pulmonary œdema. At post-mortem examination the heart was found to weigh 27½ oz. Hypertrophy of both ventricles. The mitral had a button-hole opening and was insufficient. Oedema and brown induration of the lungs. Chronic diffuse nephritis. Enlarged and pigmented liver. Death was attributed to cerebral embolism.

In 18 per cent. of my first series of 100 cases the rhythm was irregular. In 20 per cent. of my second series it was also irregular. In one there was a double rhythm; in one a quadruple rhythm. In several there was a gallop rhythm. The cardiac impulse may be ill defined or diffuse, heaving or strong, and the impulse may be carried to the epigastrium. In well established compensation the heaving is due to hypertrophy of the right ventricle. A noteworthy sign of mitral obstruction to which little attention has been given is the pretty constant relation of a strong cardiac impulse to a weak radial pulse.

There has been some effort to divide mitral obstruction into three stages based on auscultatory signs (Broadbent, 3rd edition, 1900), though as these signs are not closely associated wtih the stages it is a difficult task. It is more in accord with present methods to divide this affection into stages on an anatomical basis. This is not altogether satisfactory, but I have endeavored, somewhat roughly, in the cases used for illustration, to indicate that there may be a preobstructive stage, and early, middle and late stages.

In fact, I have already described the anatomical changes which take place in the evolution of a case of uncomplicated mitral obstruction. According to this view we have at first simply the signs of mitral insufficiency. Then when those of insufficiency give way to those of obstruction the right ventricle becomes hypertrophied and the impulse is "thumping" or strong. In a certain proportion of cases there is a thrill and a diastolic murmur, perhaps a presystolic; and a sharp, "tapping" first sound at the apex. In the last stage, when compensation fails, the presystolic murmur disappears, because the auricle has no longer strength to drive its column of blood into the left ventricle. The right auricle becoming dilated through the giving way of the tricuspid, causes pulsation in the veins of the neck. Dyspnœa, dropsy, and pulmonary œdema then supervene. This last stage is well shown in the following case:

Case XII. Mitral Obstruction; Pulmonary Oedema, etc.—————, aged thirty-seven, plasterer, was admitted to the hospital May 20, 1879. Three weeks before admission his feet began to swell. General anasarca followed, with debility, scanty urine, and pulmonary œdema. The heart was hypertrophied, but no signs were noted expect those of mitral insufficiency. No second sound was audible. Pulse 40. Under appropriate treatment he improved and was discharged. But in less than a month he was readmitted with a recurrence of the symptoms, culminating in suppression, of which he died. At the post-mortem examination there was found general anasarca, with 12 oz. of fluid in the pericardial sac. Heart hypertrophied, weighing 22 oz. Aortic and pulmonary valves free. Mitral a mass of atheroma, with a small button-hole opening, causing both obstruction and insufficiency. Lungs œdematous. Kidneys enlarged and congested. Liver atrophic, but pigmented.

In mitral obstruction there is great danger of embolism, not so much from the diseased valves as from clots that become entangled in

the interstices between the tendinous chords and papillary muscles during imperfect cardiac action.

The following points appear from my tables:

1. Mitral obstruction is usually fatal before the age of forty is reached.

2. Females are little more prone to it than males.

3. There is apt to be a marked contrast between a strong cardiac impulse and a feeble radial pulse.

4. The true presystolic murmur occurs in 15 per cent. It comes and goes; but it is usually inaudible in the last stage.

5. It is apt to have a loud, rasping or sawing quality, but may be "gushing" or "whirring." It may also be faint.

6. In about 40 per cent. there is some sort of a diastolic murmur.

7. These murmurs are best heard over a rather limited area, somewhat oval in form, having for its centre a point about over the middle of the 5th left intercostal cartilage, and about midway between the nipple and the ensiform appendix. The breadth of this area may be two to three inches and its vertical length five to seven. The murmur is sometimes heard best as low as the fifth, sixth or even seventh left space; more rarely it is heard as high as the second left rib.

8. In 10 to 35 per cent. there is a thrill over this area.

9. The first sound at the apex is short and abrupt.

10. The second pulmonary sound at the base is usually intensified.

11. Occasionally a murmur with the second sound at the base is heard over the left auricular appendix.

12. At first there is dilatation and hypertrophy of the left ventricle. Then atrophy of it, with dilatation and hypertrophy of the left auricle; then follow dilatation and hypertrophy of the right heart.

13. Mitral insufficiency must to some extent precede or accompany mitral obstruction.

14. In distinguishing the presystolic murmur of mitral obstruction from the Flint murmur of aortic insufficiency we should rely on the "long heart" and the strong impulse, or the "Corrigan" of insufficiency, rather than auscultatory signs. In case there is both aortic insufficiency and mitral obstruction a differential diagnosis is impossible, with the means we now have at our command, unless we can recognize some distinguishing qualities in the murmurs at the two openings.

Chapter VI.

DISEASES OF THE AORTIC VALVE.

Of all cardiac valves the mitral and aortic suffer most in endocarditis, but there has been a divergence in opinion as to which of these is most often affected. According to Sperling's tables, however, in 200 affections of single valves, the aortic was involved in over 40 instances, or 20 per cent.; while the mitral was affected in 157, or 78.5 per cent. On the other hand, in 100 of his combined cases the incidence on the aortic was 88, or 88 per cent.; on the mitral 98, or 98 per cent. So far as my own statistics, in combined cases, are concerned, they tally pretty well with Sperling's. In 44 combined cases the incidence on the aortic was 39, or 86.8 per cent.; on the mitral 40, or 90 per cent. From these statements it appears, therefore, that mitral disease preponderates in frequency over aortic disease, both in single and combined cases.

But as aortic disease is apt to be a rather late phenomenon in general endocarditis, which attacks the mitral first, as a rule, we look for implication of the aortic in advanced mitral disease. In my 44 cases this conjunction occurred in 37, or 84 per cent. If the tricuspid is involved, it is apt to be still later in the endocardial cycle.

Aortic disease gives rise to both insufficiency and obstruction, the latter often erroneously regarded as synonymous with stenosis. For obstruction may exist without stenosis, as shown in Case XVII. Insufficiency is the more common.

Insufficiency of the aortic was first noted, it is said, by Morgagni, but a satisfactory explanation of it was not given until 1830, when Corrigan, of Dublin, gave his masterly description of it. It is more frequent in males than in females. There are two forms of insufficiency, (1) *organic* and (2) *inorganic* or *relative,* each capable of giving the characteristic signs of the disease.

Of the organic variety one of the prominent causes that has been given is congenital malformation, but judging from my experience this condition must be extremely rare. In the congenital cases that I have seen, closure of the valves was usually perfect, notwithstanding their defective construction. Furthermore, the claim made that congenital malformation is an etiological factor is apt to

be associated with the statement that the leaflets had undergone inflammatory changes. But these changes may have occurred independently of the anomalies. In fact, there is no reason why malformed segments should incite either inflammatory or degenerative changes. In this connection it may be said that the small fenestrations so commonly seen in the leaflets do not produce insufficiency, though large ones, the result of rupture, may do so.

A more common cause is *endocarditis,* which may be of the benign or malignant variety. The latter, due to acute infective processes such as gonorrhœa, etc., is usually fatal, but the former, which is more common and usually the result of rheumatism or the continued fevers, is a slow, subacute process, originating with vegetations on the valves, which either grow into tuberous masses that will eventually snap off, or undergo fatty degeneration, or harden down into calcareous nodules causing obstruction. Again, adhesions may take place between the cusps, or between the cusps and the adjacent walls. In one of my cases a calcareous body was found attached to the edge of a leaflet, and it must have been carried backward and forward in the blood current. In another a tumor the size of a chestnut lay beneath one of the leaflets, preventing its closure.

A cause often assigned is *arterio-sclerosis,* and it is the most serious. Occurring in early middle or advanced life, it is a general systemic disturbance implicating other important organs, and indeed the whole arterial system. At first it is merely a slow sclerotic change, but with advancing age and under certain conditions it assumes a degenerative character, from which there is little hope of amelioration, but only a prospect of progressive destruction.

The first of the causes of arterio-sclerosis is to be found in the strain or tension of severe exercise. This condition is met with not only in the toiler (laboring man), but also in the athletes training for feats of strength or endurance. Alcohol is another of the causes. A third is syphilis, which produces at first hyaline changes in the vascular tunics and then thickening of the walls. Another cause is lithæmia, which produces changes in the vessels somewhat similar to those resulting from syphilis. Rupture of a valve is another. This is a rarity, but I have seen it at post-mortems. It may occur in health as the result of a fall or severe concussion; but a slight accident, such as a hurried walk, may produce it, when the valve is infiltrated and brittle.

Relative insufficiency is of comparatively infrequent occurrence.

It may occur in any form of hypertrophy of the left ventricle, but is unusual, unless associated either with a sacculated aneurism of the arch, or the fusiform dilatation sometimes found in its ascending portion (Hodgson's disease). This form of insufficiency, therefore, is of late occurrence in life, while organic insufficiency may occur at any time.

In the endocardial forms occur the usual indications of that disease, which do not differ materially from the endocardial lesions of other valves; though the intense rigidity that is seen in the mitral and the infiltration of the adjacent parts is not so common. But a very small amount of valvular infiltration may produce insufficiency, by shortening the cusps. In fact, there may be great relaxation of the ring at the base of the valves. This is seen in the relative insufficiency that is apt to attend the dilatation of the aorta from either the fusiform or sacculated aneurism.

In arterio-sclerosis, which is in most cases associated with endocarditis, there may be deposits at the base of the valves without insufficiency; or a spiculum or plate projecting out may cause insufficiency, so that a considerable amount of arterio-sclerosis may exist without insufficiency. As in endocarditis, it is the location and configuration of the deposit rather than its size which determines the insufficiency. There are great differences in its degree. In moderate cases there may be only a triangular defect; in advanced instances it may take the form of a slit or cleft. Occasionally the opening may be widely dilated; but there may be an enlargement of the leaflets to compensate for the widened opening.

In aortic insufficiency the changes in the heart, in the progressive evolution of insufficiency, are as follows in an uncomplicated case: Owing to the leak in the aortic valve some of the blood in the aorta regurgitates during diastole into the left ventricle, and meeting there the normal quantum of blood coming at the same time from the left auricle, causes dilatation of the ventricle. The latter, in order to expel this increased amount of blood, hypertrophies. Now if the insufficiency is progressive, or if stenosis is combined with insufficiency, there will be a progressive increase of blood in the left ventricle. But the increased collection of blood in the left ventricle will embarrass the left auricle, and through it the current of blood flowing into it from the lungs will be delayed, so that the right ventricle will also dilate and hypertrophy. But even if the right ventricle should enlarge when a sudden call is made on the heart, it may diminish in size when the strain has been taken

off. Hence the enlargement of the left rather than the right heart is a measure of the success of compensation. This aortic lesion makes the *big and long heart* where dilatation and hypertrophy are very prominent. In one of my cases with obstruction and marked insufficiency the heart weighed 29 ounces.

The early development of aortic insufficiency is so insidious that it is apt to be overlooked. Indeed, at first no physical signs may be present, and the attention of the patient is first called to it by subjunctive symptoms, such as præcordial oppression, palpitation, fulness, roaring in the head, sparks before the eyes (denoting an irregular supply of blood to the head), or anginoid attacks. Occasionally these symptoms are only evoked by a strain or excitement of some kind, or when the hypertrophy does not keep pace with the growing insufficiency.

If arterio-sclerosis is at fault, there may be the *facies arterio-sclerotica*. In endocardial cases, however, the face is often pale, In arterio-sclerosis there will also be general systemic changes, and it is in these cases particularly that there is apt to be pain.

On inspection in well established cases there is a *broad area of cardiac pulsation*. In children and young people there may be a bulging of the præcordium, and occasionally systolic contraction of the soft parts owing to the flexibility of the walls of the chest and the pressure of the enlarged heart, which often displaces the lungs.

The *apex* may be found in the 7th or 8th space instead of the 5th, and may extend to the line of the nipple, or in very rare cases to the axilla. In well developed cases the impulse will be strong and heaving. Sometimes there is a thrill when obstruction is present; it may be systolic or diastolic.

Percussion will demonstrate that this is the large and long heart, and as dilatation and hypertrophy are largely confined to the left ventricle, the heart will assume a more vertical position than usual. But during the temporary dilatation and hypertrophy of the right ventricle, that may occur before compensation is established, after excitement or strain, or in failing compensation, the dulness may extend an inch or more beyond the right margin of the sternum.

On *auscultation* a prolonged murmur is heard in diastole over the aortic area, which corresponds to the second right or left interspace and intermediate space, and the bruit is propagated down towards the ensiform cartilage, but it is usually heard with greatest intensity in the middle sternal region, extending sometimes to the

right, but ordinarily to the left, of the sternum. Occasionally it is continued to the apex and even beyond it, the "Flint murmur." Indeed, it may closely simulate the murmur of mitral obstruction and be produced by the blood impinging on a leaflet of the stiffened mitral, for in more than half the cases aortic insufficiency is combined with mitral disease. In dilatation of the ascending arch of the aorta (or Hodgson's disease), which is common in advanced arterio-sclerosis, the murmur may be carried far to the right, but the dilated arch will not necessarily be dull on percussion. The murmur may occur at any part of the diastole, and may entirely supersede the second sound.

The *bruit* is often described as "gushing," "swishing" or "whirring," but it may be so faint as to be overlooked. It may also be rough, or even musical. The intensity of the bruit depends on the size of the opening, and the force of the contraction of the ventricle. When extreme stenosis exists or in extreme dilatation there may be no murmurs. Other lesions of the valves may also mask them.

The *pulse* is large and quick because the arteries are dilated by the powerful cardiac impulse, which has to be short in point of time to drive the requisite quantity of blood, during the interval allowed it. But this quick discharge of blood at the aortic is followed by a sudden leak or backward regurgitation into the ventricle. Hence the characteristic pulse first described by Corrigan in 1830 and known as the *"cannon ball," "pistol shot," "trip hammer," "water hammer,"* or *"Corrigan."* It was for a time thought that the sphygmograph alone could furnish a ready diagnosis of this condition, but it is now known not to be pathognomonic, for a similar pulse has been occasionally found in aneurism and anæmia.

Another sign is the *capillary pulse.* Rub the patient's forehead with a towel until it is red and then note how the systolic action of the heart increases the ordinary redness, especially at the border of the reddened area. This capillary pulse, however, due to the left ventricle's forcing a column of blood directly into the capillaries, is seen in all hypertrophied hearts. Nor is the *venous pulse of Quincke* pathognomonic. It merely indicates that the blood is forced through the capillaries into the veins.

In aortic insufficiency there is apt to be visible pulsation of the arteries. There is also *pulsation of the jugulars* when compensation is incomplete.

Pressure with the stethoscope on the femoral will sometimes

produce a short systolic sound (*"pistol-shot"*). Such pressure in health will produce a systolic murmur, but if a diastolic is also produced (*Duroziez's sign*) it is a pretty sure sign of aortic regurgitation. Still it is not often met with.

Attacks of *angina* are more frequent in aortic insufficiency than in any other valvular lesion. The other symptoms often noted are headache, dizziness, palpitation, thrill, and pain transmitted up the neck and down the arm. *Sudden death* is more frequent in this lesion than in any other form of valve disease; and it is here that neurotic disturbances are so prominent. In fact, such disturbances should be expected, though they may be merely of the hysterical variety. As in all other forms of valvular and non-valvular disease, there are three stages.

During the *first stage*, in organic cases, as the dilatation and hypertrophy of the heart keep pace with the advance of the insufficiency, there may be no subjective symptoms, unless the patient undergoes emotional excitement, or strains his heart in some way. Then the hypertrophied heart finds itself unable to cope with the increased load, and the lungs fill up with blood, causing dyspnœa and palpitation; and such attacks are always liable to occur.

In the *second stage* compensation will have been established, and, barring such incidents as have just been described, there will be no subjective signs. This stage may last many years, fifteen or twenty, perhaps more.

When compensation is about to fail, its approach will be heralded by shortness of breath and dyspnœa, especially at night, cough and acceleration of the pulse. The pulse will be rapid, 110 to 120, or more. A new sign is at hand as soon as the systemic circulation is involved: we now note pulsation in the epigastrium, due to dilatation of the vessels in the liver.

Aortic insufficiency is recognized with comparative ease. In my 37 cases it was noted in 23, or 62 per cent. In 65 proved by post-mortems at the Massachusetts General Hospital, only 44 were recognized, or 67 per cent. The diagnosis is based on five principal points.

We have the *large and long heart*, because in it dilatation and hypertrophy are prominent, but the dilatation and hypertrophy are mainly confined to the left ventricle. The position of the heart is apt to be somewhat vertical.

In consequence of the enlargement of the left ventricle this displaces or compresses the lungs, so that it comes nearer to the

sternum than usual, and its pulsation is better communicated. For the same reason the beat is more diffuse and heaving.

The *murmur* is usually soft, but not necessarily so; it may be rough or musical, while it is conveyed downward, and is apt to be heard best in the mid-sternal region along the left sternal border; occasionally at the ensiform cartilage or at the apex. One should not pin too much faith on the murmur, however, for it may be absent.

The pulse is usually characteristic. It is full and quick, and is apt to be of the *trip-hammer* variety.

A *thrill* is comparatively rare, but it may occur if through the distortion of a valve or some impediment, the current of blood is twisted.

In general, a previous history of alcoholism, lead-poisoning or syphilis should lead to a suspicion of aortic disease, especially in middle life. If mitral disease has been previously well established, we should, in 70 per cent. of the cases, expect aortic disease.

One of the greatest difficulties is the differential diagnosis between aortic valvular disease and aortic aneurism. The following diagnostic points must be taken into consideration:

Aortic insufficiency. Bruit and thrill in area to right of sternum, increasing as we approach the aorta from above.

No dulness necessarily over dilated aorta.

May also be inequality of radial pulse, but "trip-hammer" variety the rule.

In arterio-sclerosis may be a history of syphilis. May be caused also by rheumatism, gout, lead poisoning and infections. The X-ray throws no shadow.

Aneurism. Bruit and thrill affect both arch and ventricle, and area to right of sternum, diminishing as we approach the aortic valve from above.

Dulness widespread if aneurismal sac contains fibrin. Inequality of radial pulses, but never the "trip-hammer." Syphilitic history usually. The X-ray throws a shadow corresponding to the sac.

In general, the amount of the lesion is proportioned to the hypertrophy of the heart, except when obesity or Bright's disease coexist. The prognosis depends on many considerations. If the insufficiency is of traumatic origin, such for example as is caused by a sudden strain, the prognosis may be good, but such accidents are extremely rare. Occurring in the young, before the usual degenera-

tive changes have taken place, it has a better outlook. It is also comparatively favorable, because compensation is established by the left ventricle, which alone is competent to rectify the bad effects of this lesion. But though compensation may last fifteen to twenty years or more, long life is seldom attained. In the management of a case we should remember that compensation depends on the ability of the left ventricle to do its work. Hence its dilatation and hypertrophy are to be maintained. Whenever it shows signs of irregular action or feebleness, it should be quieted and sustained. Rest in the recumbent position is at first the *sine qua non* in an acute attack, while cold compresses may be applied to the chest, and the extremities massaged, so as to draw off as much blood as possible from the heart. I am in the habit of using in these cases the bromides, or the mono-bromate of camphor; sometimes acetanalid in 2 to 5 grain doses; also Hoffman's ether and the comp. tincture of valerian in drachm doses. When the acute stage is over I use carbonated baths and exercises.

The following cases taken from my hospital records illustrate some of the ordinary varieties of aortic insufficiency:

Case XIII. Malignant Endocarditis; Aortic Insufficiency and Obstruction.— ———, fifty-six, single, was admitted to hospital May 21st, 1881. Patient had been a well man, according to his statements, up to four weeks before admission. At that time he was attacked with pain in all his joints, which were swollen and disabled. For the previous three weeks he had suffered from headache, epigastric distress, and nausea, without vomiting; and during this time had irregular chills and sweats. In the week preceding admission he had increased dyspnœa, but without cough.

Physical examination showed that the apex was in the fifth space, one-half an inch to the left of the nipple. A double mitral murmur was heard. Pulse very irregular. Fine, moist râles at the bases of both lungs. Later a presystolic murmur was heard twice at the apex, but no double murmur. Later a double murmur was heard at the base. Orthopnœa. On May 31st signs of hypostatic congestion were recognized, and a diagnosis of probable ulcerative (malignant) endocarditis was made. The patient died suddenly the same day.

At the autopsy, which, unfortunately, was not very complete, it was found that the pericardium contained half an ounce of bloody serum, while the right auricle was attached posteriorly. There was a large vegetation at the aortic valve and an ulcer extended

beneath the valves, from the aortic to the mitral. The spleen was irregularly spotted and puckered from old infarctions, but no recent ones were found.

Here, then, was an instance of aortic insufficiency in an old benign endocarditis which had taken on a malignant character.

Case XIV. Organic Aortic Insufficiency, with Relative Mitral Insufficiency.— ———, twenty-seven, was admitted to hospital April 2, 1887, with the history of an attack of acute rheumatism ten years earlier, and a recent recurrence one week before admission. On physical examination a low-tension pulse alternating between the normal and 120 to 130 was noted. Apex in the fifth space. Heart enlarged. At the apex there was a soft systolic murmur quite diffused and an aortic direct and indirect murmur. Dyspnœa, palpitation and nausea followed one another in the course of his three visits to the hospital. During the last of these he developed pleurisy with effusion, and thirty-two ounces of fluid were withdrawn.

The apex had been found at one time in the seventh space. Heart enlarged. There was much præcordial pain with anginoid attacks. The patient died Nov. 3rd, 1887, with acute dilatation.

At the autopsy there was found marked aortic insufficiency, but it was not thought that there was enough infiltration in or about the valves to indicate obstruction. There was marked mitral insufficiency without organic changes (relative variety). Heart weighed twenty-one ounces. Cardiac tissues degenerated.

Case XV. Aortic insufficiency, with Dilatation of the Orifice and Fusiform Aneurism of the Ascending Arch (Hodgson's disease).— ———, sixty-four, was admitted to hospital July 8th, 1882. Patient admits having been a moderate drinker, and having had gonorrhœa and rheumatism. On admission he was found to have œdema of the upper and lower extremities. Double aortic murmur at base. Apex in fifth space, one-half an inch outside the nipple. Rapid and irregular pulse. Patient soon developed suppression with albuminuria, with noisy delirium and dyspnœa. He died in coma on October 28th.

At the autopsy the following was found: Heart not markedly enlarged, but an adherent pericardium. Cavities dilated; all valves sound except the aortic. The aortic segments were thickened and shortened, and the orifice was markedly dilated. The ascending aorta was also dilated and had atheromatous plaques, ulcerations and calcareous deposits. It constituted a fusiform aneurism (*Hodgson's disease*). Kidneys granular, contracted and cystic. Liver

slightly cirrhotic; chronic meningitis. Death attributed to chronic diffuse nephritis.

Case XVI. Relative Aortic and Mitral Insufficiency Due to Cardiac Hypertrophy of Renal Origin.— ———, thirty-eight, was admitted to hospital Aug. 22nd, 1887. He had suffered from acute articular rheumatism, and for the past three years from palpitation and dyspnœa, but swelling of the feet and abdomen had occurred, for the first time, about a week previously. On admission he complained of sparks before the eyes, cough and bloody sputum; was pale, and had facial œdema. Pulse irregular. The heart was found to be hypertrophied. Apex one inch below line of nipple and one and one-half inches outside it. Double aortic and also a mitral regurgitant murmur. Urine sp. gr. 1012. Trace of albumin. The uræmic symptoms from which he had been suffering became aggravated, and he died about two weeks after admission.

At the autopsy it was found that so far as the aortic and mitral valves were concerned there was no organic change, except a slight roughness in the aortic, and the insufficiency of both valves was attributed to the hypertrophy of the heart from renal implication.

The following case shows incidentally how auscultatory signs may be misinterpreted:

Case XVII. Aortic Insufficiency mistaken for Mitral Insufficiency.—B.— was admitted to hospital December 11th, 1880. The patient stated that he felt perfectly well up to three weeks previously, when he was taken with præcordial distress and difficult breathing. Two weeks later he had cough and expectoration, orthopnœa, general malaise, nausea and vomiting. On examination it was found that the heart was hypertrophied and that there was pulsation of the veins in the neck. Apex to the left and below nipple. Wavy movement of the epigastrium. Expiration high pitched and prolonged. Consolidation of left lung posteriorly. Murmur at apex thought to be with first sound. Murmur heard behind and better above than below the scapula. Diagnosis of dry pleurisy and of mitral regurgitation. Patient died with œdema of the lungs December 14th. At the autopsy both lungs were found to be almost solid at their bases. The heart was enlarged. Pulmonary opening dilated. Dilatation of right heart. Fresh vegetations on border of aortic valve. Both cusps thickened. Obstruction, but no stenosis. Mitral normal.

In the absence of mitral disease we see in this case how sometimes the murmur of aortic insufficiency may be conveyed to the apex and beyond, probably in this instance through the agency

of the consolidated left lung, the result being an error in the diagnosis.

Aortic Obstruction.—In the choice between the words obstruction and stenosis I select the former, because it has a wider significance, embracing all forms of stenosis, while the latter does not embrace all forms of obstruction. As in mitral disease, there may be obstruction without narrowing (stenosis).

Aortic obstruction is a tolerably common valvular lesion. Walshe put it second in his list, and in 65 of my autopsies, in valvular diseases, I found it holding the same position. In a clinical series of 50 cases, however, I put it third. But while there is a general agreement that it is a common valvular lesion, it is not so well understood that uncomplicated aortic obstruction is extremely rare. Several of our most prominent clinicians have seen no cases, and in the pathological records of the Massachusetts General Hospital, embracing 252 valvular cases, not a single one was recorded. In my 65 post-mortems I have but one. If there were any lingering doubt as to the rarity of simple obstruction, a search throngh the literature should dispel it. Certainly painstaking investigations on my part have discovered only a very few published cases of aortic obstruction without insufficiency. It well might happen that a practitioner with a pretty wide experience in heart cases would never meet with a pure example of uncomplicated aortic obstruction.

These cases will be referred to, however, for after all it is only from uncomplicated cases that we can frame rules for diagnosis. Indeed, the inference is positive that heretofore our diagnoses have for the most part been based on phenomena not confirmed by autopsies, or on cases of aortic disease complicated by insufficiency. But while, practically speaking, obstruction is usually attended by insufficiency, the converse is not true. And this was shown in my 65 cases, where in 11, or 17 per cent., there was aortic insufficiency without stenosis (obstruction), but of course relative insufficiencies were included.

This point has a practical bearing, for given the signs of aortic obstruction, and the chances are that in the great majority of instances obstruction is combined with insufficiency.

These two forms are well contrasted in the following cases:

Case XVIII. Aortic Obstruction and Insufficiency Associated with Aortic Aneurism. Syphilis.—J., thirty-eight, painter, was admitted to hospital January 10th, 1883. The patient had contracted syphilis about seven years perviously, secondary manifestations, such

as sore throat and alopecia, occurring four or five years later. He then complained of pain in the sternum. On physical examination direct and indirect aortic murmurs were distinctly heard. The patient died five days later, during a sudden attack of cyanosis with dyspnœa.

At the autopsy the pericardial sac was found to contain 12 ounces of serum. The heart was very much hypertrophied, especially the left ventricle. Weight 24 ounces. The aortic valve was much thickened, and its segments were shortened by atheromatous deposits, there being insufficiency and obstruction. All the valves of the right side were normal.

The arch of the aorta was the seat of what was regarded as extensive syphilitic endarteritis, with ulcerations and cicatrices. At one point to the right of the aortic valve some of the arterial coats had given way and a sacculated aneurism had formed, about the size of a hen's egg. A small opening led into the sac. In the liver were small gummatous tumors.

Case XIX. Aortic Stenosis without Insufficiency.—E., forty-eight, was admitted to hospital April 4th, 1878. The patient, whose only previous disease, so far as he knew, had been pneumonia, or possibly scarlatina, learned that he had heart disease one year previously from a physician who was treating him for a cough and hæmoptysis. Shortly afterwards he found that his lower extremities were beginning to swell. Two weeks before admission the œdema had reached his scrotum, and the urine had fallen to 15 ounces per day. On examination the apex beat was found to be diffused over a larger area than normal and was two inches below the nipple. On auscultation a systolic murmur was heard at the apex, not conveyed to the left or behind (tricuspid regurgitation). At the base was a double murmur supposed to be an aortic direct and indirect. Pulmonary œdema. The patient was put under active treatment, with digitalis and elaterium, and cupped; but there was little reaction. Symptoms of uræmia supervened and he died twenty-four days after admission. At the autopsy his heart was found to be very large, weighing 31 ounces. All cavities distended. Aortic perfectly sufficient, but leaflets contracted, while the free surface exhibited a small row of white, shining, translucent bodies. Left ventricle hypertrophied. Right very little hypertrophied. Tricuspid and pulmonary insufficient, a large stream of water passing through the former. Infarcts in both kidneys. Cysts in one. Nutmeg liver. Vessels at base of brain atheromatous. The cause of death in this

case was uræmia, induced in part at least by renal embolism. In this case the only error committed was that pulmonary insufficiency was mistaken for aortic insufficiency. The extreme rarity of the former might be offered as an excuse for the failure to recognize it.

Aortic obstruction in the vast majority of instances is caused by arterio-sclerosis, which in turn often means syphilis, and it is merely an extension of the disease of the aorta back into the valves, Or rheumatism may cause endocarditis, which invades the segments of the valves, the disease beginning at the free edges, generally with the development of vegetations or papillary growths and a contemporaneous infiltration of the segments, and subsequently their contraction or distortion. Again, the leaflets may become fused together by adhesive inflammation, leaving a triangular or funnel-shaped opening. This latter is not so common as in mitral stenosis. In health the opening should admit the little finger, but in stenosis it may become so small that it will only admit a small sound, or even the tip of a slate pencil, as in one reported case.

Congenital aortic obstruction, which is a true stenosis, is only one of the numerous cardiac anomalies, the combination of which, as a rule, soon ends the infant's life. Very exceptionally, patients may live with aortic stenosis of the congenital type to advanced life, according to Eshner,[1] who has reported one that lived to the age of 90 years. The duration of life, however, is probably measured by the insufficiency that accompanies the stenosis.

Among the remoter causes are strain and injury, which almost necessarily lead to inflammation, adhesion and contraction. Other causes of obstruction are lateral pressure on the valves by something extra cardiac, as an enlarged gland.

Aortic obstruction being a late event in valvular disease, it is found, as a rule, after middle life; in exceptional cases it occurs in early life or even in infancy.

The obstruction to the escaping blood throws extra work on the left ventricle, which hypertrophies and dilates, as seen in Case XIX, the heart becoming greatly enlarged. It has been claimed (Whittaker) that it never assumes the magnitude of the heart in aortic insufficiency, but this is disproved by Case XIX, where the heart weighed 31 ounces, and this also disproves the statement of Potain that a scarcely appreciable hypertrophy with but slight displacement of the apex speaks for aortic (stenosis) obstruction, for the case given was one of pure aortic stenosis. The truth is that while the left heart hypertrophies invariably from a moderate degree to an

enormous extent, it does not on the whole average so much as in aortic insufficiency.

Basing the symptoms of uncomplicated aortic obstruction on the recorded cases[1] I have collected, including one of my own, I find the following symptoms:

They are mostly in males, usually after middle life, though one of them was 19 years of age. The pulse is slow and weak, sometimes irregular. Dyspnœa, palpitation and syncopal attacks occur. The impulse is diffused over a wide area and the apex is to the left. Usually the murmur is absent; always when stenosis is extreme. In two cases, systolic murmurs at the apex, or thereabouts, were attributable to tricuspid regurgitation.

Among other signs are pulsation of the veins in the neck and in the interclavicular notch, cyanosis and dropsy. There was no thrill in any of the cases. Right ventricle moderately or greatly enlarged; left less so. Embolism and œdema are other features.

It will be noted (from the above summary) that no thrill was found, and this may be regarded as an important negative sign. We have seen that it is a positive sign in insufficiency. The impulse, of course, varies with the stage of the disease. It is diffuse, though not so strong and heaving as in insufficiency combined with stenosis. The murmur is usually heard best in the second or third right intercostal space, is long-drawn, conveyed up the great vessels of the neck, and gradually diminishes in loudness as it asscends. Its quality depends on the character of the obstruction. If the orifice is smooth or small, there may be no murmur. A systolic murmur at the apex, due, according to Dickinson, to regurgitation from strong pressure through the mitral, was attributable according to Case XIX to tricuspid regurgitation. When there is a rough or musical murmur, there is roughness in the valve or just above it. It may be so loud that it is heard all over the heart, and cannot well be located. The apex will be low down to the left, and may even reach the seventh space, and extend two inches outside the nipple, but this is exceptional. In all cases there will be an enlarged heart with the apex carried downwards and to the left. There must be, in the early and middle stages, certainly until the orifice has diminished to a mere

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[1] Farwell, *Birmingham Med. Gaz.*, 1830.
Eshner, *Path. Soc. of Phil.*, XIV., p. 154.
Ashton, *Path. Soc. of Phil.*, XVII., p. 103.
Gibbons, *Path. Soc. of Phil.*, XVII., p. 134.
Murray, *Path. Soc. of London*, XXI., p. 98.
Owen, *Path. Soc. of London*, XXXIII., p. 72.

slit, a systolic bruit, heard best in the aortic area and carried up through the great vessels of the neck. When stenosis and insufficiency are combined, as is usual, there will be the signs of insufficiency. Engorgement of the lungs due to commencing heart failure will lead to tricuspid regurgitation, and will be evidenced by a systolic murmur near the apex, increasing as the ear nears the ensiform cartilage. Obstruction of the pulmonary artery is rare, but it may lead to confusion, because the signs of aortic obstruction are sometimes best heard over the pulmonary valve. In pulmonary valve disease, however, the sounds are very superficial.

Angina, embolism and vertigo are especially common in aortic stenosis.

Owing to the diversity of causes, the prognosis is variable. In congenital cases aortic stenosis is usually only one of the many cardiac anomalies which together soon end the infant's life; so that the consideration of this matter from the standpoint of the aortic lesion is of comparatively little importance. And yet a man with acquired stenosis of an extreme type may live to ninety, as in Eshner's case. Considerable contraction of the orifice may exist for a long time, if hypertrophy develops contemporaneously with the construction; but unfortunately, as already said, we seldom have pure cases of aortic disease. As it is generally combined with aortic insufficiency, the duration of life must be measured by the insufficiency, and the rule laid down by Fagge is probably correct that in the *see-saw* of aortic disease the louder the first sound the better the prognosis; the louder the second sound, the reverse. But a short second sound is regarded as more unfavorable than a long-drawn second sound. When there is a loud systolic murmur the probability is that there is a wide opening and little obstruction. If the patient has syphilis the prognosis, other things being equal, is worse than if he has rheumatism. And yet if he is under good management, and leads a careful life, it is better than in rheumatism. Unfortunately we are too apt to believe that a prolonged course of treatment extending over two or three years, by mercurials and the iodides, will rid the system permanently of the poison. This is a very serious error.

Chapter VII.

PULMONARY VALVE AFFECTIONS.[1]

Of all cardiac valves the pulmonary is least often affected. Very exceptionally it is a single lesion. Sperling (*Gibson's Dis. of the Heart,* London, 1898), with his large experience, has not recorded a single case. Fortunately I have brief notes of one.

In association with other valvular diseases, pulmonary affections occur in the ratio of 1 to about 10 per cent. Sperling puts it at 4 per cent. Insufficiency is the form commonly seen in adult life; stenosis in fœtal life or infancy.

Pulmonary valve diseases occurring in adult life, however, are usually consecutive to endocarditis or other valves, or to arteriosclerosis, the ratio being, according to my tables, six in a total of 177 lesions verified by post-mortems. In my office cases the ratio was put at 3 per cent., and in fifty taken from my clinic by myself and assistants, pulmonary insufficiency was at the bottom of the list. Walshe (*Dis. of the Heart,* London, 1873, p. 105) from his list makes it occupy the sixth place in a total of seven varieties of lesions. But his clinical notes, not to any large extent confirmed by pathological data, are interesting rather than convincing. Five of the cases I now record are taken from my post-mortem records as former pathologist to the St. Luke's and Presbyterian hospitals. One was furnished me by the Babies' Hospital. Temporary insufficiency is doubtless of common occurrence. For a slight diastolic murmur can be produced over the second or third left intercostal space, after the breath has been held for a brief space, when respiration is again commenced. Chronic insufficiency, however, is not common. Gerhardt, in 1890, (*Charité Annalen,* XVII, s. 255) had collected only twenty-nine cases that were verified by post-mortems, and Barié a year later (*Arch. Gen. de Paris,* 1891, Vol I, p. 650, and Vol. II, p. 30 and 183) fifty-eight, similarly supported by post-mortems from the years 1831 to 1874. Pitt, however, has carried the number up to ninety-nine[2] (*Albutt's System* 1899), while, as already said, I have been able to add six new cases, brief notes of which I give:

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[1] Published originally in the *Med News,* Sept. 6, 1902.

[2] Of these forty-four were taken from the pathological records of Guy's Hospital.

Case XX. Phthisis; fatty heart, chronic parenchymatous nephritis; tricuspid and pulmonary insufficiency (relative).—A woman of forty-two years was admitted to hospital January 23, 1884, with the usual signs of tuberculous phthisis, including cough, dyspnœa, profuse greenish-yellow sputum, night sweats and cyanosis. The pulse reached 104; temperature 102° F. She died of heart failure about two weeks after admission. At the autopsy all the heart cavities were found dilated, the walls thinned and apparently fatty. The aortic and mitral valves were sufficient, the tricuspid markedly insufficient, the pulmonary less so. The cause of death was put down to fatty heart and chronic parenchymatous nephritis. The tricuspid and pulmonary insufficiencies were due to the dilated right heart, such as occurs in the fatty heart of phthisis. The attention of the physicians appears to have been directed to the lungs chiefly, so that the lesions of the right heart escaped notice, as very frequently happens under such circumstances.

Case XXI. Aortic and mitral endocarditis; tricuspid and pulmonary insufficiency (relative).—A widow of forty-eight years was admitted to hospital June 27, 1885. The patient had enjoyed fair health until three months previously, when she was taken with cough, dyspnœa and night sweats. On examination the apex was found in the sixth space, 4½ inches from the median line. Loud systolic murmur at the apex, conveyed to the left, and even to the angle of the scapula. A loud systolic murmur heard over the second and third right interspaces, propagated up the great vessels. No aortic diastolic murmur appreciable. Pulse 128, irregular in force and frequency. Later, some enlargement of liver. Mental disturbance. Anteriorly, respiratory murmur rough; sibilant and sonorous râles. Still later, ascites developed. The patient died of heart failure. At the post-mortem examination the heart was found enlarged, weighing eighteen ounces. Cavities dilated. Vegetations at aortic and mitral, causing obstruction at both orifices. Pulmonary and tricuspid valves insufficient from dilatation of the right ventricle. The pulmonary and tricuspid insufficiencies were not recognized during life.

Case XXII. Arterio-sclerosis; tricuspid insufficiency and relative pulmonary insufficiency, due to dilatation of the pulmonary artery; Hodgson's Disease.—A sailor of fifty-six years was admitted to hospital November 23, 1886, for cough, dyspnœa and bloody expectoration, which had persisted for a month. Syphilitic history of thirty years' standing.

On examination he was found to have general œdema and emphysema. Visible pulsations in brachial, radial and ulnar arteries; venous pulsation in jugular veins. Urine sp. gr. 1002; albumin 33 per cent. Granular and hyaline casts. Fairly loud systolic murmur heard over the liver and at lower end of sternum. Diagnosis of tricuspid regurgitation made. Pulse hard as a telegraph wire. Cheyne-Stokes respiration. Relief was afforded by the nitrites. Patient died of œdema of the lungs. At the post-mortem examination the pericardial sac was found to contain eighteen ounces of clear serum. The heart weighed twenty-seven ounces and was hypertrophied, especially the left ventricle; the right was thinned and dilated. Aortic and mitral valves sufficient. Commencing aneurismal dilatation of the aorta. Pulmonary orifice dilated and insufficient. Liver enlarged and of a dark color. Diffuse nephritis.

Case XXIII. Aortic and pulmonary insufficiency, due to adhesive pericarditis, etc.—A man of thirty-four, Ireland, blacksmith. Admitted to hospital September 18, 1885. In the preceding January he had a cough and six weeks previously hæmoptysis, losing about 1½ ounces of blood. Had suffered from acute inflammatory rheumatism. On examination the heart's apex was found in the fifth space, 2½ inches from the median line. Soft systolic murmur over the aortic and also over the pulmonary area, with accentuation of the second pulmonary sound. Dulness with cavernous breathing over second and third left interspaces. Below, amphoric breathing. At right apex some signs of softening.

The patient died of cardiac failure October 3. At the post-mortem examination it was found that the heart weighed only 12 ounces. The heart substance was soft and flabby; walls thinned and cavities dilated. Slight fatty change in mitral leaflets. Aortic and pulmonary valves seat of numerous small perforations; pericarditis. It was held at the autopsy that, owing to the relaxed condition of the walls from the adhesive pericarditis, the valves must have been to some extent insufficient. Cavities in apices of both lungs. Parenchymatous nephritis, etc. A diagnosis of aortic disease had been made. This was not a satisfactory case from several points of view, but it is none the less on record as one of aortic and pulmonary insufficiency, and I therefore record it. It seems, too, as if this diagnosis accords best with the auscultatory phenomena and the post-mortem findings.

Pinhole openings in the valves are sometimes found as the result of congenital malformation. They never cause murmurs, but

good-sized openings and clefts may cause them. The pulmonary area is, of all valvular areas, the most likely to lead to error for the reason, among others, that it is so close to the aortic. In fact, as already stated, aortic murmurs may sometimes be heard best over the pulmonary area, and vice versa. Or they may be best heard above the second left cartilage or over the sternum opposite to it. But there are murmurs heard over the pulmonary area that, so far as we know, are quite independent of valvular disease, being due to anæmia, change of position, etc., etc. Hence the danger of attaching too much importance to auscultatory signs at this point.

Congenital insufficiency of the tricuspid is apt to be associated with pulmonary stenosis and insufficiency. Insufficiency may be found at any age. Barié found it between the ages of three and seventy-five in acquired cases. It does not seem to have any preference in the matter of sex.

According to Pitt, whose statistics are based on the largest number of tabulated cases, the chief cause given was ulcerative endocarditis; while next in importance were pulmonary stenosis, aortic aneurism and pulmonary dilatation, or some abnormality in the formation of the valves. In this connection it may be stated that Barié found dilatation of the pulmonary artery in sixteen, or 27 per cent. Among the causes given by Pitt for the ulcerative form were gonorrhœa, puerperal fever, pyæmia and pneumonia; while associated with the abnormalities were patent ventricular septa and patent ducts of Botalli. Stenosis causing insufficiency may also be due to adhesions of leaflets.

The *congenital* form, which is pretty certain to be associated with stenosis and cardiac anomalies, will be readily recognized by a general cyanosis of intense character, clubbed fingers and toes, and an abnormal development of the chest. In these cases the murmur is soft, because the blood stream lacks vigor and there are no ulcerations. In the acquired form there are apt to be (together with cyanosis and dyspnœa) signs of defective nutrition, as shown in my cases; for all four had chronic pulmonary disease with cough, dyspnœa and hemoptysis, and two of them had night sweats.

The most important physical signs of pulmonary insufficiency are, according to my returns: 1. Displacements of the apex. 2. Diastolic thrills in the second or third left space (in 20 per cent., Barié) from the edge of the sternum to a distance of one inch to the left of it, and conducted down the left edge of the sternum (Boyd).[3] 3. Double murmurs (in about 25 per cent.). 4. Diastolic

[3] Boyd, *Scott. Med. and Surg. Journal*, 1889, Vol. IV, p. 121.

bruits intensified by inspiration. 5. Implications of the lungs. 6. Murmurs intensified in the sitting position. 7. When a long breath is taken a jerky vesicular murmur. 8. Hemoptysis. 9. Dyspnœa. 10. Usually, but not always, hypertrophy of the right ventricle. 11. Epigastric pulsation. The pulse has no distinctive character. Often there will be no bruit at all. We must, however, discriminate against aortic disease (insufficiency) by the absence of hypertrophy of the left ventricle and by the Corrigan pulse. The murmur should be louder than the aortic.

According to Barié, the *diagnosis* was made in but thirteen out of fifty-eight cases (22 per cent.). According to Pitt, the diagnosis was made in more than half his septic cases. The ulcerative process seems to be necessary to cause murmurs. However, the diagnosis would certainly be made more frequently, if physicians would be systematic in their examination of the heart, whenever there is a suspicion of cardiac disease, making notes of all the murmurs at the four valces, as I have urged in a former chapter.

The *prognosis* is unfavorable in congenital disease. Acquired cases have been known to live to seventy-five. My four acquired cases reached ages between thirty-four and fifty-six; for after all, effective compensation may be established by hypertrophy of the right heart, just as in tricuspid regurgitation, with which it is so generally associated. And because general malnutrition, and infections like gonorrhœa, measles and syphilis, and phthisis, are potent factors, the success or lack of it in treating these maladies must largely influence the expectation of life. So that in prophylaxis and treatment, infections must be avoided and combated, and special care given to pulmonary implications.

When there is sepsis from infection, the use of the newer antistreptococcus sera offers hopes of cure; they are certainly worthy of consideration, if used in conjunction with other appropriate symptomatic measures. The lung affection, of course, should have the first attention.

Pulmonary stenosis or obstruction is one of the most frequent of the many congenital cardiac anomalies which defy classification, owing to their varieties. Uusually this valve defect is associated with abnormalities of the large vessels as well; with imperfections of the inter-auricular or inter-ventricular septa, or of valve leaflets. The two most common anomalies are patency of the foramen ovale and of the septum between the ventricles.

Usually the valve defects are curious rather than dangerous to life, for though the valves may be irregular in number or shape, or the leaflets improperly implanted, they often do their work satisfactorily. But when the valves are not separated, are twisted or missing, whether from imperfect or arrested development or prenatal inflammation, so that the calibre of the outlet is affected, the expectation of life will be seriously altered. But simple thickening of the leaflets may be due to intra-uterine disease or to senile change. If, however, the function of the valve is not interfered with the abnormality may be of trivial importance; but endocarditis of the pulmonary valve is a very serious matter, as it easily destroys these segments, which are extremely delicate, and so permanent insufficiency may be produced.

One of the most common of the associated extracardiac anomalies is the persistence after birth of the ductus arteriosus, which in fœtal life connects the pulmonary artery with the descending aorta.

Still we must recognize that, in a measure, one defect compensates another. If, for example, there should happen to be complete closure of the pulmonary artery, the patency of the ductus arteriosus would be a compensatory defect, permitting the aorta, through the duct, to supply the lungs with blood. And so, if the tricuspid happened to be closed the patent foramen ovale would allow the blood from the right auricle to pass to the left auricle. Among other minor anomalies we also occasionally find strictures, either of the pulmonary artery or one of its branches, or narrowing of the infundibulum, which lies beneath the pulmonary valves. There are on record a number of cases presenting these and other anomalies. In fact, the pathological department of any hospital caring for infants or young children would naturally have frequent instances of these defects. This is true of the Babies' Hospital of this city, with which I am connected. In a comparatively small number of cases stenosis (obstruction) is acquired. What the ratio between these congenital and acquired stenoses is, I do not know. It is a difficult matter to determine, even with pretty complete clinical and post-mortem records at hand. Many of the older reports do not make it plain. In fact, it was not attempted until the time of Constantine Paul in 1871. Besides, in many instances an acquired lesion like endocarditis is engrafted on a congenital malformation.

As a single congenital lesion, pulmonary stenosis is a great rarity. Fortunately, through the assistance of Dr. Wallstein, Path-

ologist of the Babies' Hospital, I am able to record one case, which I have already alluded to. It is the following:

Case XXIV. Stenosis of Pulmonary in an Infant.—An infant of five months died at the hospital after the following symptoms had been noted: A diffuse apex beat, and a systolic murmur at the base, heard with greatest intensity at the left of the sternum and transmitted up the neck on both sides. Diagnosis not made. No cyanosis. At the autopsy the pulmonary orifice was found one-third smaller than normal. No other cardiac lesion.

It will be noted from this case that obstruction (stenosis) of the pulmonary does not necessarily produce cyanosis. Pitt (*Albutt's System*, Vol. VII, p. 9) has collected fourteen cases of acquired stenosis. I have collected and verified fifteen, yet have failed to get access to at least one-half of the literature on this subject. The references to my cases I give here:

Ebstein, Deutsches Arch. f. klin. Med. Bd., VII., s. 281.
Peacock, Lancet, 1868, Vol. I.
Rindfleisch and Oberneier, Deutsches Arch. f. klin. Med., Bd. V., s. 539.
Mayer and Oberneier, Deutsches Arch. f. klin. Med., Bd. XXIV., s. 435.
Whitley, Guy's Hosp. Repts., III., 1857, p. 255.
Whitley, Guy's Hosp. Repts., III., 1857, p. 255.
Paget, Med. Chir-Trans., 1844, p. 182.
Crudelli, Riv. Clin. di. Torino., VII., 2 p. 37-68. Schmidt's Jahrb. 8, 1870.
Bertin, Heart Diseases, 1821.
Constantine Paul, Soc. Med. de Hop. de Paris, T. VIII.
Schwalbe, Virchow's Archiv., Bd. 119, S. 2, s. 45, 1890.
Schwalbe, Virchow's Archiv., Bd. 119, S. 2, s. 45, 1890.
Schwalbe, Virchow's Archiv., Bd. 119, S. 2, s. 45, 1890.
Wagner, Archiv. f. Heilk., 1866, s. 518.
Colberg, Deutsches Archiv. f. klin. Med., Bd. 5, s. 565.

I am inclined to think that the following case was also one of temporary stenosis, caused by extracardiac pressure, relieved by evacuation of an abscess. Otherwise I have none in my post-mortem records.

Case XXV. Probable Stenosis of the Pulmonary Artery.—A groom, age twenty-eight years, was admitted to hospital in July, 1883, with an abdominal tumor continuous with the liver and extending into the epigastric and right hyperchondriac regions. Palpable pulsation over the pulmonary area, extending up to and under the clavicle and attended with a soft systolic bruit. No other signs of cardiac disease. In the following month two incisions were made into the tumor and three ounces of pus removed at each operation. The man survived the operation, but died of progressive emaciation and sepsis. At the autopsy the liver was found to be greatly enlarged, weighing eighty-eight ounces. It apparently had

compressed the lungs, both of which were unusually dry, the left especially, and by pressure had caused the same kind of temporary stenosis of the pulmonary artery that is sometimes seen when aneurisms of the aorta by pressure alter the calibre of the vessel from a circular to a crescentic form.

In congenital disease the cause must be laid to lack of development. In the acquired form, infection, according to my tables, plays a most important rôle, the order of frequency being rheumatism, aneurism, syphilis, gonorrhœa. Doubtless further researches into the etiology of this subject will enlarge the number of causes, affecting the ratio here given. Pitt, in his fourteen cases, found rheumatism a causal factor in eight, or 57 per cent. Males and females appear to be about equally affected.

In symptomatology there is a wide difference between the congenital and the acquired forms. In the former there is usually *general cyanosis*, though not always, as in the instance I have here given. There is ordinarily a lack of physical and mental development. The patient complains of headache and is somnolent, or has hebetude; is undersized, and has a bulging chest and a protruding abdomen. There is defective development of the genitals, with clubbed fingers and toes. The eyes may be prominent. The physical signs are not very distinctive. The right ventricle is enlarged in complicated cases; both are enlarged when the left ventricle is called on for extra work. The murmur is systolic and usually, if there are vegetations, loud, it may be heard all over the præcordial area, but its intensity is apt to be greatest over the pulmonary area, *i. e.*, the second left intercostal space, close to the sternum. In addition it is continued up towards and sometimes under the clavicle. Exceptionally it may be best heard lower down. In one case[4] (Hun's) it was best heard at the fifth left sterno-costal junction.

In *acquired obstruction* we should look for an antecedent infection, and especially for venereal disease or rheumatism. As distinguished from the congenital form, there is less often cyanosis in the acquired; and there are none of the characteristics of arrested mental and physical development. But if there is cyanosis it is increased by coughing, and dyspnœa is present. The murmur is more definitely located than in the congenital form, because the force of the stream is undiminished by defects in the walls of the heart. It should be loud and rasping in the endocarditis forms. If the patient hold his breath it should be somewhat fainter. The

[4] *Albany Med. Annals*, pp. 57-66, 1897.

apex beat is apt to be forcible and diffuse, and there may be a thrill. The second pulmonary sound should be faint, but there may be insufficiency and a double murmur. The murmur is apt to be propagated from the base of the heart up toward the left shoulder, as far as the clavicle. The point of greatest intensity is usually in the second left interspace. Usually there is an attendant pulmonary or bronchial disease, of a purulent character.

When, as in the case I have given, the pulmonary stenosis is single, hypertrophy should be confined to the right heart. If for any reason the left heart has been called on for extra work it will naturally hypertrophy. There is nothing characteristic about the pulse. Some œdema, even anasarca and albuminuria, may be expected toward the end.

The diagnosis is never easy. Even in the congenital forms pulmonary stenosis is, as a rule, as I have said, only one of many anomalies; two at least of them, viz.: the patency of the foramen ovale and of the interventricular septum, capable of producing in the one a diastolic, and the other a systolic murmur. In infants it is hard to distinguish between the two; but as all heart anomalies are apt to center about pulmonary stenosis, a bold guess will sometimes be rewarded with success. In acquired cases, even when they have been most carefully studied as to every auscultatory detail, the diagnosis has seldom been made. Paul and Mayer, however, have each recorded a successful antemortem diagnosis.

The prognosis is never good. Exceptionally, congenital cases have lived to forty; usually they die before the fourth year of life, and of tuberculosis.

In the acquired form the prognosis is more favorable. One of Schwalbe's patients lived to be eighty-four, another to be sixty-eight, while Rindfleisch and Oberneier's died at sixty-five. Usually, however, they do not survive the third decade.

It is well to remember that at the birth of infants having this malformation, after removing the mucus from the mouth and nostrils, the circulation should be stimulated by slapping the surface. Artificial respiration by Sylvester's method, the author's or others, should also be resorted to; or the lungs may be inflated by the catheter, introduced through the larynx. After the circulation has been established in this way, the infant should be kept in a warm, dry room. In general, patients with this congenital malformation should live an uneventful life, in a warm, equable climate. They should have systematic exercise and their diet should be carefully regulated.

Their methods of life should be such that they have no physical or mental strain. Matrimony should be sternly prohibited.

In the acquired form infections and rheumatic tendencies should be combated; and the cardiac symptoms should be met, as they arise, with appropriate treatment.

Chapter VIII.

TRICUSPID DISEASES.[1]

Serious affections of the tricuspid are comparatively rare and, with the exception of pulmonary valve diseases, are the most uncommon. They are still more rarely single. Gibson, however, maintains that tricuspid insufficiency is the most common of valvular diseases, while Sperling makes the incidence 26 in 100 of combined lesions, and three out of 200 of single lesions. In 44 cases of valve disease as shown by post-mortems I found the incidence 12, or 27 per cent., while I have observed no single cases. Tricuspid insufficiency is usually relative, and due to many causes, cardiac and extra-cardiac.

Organic lesions of the tricuspid valve, due to endocarditis or atheroma, are usually consecutive to mitral or aortic diseases; or to general arterio-sclerosis. Insufficiency and stenosis are the two forms recognized. Tricuspid insufficiency is the most common, and the number of recorded instances must easily run up into the thousands, for in Guy's Hospital alone 405 have been recorded. These figures are in striking contrast to the published cases of tricuspid stenosis, which in 1897, according to Herrick,[2] had only reached 154; so that although tricuspid insufficiency is comparatively rare, it is as common, as compared with stenosis, as the latter is rare.

Tricuspid insufficiency may be *congenital* or *acquired; organic* or *relative*. Ordinarily we see the relative (acquired) form, which is due to stretching of the valves, from dilatation of the right ventricle consecutive to chronic affections of the lungs, or enlargement of the heart, whether from fatty changes or some other form of hypertrophy; perhaps from adherent pericardium. There are also other causes. In one of my cases the tricuspid was apparently dilated from pressure on the right auricle by an aneurism of the ascending arch, and the insufficiency was discovered during life. Tricuspid insufficiency is furthermore a legitimate sequel to organic diseases of the left heart, and it may occur in the last stages of all these diseases. The preponderance of the relative variety over the organic is well shown in the records of the 405 Guy's Hospital cases

[1] Originally published in the *Med. News*, Sept. 6, 1902.
[2] *Boston Med. and Surg. Jour.*, March, 1897, No. 1.

already referred to, where 394, or about 97 per cent., were classed as relative. My tables make it about 90 per cent.

The question of insufficiency, however, has been disputed, even at the post-mortem table, some holding that perfect competency is uncommon, because in applying the water test there will often be a little oozing through the valve. As a matter of fact, such fine distinctions have little practical value, because a slight oozing is not recognizable during life by any ordinary symptoms, and at any rate may be a temporary affair and of slight importance. For this minor degree of insufficiency may be due to faulty technic on the part of the operator, who fails to hold the organ squarely in his hand while applying the test. Practically, unless a good-sized stream of water can be passed through the valve when the organ is held properly in position, it should be regarded as competent. Besides, the ring of the valve is well supplied with smooth muscle fibres, and the somewhat relaxed condition sometimes found at autopsies may be due to general muscular relaxation which has occurred in the last hours of life, or even after death. Organic insufficiency is almost always due to endocarditis, and is a late manifestation of the general implication of the valves, which by preference first attacks the mitral and then the aortic.

Relative insufficiency is simply the result of physiological laws. When from any cause there is obstruction, or stasis, or slowing in the pulmonary veins, due either to chronic pulmonary disease or to disease of the left heart or pericardium, causing engorgement of the lungs with venous blood, the current of blood passing through the pulmonary artery finds an obstacle ahead of it. Consequently the right heart dilates, and then hypertrophies, in order to force the blood into the lungs. This dilatation has the effect of stretching the soft muscular substance of the tricuspid ring, causing insufficiency; and yet this relaxation may, after a time, be overcome by the general hypertrophy. Later, however, when the right ventricle begins to tire and contract irregularly from weariness or degeneration of its fibres, the muscular tissue of the ring will also participate in the degeneration and again become incompetent. This is one of the common incidents of heart failure, in the last stages of valvular disease.

In other enlargements of the heart, as in fat deposition and in chronic fibrous nephritis, a certain amount of enlargement of the elements of the valve may take place and even keep pace with the enlargement of the heart walls; for it is known that in valvular dis-

eases even the leaflets sometimes enlarge, in order to close the orifice which they are intended to protect. If, however, there has been any deposit in the substance of the valve of a fibrous nature, contraction, rather than expansion, must take place.

As soon as tricuspid insufficiency has become established a column of blood is forced through the tricuspid valve into the right auricle which also dilates and later may hypertrophy; but the systolic action of the right ventricle, acting on the blood contained in the right auricle, communicates its impact to the systemic veins, dilating them, provided the dilatation reaches such a degree that the venous valves are no longer competent. And so the blood in the veins pulsates. This phenomenon of *venous pulsation* is always best seen in the veins of the right side, because they are in the direct line of the blood current as propelled through the insufficient tricuspid. As venous pulsation is specially well seen in the jugulars, it is called *jugular pulsation.* For the superior vena cava and the innominate veins have no valves, so that no bar to the backward wave is felt until they have been passed; while at the right sternoclavicular articulation there is a valve in the jugular vein, which resists the wave, and in fact produces a dilatation below the valve, which is called the *jugular bulb,* where the vein is expanded into a rounded body that may be seen or felt. In many instances of tricuspid insufficiency the backward wave does not progress beyond this point; the veins are simply filled. But if the valve gives way, the wave is propagated onward into the external jugulars and subclavians, and may mount into the face.

Distinct pulsation in these cervical veins is, therefore, an important sign; and yet it is not pathognomonic. Further, one must always distinguish between a true venous pulsation and a mere *undulation* of the current in the veins which occurs apart from tricuspid disease—the *false venous pulse.*

True *jugular pulsation* is best determined by pressing down the column of blood in the cervical veins, when the impulse will be found before it, not behind it. Compression of the carotids cannot be accomplished without using considerable force, so that we are not likely to confound one with the other. To bring the bulb of the jugular vein into view, the patient should be made to cough, when it becomes distinct.

A sign that has been given by Pasteur[3] is distention of the veins of the neck with or without pulsation, when the liver is compressed

[3] *Lancet,* 1885, p. 524.

by the two hands. This action slows the blood in the inferior vena cava and causes increased tension in the superior vena cava, and so in the cervical veins. In my cases, however, the venous pulse was not noted as a prominent symptom.

Stasis in the pulmonary veins of the lungs produces cyanosis and dyspnœa; in the systemic veins œdema and albuminuria.

The degree of stretching possible in a case of relative insufficiency was well shown in one of my cases, where the tricuspid admitted seven fingers, while ordinarily it admits only two. In average cases of insufficiency it will admit three to four fingers.

In the comparatively rare form of insufficiency, the organic, the valves and their rings and supporting structures become infiltrated, thickened, distorted, and perhaps contracted; for stenosis presupposes insufficiency. Yet if the stenosis is only in the ring, narrowing may occur without insufficiency, as happened in one of my cases of aortic stenosis. Practically then, in organic tricuspid insufficiency, there may or may not be dilatation of the outlet. Atheroma rarely attacks the tricuspid valve.

One of the first symptoms is *cyanosis*. This is at first slight, but may become extreme. Associated with it is more or less dyspnœa, depending chiefly on the grade of venous stagnation in the lungs. *Oedema of the lungs* is also more or less common, and for a like reason there is œdema of the lower extremities, first seen in the feet and then slowly rising towards the trunk. When the liver and kidneys become indurated we may expect ascites and albuminuria. One of the most prominent and early symptoms is the venous pulse which has been described.

Another sign is *epigastric pulsation*, but it is appreciable only when the cardiac systole is forcible. The radial pulse is usually small, soft and irregular, and is apt to be frequent. A *thrill* is of rare occurrence. When it is felt it is probably due to an associated mitral or aortic disease.

The most *characteristic sign* of tricuspid insufficiency is the systolic murmur over the tricuspid area. It should be heard with greatest intensity over the lower half of the sternum, at the root of or over the ensiform cartilage (the nearest point to the tricuspid), to the right of the median line; occasionally as far as the right axilla or to the left of the sternum at the left fifth costosternal junction (Cabot) or just above it. It should not, however, be propagated any considerable distance to the left. The murmur is usually soft and faint. Exceptionally it is rough and harsh.

The second pulmonary sound, so far as has been noted, is weak, because a comparatively small amount of blood is propelled into the pulmonary artery. Doubtless in a very large number of these cases there has at some time been an accentuation of the second sound, owing to the accompanying mitral disease; but on the failure of the tricuspid this symptom disappears, and the weakness of the second pulmonary sound is therefore, at this period, an important sign as indicating an advance in the general valvular disease. This symptom, however, is one to which but little attention has been paid.

Undoubtedly tricuspid insufficiency produces at first dilatation and then hypertrophy of the right auricle, and this is noticeable in some cases, chiefly at the level of the right auricle. The heart in its totality is not enlarged, however, when the insufficiency has been of short duration.

In the heart failure which is so frequent an event in tricuspid insufficiency one often sees the facial discoloration, the wild expression, the projecting eyes. The patient leans forward, as this position is the most favorable for respiration, and gasps for breath, owing to the stagnation of blood in the lungs. The veins stand out in the neck. Often he can hardly speak, and only in monosyllables. The radial pulse is weak and intermittent. The heart s action is tumultuous.

Yet tricuspid insufficiency is seldom recognized during life, even under favorable circumstances. In 29 cases proved by autopsies at the Massachusetts General Hospital but five, or 17 per cent., were recognized during life. In ten cases from my post-mortem records the diagnosis was made in three, or 30 per cent. But by some the diagnosis is regarded as a refinement of comparatively little importance, in the presence of other conditions more apparent, and even more immediately serious to the individual.

The most distinctive sign, as I have said, is a systolic murmur over the lower half of the sternum and ensiform cartilage, the most common center of the greatest intensity being (according to my returns) the fourth left intercostal space close to the sternal margin. At times the greatest intensity is as low as the fifth left intercostal space, close to the sternum, and the corresponding area on the right side; sometimes as much as an inch to the right of these points, so that on the whole this area is somewhat broader on the right side than on the left. Occasionally the murmur may be carried to the right axilla, but it is seldom carried much to the left of the sternum. As the tricuspid, however, is a large and rather

indeterminate areas as compared with the mitral, which overlaps it, it is essential for purposes of diagnosis that the two sounds or murmurs in these two areas should be appreciably different in character as to pitch, quality, duration or intensity; and the propagation of the murmurs should be in different directions.

Epigastric pulsation is the next important sign. It does not occur in simple mitral or aortic disease; but we should remember that tricuspid disease may be associated with organic mitral disease in from 20 to 25 per cent.; and with organic aortic disease in from 15 to 17 per cent. It may, however, be absent.

Faintness of the second pulmonary sound is also important, especially if previously there has been accentuation of it.

The *jugular pulse,* once thought to be pathognomonic, is not so regarded now. It is seen in other conditions. It occurred in one of my cases of aortic insufficiency, without tricuspid disease. Simple *fulness of the jugulars* is, however, a noteworthy sign. Increase in the transverse dulness of the heart is not a reliable sign. It may occur or may not. It may not exist in wasting pulmonary diseases.

The tricuspid murmur may be also mistaken for the murmur of aortic stenosis; or the latter may mask the former, when it is propagated backwards, as it sometimes is, towards the apex. However, the diagnosis of aortic stenosis should be established by the propagation of the murmur up the great vessels. Simple thickening of the tricuspid without roughness, like simple oozing, gives no sign.

In general the prognosis is bad. There are cases, however, in which heart stimulants will sustain the flagging heart and carry the patient into a region of comparative safety. In fact, successive attacks may be mastered in this way, but the danger is always near at hand. Still the prognosis is not so bad in the relative variety as in the organic, for the latter presupposes implication of other valves. When there is albuminuria and scanty urine the prognosis is worse.

Case XXVI. Mitral Stenosis: Pulmonary, Aortic and Tricuspid Insufficiency.—A., twenty-six years old, single, was admitted to hospital September 18, 1880. Twelve years before admission the patient had a severe attack of rheumatism, and for about a year had suffered from more or less of palpitation and dyspnœa. On physical examination marked pulsation was noted in the epigastrium. Murmur with first sound at the apex, not heard behind or in the axilla. Want of synchronism between the two radials. Hypertrophy of heart. Cyanosis, nausea and scanty urine. The dyspnœa contin-

ued, and with it marked pulsation in the vessels of the neck. About five weeks after admission a "purring thrill" was noted in the fifth intercostal space, one and a half inches below the nipple, with some recession of the soft parts, more distinct during inspiration. Patient developed orthopnœa and died in a uræmic convulsion.

The autopsy showed general anasarca. Heart weighed 16 oz. Mitral calcareous, only admitting index finger. Aortic slightly insufficient. Pulmonary insufficient. Tricuspid admits tips of four fingers. Right ventricle hypertrophied. Walls of left ventricle thinned. Fibrous phthisis. Oedema of the lungs. Nutmeg liver. In this case the "purring thrill" of mitral stenosis had been noted, as this was the predominating lesion.

Tricuspid obstruction or stenosis, for they both appear to be synonymous, is one of the most uncommon forms of valvular disease. In 1881 Fenwick[4] had been able to collect only 40 cases; Pitt, of London, in *Albutt's System* (Vol. VII, p. 25), only 87 cases during a period of 26 years, and Herrick, of Chicago,[5] 154 cases up to 1897. Since that date, however, some additional cases have been published, bringing the total, according to my reckoning, up to 162. These figures include two by Whyte;[6] one by Devic and Teyssier;[7] three from the records of the Path. Soc. of Phil., 1898, Vol. XVIII, pp. 132, 181 and 196, by Packard, Steele and McCarthy; one by Chadbourne,[8] and one by Sir George Duffey.[9] Total addition, 8; grand total, 162.

Tricuspid obstruction must not, however, be considered as an independent disease. It is almost of necessity associated with tricuspid insufficiency, just as any valvular stenosis implies insufficiency. In fact, so far as I know, there has never been a case of stenosis without insufficiency, unless it is one reported by Duroziez, the details of which I have not been able to obtain. And in only two cases (the one above alluded to and that of Devic and Teyssier) was there no accompanying mitral stenosis. Practically, therefore, we may say that mitral stenosis is a constant attendant on tricuspid stenosis, and as the mitral disease is the older, the tricuspid may be regarded as a complication of the former. It is a very serious one.

The former idea that tricuspid stenosis was a congenital condi-

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[4] *Path. Soc. of London*, 1881, p. 46.
[5] *Boston Med. and Surg. Jour.*, March, 1897, Vol. I.
[6] *Scott. Med. Jour.*, 1899, p. 18.
[7] *Prov. Med.* Lyons, 1900, XV., p. 613.
[8] *Am. Jour. of the Med. Sciences*, 1900, p. 306.
[9] *Dublin Jour.*, 1901, p. 241.

tion has been largely abandoned, certainly in post-fœtal cases, because the evidences at post-mortem of its being last in a chain of circumstances are pretty positive, while the date of the first of the events (which, so far as the valves are concerned, is mitral disease) can be proved with considerable accuracy. The exceptions to this rule are so rare that practically they can be excluded.

There is, of course, such a thing as *congenital stenosis,* which occurs usually, if not always, in association with other congenital malformations of the heart. In these cases, however, life is rarely much prolonged after birth, so that, as in aortic stenosis, these exceptional forms are almost never seen.

A feature of great diagnostic importance in tricuspid stenosis is that the great majority of the sufferers are females. In Leudet's 101 cases published in his *Thèse de Paris,* 1888[10] (as quoted by Whittaker), 80 were women, so that the proportion of women to men was about four to one. In Fenwick's 46 cases, of which 41 were women, the ratio of women to men was about nine to one. In 12 cases that I have collected 10 were women, or five to 1. The preponderance in favor of women, therefore, is well shown, though the reason is not clear; but whatever arguments tend to show that mitral disease is by preference a feminine lesion should apply here.

Fenwick found that the average age at death was between thirty-one and thirty-six. In my collected cases the average fell in the thirties. Very few survive the forties, and a less number the fifties.

It is generally accepted that rheumatism is the most frequent causative factor. Fenwick found it in 50 per cent.; my figures, in 10 cases, accord with this ratio. In half of them we have to look for the cause in influences that govern mitral diseases.

The heart is always moderately enlarged, but there does not appear to be much enlargement of the right ventricle; indeed, it has sometimes been described as small. The right auricle is usually dilated and hypertrophied, its walls showing manifest thickening. In tricuspid stenosis, coming as it does as the last link in the chain of valve events, the marked organic changes seen in mitral stenosis, are not apt to be met with. We usually find merely an agglutination of the segments, causing a funnel-shaped opening. Ordinarily it should admit two or three fingers, but in stenosis it may admit only one, or the tip of a finger. The substance of the heart must by this time have degenerated also, and we find induration of the lungs, spleen and kidneys, with the nutmeg liver.

[10] Of the 12 cases of pure tricuspid stenosis 11 were reported by Leudet.

There are *symptoms* in abundance, but they are rarely sufficiently distinctive to warrant a diagnosis.

This disease, like all other valvular diseases, advances insidiously.

The patient will complain of palpitation. In one-half of the cases there is cyanosis (Pitt), and it is apt to be extreme, while either a fulness or pulsation in the jugular veins may be observable. With it there will necessarily be dyspnœa, and perhaps orthopnœa, each of them severe. In 75 per cent. of the cases, according to Colbeck, there is albuminuria or dropsy. On palpitation there may be a presystolic thrill, and yet the thrill can be due to a mitral stenosis. In the epigastrium there will be pulsation, at times, from an enlarged liver (50 per cent., says Colbeck), and the force of the heart's impulse will be marked. The presystolic or diastolic murmur in the tricuspid area (which, according to English ideas, means "over the fourth and fifth spaces to the left of the sternum") should be heard and was heard in five out of seven of Colbeck's cases, though at irregular intervals. Some locate the murmur over the fifth or sixth right cartilage or over the ensiform; others, again, over the lower half of the sternum and as much as an inch to the left of it; still others, at the root of the ensiform cartilage or to the right of it. That there is some kind of a bruit was noted in the tricuspid area in 20 only of Fenwick's 32 cases. It should differ in pitch, quality and duration and perhaps intensity, from the associated mitral murmur, and this was noted in Shattuck's case where the diagnosis was made *intra vitam*.[11] In about one-quarter of the cases no bruit has been detected. In about one-quarter of the cases, also, there has been aortic stenosis. Mental confusion or hebetude have been noticed.

Thus far the *diagnosis* appears to have been made only in six instances (those of Colbeck and Shattuck) out of the 162 which I have alluded to. Of the greater number it may be said, in the first place, that many were not examined thoroughly, because there was no opportunity to do so; and second, in the face of the manifest mitral, and perhaps aortic, lesions, no special interest was taken in those of the tricuspid. Owing, also, to the inconstant character of the murmur and usually its entire absence, it is not so strange that the diagnosis has been seldom made. Indeed, for all time this lesion will be apt to escape notice, if we are to depend on specific auscultatory symptoms.

More constant than these are: (1) A coexisting mitral stenosis; (2) an enlarged and dilated right auricle; (3) palpitation, cyanosis,

[11] *Boston Med. and Surg. Jour.*, Vol. 124, 1891.

dyspnœa and œdema; (4) the epigastric pulse, and (5) a previous history of rheumatism.

If, then, a woman under forty has these symptoms and in the tricuspid area a diastolic murmur, the pitch, quality or other characters of which distinguish it from the mitral murmur, it will be safe to make the diagnosis of tricuspid stenosis.

The *prognosis* is worse than in any other form of valvular disease.

In congenital cases patients seldom live more than a few days. In acquired cases much depends on the condition of life. A tricuspid lesion that would prove rapidly fatal in a working man may be maintained a fairly long time by a person in easy circumstances. Pregnancy is a dangerous epoch for women.

In general, the louder the murmur the less the danger. With considerable stenosis there is apt to be a mild bruit. Though patients rarely attain the age of forty years, in the case that follows seventy was reached:

Case XXVII. Mitral and Tricuspid Obstruction with Insufficiency: Pulmonary Embolism: Pleurisy, with Effusion.—L., seventy years old, was admitted to hospital January 3, 1881. On admission the patient stated that up to a week before admission he regarded himself as well. Then he suddenly developed dyspnœa. Slight cough and œdema of legs.

On physical examination an occasional squeak was heard in the right parasternal line in the third interspace. Patient's sputum streaked with blood. Jugulars enlarged, almost pulsating. Heart sounds at first indistinct. Cardiac dilatation. Impulse diffuse. Systolic murmur loudest over ensiform cartilage. Tubular breathing. Pleuritic effusion. Retraction of soft parts at third left interspace with inspiration.

At the autopsy the right pleural cavity was found full of serum and the left nearly full. Infarctions of lung. Heart dilated and weiging 23 oz. Aortic and pulmonary normal. Right cusp of mitral thickened and bound down, orifice admitting seven fingers. Valves of tricuspid thickened and restricted (stenosis).

Chapter IX.

MYOCARDIAL AFFECTIONS.

Bertin, in his little book on Heart Diseases, published in 1821, made this quaint remark: "General inflammation of the substance of the heart is one of the maladies about which there is the most melancholy obscurity."

Bertin, however, did not distinguish between inflammatory and other diseases of the heart walls, for at that early period such distinctions were impossible. His successors, even down to the present day, have not wholly relieved us of this obscurity, largely because conflicting theories have been advocated as to the nature of these diseases. For example, Rokitansky[1] and others put the seat of parietal diseases in the muscle tissue, Bristowe[2] in the interstitial, while Huchard[3] as late as 1893 claimed that the cardiac vessels were to blame. Besides, the relations between muscle degenerations or metamorphoses and inflammations have always been hazy, while the clinical phenomena at our disposal have never been equal in amount or quality to the pathological data. Again, important discoveries have been overlooked. Hayem, as early as 1869, called attention to the fatty changes in the heart muscle of typhoid.[4] Germain Sée[5] in 1883 confirmed these views, while Romberg[6] in 1891, extending his investigations into the condition of the heart muscle in scarlatina and diphtheria, found a granular and fatty change, together with a small-celled infiltration of the connective tissue and multiplication of muscle nuclei. Hayem's work has lately been confirmed by Dehio[7] and Bollinger.[8]

Now that the discoveries of Hayem and Romberg have been recognized, the whole subject of fatty degeneration of the heart is comprehensible, and can be placed on a sound basis—the pathological.

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[1] Rokitansky, *Path. Anat.*, London, Vol. IV., p. 191.
[2] Bristowe, *Pract. of Med.*, London, 1876, p. 516.
[3] Huchard, *Maladies du Coeur*, Paris, 1893, p. 196.
[4] Hayem, *Arch. de Phys. Norm. et Path.*, 1869, II. p. 699.
[5] Germain Sée, *Maladies du Coeur*, Paris, 1883, p. 199.
[6] Romberg, *Deutsch. Arch. f. Klin. Med.*, Bd. 48 & 49, 1891-2.
[7] Dehio, *Deutsch. Arch. f. Klin. Med.*, 1898, I, s. 1, 62.
[8] Bollinger, *Path. Anat.*, I., p 74, New York, 1898.

But there are other matters which are still obscure, and I know nothing more confusing in cardiac pathology than the use made in text books and journal articles of the term *"myocarditis."* For no sufficient reason, I feel sure, Sobernheim[9] in 1837 introduced it, intending that it should be used to include all diseases of the heart walls, in lieu of Corvisart's[10] *"carditis,"* proposed about twenty years earlier.

Corvisart was an exceedingly acute physician, indeed, a man who made very important contributions to cardiac pathology, and there was no good reason for allowing his word "carditis," which included diseases of the heart muscle, interstitial tissue and vessels, to be supplanted by "myocarditis," which, while it was intended to cover all these affections, actually referred only to muscle inflammation. Clinically, also, it has led to misunderstandings, because with some it has meant simply fatty degeneration of the heart; with others disease of the coronary arteries.

Of course, it is not to be denied that inflammation of the heart muscles plays an important rôle in parietal changes, and Rokitansky's contention was correct, in so far as he maintained that there was a myocarditis, or muscle inflammation; but he was wrong in holding that it had a preponderating influence. Bristowe's and Huchard's theories are equally correct, but neither one of these authorities appears to have realized that most of the changes in the myocardium are not inflammatory at all; some being physiological, like the hypertrophy of the heart in pregnancy, or the changes of senility; while others are nutritive, that is, dependent on disordered nutrition or innervation, and so forth.

When pathologists differed as to fundamentals, it was quite natural that clinicians should abandon the pathological classification and adopt a symptomatic one. It is in this way that Ebstein's[11] *insufficientia myocardii, debilitas cordis and myasthenia cordis,* and the *"weakened heart"* came into use. But while these terms attract us by their simplicity, and relieve us from the necessity of espousing this or that pathological theory, they are essentially unsatisfactory, because they leave us in doubt as to what the matter really is. They are also unnecessary, if it can be shown, as I believe is quite possible, that a pathological classification is practicable. And if this is possible, it is the proper one, as all should admit. Abandoning, then, the words *"myocarditis," "carditis"* or *"pancarditis,"* which latter

[9] Sobernheim, *Diagnostik d. i. Krankheiten*, Berlin, 1837, s. 118.
[10] Corvisart, *Les Maladies du Cœur*, Paris, 1818, p. 64.
[11] Ebstein & Schwalbe's Handbuch, Vol. I, s. 729.

has been suggested, I purpose to show that the conditions mentioned are to be placed under the simple heading "Myocardial Affections," of which there are the following varieties, viz.:

1. Fatty changes ..... { *a*. Degenerations. *b*. Depositions.
2. Simple hypertrophies as caused by
   - *a*. Severe exercise ..... Voluntary.
   - *b*. Pregnancy. } Involuntary.
   - c. Vascular obstruction, including aneurisms and atheroma. } Involuntary.
   - *d*. Unusually small vessels..... { Congenital. Acquired. } Involuntary.
   - *e*. The emotional or neurotic heart. } Involuntary.
   - *f*. The heart in mechanical injury. } Involuntary.
3. Simple dilatations as caused by
   - *a*. Infections.*
   - *b*. Anæmia, etc.
4. Atrophies of the heart.

* Tubercles are occasionally seen in the heart substance.

HYPERTROPHIES OF THE HEART.

The normal adult male heart averages from 10 to 12 ounces; the adult female heart from 8 to 10; the size of the organ in health varying somewhat with the dimensions and development of the body, and the work that it is called upon to do.

Whether in animals or men, however, if the organ is called on for extra work, it will enlarge in order to accomplish it. Familiar examples reminding us of this truism are well known. A horse or dog that is continually forced to work at a high rate of speed will sooner or later develop hypertrophy of the heart, and may die of it suddenly, at some time or other. Such instances are probably known to all of us. Again, among the athletic youth of the present day, it is a regular event for those who train hard, to have some hypertrophy of the heart walls; so that it is to be expected in professional boating or baseball men, and even in tennis experts. I have recently had under my care a case of hypertrophy, due, undoubtedly, to the strain of tennis tournaments. We also have all the evidence that is required, to show that professional mountain climbers, and miners who have to climb long and steep ladders will very frequently contract hypertrophy of the heart; though, doubtless, in these particular instances there are other contributing factors, the mountain climbers being compelled to breathe a highly rarified air and the miners one vitiated by coal-dust or smoke. So, in many other instances, two or more causes of hypertrophy coexist.

At first, the normal heart dilates under the strain, but it contracts

again when the strain is over, and is usually no worse for it. If the strain is prolonged, however, the heart contracts less readily, and if the strain becomes continuous, the walls of the heart gradually thicken by the enlargement of the muscle bundles—perhaps by an increase in the muscle cells—until the organ is so re-enforced by additional muscle-tissue that it is competent to do the work it has before it. This new development of muscular tissue in the heart, fitting it for larger work, superinduces a condition of so-called "compensation." But the demands made upon the heart, whether in the normal or compensated condition, may be too great, and some fibres will overstretch or even break. Hence *"heart strain."*

The strain may occur in the substance of the walls, in the papillary muscles, or in the chordæ tendinæ, the heart dilating suddenly and having no power to contract, and the patient dying then and there with cardiac paralysis. Such an accident occurred in Case XXVIII. Or, the patient may have a season of arrythymia and then recover wholly or in part, in which case he will have a permanently strained heart, "the *irritable heart*" which Da Costa[12] saw so frequently among soldiers.

In the pregnant woman there is a *physiological hypertrophy* of the heart that develops *pari passu,* I believe (though it has been denied), with the increase of the blood required for the nourishment of the uterus, its contents and the breasts; but as soon as parturition has been accomplished, there begins at once a gradual diminution in the size of the heart, to correspond with the diminished volume of blood needed by the parturient woman.[13] Reasoning by analogy, therefore, we have a right to assume that as the heart develops in pregnancy and then retrogrades, a similar process may take place in the athletic heart; and this is probably true, the enlarged hearts that remain, as permanent fixtures, in these men, being instances of strain from which they have not altogether recovered.

Another physiological increase in the heart may presumably occur in gluttons or drinkers who habitually consume more than is required by the wants of nature, so that they create a surplus of blood in the system, requiring an enlargement of the heart to propel the increased volume of blood.

There are many varieties of *pathological hypertrophy,* using these terms to mean hypertrophy following lesions of the organ.

I have in my records many examples of hypertrophied hearts,

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[12] Da Costa, *Medical Diagnosis,* Phila., 1895, p. 457.

[13] An enlargement of the heart due to renal disease in pregnancy has also been described.

the majority of them hypertrophies of the left ventricle, due to defects in the aortic or mitral valves. It is quite common in these cases for the heart to weigh 15 to 20 ounces; and not very uncommon to find it 20 or 30 ounces; while it may have a much greater weight.

Two of these cases were due to arterio-sclerosis, and are worthy of brief notice.

Case XXVIII. Hypertrophy of the Heart. Arterio-sclerosis.—One of these patients, a printer, 44 years of age, entered the hospital with extreme dyspnœa and palpitation. Aortic and mitral organic murmurs were noted, with pulsation of jugulars. Pulse varied from 36 to 84. He died after being under observation about three months, having developed general anasarca. At the autopsy large atheromatous plates were found in the aorta, and the mitral was diseased. The heart was enormously enlarged, chiefly in the left ventricle, and the hypertrophy (the heart weighed 32 ounces) was ascribed to general arterio-sclerosis. Rupture of chordæ tendinæ was the immediate cause of death.

Case XXIX. Cor. Bovinum; Arterio-sclerosis.—In another case a laborer, 67 years of age, of full habit and robust, but syphilitic, was admitted to the hospital January 21, 1887, suffering from chronic diffuse nephritis and uræmia. He lived but a few days. At the post-mortem examination many signs of constitutional syphilis were noted, such as syphilitic meningitis and cirrhosis, in addition to the evidences of skin syphilis. On the heart were several milk patches, but the valves were free and sufficient, though slightly marked by fatty changes. The hypertrophy was thought to be due to the arterio-sclerosis. The heart weighed 58 ounces, free of its membranes. This *cor bovinum* is the largest I have known, and one ounce heavier than the famous heart, weighing 57 ounces, that was placed in the Museum of the College of Physicians and Surgeons of this city by the late Prof. Alonzo Clarke.

There is also a form of large heart seen in hysteria and various nervous affections, so that it might be called the *neurotic heart*. It is apt to be complicated with valvular difficulties, but occasionally we find one in which no valve lesions are recognizable.

I have seen such a case in Graves's disease when there was no valvular disease, but a moderate swelling of the thyroid associated with tremor, paroxysmal tachycardia, acute dyspnœa, anginoid attacks, epigastric pulsation, and obesity. The heart was very large, and the apex beat was 5¾ inches from the median line to the left.

(Case LXXV.) This form of hypertrophy was probably complicated with fat deposition.

The tobacco heart is also apt to be enlarged. Enlargements of the right ventricle are less common than those of the left, but they will occur, whenever there is obstruction to the pulmonary circulation. At various times a form of enlarged heart due to a *congenitally small aorta*, or *small arteries*, has been described. Morgagni[14] wrote of it, and several writers have since then called attention to it; I fancy it is of rare occurrence. However, Fräntzel[15] has given the record of a case associated with congenital stenosis of the aorta. Perhaps these cases are merely anatomical curiosities.

Sometimes when there is a general hypertrophy, the hypertrophy of the right ventricle will be most marked. This occurred in a case which I saw with Dr. W. N. Hubbard of this city.

Case XXX. Hypertrophy and Dilatation of Heart; Scoliosis.—The patient, an unmarried woman of middle age, with scoliosis of marked type was taken down with an acute attack of nephritis, in association with chronic endocarditis and anasarca. At the post-mortem examination the heart was found to weigh 14½ ounces. It was hypertrophied and dilated and the right ventricle especially was thickened. Mitral and aortic valves were slightly affected. Hydropericardium and hydrothorax. Nutmeg liver. In this case the special thickening of the right ventricle, when one should expect the left to be most thickened, was probably due to the obstruction of the pulmonary circulation, due to the deformed and contracted thorax.

It is usually easy to determine if a heart is enlarged. Bulging of the pericardium, with or without pericardial adhesions, is common in large hearts. On palpation, the impulse at the apex is diffuse and heaving; it may be in the 5th, 6th, 7th or 8th interspace; in the line of the nipple, or up to three inches beyond it. In medium grades it is in the 6th space, and line of the nipple. Percussion reveals an increased area of dulness. Beginning in the 2d interspace or over the 3d rib, it may extend from one-half inch to three inches beyond the left mammillary line, and perhaps an inch and a half beyond the right border of the sternum. This dull area is more ovoid than in health. There should be little difficulty in determining by percussion whether the heart is enlarged, though it is impossible to distinguish between simple hypertrophy and simple dilatation

[14] Morgagni, *De Sedibus et Causis. Epist.*, XVIII., Art. 2 et 4.
[15] Fræntzel, *Krankheiten des Herzen*, Berlin, 1888, s. 151.

by percussion alone. If such a differential diagnosis is necessary, rational signs must be utilized to settle the question.

In making a diagnosis we must carefully differentiate from the heaving impulse of palpitation. Then the increased area of dulness may, it must be remembered, occur in pericarditis with effusion, aneurism, mediastinal tumors, and localized pleurisy. In this regard, assistance will be furnished by calling to mind that there are *three stages* of hypertrophy. In the *first* there is the period of dilating compensation, where there is an irregular heart action, increase in strength of pulse, and a tendency to accentuation of the second pulmonary sound. When the hypertrophy has reached the stage of full compensation, normal action in heart and pulse has been established (*second stage*). In failing compensation (the *third stage*) we have increased *dilatation,* as shown by a feeble impulse at the apex, soft and irregular pulse, and cardiac distress.

To a certain extent, hypertrophy is a benign process, designed by nature to relieve another abnormal condition; and so long as the bodily health is maintained, and the work of the heart is not excessive, but proportioned to the strength of the individual, there is no need for alarm. But if the primary disease, be it valvular, pericardial or vascular, so increases, that the heart is strained in maintaining its equilibrium; or the patient has insufficient nourishment, or is overworked, the heart will dilate (failure of compensation) and collapse will ensue.

In directing the proper course of treatment these facts must always be borne in mind.

At this point it may be well to make a brief statement about the vexed subject of dilatation and hypertrophy, and their relation to one another. The facts, I take it, are these: There is a close relation between the conditions, and they often coexist (*eccentric hypertrophy*), but not always. In hypertrophy there may or may not be dilatation. For example, in the early stages of arteriosclerosis, where there is no increase of the blood, but more force must be applied, there will be hypertrophy without dilatation (*concentric hypertrophy*). In the enlarged heart of the beer drinker, who has created new blood, there is dilatation of the heart chambers, to accommodate the increased amount of blood to be driven and the heart hypertrophies in order that it may be able to drive it. In dilatation, too, there may or may not be hypertrophy. For example, in the breaking down of compensation in the hypertrophic heart there will be dilatation, but in the dilated heart of accidental strain

to a healthy heart there will be no hypertrophy. A similar dilatation without hypertrophy will occur in the stage of softening of the heart which attends and follows the toxæmias, such as typhoid, etc. Acute dilatation often occurs probably after prolonged strain. as in bicycle riding, running, etc.

At post-mortem examinations this condition may be deceptive, and the apparent encroachment on the chambers in concentric hypertrophy may be due to the fact that the patient died while the heart was in extreme systole. So, on the other hand, appearances may be equally deceptive as to dilatation of the heart, which may be because the patient died when the cavities were dilated in diastole, or because of changes in the substance of the cardiac muscles, due to post-mortem relaxation. Owing to these circumstances, the relation of dilatation to hypertrophy is often a difficult matter to decide, even at a post-mortem examination.

Atrophy of the heart is less common than hypertrophy. It may be a family peculiarity, or it may be due to arrest of development, or it may be acquired; or the result of the physiological changes of senility.

Church has recorded the case of an adult, aged 47, where the heart weighed only 3 oz., 1 dr., while Bramwell saw one weighing only 2 oz., 12 dr., 11 grains in a woman, the mother of several children (*Bramwell's Dis. of the Heart,* N. Y., 1884, p. 631). There may be total or partial atrophy. When people die of starvation or of extreme old age the heart is apt to be atrophied, but atrophy may result from arteriosclerosis, phthisis, cancer, diabetes, or in fact, any wasting disease. In many of these cases, especially in death from starvation or wasting diseases, the heart is apt to have a brown color. (*Brown atrophy.*) There is also a special variety of the small heart described by Virchow as associated with the small vessels of anæmia. He called it *hypoplasia cordis.* But a heart may be atrophied as to its muscle elements, and still be large, for the affection may be associated with a fat deposit. In some instances of atrophy of the heart the organ is so shrunken that the tortuous vessels stand out in relief on the surface, while the whole surface is thrown into folds. This variety has been called the *"withered apple heart."* Bramwell has a good plate of it. On microscopic examination the muscle elements are found to be shrivelled, and yellow granules or pigment are disposed around the nuclei and drawn out in a line with the fibres. While there are the usual signs of cardiac disease in the atrophied heart, there are none which

are distinctive, except that the heart may sometimes have a smaller area of dulness than usual, with the impulse nearer the median line and higher up than normal. The other symptoms common to atrophy and other forms of cardiac disease are turns of fainting, spots before the eyes, singing in the ears, irregular pulse, palpitation, precardial distress, confusion of mind. The following case is an example taken from my records:

Case XXXI. Atrophy of the Heart: Carcinoma.—A working man of 59, anæmic and cachectic in appearance, entered the hospital in 1884, and after a stay of about nine months, died of progressive emaciation (and in part starvation), due to primary carcinoma of the omentum, and secondary implication of the mesenteric glands, liver and kidneys. The heart was found small, soft and fatty. It weighed only 8 oz.

Case XXXII.—Another case on my records occurred in the practice of Dr. R. E. Van Giesen of Greenpoint, and illustrates the atrophy that sometimes is found in sarcoma. The pleura and mediastinum were involved in a sarcomatous growth. The pericardium varied from ½ inch to 1¼ inches in thickness, and the inner surface was hairy. The heart weighed not more than 5 oz. The patient was 22 years of age and well developed, and the body was well nourished.

Arteriosclerosis is well seen in the gouty kidney and in the physiological changes incidental to old age. There is a degeneration of the cell elements of the capillaries and smaller vessels, the protoplasm undergoing what is known as hyaline, then fatty, and later, atheromatous change. But as I have intimated, the hard pulse of old age may be regarded as physiological. The causes, as far as we know, are syphilis, alcoholism, Bright's disease, hard work, diabetes, gout, lead poisoning and emotional conditions. The changes produced in the walls of the vessels are caused by the toxins carried by the blood vessels. Nature, however, comes to the assistance of the individual whose vessels are so thickened by disease, and the left heart hypertrophies in order to compensate for the increased work it has to do in driving the requisite amount of blood through the thickened and tortuous vessels. The old theory that the arteries are thickened by the blood forced against the walls of the vessels is now maintained by few pathologists.

When arteriosclerosis attacks the coronary vessels of the heart we encounter a special phase of the malady. The coronary arteries are terminal, like those of the brain and kidneys; that is, they do not

anastomose with their fellows, so that any occlusion of a coronary artery, whatever its degree, diminishes proportionately the vascular supply within the area of its distribution, while the nutrition of the heart wall is correspondingly affected.

When such an area has been deprived of its blood the part undergoes what has been called *"anæmic necrosis,"* a condition that was formerly known as *myomalacia cordis.* This starved area has been called a *white infarct.* Whatever the result of the process, the heart wall is left, of course, unsound. The result of a healed white infarct is a fibroid area, due to the deposit of fibrin as a substitute for the dead tissue. Such a heart would also be called sclerotic.

Sclerosis may lead to cardiac aneurism, the sclerotic or fibroid tissue yielding under the contractions of the organ so that the heart walls bag out.

It is claimed that *embolism,* or even *thrombosis,* in a coronary artery will produce *sudden death.* I have never seen such an instance, however; and it would be very difficult, I fancy, if not impossible, to prove it.

Abscesses of the heart occur, but are extremely rare.

In *misplaced hearts,* the statements of Bouvier,[16] Adams[17] and Bradford[18] that heart affections are superinduced by deformities of the vertebræ have found support in the experiments of Neidert,[19] who, in 31 cases of Pott's disease and lateral curvature, found that most of the bad cases died early of heart failure. Spinal deformities cause mechanical embarrassment to the circulation, leading at first to hypertrophy, possibly to distortion of the valves, and eventually to heart failure.[20]

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[16] Bouvier, *Leçons Cliniques,* Paris, 1858, p. 145.
[17] Adams, *Curvature of the Spine,* London, 1882.
[18] Bradford, *Orthopædic Surgery,* 1890, p. 14..
[19] Neidert, *Inaug. Diss.,* Munich, 1883, V. & H.., Jahrb, 1886, s. 371.
[20] Displacements of the Heart in Lateral Curvature, *N. Y. Med. Jour.,* Sept. 30, 1899.

Chapter X.

THE FAT HEART.

Corpulence and *the fat heart* are so closely related that a consideration of one involves the other. Chambers found that in thirty-six corpulent people twelve had the fat heart, while Quain's observations were that patients with fat hearts were invariably corpulent. We may infer, therefore, that corpulence disposes to fat heart. Probably the one is essential to the other. The term fat heart means merely that the heart is burdened by an excessive deposit of fat; not that it has undergone fatty degeneration. The fat heart is said to be in a condition of *infiltration,* pathologically speaking. Fatty degeneration of the heart is a more dangerous affection, but it may be a sequel to fat infiltration. If they coexist, which sometimes happens, the prognosis is vastly worse.

Corpulence or obesity consists in an excessive deposit of adipose tissue in parts of the body which are comparatively free from fat in health. It is caused essentially by nutritive disturbances; or to put it in another way, it is the result of a loss of the equilibrium between assimilation and disassimilation.

Obesity is a serious matter in many ways. First of all, it is a positive discomfort to the patient, for locomotion is made difficult, digestion is disturbed, and the faculties are often dulled. Obese people, too, have a constant tendency to constitutional disorders, such as gout, rheumatism and diabetes. There is also the ever-present danger of some serious illness, accident, or surgical operation, which they may not be able to survive; as corpulent people are deficient in vital power. Fat infants and children, in my experience, seldom reach adult life, while in advancing years the obese may be totally unable to go about. Miles Darden, whose height was seven feet six inches, and who weighed over a thousand pounds, had to be transported in a wagon during the last four years of his life. Corpulence may be a disturbing factor in social life. It is said that in Albania, corpulence, in the male, is a proper ground for divorce.

Excessive weight has been treated successfully from very early times, the Greeks employing trained men to reduce their athletes, but corpulence, as a disease, was not plainly shown until the present century, when English physicians, such as Wadd (in 1825), Cham-

bers (in 1850), F. Harvey (in 1864), and Quain (in 1880-1885), wrote up the subject. Harvey was the physician of Banting, and his method, known as the Banting system, became widely known and was extensively practiced.

Adipose tissue is chiefly stored in the subcutaneous connective tissue beneath the serous membranes or in the inter-muscular septa. The largest deposits are beneath the skin of the abdomen, in the mesentery, in the buttocks and thighs, and in the back of the neck. In women the excess of fat is usually in the thighs and buttocks. A certain amount of fat is normally contained in the connective tissue corpuscles, in the form of minute specks. According to Michael Foster, these specks coalesce into droplets, these again into drops, until, as the protoplasm of the corpuscles diminishes and the oil globules unite, the original connective tissue corpuscle is converted into a fat cell. The remnant of the protoplasm is then gathered about the nucleus. To a moderate extent, fat tissue is natural, as it is a normal constituent of the system; and within physiological lines ordinary fat tissue may be increased so as to subserve a useful purpose, constituting a reserve store upon which the body may draw for nutriment in periods of prolonged vital strain.

But obesity has a progressive tendency, for as the bodily weight increases, and with it the dyspnœa and palpitation which necessarily follow exertion, there is a further hindrance to the oxidation of the blood, which is still more increased, when the heart becomes involved in the fatty process. Corpulence also begets plethora, and it in turn hemorrhoids, varicose veins, hæmorrhages, vertigo, headache, disturbances of sight and hearing, dulness of the intellect and dyspepsia; all of which may be attributable to passive congestions. It must not be supposed, however, that all corpulent people are so affected. Sam Johnson, the author of *Rasselas*, and David Hume, the historian, were corpulent, but led, for the most part, intensely active, intellectual lives, notwithstanding this malady.

Obesity may occur at all ages. I have seen it in infants under one year. In a number of cases I have ascribed it to a surfeit from artificial feeding with food containing too much cream, or a superabundance of starchy material. In two instances (see Cases XXXIII and XXXIV) the deficient oxidation of the tissues is shown by diminution in their normal percentage of urea. Both of these patients were lithæmic, as the diminution in the excretions of urea would indicate. Lack of active exercise, too much sleep, and a secluded life also tend to corpulence. The obesity of some women of the

East is explained by Charles Robin on the ground that they take little exercise, eat all day long, and sleep a great deal. Obesity is also hereditary, while races living in a low, cool and moist climate are especially prone to it. Drinking any liquid in excess also induces corpulence. Fermented liquors and the alcoholics are special causes. Overeating may likewise cause it. Most persons eat more than is good for them. Persons who drink water in excessive quantities are usually corpulent, especially if they drink much at their meals; perhaps because the increased amount of water interferes with digestion and assimilation. Besides, if the gastric juice is diluted, the blood is made more liquid, and the red corpuscles are in a measure dissolved.

Fat tissue appears to be chiefly formed both from the carbo-hydrates, and also from the surplus carbon of the proteids or albuminoids; from pure fat taken as food; and from water or other liquids. Some authors hold, however, that fat taken as food does not make fat tissue. Ebstein, of Gœttingen, maintains this view. The truth appears to be that when fat (or an albuminoid) is eaten in small quantity, no fat is stored up; but when the fatty food or albuminoids are increased to a point where the carbon is no longer burned, it is retained in the system as fat.

We are not very fully informed as to the pathological findings in the corpulent after death. I, myself, never gave the matter much attention, although I have made a good many post-mortems on corpulent people. But after death, the tissues of the corpulent are apt to be soft and flaccid, and decomposition rapidly ensues. In a case of fat heart occurring in my pathological service in St. Luke's Hospital, where death was *sudden* in a man only thirty-three years of age, the left ventricle was found hypertrophied; there was œdema of the lungs; the spleen was large and soft; the liver fatty; while the liver, kidneys, and portions of the stomach were congested.

The diagnosis of corpulence is simple, but it is generally admitted that the presence of a fat heart cannot be positively determined by physical signs. It is a matter of inference. But most agree with Quain, that where the pulse is small and weak, the first sound of the heart feeble, the impulse weak and the heart's area enlarged in a patient who is corpulent, it may be pretty certain that there is a fat heart. Henry Kennedy, of Dublin, in opposition to Quain, based his diagnosis on a large, full pulse, not increased in frequency, an enlarged area of heart dulness, and possibly a soft systolic murmur over the aorta, with the first sound. In my experience a fat

heart is often accompanied by valvular lesions, and I think that my experience will be found borne out by a study of reported cases. Hence it is that Kennedy may, in his cases, have found a full pulse which was due to valvular lesions. In this connection, however, I ought to say that during the treatment for fat heart previous murmurs will sometimes disappear, a fact indicating to my mind that these particular murmurs were probably due to a relaxed condition of the heart chambers or ostia, and not to an organic valvular disease.

Obesity is a disease that can be successfully treated in most cases, if the patient has a fair amount of vitality; and even in the feeble, the dangers attendant on a scientific course of treatment are small as compared with the risks in neglecting it. According to Maccary, as quoted by Worthington in his excellent *Thèse de Paris* (1875), the methods of the ancients comprised venesection, the use of purgatives, exercises, friction, diet, and stimulation of the several emunctories of the system. These methods, however, were probably not applied to the very young, the very old or the feeble. It seems hardly worth while to discuss the topic of *venesection*. Drugs, however, are still very extensively used. *Liquor potassae* was recommended by Chambers in 1850. The dose was from one-half drachm to one and one-half drachms. The theory of the action of this drug is that "it increases the vital power of metamorphosis by saponifying, in part, the fat contained in the blood, enabling it to be burned off as carbonic acid.[1] It is no longer used. Probably no stomach could stand its administration for any length of time. Its effect, if any, was to prevent digestion. In other words, it was one of the many "starvation cures."

Fowler's solution, in five minim doses three times a day, has been used. I have known it to be tried, but never with success. It is uncertain in action, and may increase the weight.

Fucus vesiculosus, or bladder wrack, a species of seaweed found in the Atlantic Ocean, was at one time used, on account of the iodine and bromine it contains. It was given in a decoction of two to four drachms to the ounce. The taste is very offensive and the stomach is greatly irritated, so that gastric catarrh may be produced. The kidneys, however, are urged to great activity. Some have simplified this latter method by giving tincture of iodine in doses of two to four drops in a wineglass with lemon juice; but this treatment also seems to produce catarrh of the stomach.

[1] *U. S. Dispensatory*, 1880, p. 862.

Bromide of ammonium in doses of five to thirty grains per day has been recommended. It is unpleasant to the taste and irritating to the system in many ways. In line with this is the treatment by vinegar. It reduces the flesh, but produces nervous disturbances. According to Brillat-Savarin (Worthington), it caused the death of a young girl of eighteen who insisted on taking a wineglassful every day.

The *Banting method* was at one time widely employed. Banting had tried the waters of Leamington, Cheltenham and Harrogate; had taken plenty of outdoor exercise of a vigorous kind; had tried Turkish and vapor baths, and had used liquor potassae as recommended by Chambers, but with no effect. His physician, Dr. F. Harvey, then put him on a regular diet that consisted of four meals a day. He took eleven to fourteen ounces of meat, game, poultry or fish (pork and salmon excepted), tea without sugar, rusks and toast in small quantity, all vegetables except potato; four to seven glasses of claret and two to three ounces of fruit. Hot drinks of "grog" at night. He is said, however, to have also been ordered a mysterious black draught on rising, the ingredients of which I have not been able to discover. On this system, kept up for something over a year, he fell off from 202 to 156 pounds, losing 46, or at the rate of 3 to 4 per month. The case, as described by the patient, is somewhat lacking in details from a medical point of view. The loss of weight per month was rather small—and the dietary, especially as to alcoholics, was, to say the least, liberal.

The *permanganate of potassium* in doses of one-fourth to one grain before meals has been recommended by Bartholow. It is said, at any rate, to relieve the acute gaseous dyspepsia of the corpulent.

Chambers' system consisted in a diet of two meals each day, active exercises, rubbing, salt baths, alkalies such as liquor potassae in doses of one-half drachm to one and a half drachms, purgatives, and even bleeding.

The treatment at the baths of Marienbad, Tarasp and Carlsbad is due to the use of Glauber's salt, which reduces by causing watery discharges; but it is apt to be so violent in its action, owing to the very short time allowed for the treatment given, that it may cause debility, palpitation, and even chronic diarrhœa.

According to Worthington, *Trousseau's plan* was to allow his patient lean meat, fresh vegetables and fruit in their seasons, but to forbid him fat meat, butter, oil and milk. The amount of bread and milk taken daily was to be diminished to a point as low as his vitality permitted. The patient was to be weighed every two weeks,

and he was expected to lose at the rate of one and one-half to three pounds per week. Exercises in the open air on foot or on horseback were ordered. In addition, he prescribed baths containing five to six ounces of the bicarbonate of soda. The same drug was given internally to the extent of thirty grains per day.

The *plan pursued by Ebstein*, of Goettingen, is about as follows, the rules being modified somewhat according to the case:

1. Breakfast—6:30 A. M. in summer and 7:30 in winter. Large cup of black tea without milk or sugar. Two ounces of white or brown bread. Plenty of butter.

2. Dinner.—2 P. M. Soup; four to six ounces of meat, with fat gravy; plenty of vegetables of all kinds excepting beets, carrots, turnips and potatoes. A little sweet fruit after dinner. Salad or stewed fruit. No sugar. Two or three glasses of light white wine. After dinner a large cup of black tea. No milk or sugar.

3. Supper.—7 P. M. A cup of tea. One egg, ham fat—in fact, any fat meat; sausage, smoked or fresh fish. Two ounces of white bread; plenty of butter. Perhaps a little cheese; a little fresh fruit. This diet to be kept up indefinitely.

Ebstein, as I have already stated, holds to an idea, opposed by most, that the eating of fat does not produce fat.

Oertel, of Munich, had a somewhat similar plan, but prescribed a peculiar course of exercises, and sometimes resorted to violent diaphoretics. He restricted the amount of liquids and solids, limited carbohydrates and fats, ordered prolonged walks, increasing the distances daily, making his patient ascend greater and greater heights (*Terrain cur*). In the winter, or whenever his Terrain cur was impracticable, he used injections of the hydrochlorate of pilo-carpine in doses of one-third to one-fourth grains twice a week.

His dietary was as follows:

Morning—Tea or coffee, four ounces with milk and sugar. Bread, two ounces (roll).

Mid-day—Beef, ten to twelve ounces; an egg. Vegetables, two to three ounces. Farinaceous food, one to five ounces. Fruit, three to four ounces. Salad, two ounces. Austrian red wine, three to four ounces.

Afternoon—Coffee, four ounces, with milk and sugar.

Evening—One to two boiled eggs, five ounces of meat or six ounces of game or fowl; one to two ounces of bread; salad; two to ten ounces of wine; Moselle preferred.

Among the newer remedies that have been used in this country

is *phytoline*, the active principle of the phytolacca decandra or pokeberry, which if taken in ten-drop doses before and after meals, is said to reduce without dieting, and at the rate of five to twenty pounds per month. The drug apparently acts on the subcutaneous fat, causing its absorption.

Thyroid extract is also extensively used. It produces emaciation, but is often poorly borne by the stomach, and is apt to be depressing. In one of my cases with hereditary ataxia it greatly aggravated the ataxic symptoms. The *grape cure* is another means of reducing flesh. The patient is restricted to unfermented grape juice for several weeks. At first he takes it in excessive quantities, then the amount is gradually reduced to the least amount compatible with vitality. Then it is slowly increased until enough is taken to fairly sustain the bodily activities. It is merely a sort of "starvation cure." The *teas* which are now widely advertised, but whose ingredients are not generally known to the public, are chiefly composed of senna leaves, with a varying quantity of chelonia, couch grass and coriander seed. To be effective the dose should be sufficient to produce two or more very watery movements daily.

Dancel, the French surgeon, who with Trousseau wrote a treatise on obesity, used the hydrogogue *scammony* (the activity of scammony is due to its resin), of which the dose is five to ten grains. At the same time he reduced the quantity of food and drink.

There are many *baths* in Europe that are resorted to for the cure of corpulence. First in order of repute are the cold Glauber's salt waters of Marienbad, in Austria, and Tarasp in the Engadine. But if patients have cardiac difficulty, asthma or diarrhœa, the hot Glauber's salt waters of Carlsbad are better; or the hot alkaline muriatic waters of Ems; the bicarbonate of soda waters of Vichy, in France; or the alkaline-saline of Brides in France, on the Italian frontier. In mild cases patients are usually sent to take the saline waters of Kissingen or Homburg, but even these latter may prove to be too strong. Some years ago I had a patient weighing 220 pounds under my charge, who lost fifty pounds' weight at Kissingen, but his nervous system was so deranged that he told me he had felt "as if he would lose his mind."

In fact, any effort to reduce the weight too rapidly, as is often done at the Continental spas, is apt to be harmful. It is not desirable to lose flesh in this way; nor is it always well to reduce the weight to the standard shown by our American tables. It is true

that professional trainers can do it, but they usually have little weight to take off, and the subjects are men of exceptional vigor and physique. It is said that De Graefe, in 1820, reduced a butcher from 306 to 150 pounds in nine months, but the man was a pugilist. Corpulent people should not be treated in this way.

The object in *reduction methods* is to take off the weight so that patients are relieved from disturbances attendant on the malady by means least calculated to disturb their equilibrium. There should be nothing disagreeable about such a reduction course. On the contrary the patients should enjoy it, and feel as each day or week passes by, that they are gradually returning to their normal state, and that their faculties are growing to be keener for the rational enjoyments of life. It is best to let it be known, at first, that the course may be a long one, lasting months, perhaps even a year or more. Banting's course took over a year. The patient should be mainly restricted as to foods that contain sugar, starches and fats, for there can be no question that fat to some extent produces fat. Oxidation should be increased by resistance exercises daily, and by baths that stimulate the skin, and so improve the circulation. Enough water should be taken to bring the urea up to the normal amount excreted; no more is necessary. Tea and coffee should be taken, if at all, in moderation, because they seem to retard oxidation. Acid fruits and drinks should be taken sparingly, because an excess of them produces indigestion. Sometimes all fruit should be forbidden. In general, however, small fruits may be taken with discretion, in their season. Sometimes the amount of both liquids and solids has to be much reduced. Laxatives should be taken, if necessary, so to produce full fæcal movements daily; and stomachics, if indigestion is acute. But the vitality of the patient should never be reduced. It should constantly increase. A patient under proper treatment may be made to lose from four to ten pounds per month, without disagreeable sensations of any kind.

The following are illustrative cases:

Case XXXIII. Corpulence; Fat Heart; Aortic Obstructive and Mitral Regurgitant Murmurs; Oedema of Extremities.—The patient, a physician, 76, weight 357 pounds, had been corpulent for many years and had suffered from acute rheumatism, and chronic eczema of the lower extremities. For several months his health had been failing; he was having constantly increasing dyspnœa, and he was unable to carry his great weight. He was wearing rubber bandages for his eczema and taking arseniate of soda and Arkansas Lithia

water. Pulse feeble and intermittent, usually absent in the left wrist. Impulse at the apex barely appreciable. Oedema of the extremities. The patient was also taking tincture of digitalis, in ten minim doses, three times a day. Respiration after walking 32 to 44, panting and labored. Acute gaseous indigestion. Cannot walk half a block. Face pallid; apex outside nipple; murmur at apex, with first sound, carried round to left; with first sound carried up great vessels. Urine five pints per day; no albumin. The patient was put on the modified Nauheim resistance exercise treatment, with baths. At first the exercises were mild, with lengthy intermissions, and the baths warm and salt; temperature, 95°; duration, five minutes. At the end of the second week they were carbonated and given immediately before bedtime. Digitalis stopped; sulphate of strychnine one-sixtieth of a grain three times a day; arsenic reduced in quantity. Examination of urine (by E. E. Smith, Ph.D.): Specific gravity, 1,020; trace of albumin; urea, 1.61 per cent., 8.34 grains to the fluid ounce; a few uric acid crystals; a moderate number of hyaline and a few epithelial casts; sugar absent.

February 25th—Pulse 75 to 84. Respiration 20. Patient evidently better. Ordered Carlsbad Salts twice a week. Two meals only, with eight ounces of meat at each meal. Patient had previously accustomed himself to two meals a day. Weight now 350. Strychnine one-thirtieth of a grain three times a day. Arsenic stopped.

March 10th—Urine re-examined (E. E. Smith, Ph.D.). Sp. gr., 1020. Indican; trace of albumin; no sugar; urea, 1.55 per cent., 7.05 grs. urea to the ounce. Neutral. Mucus and pus, traces. A few hyaline casts.

March 16th—Girth, 60½ inches. Patient walks a little farther each day. Temperature of bath, 93°; seven to nine minutes' duration.

March 19th—Weight 348½ pounds. After baths and exercises a fall in the pulse rate.

March 23d—Pulse has been ranging as follows: Before exercises, 81 to 88; after, 77 to 82; before the bath, 80 to 90; after, 80 to 86. Patient is drinking Londonderry Lithia Water.

April 2d—Weight, 343 pounds. Water, five pints. Sp. gr., 1820. No albumin; phosphates.

April 20th—Pulse before exercises, 77 to 87; after, 76 to 79; 13 resistance movements daily, total duration 35 minutes. More force used. Now takes Carlsbad salts daily in larger doses, caus-

ing two liquid movements. Milk is discontinued. In place of it Bethesda water, with a little lemon juice, to aid in satisfying thirst.

April 23d—Weight, 338 pounds. Greatest girth, 50½ inches. Takes no breakfast. In place of it a glass of hot water. Takes Apenta water at breakfast time, sometimes followed by hot water. After two months' treatment the patient reported of himself as follows: "Two months' treatment shows a loss of nineteen pounds in weight, and a reduction of ten inches in measure about the waist, with marked increase of strength and a greater freedom in breathing."

May 2d—Resistance exercises, thirty to thirty-five minutes. Carbonated baths have been gradually increased to their full strength, but are now suspended.

May 7th—Has gained three pounds, but lost nothing in girth. Ordered baths again with one-half per cent. carbonic acid.

May 14th—Weight, 332½ pounds. Lost six and one-half pounds in seven days. Bath now every other night.

May 26th—Gained two pounds last week. Rubber bandages now removed. Patient walks easily. Ordered to take only one meal per day for one day, and two meals on the alternate day.

June 4th—Weight, 327½ pounds.

June 11th—Patient going to the country is directed to take special resistance exercises daily. To take hot or cold water, one or two goblets with orange juice, before breakfast. Then to take a hearty breakfast and a light supper. To eat only twelve ounces of meat, fish or fowl daily, avoiding starchy and sugary food. To eat sparingly of small fruits; to avoid fat in every form, including butter, milk and gravies.

In January of 1899 the patient reported that under this treatment his weight had fallen during the summer to 317 pounds—a loss of forty pounds. During all this time he had attended to his daily professional routine of business, while in the city; and made long trips out of town in consultation cases, where he was obliged of necessity to walk long distances in going to and from his train. Among the noteworthy features of this case is that, under the treatment, the eczema and œdema of the legs disappeared, and that he gradually gained in strength, so that he was able to do more professional work, during the treatment, than he had done for a long time previously.

Case XXXIV. Obesity; Fat Heart; Temporary Diabetes.—A lady of about sixty-five came to me on October 4, 1898. She weighed 181 pounds, was nervous and anæmic. Skin bathed in

perspiration. Color dusky. Pulse 100 to 104. Weak impulse. Heart enlarged. No organic murmurs. Urine examination (by E. E. Smith, Ph.D.): Sp. gr., 1027. Faint trace of sugar by several tests; no albumin. Urea, 1.75 per cent., 7.95 grains to the fluid ounce. A little pus. A few uric acid crystals. Moderately large quantity of hyaline casts.

October 29th—Second examination. Urine, 60 oz. Sp. gr., 1018. Alkaline; no albumin. Sugar absent. Urea, 2.28 per cent., 10.40 grains to the fluid ounce. Pus absent. Moderate number of hyaline casts.

November 2d—Ordered anti-lithæmic diet. Resistance exercises.

November 14th—Resistance exercises and massage. One-half per cent. carbonic acid bath, at 97°, five minutes. Average pulse before exercises and bath, 90; after, 81.

November 22d—Weight, 178

November 28th—Weight, 177.

December 21st—Weight, 171.

The treatment was now stopped by an attack of influenza, the patient leaving town subsequently for a short trip to the country. From a health resort, where she had been in the habit of going, she reported on January 24th: "The doctors here think me much improved." Loss of weight, about five pounds a month.

Case XXXV. Obesity; Fat Heart; Dyspepsia.—A gentleman weighing 237 pounds, height 5 feet 6 inches, came under my care in December of 1898. Pulse 100, no intermissions. Apex beat feeble, difficult to locate; heart enlarged. Patient a smoker and lithæmic. No organic murmurs. Pain at apex, giddiness of head, acute gaseous dyspepsia, and dyspnœa. Apex four and three-fourths inches from the median line, and three-fourths of an inch below the intermammillary line.

December 28th.—Dyspepsia and regurgitation. Under subgallate of bismuth, Carlsbad salts, anti-lithæmic diet, and abstention from smoking, these disappeared.

January 27th.—Patient took the modified Nauheim course of resistance exercises and baths. Apex now about four inches from the median line, and three-fourths of an inch below the intermammillary line. Takes no medicine. Weight, 217 pounds stripped. Had lost about ten pounds in a month. Pulse of better quality; dyspnœa gone, and the patient able to walk fifty-five blocks in a single day.

By methods such as are given here it is possible to reduce the

weight, without detriment to the general health; and we have a right to assume that the deposit of fat in the heart is, measurably at least, diminished with the loss of the visible adipose tissue.

Chapter XI.

FATTY DEGENERATION OF THE HEART.[1]

Fatty degeneration of the heart was first correctly differentiated from the fat heart of obesity by Laennec[2], and a little later by Andral[3], who called it *ramollissement du coeur.*

The source of this degeneration appears to have been discovered by Hayem,[4], at least so far as typhoid fever is concerned. In 1883 Germain Sée[5] confirmed this view, while in 1891 Romberg,[6] after an elaborate study of the heart substance in typhoid, scarlatina and diphtheria, discovered in all of them a granular and fatty degeneration of the heart-muscle, together with a small-celled infiltration of the adjacent connective tissue. In 1898 Bollinger[7] adopted this view and described three degrees of fatty degeneration, though he did not intimate that they might be three stages in the one process. In the same year Dehio[8] confirmed Hayem's views, so that we may now regard the matter of fatty degeneration of the heart as fairly understood, pathologically. It remains for us to attach, if possible, clinical phenomena to the successive stages of its development.

It is very important to put this matter on a sound clinical basis, for how often sudden death, from heart failure, strikes down a person who, up to the time of the attack, appeared to have excellent health. In fact his physician, after examining his heart and, finding no signs of valvular disease, may have been content with the diagnosis of a "weak heart," and yet the heart substance may have been so profoundly diseased that it only needed a sudden, violent, or even prolonged, "strain" to cause hyperdilatation and cardiac paralysis.

I described such an instance at a meeting of the New York Pathological Society some years ago:[9]

Case XXXVI.—A gentleman, seventy-three years of age, who had led an active life up to a year before his death, was suddenly taken with dyspnœa, after some hill-climbing in California. Up to this time he had never had heart-symptoms. He recovered promptly,

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[1] Originally published in the *Med. News*, Feb. 2, 1901.
[2] Laennec, *Dis. of the Heart and Lungs*, London, 1846, p. 607.
[3] Andral, *Path. intern.*, Paris, Vol. I., p. 324.
[4] Hayem, *Arch. de phys. norm. et path.*, 1869, Vol. II., 1, p. 699.
[5] Germain Sée, *Maladies du coeur.*, Paris, 1883, p. 199.
[6] Romberg, *Deutsch. Archiv. f. klin. Med.*, Bd. 48 u. 49, 1891-92.
[7] Bollinger, *Path. Anat.*, New York, 1898, Vol. I., p. 74.
[8] Dehio, *Deutsch. Archiv. f. klin. Med.*, 1898, LXII., s. 1-62.
[9] Satterthwaite, *Trans of N. Y. Path. Soc.*, Oct. 22, 1879.

however, from this attack. Later he took a 25-mile sleigh-ride and had another attack, but was restored by stimulants. Five weeks before his death he lost consciousness in a street-car, and was taken home with some difficulty. Stimulants, however, again revived him. An examination by the family physician showed that he was anæmic and weak; the pulse could not be felt, and the apex beat was barely appreciable. He did not rally. At the post-mortem examination, which I made, there was a marked blanching of the surface of the body. The lungs were œdematous and the kidneys pale and fatty. The heart was not enlarged, but it was pale and flabby.

Microscopic examination of the cardiac muscular tissue showed that along the inner wall of the left ventricle the striations were quite gone, though they were fairly well marked at the periphery of this ventricle. There was no valvular disease. In this patient the initial "strain" to the degenerate heart-muscle seemed to be directly traceable to the hill-climbing in California. There was apparently no other lesion to cause death.

The weak heart of fatty degeneration is common enough at all periods of life. In infants and children it occurs during and after the eruptive fevers and diphtheria, acute rheumatism, pneumonia, influenza, or, in fact, any acute febrile attack of toxæmia. In senility, at whatever age this may happen, whether early or late in life, it is a phenomenon to be expected. In fact, whenever there is a prolonged fever, toxæmia, dyscrasia or mechanical injury, fatty degeneration of the heart may occur. Very often the so-called "weak heart" is in reality the heart of fatty degeneration. And yet, to judge from individual experience, as derived from intercourse with physicians, and from our current literature, the matter is not so understood by the profession at large. In consequence fatty degeneration may be recognized too late for treatment, as in the case just cited.

The *early*, or *preliminary* stage is the one in which the prognosis is the most favorable for complete recovery; in the *second*, or *intermediate* stage we should be able to hold the disease in check; in the *third*, or *final*, stage palliative treatment alone is possible.

If, however, we are to grasp the matter intelligently, we must first of all look to the minute structure of the heart-muscle. Though involuntary, it is made up, as we all know, of muscle-cells marked by longitudinal and cross striations. The body of each cell is cylindrical and contains one or two nuclei. The nucleus is vesicular and oval, and is placed near the centre of the cell-body. The cells are

not only joined at their ends with contiguous cells; but, through branches, with other cells, making up a reticular network. The apposition of the cells is effected by a cement substance, that is not seen under ordinary conditions; and the network holds in its meshes vessels, nerves and fat.

In inflammation of this muscle the cells become opaque, owing to their infiltration with granular matter, or at least with something presenting that appearance (cloudy swelling), and the particles have been termed *albuminoid*, because they disappear to some extent under the action of weak acids or alkalies. One theory that has been proposed is that this *albuminoid* material is formed from the cytoplasm of the cells. Another theory supposes that the granules represent the toxic elements of the disease; another that they are effete matters being eliminated; another that they are micro-organisms; another that they are fatty particles in a stage of minute deposition. It is certain, however, that some of them are pigmentary, and also that in poisoning by phosphorus, arsenic and in typhoid fever, many are either fatty or of some closely allied substance. So much for the first stage.

In the second, or intermediate stage, the muscle cells are clearly seen to be studded with oil globules, which appear to occupy the same position as the "albuminoid" and fatty particles of the first stage.

In the final, or third stage, the fatty globules reach a higher degree of development, while occasionally the muscle-cells are pulled apart (*segmentation*), or broken into fragments (*fragmentation*), as the result of hyperdilatation of the heart chambers. In this stage there is an engorgement of the blood-vessels of the internal viscera, limiting their functional activity and causing hyperæmia, œdema, thrombosis or embolism. Renaut[10] has gone so far as to classify segmentation as a variety of chronic myocarditis, which he has called *myocardite segmentaire chronique*. At first, this view met with opposition from Ziegler and Recklinghausen, who regarded segmentation as an agonal manifestation. Hektoen,[11] of Chicago, however, after examining eighty hearts to test the matter, came to the following conclusions: *Segmentation* and *fragmentation* do occur in heart-muscle *during life* in consequence of irregular contractions or injuries, usually associated with disease of the heart-walls. If the segmentation or fragmentation is limited in area, it is not immediately fatal. Consequently we are forced to believe that both segmentation and fragmentation are real events in heart-strain, con-

[10] Renaut, *Gaz. Med. de Paris*, 1900, p. 100.
[11] Hektoen, *Trans of Path. Soc. of Phila.*, 1898, p. 267.

tributing, of course, to the fatal issue, though they hardly seem to be worthy of a separate classification.

Reverting for a moment to the subject of the so-called albuminoid change, a theory has been advanced that in exhausting diseases associated with prolonged fever in which the cardiac muscle is actually starving from lack of nourishment it makes a demand, in its hunger, on the store it has already accumulated, viz., the metaplasm of the muscle-cell; but if this is exhausted and more is needed, it is compelled to consume its own substance. Now the reserve stock in the meshes of the muscle-cells is made up of carbohydrates, and there is no harm done to the cell by their consumption. When, however, the living tissue of the cell has to be sacrificed, there is a loss of the proteids or albuminoid substances of the cell, and the damage done is directly proportionate to the amount consumed; and yet the combustion is incomplete, a granular *débris* being left behind in the cells. But consumption may proceed a step further, so that both the fatty matters and the fluids of the protoplasm are burned, though the combustion is still incomplete, and a further residue of oil is left behind in the cells. Still, even now, a restoration of the cells is possible, provided only the nucleus has been left unimpaired. Death of the nucleus, however, means death to the cell.

Quite a different view has been given by Huchard.[12] It is that the first stage of fatty degeneration is due to the irritation of a micro-organism or toxin, which, if sufficiently virulent, causes inflammation and perhaps, ultimately, necrobiosis of the cell. This last theory is plausible, but it does not apply to the fatty degeneration seen in retrograde changes, such as occur in the parturient or athletic heart and in adherent pericardium, where the fatty degeneration of certain portions of the cardiac walls appears to be due directly to lack of use; for when certain fibres are no longer needed they are pretty sure to degenerate and eventually disappear.

So much for the theories of the production of fatty degeneration. Accepting the dicta of Hayem, Romberg and Huchard, the acute stage is essentially a fatty one; that is, so far as the acute softening in typhoid, scarlatina and diphtheria is concerned.

Fat deposition or accumulation, in the heart, the fat heart of obesity, is quite another condition etiologically and pathologically. Fat accumulates in the connective or interstitial tissues lying between the muscle-bundles. It is the result of overfeeding either with fatty food, or any food material that is in excess of bodily re-

[12] Huchard, *Phila. Med. Times*, March 24, 1900.

quirements; but clinically the two processes are to some extent allied, for the muscle-tissue may suffer atrophy in the fat heart from pressure of the fat tissues; or the fibres may be so separated or overstretched that their integrity is impaired, and they will degenerate in consequence. Fat deposition and fatty degeneration may, therefore, coexist in the same heart; and yet while fat deposition is apt to cause fatty degeneration, fatty degeneration never causes fat deposition.

To the naked eye there is little difficulty in recognizing fatty degeneration in the heart-muscle by its pale yellow color, well shown beneath the lining membrane of the left ventricle or papillary muscles, in nearly every heart in which there is chronic valvular disease; but the eye will often fail to recognize it in the softened heart of typhoid fever or diphtheria, for the tissue will not be yellow, but of a muddy, brown color; or, if there is a fibroid element, the color may be violet-brown or violet. It occurs in localized areas presenting a mottled appearance; or is diffuse and the special area of softening "mushy," so that in handling the heart the fingers sink into it. The left ventricle is chiefly affected, at first.

Fatty degeneration taken in the broad sense that I have indicated must be quite frequent. Roemer, of Tübingen, between 1870 and 1890, saw at his clinic 291 cases of valve lesions, in which 230 showed symptoms of disturbed compensation, while at the same time there were 234 cases that he called myopathic heart-failure, without any lesions of the valves. Now, as most of these 234 cases were probably instances of fatty degeneration, heart-failure without valve lesions was about as common as heart-failure with valve lesions.[13] Chronic fatty degeneration of the heart is usually associated with cardiac hypertrophy, atrophy, fibroid disease, fatty deposition, chronic endo- and pericarditis, and some other cardiac or arterial disease. For example, Quain found hardening or calcification of the coronary arteries in 13 out of 33 cases of fatty degeneration; Markham in one out of 12 (Whittaker); so that arteriosclerosis may be regarded as one of the causes of fatty degeneration. It is an unfortunate idea prevailing in some quarters that coronary disease has a preponderating etiological connection with fatty degeneration. This is not true, though it is not at all uncommon for me to have a case of fatty degeneration turned over to me with the label "coronary disease." With some physicians, I might also add,

[13] Whittaker, *Twentieth Century Practice*, Vol. IV, p. 340.

the term "myocarditis" means fatty degeneration, though they are obviously two distinct processes.

We usually meet with the chronic variety of fatty degeneration in middle life, though it may of course occur at earlier periods. In 88 cases published by Hayden,[14] fatty degeneration was found most commonly between sixty and seventy, less often between forty and fifty years of age.

It is extremely important for us to recognize the early or premonitory stage, as bearing both on prophylaxis and treatment. Unfortunately this is not always possible. In mild cases the physical signs may be absent, for there may not be any appreciable dilatation, and the physical signs depend on this circumstance. Hence the importance of outlining the heart by percussion; for variations in outline from the standard will, in some degree, indicate whether the right or left ventricle is dilated; and the position of the apex will aid greatly in determining whether the heart is enlarged. Besides, the impulse at the apex will be more or less feeble, and the pulse will be intermittent and rapid. The sounds of the heart will also be weak. There may not be any murmurs, or, in the absence of valvular disease, there may be systolic murmurs due to muscular insufficiency. If there is hypertrophy of the right ventricle, following dilatation of the left ventricle, there will be an accentuation of the second pulmonary sound.

There may also be præcordial pain or distress and spells of dizziness or fainting. This stage may last a few days, weeks, or months; or longer, after diphtheria and influenza. A few cases taken from my pathological records while pathologist to the St. Luke's and Presbyterian Hospitals illustrate the subject of fatty degeneration:

Case XXXVII. Acute Pericarditis; Empyema; Fatty Degeneration of the Heart.—A drinking man, thirty-eight years of age, entered the hospital with an empyema, the abscess discharging in the mammary region. Pulse 100; respiration 31 to 36; temperature 102° F.; dyspnœa, præcordial pain, cough, mucous expectoration and night-sweats. After battling with the attack for three months the patient died of cardiac failure. At the post-mortem examination, the opposing surfaces of the pericardium were found to be acutely inflamed and "hairy." The pericardial sac contained three ounces of clear serum. The heart was dilated and in a condition of acute softening. Fifteen ounces of purulent fluid were taken from the pleural cavities. This was an example of fatty degeneration

[14] Hayden. *Dis. of the Heart*, Dublin, 1875, p. 66.

of the heart in the first stage, due to a combination of two causes, acute pericarditis and empyema. The cardiac symptoms were not clear, being obscured by those of the empyema.

Case XXXVIII. Yellow Fever; Fatty Degeneration of the Heart. —This case also illustrates fatty degeneration of the first stage, in a marked manner. M. C., thirty-two years of age, widow, stewardess, was admitted to the hospital July 25, 1879, with yellow fever. Her illness dated back only five days, when she was taken with frontal headache and general "soreness" of back and legs. The initial chill was followed by nausea, diarrhœa and fever. Pulse 108, and weak; temperature 105° F. On the ninth day she vomited dark matter, became delirious and had albumin (50 per cent. by volume); scanty water. At the post-mortem examination the heart was found to weigh ten ounces and was soft, flabby and fatty. The valves, however, were free and normal. This fatty condition of the heart had previously been noted by Dr. H. D. Schmidt, of New Orleans, as a characteristic of yellow fever, with at the same time fatty degeneration of other internal organs, including the brain. This case I regard as another instance of fatty degeneration in the first stage. I made the microscopic examination of the heart.

In the *second stage* the signs of a dilated heart are more pronounced. The left ventricle may reach to the left nipple or extend beyond it; the right ventricle an inch or more beyond the right border of the sternum. The apex may be in the fifth, sixth or seventh space. The pulse will usually be soft, intermittent and infrequent. There may be, and often is, a lack of harmony between the heart and pulse-beats. These manifestations are apt to be seen (though not always), in persons who have passed middle life, and are inclined to be stout, but are anæmic in appearance. The apex beat will be difficult or impossible to locate by palpation. There will be dyspnœa and some precardial oppression, with occasional attacks of dizziness. Abdominal symptoms will always be in evidence; occasionally gastric crises alternating with anginoid attacks. Cyanosis will occur at times. Such patients will usually be irritable or whimsical, or nervous about trifles, and always concerned about their health. There will be a disinclination to undertake anything new, even to walk, and the gait will be uncertain. Occasionally there will be hemorrhages, usually in the form of epistaxes. Local œdema of face, hands and feet will occur at times. The urine will contain a little albumin, and often a little sugar. This stage may last from two or three, to ten or fifteen or even twenty years,

depending largely on the degree of the degeneration and the care the patient takes of himself.

The *third stage* is ushered in by symptoms that indicate secondary implication of other organs. There may now be pseudo-apoplectic attacks, due to cerebral embolism or thrombosis referable to imperfect heart-action. Attacks of heart-failure are more frequent and more alarming. Albuminuria is more in evidence and less amenable to treatment. In fact, symptoms referable to chronic nephritis are the rule. Gastric disturbances become so pronounced that the patient is afraid to eat for fear of the distress it occasions. Local œdema passes over into general. *Thrombosis* may be a noteworthy feature. In one of my cases thrombi distending the right external jugular vein were distinctly felt. A low form of meningitis from effusion at the base may be one of the final events. The mind will be disturbed and there may be, at times, Cheyne-Stokes breathing, though this is not necessarily a sign of immediate dissolution. Until stasis occurs in internal organs, however, we should not despair; but where this takes place, and this condition characterizes the third or final stage, the end cannot be far off. With great care life may be prolonged a few months. Death may occasionally occur, however, not from any of the above causes, but from *rupture of the heart*. Quain has seen it in 28 out of 83 cases.

This stage is illustrated by the following three cases:

Case XXXIX. Syphilis; Chronic Nephritis; Fatty Degeneration of the Heart.—A man, forty-six years of age, entered the hospital with the signs of general syphilis. He had cough, jaundice, dyspnœa and swollen feet. A soft systolic murmur was heard at the apex. The patient died fifteen days after admission, of chronic nephritis. At the post-mortem examination the pericardial sac was found to contain ten ounces of clear serum, and there were several milk patches on the heart. It weighed eighteen ounces. There were no valve lesions. The liver was large and fatty. The kidneys were the seats of chronic nephritis. At the autopsy, the murmur was held to be muscular, and due to fatty degeneration of the heart. Syphilis was pretty certainly the remote cause of the fatty degeneration.

Case XL. Arteriosclerosis; Cerebral Hæmorrhage; Fatty Degeneration of the Heart.—The following is another of my cases: A woman, thirty-eight years of age, who said she had suffered from "malaria," was admitted to the hospital with œdema of the lower extremities. She complained of attacks of palpitation, with præcordial pain, tonic spasms and dyspnœa. The pulse was intermit-

tent, and the aortic second sound accentuated. The patient died of cerebral hœmorrhage. The autopsy disclosed general arteriosclerosis with chronic diffuse nephritis. The heart was dilated and soft, but the valves were free and sufficient. The cause of the fatty degeneration in this case with arteriosclerosis, and the death by cerebral hæmorrhage illustrates one of the several ways in which the end comes at last.

Case XLI. Adherent Pericardium; Fatty Degeneration of Heart.—A woman, sixty-three years of age, with a history of articular rheumatism, was admitted to the hospital suffering from cough, hemoptysis, dyspnœa and chronic nephritis. She also had a systolic murmur over the apex, and over the aortic and pulmonary areas, with accentuation of the second pulmonary sound. She died of cardiac paralysis and chronic nephritis. At the autopsy the heart was found to weigh twelve ounces. The valves were free and sufficient; but the cardiac walls were soft and flabby, and the cavities of the heart dilated. The heart and pericardium were united by an old adhesive pericarditis. The liver was large and fatty. In this instance we see the fatty degeneration of the heart always found in adherent pericardium, though the toxins of rheumatism may be assumed to have been early factors in causing degeneration, independently of the pericarditis, which operated, in a purely mechanical manner, to produce the fatty change. This case is another instance of fatty degeneration in the third stage.

If, then, we realize that this affection of the cardiac walls is incidental to infective diseases, continued fevers, septic or suppurative processes, toxæmias, dyscrasias, hypertrophies and atrophies, we should be on our guard to protect the patient against the inherent damage to the heart resulting from these several conditions. We should thus hope either to prevent a break-down of the cardiac walls, or certainly to postpone it. In infants or young children convalescing from eruptive or continued fevers we should watch carefully their "weak hearts." With them rest in bed and minute doses of iron and strychnine, perhaps in conjunction with some malt preparation and cod-liver oil or quinine, will usually be sufficient, in uncomplicated cases, provided the diet is carefully regulated, and hurried movements, or any form of muscular strain or nervous excitement are avoided.

In young people, such as are anæmic or tuberculous, gymnastic exercises are appropriate for the intermediate stage, provided they are given under suitable medical direction and are not carried to

the extreme that is common at the present day, in gymnasiums and outdoor games. There are plenty of schools for physical instruction, under medical supervision, where those convalescing from illness with weak heart-action can be greatly improved. On the other hand, I cannot too strongly condemn the class exercises of the day, if the patient has heart-weakness or in fact any form of heart-disease. Class work calls for uniform movements, usually rapid, which give few intermissions for breathing; so that it is only suited for those whose hearts are sound.

In fact, every case of weak heart should be treated by itself, whether in the young or old, and the exercises should never be carried to the point of increasing the rapidity of the heart's action beyond its normal range, which of course differs with each individual; for although we have adopted an artificial standard for the rapidity of the pulse, one man's normal pulse may be 60 or less, and another's 80 perhaps, or more. Systematic exercise of a specially prescribed variety should be insisted on, because habits of indolence tend to a fatty heart, and will increase the difficulty. In fact, oily matter of any kind should be consumed by muscle work. At the same time, patients should be put on a specially restricted diet. It prevents gaseous distention of the stomach and intestines, which provokes cardiac irregularity.

When young or old people have not the strength for ordinary gymnastics they should have resistance exercises, with or without massage. These exercises are more thorough than massage, and tend to rid the muscle-cells of their granular contents, whether they are toxic or otherwise. But with delicate patients massage and exercises may be too severe a tax on the strength, producing too profound a reaction. The treatment should always have the effect of toning up rather than relaxing the individual. Carbonated baths, of course, are to be used in conjunction with resistance exercises, in the intermediate stage of the disease; but baths and exercises are of little or no use in the third stage. And yet while they may not do any good, they may not do any harm. Massage, however, is often very grateful.

Roemer, quoted by Whittaker,[15] reports that in 234 cases of heart-weakness without valve lesions, where 81 of them were treated by the mechanico-dietetic improved Nauheim plan, 46 recovered and 17 were improved. But neither exercises nor baths should be instituted, until a careful physical examination of the patient has

[15]Whittaker, *Loc cit.*

been made. If, for example, he is suffering from a violent attack of palpitation, rest in bed in a quiet room, and a restricted diet, should be ordered. Milk is excellent if it agrees with the patient. In ordinary cases of fatty degeneration the patient, if anæmic, should be put on iron; if tuberculous, on malt preparations and creosote or cod-liver oil; if arteriosclerosis, on the iodides; if rheumatic, on antilithæmic diet and remedies, in conjunction with the hot-air treatment.

In the *third stage*, when all hope of cure by any of the above measures is at an end, and it is merely a question of prolonging life and alleviating suffering, digitalis is appropriate, if there is an effusion referable to the failing action of the heart, through inaction of the kidneys. Merck's digitaline (in 1/100 grain doses every few hours), I have found excellent; but digitalis should not be continued for more than a week or so at a time, and strichnine should follow it to maintain its effects. Other remedies that are useful in this stage are nitroglycerine, caffeine, strophanthine and sparteine. Attacks of difficult breathing, if not severe, are relieved by oxygen gas, while sudden attacks of true or false angina are best treated by the nitrites. And yet in the third stage it must be remembered that the degenerate heart has a remarkable capacity for recovery, even without the so-called heart stimulants. If given, their action should be constantly watched and they should only be used for the briefest possible period. These remarks are especially applicable to digitalis, strophanthus, scoparius and nux vomica.

The *prognosis* of fatty degeneration varies with the stage, the individual, his environment, and the influence of the disease which has produced it; for fatty degeneration is not strictly a disease *sui generis*, but a degenerative process superinduced by a number of diseases. For example, the fatty changes attendant on infective diseases of toxæmias should disappear entirely, provided the heart has had rest during the stage of convalescence. On the other hand, if during convalescence the patient has been called upon to do an improper amount of physical work, dilatation may ensue and fatty degeneration be produced. In infants and young children fatty changes in the heart incidental to infective diseases, or as sequels of them, are quite common. On the other hand, a moderate amount of fatty degeneration is not inconsistent with an average life; in fact, there is a certain amount of fatty degeneration in most adult hearts otherwise sound, reflecting probably the methods of life in what

we call civilized communities, where the intellectual rather than the physical parts of our system are most in use. This is well shown in the hearts of athletes; for when hypertrophy has been caused by severe training, there seems to be always a certain amount of fatty degeneration coincident with the gradual return of the heart towards its normal size, which retrograde process takes place when athletics are given up for the ordinary routine of a business or professional life. In such cases, however, when the enlarged heart fails to contract to the normal size, we may infer that the delay is owing to some other organic heart affection, such as valvular disease or mechanical injury or an old strain.

In the middle period of life, when degenerative changes are the rule, a weak heart may be toned up and restored to a fair degree of soundness by appropriate treatment; though it is one thing to make an organ sound physiologically and quite another to have it sound pathologically. And yet it is always a triumph of medical art to restore the functions of any organ.

In the final stage when *anasarca* supervenes and the kidneys, from hyperæmia or structural changes, secrete a lessened amount of urine, containing albumin and granular casts; and there is transudation of fluids into the bronchi, lungs, or chlylopoetic tract, interfering with the functions of these organs, the prognosis cannot be favorable. The fatal issue is certainly near at hand and, though the heart may be sustained and the kidneys forced to do better work, it is a struggle in which Nature will eventually triumph most likely by thrombosis or embolism; perhaps by apoplexy, meningitis or uræmia.

Chapter XII.

SYPHILIS OF THE HEART.

Before Ricord's[1] time cardiac syphilis had been recognized, but he was the first to describe it clearly. Shortly afterwards Virchow[2] confirmed Ricord's statements. A few years later Lancereaux[3] classified it under four types. But grave doubts were still entertained of its existence, and they are only now being successfully overcome, though a great deal has been written on the subject since Ricord's time.[4] The instances, however, that are conclusive, from a pathological point of view, have been comparatively rare. The naked-eye appearances in syphilis show nothing that is positively distinctive, if we except gummy tumors, and they are seldom detected. Indeed, so keen an observer as Fagge[5] had failed to recognize more than four cases up to 1886, notwithstanding his long pathological service at Guy's Hospital. Only one of them was classed as a gummy tumor, three being fibroid infiltrations of the heart walls. In my own pathological records I have only three cases put down to cardiac syphilis, and am still in some doubt as to their true character. And yet I believe many syphilitic manifestations are overlooked at post-mortem examinations, simply because the naked eye evidences are inconclusive. However, the researches of Mracek[6] and Kundrat, of Vienna, should set at rest forever the question of the existence of this cardiac lesion, for in 1893 he published a series of 102 cases in which the evidences were established by autopsies. His table indicates the different ways in which he found that syphilis may affect the heart. As taken from the literature, including ten cases of his own, it is as follows:

Myocarditis, gummatous	10
" fibrous	9
" gummatous and fibrous	8
Endocarditis	2
Pericarditis	1
Diseases of vessels	3

[1] Ricord, *Lettres sur la Syphilis*, Paris, 1856, p. 349.
[2] Virchow, *La Syphilis Constitutionelle*, Paris, 1860, p. 117.
[3] Lancereaux, *La Syphilis*, Paris, 1866, p. 384.
[4] More than a hundred contributions are to be found in the literature.
[5] Fagge, *Principles and Prac. of Med.*, 1886, Vol. II, p. 34.
[6] Mracek, *Arch. f. Derm. u. Syph.* (Ergaenzungshefte), s. 279 u. 337.

Myocarditis with or without endocarditis	15
Peri- and endocarditis	1
Diseases of myocardial vessels and myocarditis	1
Diseases of ganglia, etc	11
	61

It will be observed that he found gummas in 30, or about 50 per cent., and as many or most of the other forms may be logically attributed to gummas, it follows that the lesion is the one par excellence of the disease. This preponderance of gummas points out another interesting fact, viz., that the disease is usually a manifestation of late syphilis, indeed, of the third stage. In fact, it may be expected as a late event, perhaps as much as eight to ten years, or even more, after the initial lesion.

In rare cases, however, it may occur in childhood and early youth, and is sometimes congenital. In 150 autopsies on infants with hereditary syphilis Mracek found cardiac syphilis in four. Fischer[7] has also reported a case; but they are extremely rare. Wollstein, Pathologist of the Babies' Hospital of this city, in her large experience has never seen a single one.

As one might suppose, it is more common in the male sex, and is usually seen between the ages of 20 and 40. The gumma varies in size. It is usually found in the ventricles, but may also occur in the auricles, septa or papillary muscles; or in fact anywhere in the heart. It is usually multiple, and the nodules, as we commonly recognize them, are from the size of a pea upwards. It may dissolve or break down and discharge into the cavities of the heart or outside of it, causing a cardiac aneurism. If cicatrization follows a gumma, there will be atrophy of muscle fibres in the adjacent territory; and if there is a general sclerosis extending from the thickened vessels, there will be general atrophy of the muscle. If the gumma is near the periphery of the organ, the sclerosis will extend toward the surface, and may leave a point of thickening (milk patch) there. In the same way valvular deformities may be produced by the deposit of a gumma in the valve. Its dissolution, with subsequent puckering, from loss of substance in the valve, or a defect due to a gumma in a tendinous chord or papillary muscle, may cause distortion of the valve and incompetence or obstruction, or both.

The gumma at first is of a pearly gray color, and enclosed in a pretty firm fibrous capsule. If hæmorrhage ensues, it becomes red

[7] Fischer, *Muenchner Med. Woch.*, 1904, 51, s. 652.

or yellowish, provided it undergoes change or breaks down and discharges. Under these circumstances, the central material is fatty or sticky, the latter when it undergoes a mucoid rather than a fatty change. It is this mucoid substance which sticks to the fingers and gives its name to the gummy tumor.

But whatever the change is, that is, whether it undergoes absorption or discharges its contents, the capsule contracts, hardens, and assumes a whitish color; and if the material has been discharged from the center, it leaves a depression, looking like a depressed cicatrix, from which bands of fibrous tissue radiate outwards; while the center is apt to be stained of a yellow color.

In general these peculiar changes depend on larger or smaller gummy tumors, originating in the walls of the vessels and spreading throughout the organ, along the line of the vascular network. The process appears to be originally an arteritis, which begins in the substance of the vessel or its periphery. It is an axiom that syphilis loves arteries, so that, in general, to them, rather than elsewhere, we must look for the original focus of the syphilitic manifestation.

It follows from all this, that gummy tumors and even aneurisms are apt to be multiple; but, as already said, the difficulty of recognizing any of these conditions, except the gummy tumor or the fibroid infiltration immediately connected with it, is very great, so that endocarditis, pericarditis or myocarditis occurring in syphilis, and as seen at autopsies, is likely to be attributed to anything but the constitutional disease.

In fact, the problem of determining whether any fibroid infiltration, unless connected with a gummy tumor, is really syphilitic is to my mind practically unsolved at the present time. Another reason for failure in recognizing cardiac syphilis, clinically, is that it is one of the latest phenomena of syphilis, occurring many years after all external manifestations of the disease have disappeared. Neither physician nor patient may have any suspicion of it. In fact, Weber's[1] cases go to prove that the cardiac lesion may occur so long after every external sign has gone that nothing except specific treatment will reveal its true nature. In many cases of heart syphilis we naturally expect to find sclerosis of the coronary arteries or their branches, for we know that syphilis is a common cause of arteriosclerosis, and the most frequent one of aneurism. We must be careful, however, not to impute all coronary diseases to syphilis. It is true that these affections are often associated with syphilis.

[1] Weber, *Post-Graduate*, Nov., 1903.

There are, of course, other causes of sclerosis. Syphilis may also affect the nerves and ganglia of the heart, as microscopic examinations have shown.

The diagnosis, in any instance, is mainly based on a previous history of syphilis, usually in a patient who has been treated according to the regulation methods with both iodides and mercurials, until the manifestations have disappeared, so that there are no visible or palpable signs of the disease remaining. In fact, it may have lain dormant ten, twenty, thirty or more years, according to Weber's experience. Some of the signs are as follows: Arteriosclerosis, a weak, intermittent and perhaps frequent pulse, dilated heart or angina, disease of the aorta, possibly aneurism, occasionally a valvular disease. If, in such instances, a course of iodides and mercurials greatly ameliorates or relieves the symptoms, and other possible causes of cardiac affections are excluded, the diagnosis is practically made.

The late Dr. Whittaker,[9] in an excellent article on this subject, tells us how he made a diagnosis under these circumstances and cured his patient. The following is an illustrative case from my records:

Case XLII. Syphilis; Tubercular Phthisis; Locomotor Ataxia; Mitral Disease.—Mr. A., married and about 35 years of age, consulted me first in February, 1897. He had contracted syphilis about seven or eight years earlier. I found him weak, emaciated and ataxic, walking with great difficulty and hardly able to get up or down stairs. Pulse hard and frequent, 125—130. Respiration 20. Temperature rising daily to 101° F., and more. Cough and abundant muco-purulent expectoration; occasional bacillus of phthisis in sputum. Physical examination showed cavities in both lungs and a dilated heart. Apex in 6th space, and outside of nipple. Mitral regurgitant murmur carried to axilla. Patient had been taking digitalis daily, under medical advice, but it was stopped at once. Under treatment by iodides and hydriodic acid he improved so much that a modified series of baths and exercises were given him (Nauheim plan), with electricity. Later he was kept on mercurials and iodides for a period of two years, during which he had no heart stimulants. Under this treatment the lightning pains disappeared, and he gained in health and strength to such an extent that in the autumn of 1897 he was able to resume his business, continuing at it, with only occasional interruptions, up to the date of his last illness in March of 1899. At that time he was taken down with an

[9] Whittaker, *Twentieth Century Med.*, Vol. IV., p. 369.

attack of acute gastritis, to which he succumbed after a few days' illness. The attack was superinduced by causes that were apparently in no way related to the specific disease or cardiac manifestations. Whether in this instance the endocarditis was syphilitic or not I do not know. So far as my records go, it was, except for tuberculosis, the only predisposing cause of which I was aware.

Without the anti-syphilitic treatment the patient was unable to attend to his business, but under the alternate use of both iodides and mercurials, more especially the latter, he led a fairly active life. In this connection it is interesting to know that Schuster,[10] of Nauheim, and others have noticed the connection between tabes dorsalis and heart syphilis. I have now under my care two gentlemen, one a physician, who have both had tertiary syphilis and have been treated for it, one by a well-known practitioner. In neither case did the Nauheim method give the usual relief until the patients were put on specific treatment, when the improvement was comparatively rapid.

We are told by Huchard[11] that *angina pectoris* is a most important sign of cardiac syphilis. In 110 cases collected by him, 32 had a syphilitic history. If it is true that angina is essentially a disease of the coronary arteries, judging by analogy, syphilis, which predisposes to coronary disease, should be an important cause of angina. The therapeutic inference is obvious.

Case XLIII. Syphilis; Cirrhosis; Fatty Degeneration of the Heart.—The following is one of my hospital cases of general syphilis, with fatty degeneration of the heart, in which syphilis figures as the only predisposing cause: A man of 46 entered the hospital with swollen feet, cough, jaundice and dyspnœa. A soft systolic bruit was heard at the apex. The heart was found to be enlarged. At the post-mortem examination the pericardial sac was seen to contain 10 ounces of clear serum, and there were several milk patches on the heart. There were no valvular lesions. The liver was cirrhotic and there was chronic diffuse nephritis. At the autopsy the murmur was attributed to the fatty degeneration of the heart. It is in cases like these where the cardiac lesions are possibly due to syphilis, that we still lack conclusive proof. It is to be hoped that we may soon have some method, chemical or biological, by which syphilis can be recognized at any stage. It is not unlikely that the milk patches in this instance were continuous with syphilitic sclerosis of the heart walls.

The occurrence of syphilis as a factor in heart disease is probably

[10] Schuster, *Deutsch. Med., Woch.*, Oct. 8, 1903.
[11] Loc. Cit., p. 798.

not only more frequent than has been supposed, but the actual cause of death in many instances. Runeberg's[12] statistics, which have been widely read, indicate this: He found in the experience of a single life insurance company that out of 734 deaths, at least 84, or about 11 per cent., were of persons who had contracted syphilis. Twenty-two of these deaths were attributed to progressive paralysis, and 33 to disease of the central circulatory system (that is, of the heart and aorta), 24 of the latter dying of syncope; so that the danger of death from a syphilitic heart or aorta was greater than from syphilitic disease of the central nerve system, and *sudden death* was the rule.

In looking over my private cardiac cases I find that syphilis was *positively* present in about 5 per cent., and *probably* in another 5 per cent.; the inference being that in cardiac disease we should suspect that syphilis is a factor in at least 10 per cent.

The prognosis is bad, but not altogether so. If the diagnosis can be made early and the proper treatment instituted, some success may be expected. Even in advanced cases where, for example, there is tabes dorsalis, improvement can sometimes be effected, as is shown in Case No. XLII. Yet, notwithstanding that we may be able to remove the deposits by medicine, a something will remain, so that if the part resumes its physiological activity it still may not be sound pathologically.

We are obliged to conclude, therefore, that cardiac syphilis is more common that has been supposed. Like syphilis of the lungs, it exists, and the physician who fails to appreciate either of them falls short of his duties as a practitioner of medicine. In fact, neither heart nor lungs should be examined without always holding in view the possibility of syphilis, as the cause of the disease. Where it may not be possible to make a positive diagnosis, a probable one can often be reached. Appropriate treatment will confirm it. Cardiac syphilis is an insidious disease, and its manifestations are neither pronounced nor distinctive. For this very reason physicians in making inquiries and in physical examinations should pay particular attention to the subject of syphilis. A cure may be possible, while relief is probable.. Iodides and mercurials are the proper remedies, but mercury is the sheet anchor. Sometimes both of them must be given for months and even years, with brief interruptions of a few weeks or so. If in such cases the physician fails to recognize the existence of syphilis, he should not be surprised if his patient is carried off, without warning, from sudden heart failure.

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[12] Runeberg, *Deutsch. Med. Woch.*, 1 u. 2, 1903.

Chapter XIII.

DISPLACEMENTS OF THE HEART.

Displacements of the heart are indicated in a general way by the position of the apex beat. This, in the well developed adult (in the standing position, with respiration suspended) is about 3½ inches from the median line and in the 5th left intercostal space. More accurately, however, under similar conditions and in the male it lies midway between the nipple and the tip of the xiphoid appendix, and the distance of 1½ to 2 inches inside the nipple line. In persons of slight build the apex may be only from 2 to 3 inches from the median line, however, so that the method of fixing 3½ inches from the median line as the normal point for the apex, is obviously improper.

A certain amount of motility of the heart is physiological, because while it is fixed firmly at its base, by the great vessels, the organ itself hangs loosely in the pericardial sac. Thus if the patient, after lying on his back, turns over on the left side, the apex will usually be displaced an inch or more to the left, and sometimes as much to the right, if he turns over to that side. In forced inspiration, also, the diaphragm descends, carrying the heart downwards, while in expiration, the diaphragm moves upwards, and the apex goes up with it. This excursion of the diaphragm may be as much as 2 inches; but, strictly speaking, there is no rotation of the organ.

In some cases the impulse is best felt if the patient leans over forwards. For example, in pericardial effusions, where the impulse is wanting it is natural that gravity should cause the heart to displace the fluid, and as a matter of fact, when the patient leans forward, the apex comes nearer to the anterior wall of the chest.

Displacements have been divided into the (1) *intrinsic* and the (2) *extrinsic*. In the intrinsic the displacement is due to the enlargement of the ventricles, the extrinsic to external causes. Undoubtedly under certain conditions, as in uncomplicated aortic stenosis, which has not been compensated, the dilated left ventricle operates to displace the apex to the right. So in well developed lobar pneumonia, dilatation of the right ventricle will tend to displace the apex to the left. Such a state of affairs, however, is short-lived, and limited to the duration of the conditions named; nor is the displacement of high degree. Yet, in exceptional instances, as in

extreme general dilatation, the apex may assume a very unnatural position. On the other hand, in the extrinsic form there are very high degrees of displacement. Here the heart may be displaced in any direction, *upward* or *downward, laterally, forward* or *backward*. The vertical and lateral displacements are the most common, however.

The heart is *pressed upward* by gastro-intestinal distention, ascites, gas in the peritoneal cavity, abdominal tumors, the pregnant uterus, even by an enlarged liver[1] or spleen; or it may possibly be drawn up by the fibrous contractions of pulmonary or pericardial disease. Gibson has reported the case of a diabetic, where the upper margin of the organ corresponded to the lower margin of the 4th left rib. In this case the lungs were also pushed upward.

Often in stout people simple distention of the stomach and intestines so presses up the heart as to cause annoying dyspnœa. But in these, and other slowly moving precesses, the embarrassment of the heart does not always cause immediate danger to life. On the other hand, in acute processes, such as sudden distention of the peritoneal cavity from rupture of the intestine, great distress may ensue and and death result, unless surgical relief is promptly given. Fortunately in many chronic cases, like the puerperal and in ovarian dropsy, the abdominal walls easily yield and distending forwards, relieve the upward pressure.

In some cases there is *downward displacement* of the heart, due perhaps to thoracic aneurism, pulmonary disease, tumor or pleuritic effusions, gastroptosis, or general ptosis of the abdominal organs. (Glénard's disease.)

Lateral displacements are very common. They may be produced by pleurisy with effusion, collapse or destruction of lung, cavities, pulmonary diseases, curvatures of spine, tumors, or congenital displacements of viscera. They may not cause much distress, but often are sources of constant pain. Pleurisy with effusion is a very frequent cause. As it comes on slowly or rapidly, so the danger to the heart is inconsiderable or great. The heart may be moved to the left or the right. In right-sided pleurisy the apex is carried towards the left axillary line. In left-sided pleurisy, however, as a rule, the apex seldom extends beyond the middle line. In one instance, however, recorded by Walshe, the apex was carried under the right nipple.

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[1] In one of my cases of enlarged liver the apex beat was seen to be above the level of the nipple.

The heart may also be carried forwards by thoracic aneurism or by tumors of the posterior mediastinum. It may be displaced backwards in pericarditis with effusion, in pulmonary or pleuritic diseases, and in tumors or abscesses of the anterior mediastinum.

In any case, if the displacement is gradual the subjective symptoms may be insignificant; if sudden, there may be præcordial pressure, even angina, palpitation and dyspnœa, causing collapse and possibly death.

Lateral displacements are also easily recognized by the position of the apex beat. Percussion and the discovery of the apex, either by its impulse or by the stethoscope, makes the diagnosis easy. It may be confirmed by the X-ray, as in Case XCV. But prognosis and treatment depend on the causes. Intrinsic displacement calls for the treatment of the underlying valvular condition. Acute dilatation offers good ground for hope, if the condition is recognized early and the proper measures are taken. In chronic cases more or less relief is to be expected. These matters are discussed at length in the chapter on the General Management of Heart Diseases.

The prognosis of displacement due to extrinsic causes depends on the success which attends the management of the underlying diseases. The operation of thoracentesis will give temporary relief in pleural effusions, perhaps it will be curative. Tapping for ascites will also give relief. So also will the removal of an ovarian tumor, or a normal parturition, where displacement is the result of pregnancy.

The prognosis when displacement is caused by tumors or aneurisms is unfavorable. In chronic lung affections no permanent gain can be expected by treatment; nor in chronic incurable conditions, like an enlarged liver. Where the displacement is due to enlargement of the spleen, the prognosis is better. In curvature of the spine much relief is afforded by suitable exercises. The following are illustrative cases of extrinsic displacement:

Case XLIV. Uræmia: Adherent Pericardium: Sero-Purulent Pleurisy: Displacement of Heart to the Right.—C. B. was admitted to hospital Jan. 3rd, 1881. He was emaciated, anæmic and dyspnœic. Oedema of feet. Scanty urine. Temperature normal. Pulse 112. Respiration 40. On physical examination a large amount of fluid was found in the left side of the chest. Apex of the heart to the right of the sternum. Under treatment the urine increased to an average of 18 ounces per day.

An operation revealed a sero-purulent fluid in both pleural cavities. The patient died of uræmia. At the autopsy 100 ounces of

a sero-purulent fluid was taken from the left pleural cavity, and 15 from the right. Adherent pericardium.

Case XLV. Chronic Pericarditis and Endocarditis: Pleurisy with effusion: Displacement of Heart to the Left.—A. H., admitted to hospital Aug. 2, 1885. He was emaciated and anæmic, complained of dyspnœa, dry cough and debility. On examination, the signs of pleurisy with effusion were found; the right side, in the line of the nipple, measuring 18½ inches, to 16½ of the left. There were also indications of pericarditis. Apex of the heart in the 5th space, in the line of the nipple. Aspiration drew off 50 ounces of clear serum from the right chest. The patient recovered from the aspiration, but died 10 days later of heart failure, due, it was thought, to the pericarditis. The pericardial sac contained 34 ounces of clear serum; in the right pleural sac were 32 ounces of a similar serum.

Displacements of the thoracic and abdominal viscera are also natural sequences of lateral curvature, and inasmuch as the deformity is usually most marked in the dorsal region, it is the contents of the thorax that are apt to suffer most.[3] In the early stages of the disease, or in slight cases, the alterations of position or of function in the thoracic organs may be inconsiderable, but in advanced degrees of the deformity unnatural positions of these viscera may so seriously affect their functions, as to disturb both respiration and circulation.

The large experience of Adams[4] led him to say, in speaking of the effect of lateral curvature on the general health: "While no affection can possibly be more variable as to its general symptoms and its influence upon the health of the patient than lateral curvature of the spine," and while "no symptoms such as would interfere with the general health of the patient may be present, even up to the middle period of life, although the evil day is put off, it nevertheless arrives sooner or later, and the patient suffers from functional disturbance of the thoracic or abdominal organs to an extent which leads to the belief that serious disease of these organs exists."

In regard to the functional derangement of the thoracic organs, he further says: "In slight cases of lateral curvature of the spine, when associated with general debility and occurring in girls, it is by no means uncommon for the patient to suffer from palpitation of the heart, with a disposition to fainting, etc., with feeble and irregular pulse." Adams adds: "The opposite view, however, is gener-

[3] See *New York Med. Journal*, Sept. 30, 1899.
[4] *Curvature of the Spine, London*, 1882.

ally taken by those who see but few cases of spinal curvature, and the enfeebled condition of the general health, together with the associated functional disturbances, are regarded as the primary and essential affection, to which the spinal curvature is merely secondary; but," he continues, "in the more severe cases of lateral curvature of long standing, where the heart becomes displaced and to some extent, perhaps, embarrassed by the contraction and deformity of the chest, palpitation, with some irregularity in its action, may become a more prominent symptom and lead to the suspicion of the existence of heart disease." Adams also quotes John Shaw, of London, who stated that he "had been consulted by several patients who had been treated for disease of the heart, though all the symptoms were caused by distortion of the spine," and adds, "I have known several patients about the middle period of life, afflicted with severe spinal curvature, so impressed with the idea that they were suffering from disease of the heart that the highest medical authorities failed to remove such impression." And he concludes: "Many similar cases, and several in which the symptoms referable to the interference with the functions both of the heart and lungs have been much more severe, have been under my observation in hospital and private practice." As an illustration of the unnatural division of the thoracic cavity in lateral curvature, he alludes to a specimen in the collection of the Royal College of Surgeons in London, where the space between the bodies of the vertebræ and ribs was reduced to three-fourths of an inch.

Bouvier[5] also speaks of a case where the heart could scarcely find room for itself in the narrow space left by the lung, and in fact it appeared to be forcibly applied to the thoracic wall.

These statements from well-known orthopædists, widely separated in locality and spheres of influence, go to show that there is an established belief that lateral curvature tends to disturb the functions of heart and lungs.

Indeed, Adams appears to approve of Bouvier's statement that "individuals thus affected rarely live to old age, and fall victims either to phthisis or heart disease." How far this is true I am unable to say, but I have seen a number of cases in which it seems to me that the curvature was the principal factor in determining cardiac and, associated with it, pulmonary disease. Conversely, I may add, I have seen improvement in the curves of the spine, and correspondingly, an improvement in the position of the heart, so favor-

[5] *Leçons cliniques,* Paris, 1858, p. 145.

ably affect the circulation, respiration, and general health that I am disposed to believe, with Adams, that the lateral curvature is, in some cases, at least, the *fons et origo* of the functional disturbance, rather than the cachexia which is so apt to attend it. My experience has been chiefly, but not altogether, confined to girls between the ages of twelve and twenty-four. Lateral curvature is comparatively infrequent in boys and young men. The chief curve I have usually found in the dorsal region and to the right. The heart has in all these cases been displaced, the apex varying in position from a point three-fourths of an inch to the right of the nipple to a point an inch to the left of it. I have found the usual difficulty in determining the causes. Phthisis, pleurisy, especially of the suppurative form, post-scarlatinal paralysis, rickets, and the carrying of unusual weights have appeared to be determining causes. While pain is an acknowledged sign of curvature, I have not found it constant or confined to any special locality. It may be referred to the spine, or to the parietes of the chest or elsewhere. It is usually on the side of the principal curve. Palpitation and dyspnœa are not uncommon.

My general plan of treatment, varying according to individual cases, has been the employment of—

1. Resistance exercises with forcible pressure.
2. Carbonated brine baths.
3. Massage.
4. Faradism.
5. Nutrients.

My system of exercises is a little on the lines of the system laid down by Bernhard Roth, of London.[6] I give his plan in brief in order to compare it with my own. It is as follows:

1. The patient lies on the back, with arms to the sides; hands supinated. Several deep inspirations are then taken.
2. The patient in the same position extends the arms above the head and inspires deeply several times.
3. In the same posture the patient rotates the head, and flexes it laterally.
4. In the same position the arm is circumducted.
5. In the same position one hip is circumducted, then the other.
6. In the same position the patient extends the arms forward and backward.
7. The patient lies prone and circumducts one hip, then the other.

[6] *British Med. Journal*, May 13, 1882.

8. The patient now sits on the couch and the ankle is circumducted.

9. The same, but the operator resisting.

10. The patient sits astride a narrow bench or couch prepared for the purpose and flexes the trunk, the operator resisting.

11. The patient grasping pegs on a pole, the operator rotates the pelvis.

12. The patient lies on a couch with the head projecting, while the head is flexed by the operator.

These exercises appear to be in the right direction, but are mild as compared with my own. They would be chiefly applicable, I think, in the earlier stages of the disease or in slight curves. I should prefer the more elaborate and scientific method laid down by Dr. Sayre[7] to Roth's system. Assuming that the scheme of exercises should vary somewhat according to the nature and extent of the curve, I now give my ordinary method in the fully developed double curve.

The system is mainly that of Professor Hartelius, of Stockholm, as modified by Dr. H. V. Barclay and myself. The first step before each exercise is to make the patient assume a posture that in itself tends to reduce the deformity, and have this position maintained so far as possible during the exercises.

1. The patient, usually a young girl, standing with the hands to the side, raises both arms (fully extended) laterally upward, so as to be parallel. At the same time she raises herself on her toes. The arm movement elevates the scapulæ and the ribs, pulls on the spinous processes of the vertebræ, turns them upward and toward the median line, and in so doing elongates the spine. Put a patient with this special deformity on a measuring block, let her go through this motion, and the spine will be seen to elongate perceptibly. I have seen it elongate two-tenths of an inch. This movement of raising the body on the toes tends not only to elongate the whole body, but also to correct the position of the pelvis.

2. The patient, resting against a suitable support, such as the horizontal bar of the gymnasium, applying the front of the pelvis to the bar, extends the arm corresponding to the dorsal concavity upward, the operator resisting. The low shoulder is thus raised, while the dorsal and all compensatory curves in lumbar and cervical reigons are more or less straightened out.

3. The patient, standing in the same position and with the same

[7] *N. Y. Med. Journal,* Nov., 1881.

support, places behind her head the hand of the arm corresponding to the dorsal concavity. The other hand grasps the hip of the other side. Now the patient flexes her body at the hip joint, and then erects herself, carrying the body a little beyond the vertical line. The operator stands behind, places his hands on the convexity of the dorsal and lumbar curves and makes lateral pressure, resisting the erection of the body.

By this method the patient, having her spine supported, and in a measure straightened by the operator, the erectors of the spine and the lumbar and gluteal muscles, are brought actively into play.

4. The patient now suspends herself by the hands from the ordinary horizontal bar, which is raised at one end a peg or two higher than the other, so that when the patient hangs by both hands the low shoulder is raised. The weight of the body is now thrown to the side of the convexity, taking the strain off the muscles over the convexity, while the spine is more or less straightened by the weight of the body and extremities. In this position the patient flexes the head backward, the operator resisting. This latter movement tends to reduce the secondary curve in the cervical region, by bringing the muscles of the neck into play equally on both sides.

5. The patient now places herself prone on a flat table with the arm corresponding to the dorsal concavity stretched forward. She then raises the corresponding leg and opposite shoulder. This exercise flattens the "hump" in the back. Under this combined movement the dorsal curve recedes very perceptibly during the exercise in the direction of the median line.

6. The patient lies in a semirecumbent position on a couch with the legs hanging over the extremity. The arm on the side of the dorsal concavity is placed behind the head; the other hand grasps the opposite hip. The operator now fixes the patient's knees, while she erects her body.

7. No. 7 is a repetition of No. 3, but in a sitting position, the knees being supported in front. This support steadies the pelvis.

8. The patient on the couch, with the upper hand over the head. lying on the side corresponding to the lumbar concavity, raises the leg as far as possible. This movement, which may be resisted, tends to eradicate the lower curve.

9. The patient sitting on a chair with her body bent forward, with spine as straight as possible, extends the arm corresponding to the dorsal concavity upward, while the other arm is carried down. These movements are done simultaneously, the operator resisting

each movement. Then the arms resume the original position without resistance. The muscles of the cervical region are exercised by the ascending arm and the dorsal muscles by the descending arm.

10. The patient in the standing position raises the straightened arm corresponding to the dorsal concavity to the perpendicular. The other arm, still straight, is carried backward and inward. These movements, like those of No. 9, tend to straighten dorsal and cervical curves. They should be carried to the limit.

11. The patient, suspended from the bar by the hands, raises both knees. The abdominal and psoas muscles are brought into play, the body being held in a favorable position.

12. The patient, suspended from the bar, extends both legs toward the side of the lower convexity. This exercise tends to straighten the lumbar curve.

13. The patient, standing with leg of side corresponding to the lumbar convexity, in front, and leaning forward, carries both arms backward and inward to the limit. This exercise carries the shoulder blades toward the spine, and tends to remedy their false position.

14. The patient, lying prone across a narrow table, with the arm corresponding to the dorsal concavity extended, the other hand on the corresponding hip, both ankles being fixed by the operator, erects the trunk several times successively.

15. The patient, suspended from the bar, separates the extended legs, under resistance. This movement tends to lessen the lower curve.

16. The patient, lying on a table, and on the side corresponding to the lumbar concavity, with the trunk extended beyond the edge of the table, the ankles being fixed by the operator, with the arm of the depressed shoulder extended over the head, carries the trunk upward to the limit. This exercise is very effectual in straightening the lumbar curves, while the extended arm tends to straighten the dorsal curve.

In the beginning, or with feeble patients, these movements may require assistance by the operator.

In the majority of cases there is advantage in using physical force in the reduction of the deformity. The "hump" should be pressed down forcibly when the patient is lying prone on a hard surface. I have never known this method to give pain; in fact, is is agreeable to the patient.

Forcible correction, the *redressement forcé* of the French, has been recommended and resorted to by various orthopædists. Indeed,

there are, as is well known, appliances adapted for forcible correction. Barwell[8] has described a method which he calls rhachilysis. My experience indicates that if the operator is strong enough, and can give the requisite time, forcible correction can be accomplished with a fair degree of success by manual means without the assistance of any mechanical appliance. But it is not unlikely that mechanical contrivances, for forcible correction, may be used with advantage in correcting certain classes of these deformities, in conjunction with resistance exercises and manual pressure.

If there is also Pott's disease, or a reasonable suspicion of it, and such cases will confront us, forcible correction would, of course, be a very improper procedure in connection with resistance exercises.

Forcible reduction rests the overstretched muscles over the convexity and reëstablishes the tone. The projecting capulæ will sink toward the normal position.

Massage to the muscles of the back is not only a valuable adjunct, but is enjoyed by the patient. It should be given when the patient is lying with the back bared upon a well-upholstered couch, and at the end of the exercises.

The faradaic current may be applied occasionally during the entire course of treatment. It should be employed for a few minutes only, so as to actively contract the muscles of the back. As in any disease, each patient must be treated with a view to the individual case, but the methods described are those that are applicable to ordinary uncomplicated cases with the S-shaped curve.

The use of carbonated brine baths is of material aid in the treatment, the results being more rapidly attained when baths and exercises are combined. In private practice they should not be neglected.

I advocate the use of the warm carbonated brine baths of moderate strength during the entire course of treatment. The strength of the brine should be from three-fourths per cent. to one per cent., and of the carbonic-acid gas from one-fourth to one-half per cent.

The stronger carbonated baths applicable for chronic valvular diseases of the heart are not advisable.

In a majority of cases I use (to supplement the treatment) tonics, such as iron and strychnine, and nutrients, such as cod-liver oil and the malt extracts, and I continue them for long periods. Some preparation of malt I find desirable in nearly all of these cases.

The following are illustrative cases:

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[8] *Lancet*, April 27, 1889, p. 831.

Case XLVI. Spinal Curvature; Anæmia; Cardiac Displacement; Corpulence.—Miss H., aged twenty-four years, Havana, Cuba, a young lady of large build; height five feet six inches and a half, weighing about a hundred and sixty-three pounds, came to me for treatment July 10, 1898, wearing a spinal brace.

September 1, 1898.—On examination it was found that she had, on standing, the usual lateral curve to the right in the dorsal region;

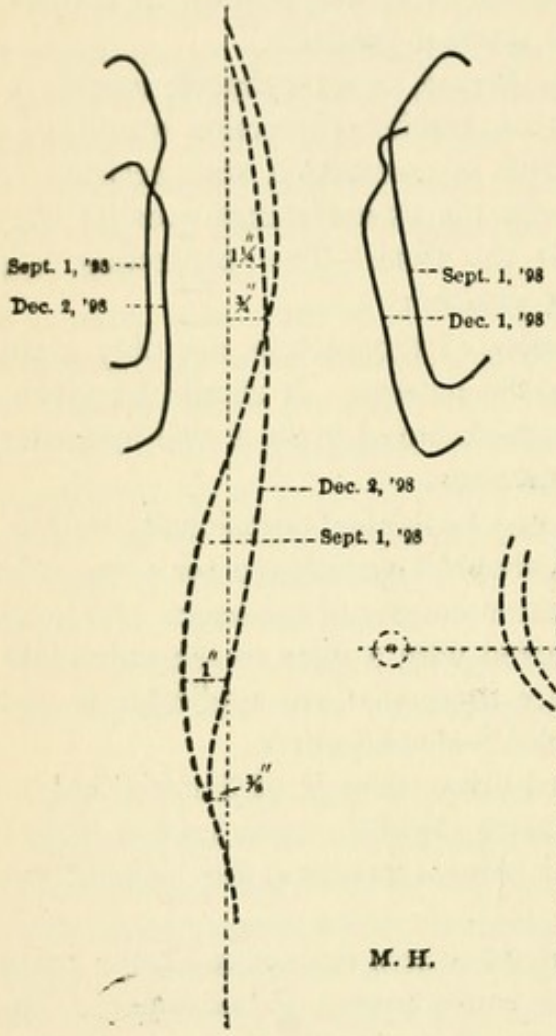

Fig. 16.

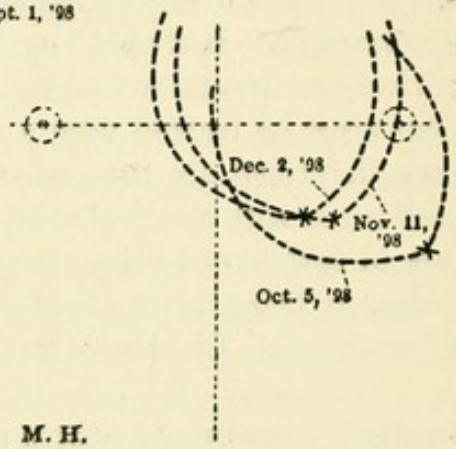

Fig. 17.

pelvis rotated and tilted. Left breast unduly prominent. Pale and anæmic. An excess of flesh, chiefly about the waist and hips. Short-winded on slight exertion, with deviation of about an inch and a half from a straight line in the interscapular region, and deviation of an inch to the left in the lumbar region. Apex of heart an inch below the nipple. Patient given iron and strychnine and the brace removed.

October 19.—One-half per cent. carbonated baths given twice

a week. Resistance exercises daily under direction. Massage and electricity to spinal region.

November 11.—Three-fourths per cent. carbonated baths once a week.

December 20.—Under this treatment, continued for three months, though the course was interrupted by an attack of influenza, the spinal deformity was so far rectified that the greatest deviation from the normal line on standing was only three-quarters of an inch in the dorsal region, and in the lumbar region three-eighths of an inch.

January 2, 1899.—Apex three inches from the median line and one from the intermammary line. The patient was now suddenly summoned to Havana. She also lost about fifteen pounds in four months, a little less than four pounds a month, and was greatly improved in her physical condition.

It will be seen by Fig. 16 that while the spine was being straightened the heart (Fig. 17) was gradually carried inward toward the median line. The apex was brought inwards 2⅝ inches.

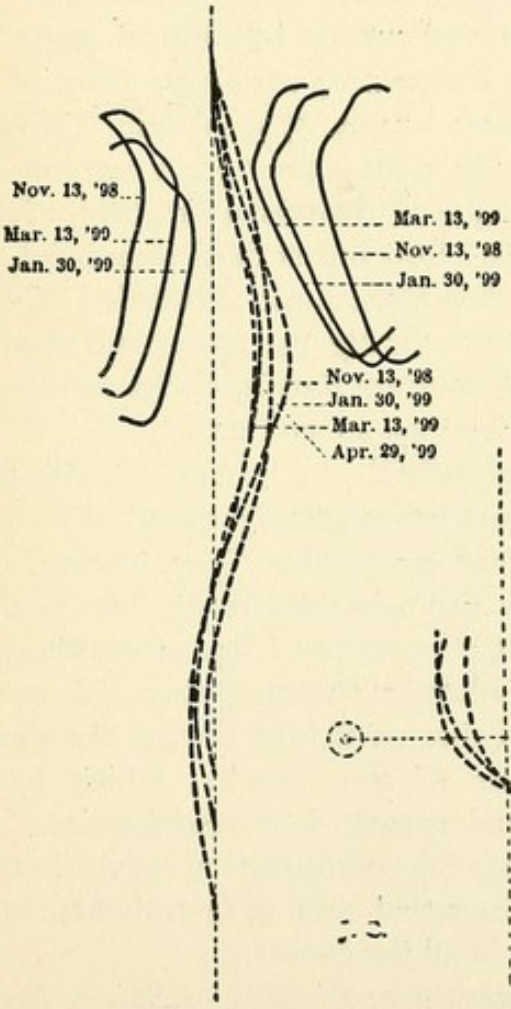

Fig. 18.

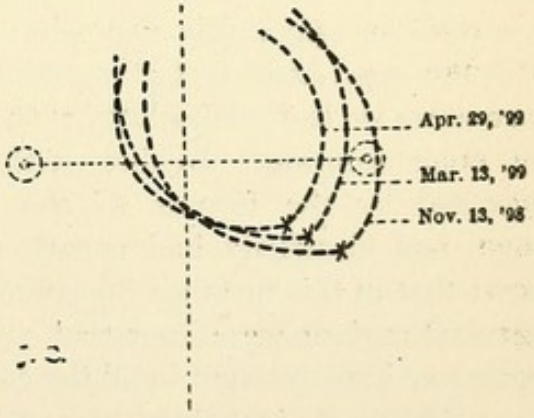

Fig. 19.

After the treatment had been begun the brace was entirely dispensed with.

Case XLVII. Lateral Curvature; Cardiac Displacement.—J. C., a patient sent to me by Dr. E. S. Holt, November 13, 1897. Age, 16 years; weight, 113¼ pounds; height standing, 58½ inches; sitting, 31 inches; chest, 29—31.5. Curvature of the spine noted about five years ago. Had pneumonia and pleurisy since the curvature began; had sore throat often, so had brothers and sisters; was feeble as a child; had the common dorsal curve to the right. Left breast prominent. Often had pain in left side of chest. The left hip higher than the right. The anterior superior spine of left side lower and more forward. Not short-winded; no thoracic pain, but insomnia. Had worn a brace about two years. An hour of resistance exercises were given her daily under my directions for about nine weeks, and then afterwards three times a week. The improvement in the curve and in the position of the heart are shown by Figs. 18 and 19. Patient broadened the chest under treatment an inch and a quarter, and had an inch and a fifth more expansion. Lost two pounds in weight, but improved greatly in physical appearance.

Case XLVIII. Spinal Curvature; Moderate Cardiac Displacement.—K. G., aged 13 years; height, 57.5 inches. Two years ago noticed a crook in her back; thought it came from carrying an infant, possibly from a fall that caused depressed fracture of her skull. Often had pain in right side. Left breast most prominent. Left hip highest; left anterior superior spine most forward. Wore a brace about six months; took it off during the gymnastic treatment and felt better without it. The diagram illustrates the results under resistance exercises. (See Figs. 20, 21 and 22.)

In this case, and in Case XLIX, the tracings, kindly taken by a well-known surgeon of the Orthopædic Hospital, must be studied in connection with other measurements. The tracing of May 8, 1899, shows an apparently excessive deformity, but measurements made in this way, as in fact all systems of measuring these deformities, are more or less misleading, even where they are, as in this instance, quite accurate. In this particular tracing the deformity is exaggerated by the fulness of the muscles, which by exercise, massage, and electricity had greatly increased in size. But Fig. 20 shows that in this instance the compensatory curves in the lumbar and cervical regions were improved, and, in fact, that there had been a progressive improvement in all the curves.

Case XLIX. Spinal Curvature; Rigidity of Spine; Rickets; De-

bility.—L. F., aged 16 years; height, 52.6 inches standing, 28 inches sitting; weight, 81 pounds. The following history was kindly furnished by Dr. Ethel D. Brown, under whose observation she came,

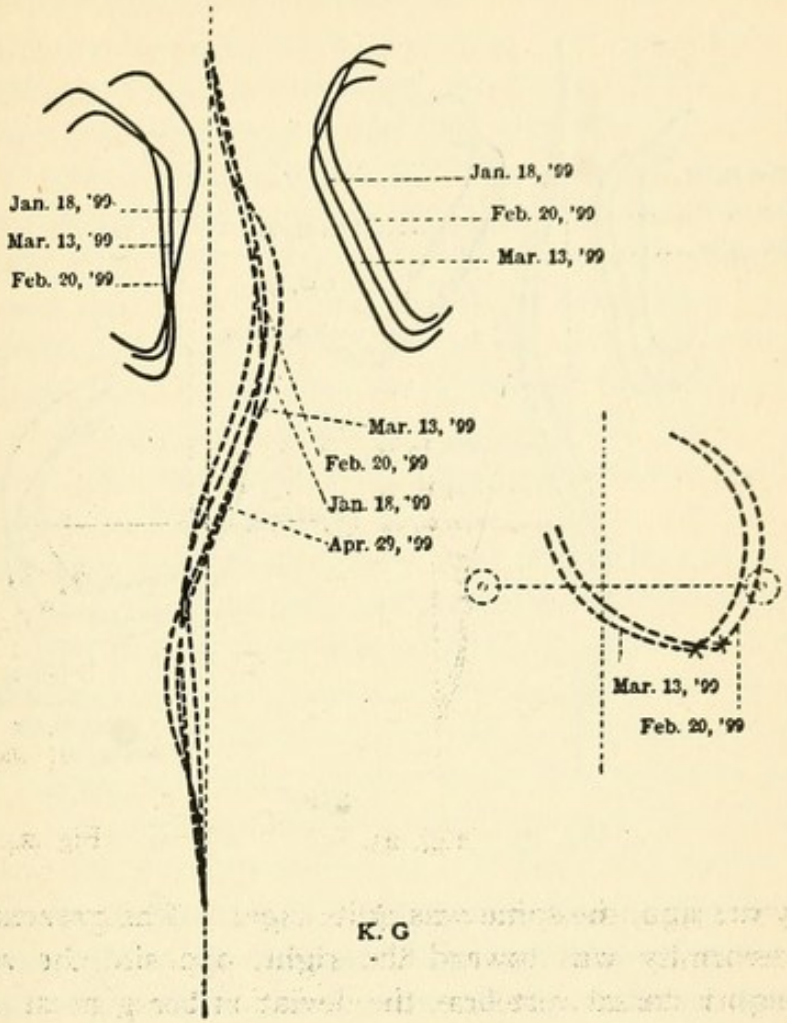

FIG. 20.

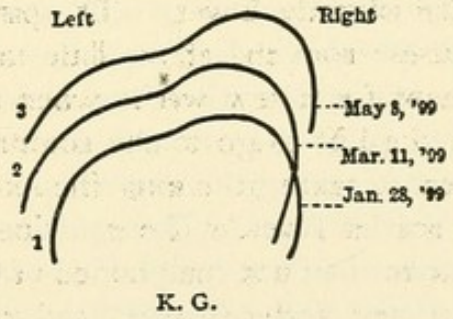

FIG. 21.

FIG. 22.

and by whom she was first seen February 14, 1895. Patient as a baby was weak, and sickly as a young child. The trouble had only been noticed by the family a few weeks earlier than the date mentioned.

The first symptoms she complained of were of pain in the lower part of the spine, and a tired feeling. Then the mother noticed that one shoulder was larger than the other. Even at that time, ten

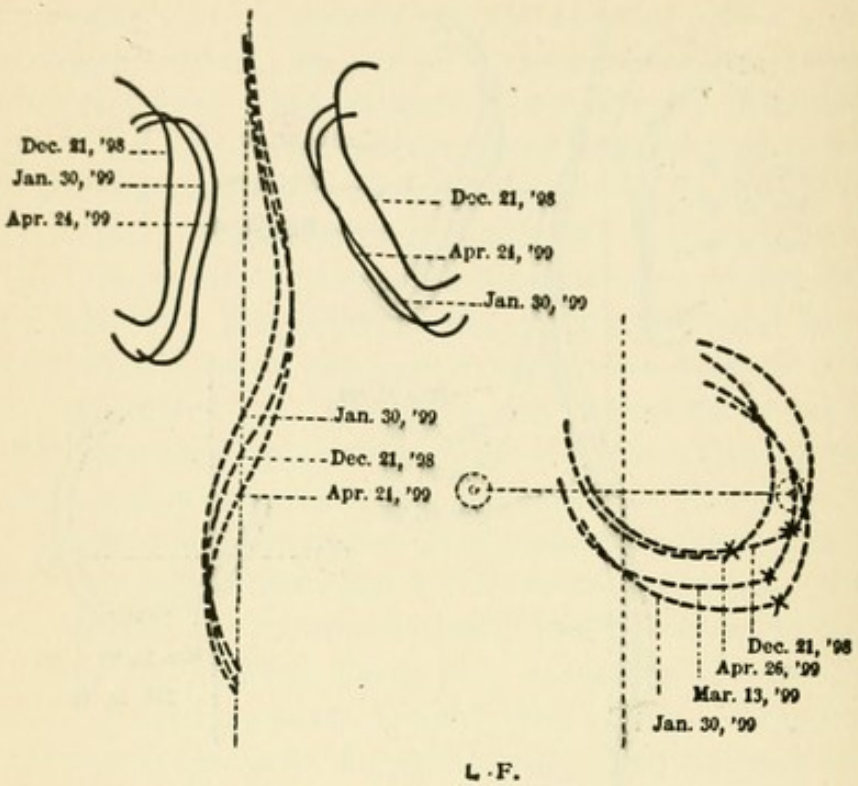

Fig. 23. Fig. 24.

years ago, the spine was quite rigid. The greatest convexity of the deformity was toward the right, opposite the sixth, seventh and eighth dorsal vertebræ, the deviation being at the highest point half an inch from the median line, approximately. Considerable rotation. Left shoulder slightly lower. The patient was put on tonics and given exercises from the above date until the autumn, three times a week, except for a few weeks when she was in the country. In the autumn she left to go to the country, the parents preferring to have her wear a brace, to taking the exercises.

Patient had had scarlet fever. The principal cause of the disease was rickets. Patient had not complained of palpitation, was not short-winded, but suffered from frontal headache. Pelvis rotated and tilted. The crest of the left ilium was about an inch higher than the other, while the anterior superior spine was turned downward and thrown forward. On January 13th patient was wearing a brace. She wore one for a while, when tired, but has now totally dispensed with it. The patient was put on maltzyme, and later on maltine, and has been gradually improving in her general health.

May 16, 1899.—Height, 53 inches; weight, 82¾ pounds. Patient has gained an inch and a half in the chest measurement, and two inches and three-quarters in expansion. The diagrams No. 23 and No. 24 show the changes under treatment. The curves are straightening a little, while the heart's apex has moved inward.

This case was selected as a bad one, in which little, if anything, was to be expected; indeed, such was the deficiency in height, weight, and bodily strength, and so great the rigidity of the spine, with exceeding deformity, that the patient was thought a typical one, in which to test the value of this method in bad cases. In fact, an English authority,[9] when asked whether his exercises would benefit this particular case, replied that he would not think of using them.

The case is also remarkable in several other ways. No baths were given. The treatment was merely resistance exercises with forcible pressure and nutrients.

In measuring a spinal deformity I let the patient stand with the feet together, hands to the side, and shoulders thrown back, the head and body being held in as erect a position as possible. I then trace the spinous processes, the angles of the scapulæ, and the levels of the crests of the ilia on the bare skin with a dermographic pencil and take off a tracing with the ordinary vegetable-fibre tissue paper used by artists. Of course, antero-posterior curves do not show by this method, and yet, I may incidentally add here, that antero-posterior curves are apt to be greatly improved during treatment, as shown by the "popping up" of spinous processes which at first may have been buried out of sight.

But while I have adopted the method of measuring the amount of curves as mentioned, I have not relied wholly on it, but have, as already stated, supplemented it by the lead tracings, as shown by Fig. 22 and also by photographs and X-ray pictures. As regards the scapulæ, it will be seen from the paper tracings that the results of this treatment are that the inner borders are brought nearer the spinal column, and assume more nearly the vertical or normal line.

It is quite unnecessary, I feel sure, for me to state here a fact widely recognized by orthopædists, that every system of measurement heretofore devised has proved more or less unsatisfactory. A few of the objections are as follows: Photography rarely gives one a sufficiently distinct picture of the deformity. X-ray pictures are difficult to make. Lead tracings may exaggerate the deformity, and are therefore deceptive.

The method I employ is also apt to be misleading, I will admit,

[9] Roth.

especially if the patient does not stand as erect as possible; but it is a simple one, and if carefully carried out, is reasonably accurate.

It must not be thought that I am inveighing against spinal supports. I have used them in lateral curvature at various times in my practice, and have been satisfied with the results they have given. But a support is palliative rather than curative. Often the patient finds that he is gradually getting worse, notwithstanding his support. The method I have advocated has, I hold, a much wider application, and is more scientific. It is more or less applicable to all stages of the deformity, and is capable of yielding good results, in remedying both the curvature and the displacement of the viscera, provided the patient is willing and able to coöperate with the physician. While little benefit may be obtainable by this method in old cases, or where there is considerable rigidity, I am satisfied that it is capable of doing something for circulation and respiration, even in them.

Gymnastic exercises, however, such as are given for lateral curvature in the ordinary perfunctory manner in schools and gymnasiums, will not accomplish much, if the patient is a delicate subject, or the case is advancing rapidly. Special exercises adapted to special deformity are requisite. Resistance exercises also are needed, and with them forcible pressure.

Briefly, treatment by the method advocated is the one that gives the largest promise of good results in lateral curvature. Spinal supports are chiefly useful in mild cases as temporary expedients, or as a last resort. Three of the patients whoses cases are here given are still under my care, taking from time to time courses of exercises, which invariably relieve their cardiac symptoms.

Chapter XIV.

PERICARDIAL DISEASES.[1]

Inflammations of the pericardium are closely related to the parts it covers. In a record of forty hospital and private cases, thirty-three were associated with diseases of the heart itself, or with the roots of the great vessels contained in the sac. Pericarditis is most frequently due to some constitutional disease, like inflammatory rheumatism or Bright's disease; less often to a distinctive disease, such as tuberculosis, septicæmia or scarlatina. There may be other causes, such as cold or an injury. From an etiological point of view, it is convenient to divide pericarditis into the *primary* (so-called idiopathic form), and the *secondary*.

The primary form is rare, but cold is certainly a cause of it, and so are injuries of various sorts. It is a well-known fact that needles, pins and other foreign substances, that have been accidentally swallowed, may find their way into the pericardium, causing acute pericarditis.

Secondary pericarditis occurs in from 20 to 70 per cent. of acute rheumatic affections. Next in line, as a casual factor, is chronic nephritis. Following in order of frequency is sepsis, usually from puerperal fevers or bone diseases; then come pneumonia, scarlet fever, measles, typhoid, smallpox and malignant endocarditis. So much for the infective diseases. Or it may be due to such dyscrasias as scurvy, diabetes or alcoholism. Then, it is not uncommon to have the disease a continuation of more remote affections. Thus, pleurisy may extend into the pericardium, producing pericarditis.

In forty of my cases, as taken from my tables, the causes are given in thirty-six. They are as follows: Rheumatism, thirteen; chronic nephritis, eight; tuberculosis, three; pneumonia, two; septicæmia, two; extension of disease, two; alcoholism, two; syphilis, one; scarlatina, one; malignant endocarditis, one; cold, one.

Pericarditis is found at all ages. It even occurs in fœtal life. For some reason, which is not apparent, males are more subject to it than females. Of my forty cases there were twenty-nine males, and only eleven females.

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[1] Originally published in the *Med. Times*, April, 1899.

It is common to make the following divisions of pericarditis:

1. Acute, plastic or dry.
2. Pericarditis with effusion.
3. Chronic adhesive pericarditis (adherent pericardium).

As a matter of fact, these varieties run into one another.

Pericarditis may occur in small or large areas. It may be confined to a very limited space, or may involve the whole serous membrane. A few threadlike bands may stretch over from the visceral to the parietal layer of the pericardium; or the two opposing surfaces may be covered, rub against one another, or become attached. Friction may cause portions of the fibrine of which the plastic deposit is formed, to be separated. These portions will then fall to the bottom of the cavity, to be absorbed, or remain and undergo some sort of degeneration. In tuberculous pericarditis there will be a complete adhesion, eventually, between the opposing surfaces, as the disease progresses. In some cases, and especially in tuberculosis, we may also find an adhesion between the outer surface of the pericardium and the pulmonary pleura.

Acute plastic or dry pericarditis is usually a secondary form of the disease. It is almost always due to some antecedent or concomitant affection of the lungs or heart; or to some general dyscrasia.

Owing to the constant friction of the opposing surfaces of the pericardium, the membrane is apt to have a rough appearance, due to the rubbing of the filaments of fibrin that extend across the sac. When these filaments have any length, they give a "hairy" or villous look to the membrane: hence the name *"hairy heart," cor villosum.* Sometimes serum or pus becomes lodged in the interstices between the fibres or in the meshes of this imperfectly formed connective tissue, giving the whole a honeycombed appearance. At first the heart muscle will retain its integrity, but if the attachment continues, the muscular tissue will begin to disintegrate, at first along the border of the pericardium. In adherent pericardium of long standing, there is invariably myocardial degeneration, a condition which is, of course, irremediable. Where there has been an attachment between the visceral and parietal layers of the pericardium and they become detached, the point of separation is apt to be marked by a deposit of fibrous tissue, the *"milk patch."* There are other ways, however, in which this patch or spot is produced.

Sometimes there are few or no *objective signs.* The patient may, however, complain of præcordial weight or distress; or refer to a pain in the ensiform cartilage. The pain may be steady or periodic,

slight or intense. It may extend up the neck, or down the ieft arm. As soon as effusion occurs, there is a diminution of the pain. It may be caused at first by the friction of the opposing rough surfaces. Distention of the sac with fluid, however, will cause a return of the pain. Occasionally the pain is anginoid in character. Usually there is some fever, but the temperature seldom rises above 102 degrees, though it may reach 108 or more before death. In a single instance, associated with broncho-pneumonia, I knew it to reach 110 at death. Ordinarily the nervous symptoms consist merely of headache; but there may be melancholia or delirium. Among other phenomena I have noted dizziness, faintness and even cyanosis.

Rheumatic pericarditis usually occurs, if at all, during the first or second week of inflammatory rheumatism, but it may precede the general rheumatic attack.

The first of the physical signs is difficult inspiration, but this sign may be absent. Palpation over the pericardium, however, may reveal a fremitus. If felt, it is usually over the right ventricle, the most anterior portion of the heart. There may be a thrill. The heart's action is apt to be irregular, even tumultuous. Auscultation may reveal friction sounds, the most distinctive of the signs. It is a double sound, not perfectly synchronous with the heart, and somewhat more prolonged. Yet these friction sounds may be mistaken for endocardial murmurs by experienced clinicians. I have known several such cases, as the autopsies have shown. Although usually double, the friction sounds may be single or triple. They are in every case transitory.

The sound may also have a peculiar rubbing or creaking sound, like that of new leather, or it may be musical. It is always superficial, close to the ear, and intensified by the pressure of the stethoscope on the fourth and fifth interspaces.

Friction sounds may be heard up and down the sternum. They are often best noted over the aorta, but may be heard at the apex. Unlike valvular murmurs, they are not transmitted in definite directions, while the intensity depends on the position of the patient. They are best heard when he takes the upright posture. The pulse may be feeble, rapid, irregular, paradoxical or dicrotic. It may be as slow as 56 or as rapid as 130, in my experience.

Another symptom is the epigastric pulse, but this is not distinctive. It is seen in Graves' disease, and in other nervous conditions.

The great difficulty in the diagnosis of acute plastic pericarditis

is that it is apt to be obscured by attendant pulmonary or cardiac diseases, which may completely mask or overshadow characteristic symptoms.

The course of an attack of acute pericarditis will vary. It may last only a few days; or it may take on the sub-acute form, passing into pericarditis with effusion.

On the whole, the friction sounds of pericarditis are not difficult to recognize, and should not be mistaken for organic valvular murmurs; though, as mentioned, this mistake has sometimes been made by our most prominent clinicians. It is more difficult sometimes to distinguish between pericardial and pleuritic friction sounds. This danger, however, can be eliminated by making the patient take a deep inspiration and then hold his breath. By this act the lungs, if not attached by adhesions, will be retracted widely apart, leaving the pericardium quite uncovered by pulmonary tissue.

Pericarditis rarely kills directly. It is the secondary effects of it, that are to be feared. The fibrine may be absorbed, and that is the end of it, so far as danger to the integrity of the heart is concerned; or it may survive as a scar on the heart's surface, known as the "milk patch." But if, as the inflammation subsides, the two opposing pericardial surfaces unite or cohere, the result will necessarily be hypertrophy and dilatation of the heart, and degeneration of its substance.

In *tubercular pericarditis* there is great thickening of both the parietal and visceral layers of the pericardium, and the acute disease always goes on to the chronic form. Eventually there is more or less a completely adherent pericardium.

The treatment of *acute dry pericarditis* is simple. The patient should be put at rest. Sedatives, such as the bromides, serve a useful purpose. Rubefacients, blisters and dry cups are also indicated. More than these are rarely necessary.

Pericarditis with effusion presents special features. It is usually the sequel to the acute form just described. The effusion may be serous, bloody or purulent, and the amount will vary from 1 to 70 or more ounces. In my experience it is usually from 8 to 15 ounces. This form of pericarditis is common in pleurisy, pneumonia, endocarditis and acute rheumatism. Although, as I have said, it is apt to be a sequel to the acute form just described, the onset may not be appreciated, owing to its insidious character; but there may be pain, which is usually increased by pressure. Dyspnœa and palpitation are also symptoms, when the effusion is extensive.

In such instances the patient will show signs of restlessness, or his countenance will be anxious. The pulse may become rapid—it may reach even to 200, 300 or 400, and be paradoxical; it may be very weak or disappear on inspiration. There may be distention of the veins of the neck. In rheumatic forms it is not unusual to have cerebral symptoms; indeed, there may be delirium and coma, but these will be due to the intercurrent disease.

With ordinary cases of moderate effusion, in adults, there will be no external manifestation of the effusion; but in children, if the effusion is large, there is *bulging of the præcordial region;* and, perhaps, coincidentally, œdema of the chest-wall. On palpation the apex beat may not be found. If a friction sound has been heard, it will usually disappear as the effusion extends; or it will remain for a while, exclusively at the base. On percussion there will be an area of increased dulness about the base of the heart. The shape of this area is peculiar. Unlike the area of cardiac dulness, the outline will be that of a pear or truncated cone, with the base below the sixth rib and the apex at or above the second interspace. I have know, in an exceptional instance, the transverse width of the pericardial effusion to be eight inches, and reach from nipple to nipple. As the effusion increases, the heart sounds become more indistinct. Whenever the pericardial friction sound is distinctly heard, the physician should anticipate a possible effusion, and at once demarcate the heart's area with a dermographic pencil, so that any increase in the area will be shown, and he may thus be able to note the progress of the effusion.

The effusion may extend rapidly, causing suffocative symptoms, or may subside very rapidly. Even exudations of pus, if small, may sometimes be absorbed. Such septic cases, however, sometimes terminate fatally, in a few hours. On the other hand, large serous effusions developing slowly may be borne fairly well for several weeks, giving ample time to institute remedial measures.

In general, serous effusions disappear of themselves without special treatment directed to the effusion. In the purulent or hæmorrhagic forms, of which I will speak later, the disease is usually fatal, because the constitutional and concomitant diseases, which are the more serious, are of a fatal character. In rheumatic cases the prognosis is apt to be favorable. In other cases of serous effusion the prognosis is more uncertain.

It is not always easy to distinguish between dilatation of the heart and pericardial effusion, especially in stout people and in women

whose breasts are large. In dilatation, from whatever cause, there is a waving impulse of the heart, and the apex is better felt generally; though in many cases of fatty heart it may not be appreciable. A point of great importance, however, is the configuration of the dull area. A skillful percussor can in most cases outline the heart's borders sufficiently well to show the general contour. In enlargement, the heart is shaped like a pear, with the small end directed downward and outward; while in effusion, when there are eight ounces or more, the form of the sac, as marked out on the chest wall, will be pear-shaped, with the base directed downward ond outward, and the small end directed toward the manubrium. Change of position in the patient makes very little difference in the area of dulness in enlarged hearts, while in pericardial effusions a considerable difference is generally apparent. If physicians would practice demarcation of the heart by percussion, it is probable that they would learn to avoid the mistake that is sometimes made of confounding hydro-pericardium with a large heart. The greatest difficulty is often found in distinguishing a pericardial from a pleuritic effusion. We should remember, however, that a large pericardial effusion will compress the lung, much as a pleural effusion. In both, the lungs will be compressed if there is much fluid, but in pericardial effusions compression will be mainly exerted on the lower lobe of the left lung, forcing it towards the left, and if this lobe is much compressed there should be increased vocal resonance and bronchial breathing, heard from behind.

Both in pericardial and pleural effusions the respiratory sounds are feeble whenever the action of respiration is interfered with. It is well to remember that in rheumatism the effusion is apt to be serofibrinous; in tuberculosis it may be fibrinous or fibro-purulent; in infective diseases, such as malignant endocarditis, it will probably be more or less hæmorrhagic. In the effusions of old people there is also a tendency to the hæmorrhagic form. Some curious mistakes are shown by my hospital records. Thus, I have known enlargement of the left lobe of the liver to be mistaken for pericarditis with effusion. So small an amount of liquid as 4 to 6 oz. will not ordinarily be detected, and yet it may be assumed that if effusion takes place in a case of acute heart disease, the amount of liquid in the sac will be at least from 4 to 8 oz. Unless we resort to aspiration, however, there is no way of distinguishing between pericarditis with a serous effusion and hydro-pericardium, due to dropsy of the sac.

In the matter of treatment, absolute rest in bed is the first indi-

cation. In extreme cases, wet cups to the præcordium may be advisable for plethoric people, but my experience tells me that they should seldom be resorted to in any instance. Dry cups and rubefacients are in order, however. The ice or cold-water bag are recommended by continental physicians, but at best they should only be used for temporary relief. The diet should be carefully regulated, so as to prevent gaseous distention of the stomach, which adds greatly to the pericardial distress. The physician should address himself promptly to the kidneys and skin, in order to relieve the system of its superfluous fluids, and also to the heart itself; and digitalis, which is both an established heart stimulant and diuretic, will here hold the first rank. It should be given in doses of 1 to 2 grains of the substance two or three times a day, or by tincture or infusion. Acetate of potash has achieved an excellent reputation in cardiac effusions. It is both a diuretic and a diaphoretic. From 10 to 30 grains may be given every four hours. In the smaller dose it is a diaphoretic. A common remedy is the pill of calomel, digitalis and squill, one grain each, to be taken three times a day. An excellent remedy is Basham's mixture, consisting of the chloride of iron, dilute acetic acid and the acetate of ammonia. It is both a diuretic and diaphoretic, and never produces alarming symptoms. If the disease has taken on a sub-acute form, iodide of potassium, in 7 to 10 grain doses three times a day, will tend to absorb the fluid. It will sometimes act very efficiently, though slowly, in very large serous effusions. Ordinarily alcoholics, and perhaps diffusible stimulants, or heart tonics, like strychnine, in doses of from 1-60 to 1-25 grains, may be found desirable. In great præcordial pain and distress opium may be necessary; or trional, in doses of from 15 to 30 grains. Paracentesis may be applicable in some cases. In sixty cases collected by Roberts there were twenty-one recoveries after the operation. There are different views as to the proper site for the puncture. Some recommend introduction of the aspirator needle in the fourth or fifth interspace, 2 to 2½ inches from the left edge of the sternum (MacDonnell). Roberts insists that the best spot is the fifth left interspace, 2 to 2¼ inches from the median line of the sternum. Bristowe says the preferable site is at the inner end of the fourth or fifth left interspace, close to the sternum. He suggests the use of a fine aspirating needle, first, to determine the character of the liquid. Rotch recommends the fifth space, to the right of the sternum. Others advise the third left space, 1½ inches from the sternum. The important point is not to injure the internal mam-

mary artery, which runs along the edge of the sternum until it reaches the sixth rib, where it bifurcates, continuing downward and outward in the sixth interspace. The operation should be done slowly and carefully. It is not demanded except in rare cases. According to MacDonnell, the normal pericardium can only me made to contain, under pressure, about 10 oz. of liquid, but according to Stokes, 36 oz. may not produce dysphagia, and yet a sudden discharge of a few ounces of blood or stomach contents into the sac may cause sudden death. Sometimes there may be *pus in the pericardium*, either by itself or intermixed with the serum and fibrine or blood. It is always secondary, and due to some deposit of pus elsewhere, as, for example, from an abscess of the liver, pyæmia, or septic peritonitis. In fact, if any accumulation takes place in the pericardial sac in the course of these affections, is is pretty apt to become *purulent*. The remedy for such a condition is incision, free drainage, and antiseptic injections. It is hardly worth while to expect that a cure will follow aspiration, even if it is frequently repeated. Some little success has attended the operation by thorough drainage. While a very small amount of pus in the pericardium may be absorbed or calcified, a large amount will inevitably lead to a fatal issue, unless free incision be resorted to. When blood is effused in the pericardium, it is the result of injury to the pericardium, heart or great vessels, or it may be the result of disease in some adjacent part, such as pneumonia. It is apt to be associated with aneurism of the first part of the aorta. The only way of determining whether blood is present or not, is by aspiration. The amount is usually small. Large or sudden hæmorrhages may cause the heart to stop suddenly. While this kind of effusion has in some instances been relieved by aspiration, the accumulation is so rapid and unexpected that the patient may be dead, before his malady is even suspected.

The pericardium usually holds from half an ounce to an ounce of a greenish-yellow serum. When a considerable quantity is present in dropsy cases, we have *hydro-pericardium*, or pericardial dropsy. This may be borne satisfactorily, if the effusion is not too rapid. In the following case of acute hydro-pericardium it appears to have caused death, though the fluid was only eight ounces in amount.

Case L. Acute hydro-pericardium: Meningitis.—L., 30, Ireland, was admitted to hospital Sept. 23, 1881. He had been made to walk to the station-house under the supposition that he was intoxicated. He was then taken to the hospital in an ambulance. On

admission he was found to be moribund. Pulse almost imperceptible. Heart sounds indistinguishable. Apex beat faintly felt, one-half inch to the left of the nipple. Cardiac dulness increased. Urine negative. He was temporarily revived, but died suddenly on the same day, after making a slight exertion. At the post-mortem examination the pericardium was found to contain eight ounces of a clear serum; but there were numerous ecchymotic spots beneath the visceral and parietal layers of the pericardium, especially over the roots of the great vessels. There was also a small extravasation of blood in the cardiac muscle near the apex, just beneath the visceral pericardium. On the heart were a number of milk patches, and some thick fibroid deposits of comparatively recent date, with fibrous bands stretched across the upper part of the pericardial sac. The heart was hypertrophied and weighed 24 ounces. Its cavities also were all dilated. Some tricuspid insufficiency (relative), but otherwise no valvular disease. Muscular substance pale, soft and flabby. Liver enlarged and congested. Serous effusion within calvarium and spinal canal. Meninges of brain thick and opaque.

The sudden death was attributed, and correctly, I think, to the acute hydro-pericardium.

The following is a case of acute fibrinous pericarditis, or *"Hairy Heart":*

Case LI. Acute Articular Rheumatism: Acute Pericarditis: Hairy Heart.—M., 39, was admitted to hospital Nov. 19, 1883, suffering from acute inflammatory rheumatism. Heart's action irregular. About the 18th day double friction sounds were heard over the pericardial region, and continued for three days. Notwithstanding active treatment by means of the salicylates, digitalis, and external applications, including blisters to the pericardium, acute delirium supervened, and he died on the 11th day after admission, in semi-coma. At the post-mortem examination the pericardial sac was found to contain 15 ounces of clear serum. Both visceral and parietal layers of the pericardium were covered with the papillary growths characteristic of the "Hairy Heart." At the apex the two pericardial surfaces were connected by a few bands.

The following is a case of acute purulent pericarditis:

Case LII. Lobar Pneumonia: Endocarditis: Pyo-Pericardium: Pleurisy with Effusion.—R., 21, male, was admitted to hospital Feb. 15, 1877, suffering from lobar pneumonia. Pulse 92, temperature 101° F. The patient did well for five days, but on the 20th the temperature and pulse rose, the former to 105° F., the latter to 130.

On the following day he was delirious, and died, suddenly, in the evening. At the post-mortem examination was found some adhesive inflammation of the pericardium, which contained two ounces of pus. Heart dilated and hypertrophied. Myocardial degeneration. Vegetations on the aortic valves. Liver large and congested. Pneumonia in the stage of resolution. Pleurisy with effusion. The precise cause of the sudden death was not apparent. It was probably due to the pneumonia.

The following is a case of chronic fibrinous pericarditis (adherent pericardium), showing how it may pass unnoticed, even under the eye of an experienced clinician:

Case LIII. Lobar Pneumonia: Adherent Pericardium.—C., 36, was admitted to hospital March 21, 1888, after exposure to cold and wet. A few days previously he had a chill, with pain in his side, and on the next day cough and a brownish expectoration, with some dyspnœa. On examination he was found to have pneumonia. Heart's action feeble. Temperature, 104½° F. Albumin in urine, 5 per cent. During the few days he was in the hospital his heart never acquired any additional force. He died of heart failure. At the post-mortem examination the pericardial sac was found to be completely obliterated by old adhesive inflammation. Cardiac hypertrophy and dilatation. Red hepatization of left lung. Some pleurisy with effusion on both sides. Enlarged spleen; hob-nailed liver; chronic nephritis. The adherent pericardium was only one of the contributing causes of death which was due primarily to pneumonia and uræmia.

Hydro-perocardium occurs frequently in chronic Bright's disease, and sometimes in scarlet fever. The diagnosis is a matter of inference, unless aspiration is resorted to; but the clinical history of the case will generally suggest the character of the effusion. Its treatment has been considered under that of pericarditis with effusion.

Pneumo-cardium, or air in the sac, is of very rare occurrence. The causes are, as far as we know, cancer, ulcer of the stomach, and perforation, as from wounds. If there is a large collection of gas, the action of the heart is naturally interfered with, causing dyspnœa, cyanosis and collapse. The ordinary area of dulness is replaced by areas of metallic, or cracked-pot sounds, over the gas. Splashing may possibly be heard. The sounds are like those of a circumscribed pneumo-thorax, but we may distinguish the two in this way: When respiration is suspended the sounds persist. Recovery has taken place in a few rare instances. The treatment is surgical. In

chronic pericarditis, or the *adherent pericardium* (for this is the only form that can persist and be compatible with life), the attachment may be partial or general, as I have already said. Slight attachments, especially over the great vessels, are common and have no great significance. Occasionally a fibrous band or two persist after an attack of acute pericarditis, but they will not excite attention unless the heart's action is interfered with. When there is such a general adhesion that the heart and pericardium are practically united, the consequences are always serious. Several things happen. At first the heart enlarges to overcome the embarrassing effect of its attachment to the pericardium; then it dilates, and later undergoes more or less true fatty degeneration. Obliteration of the pericardial sac, which is not uncommon, especially in old rheumatic cases, is necessarily followed by degenerative changes in the myocardium. There are no distinctive signs, but merely those of advanced heart disease, such as dyspnœa, palpitation, a sense of præcordial constriction, cyanosis and, perhaps, dropsy. In these cases we find an enlarged area of cardiac dulness, with an enlarged area of cardiac impulse, which may extend from the third to the sixth interspace. There is no characteristic wave of the impulse in adherent pericardium, but *retraction of the apex* during systolic is an important sign. Friedreich's sign is also to be mentioned. It is collapse of the veins of the neck during cardiac diastole. It is noticeable in adherent pericardium, that the dulness on percussion may reach the first interspace. On auscultation, the pulse may have a galloping rhythm, or may be paradoxical.

In my experience, however, as derived from a study of my hospital cases, adherent pericardium is seldom[2] recognized during life; and my cases have passed under the observation of our best clinicians. Assistance in diagnosis may be derived from the fact that two-thirds of these cases have been associated with acute articular rheumatism. Pleurisy and tuberculosis are also manifest causes. In many of my cases there was dyspnœa and præcordial pain; the heart's action was weak, and the sounds were muffled. Other signs that I have noted in adherent pericardium were cough and an infrequent pulse. A soft systolic bruit at the apex was not uncommon. Sometimes creaking sounds were heard over the pericardium, as if old adhesions were being stretched.

[2] In fact, in the many cases I have seen at post-mortems, I do not remember that the diagnosis was made in a single instance.

Chapter XV.

FUNCTIONAL CARDIAC DISEASES.

PALPITATION; ABNORMAL RHYTHM;[1] THE FREQUENT PULSE.[2]

Some peculiar cardiac phenomena are conveniently grouped under the titles of Palpitation, Abnormal Rhythm, the Frequent and Infrequent Pulse, and Graves's Disease. They are also sometimes classed under the Cardiac Neuroses—which indicates the paramount implication of the nervous system in their production, without necessitating the adoption of any set theories as to the source or *modus operandi* of the nerve influences. Three of these neuroses will be here described.

Palpitation is the most common of them. It is a subjective phenomenon, a beating of the heart felt by the patient as a disagreeable or oppressive sensation, although the pulse is not necessarily increased in frequency. These characteristics distinguish it sharply from the frequent pulse of so-called tachycardia, which is not necessarily appreciated by the patient.

The cause of palpitation is not always apparent. It may be due to disease of the heart substance, as in the cardiac hypertrophy of Bright's disease; or to functional disturbance of the cardiac nerves, as in neurasthenia; or to some defect in the quality or quantity of the blood, as in anæmia.

In general, we recognize three kinds of causes: the *direct*, the *reflex*, and the *toxic*. The direct are subdivided into the *organic* and the *functional*. For example, in diseases of the spinal cord involving the roots of the pneumogastric; and in pressure on or disease of this nerve, the cause is direct and organic, and many examples of this variety have been observed. On the contrary, in the palpitations of the emotions, such as are due to sudden alarm, distress, apprehension, or even pleasurable sensations, the cause is direct and functional. Palpitations of this kind are common enough in every one's experience. Reflex palpitations also are many and various. Most noteworthy of all, perhaps, are those produced by gastro-intestinal intoxications, the result of absorption of certain

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[1] Allorhythmia, arrhythmnia.

[2] Originally published in *Internat. Clinics*, Vol. 4, Ser. 13, p. 83.

toxins into the system. On the other hand, distention of these organs, the result of disordered functions, may cause pressure on the heart or large venous trunks, mechanically disturbing the heart's action and causing palpitation; or venous congestion due to the various sorts of enteroptoses may bring on a seizure. Diseases of the kidneys, gall-bladder, ovaries, or uterus are also reflex causes, and the cessation of palpitation, when affections of these offending organs have been successfully treated, offers *a priori* evidence in favor of this view.

A special toxic agent is alcohol, and tobacco is prominent in this relation to a marked degree, so much so that the term *"tobacco heart"* is well known and in general use. Coffee and tea, to a less degree, produce palpitation. In fact, palpitation is usually the result of nervous influences, rather than organic heart disease.

Palpitation implies an irregular and sometimes tumultuous action of the heart. If slight, it may only cause a mild discomfort, but the impulse can be so violent as to shake the whole body. While in healthy persons nothing short of a sudden alarm of fire, the shock of a railway accident, etc., will bring on an attack, in neurotic individuals circumstances of an apparently trivial nature, such as a discordant voice, a ring at the bell, or mere apprehension that an accident or misfortune is impending, are sufficient. Under these circumstances the heart may seem to actually stand still.

But palpitation is not a disease. It is merely one of many phenomena, such as a feeling of constriction in the throat, embarrassed respiration, pallor or flushing of the face, præcordial oppression, perhaps cerebral congestion, which are frequently associated together. Lassitude and exhaustion are the sequels of severe attacks. Women, having a feebler nervous temperament, are more subject to it than men. The pulse may run up to 150, or more, a minute. When seizures are induced by gastro-intestinal intoxications they are attended by dyspeptic symptoms, often with distention and eructation of gas.

So far as the heart is concerned, no change will take place in it, provided the attacks are mild and infrequent. If severe or prolonged, however, the organ becomes dilated. But palpitation of itself does not produce murmurs.

In the treatment it is, of course, essential to recognize the cause. If the palpitation is due to Bright's disease, remedies that tend to improve the condition of the kidneys are indicated—such as iron

and spirit of Mindererus, which are well combined in Basham's mixture, as follows:

℞ Tincture of iron chlorid..... 3 drams
Dilute acetic acid..... 2 drams
Simple syrup ..... 1½ ounces
Solution of ammonium acetate, sufficient to make 4 ounces
Mix. S.—One teaspoonful 4 times a day.

Excesses of all kinds must be abstained from. Over-eating and over-drinking are to be remedied by regulating the diet and giving saline laxatives, especially those containing sodium phosphate. Ordinary constipation is successfully combatted by stomachics, or by the use of dilute nitro-muriatic acid combined with a bitter tonic, such as gentian. Either of the following formulas will be found serviceable:

℞ Bismuth subgallate ..... 2 grains
Powdered cubebs ..... ½ grain
Lactopeptin ..... 1 grain
Sodium bicarbonate ..... 1 grain
Mix. S.—One powder to be taken after each meal.

℞ Sodium bicarbonate ..... 1 dram
Powdered rhubarb ..... ½ dram
Peppermint water ..... 2 drams
Distilled water, sufficient to make..... 4 ounces
Mix. S.—Two teaspoonfuls every 4 hours.

During an attack the patient should be made to lean back in a chair or recline on a lounge or bed; cold applications should be made to the chest, and as much fresh air as possible should be given. Aromatic spirits of ammonia in 15- to 25-drop doses, in half a glassful of water, or Hoffman's ether, in half-dram doses, are also excellent. In prolonged cases the bromides are useful. Better than potassium or sodium bromides are the mixed bromides. A combination of lithium bromide 5 grains, ammonium bromide 10 grains, and potassium bromide 20 grains, is more sedative than any potassium or sodium salt singly.

In exceptionally severe cases morphine in one-quarter grain doses may be necessary. Electricity, while capable of doing good if currents of moderate strength are used, and for a few minutes only—the positive pole to the pneumogastric of the neck and the negative pole over the sternum—may be dangerous and should be used as a last resort. The advantages are more than offset by the disadvantages, for a strong current may stop the heart's action entirely. A simple and effectual remedy is counter-irritation to the chest. If the palpitation is due to valvular disease, the treatment recom-

mended for the several forms of this disease is applicable. If it can be positively determined that the palpitation is due to reflex influences, as from a diseased ovary, a floating kidney, etc., appropriate treatment of these organs is essential. After the seizure has passed, it may be necessary to enjoin absolute rest; but in some cases tincture of strophanthus or of digitalis, in 3- to 5-minim doses, may be given until the normal action of the heart has been restored. Usually, however, a simple sedative or cardiac stimulant like camphor monobromate in 1- to 2-grain doses will be safer and quite as effective. A good example of palpitation in organic heart disease is given in Case LIV.

ABNORMAL RHYTHM.

Sometimes the normal rhythm of the heart is lost. This may be due to many causes, such as mental or neurotic disturbances, organic heart disease, *reflex* influences (such as from injuries to distant parts), or *toxic* agencies (such as uræmia,) etc. Two or more of these may co-exist. There are several varieties, such as the *paradoxic* pulse of Kussmaul, which is less full during inspiration, sometimes stopping at the end of a long inspiration. It has been noted in fibrous pericarditis, diseases of the mediastinum, and obstruction of the air-passages. Other varieties have been described in a previous chapter.

THE FREQUENT PULSE.

The *frequent pulse*, like palpitation, is not a disease, but a symptom. It may be physiological or pathological. If merely the accompaniment to or sequel of violent exertion, in which the heart is called on for increased energy, within normal bounds, the frequent pulse is physiological. But from whatever cause, so far as we know at present, increased frequency must be laid to nervous influences.

Experiments on animals show that irritation of the accelerator nerves increases the pulse-rate. Similarly, section of the pneumogastric, or injury to its nucleus, increases the pulse-rate to 150 a minute or over. It produces the continuously frequent pulse. On the other hand, pressure on the pneumogastric will, in some persons, arrest a frequent pulse. Curiously, according to Martius's experiments, when an injury to the pneumogastric causes a frequent pulse, the rhythm is always normal unless affected by reflex influences. However, according to the recent experience of Hoffman, in cases of paroxysmally frequent pulse with abrupt onset, sudden ending,

and arrhythmia, the cause may be the *interpolation of beats.* These interpolated beats, in his experiments on frogs, originated at the venous ostia, as the result of electric stimulation of the veins, causing double the number of ventricular contractions. These experiments seem to have been confirmed by Hering and Gerhardt.

To appreciate the situation better, it may be well to review some of the points bearing on the nerve supply of the heart. First of all, there are three sets of nerves that influence the heart: (1) The pneumogastric slows it and lowers arterial pressure. (2) The sympathetic supplies the accelerator, quickens the action of the heart, and also furnishes nerves that regulate the calibre of the arteries (Mackenzie)[3] (3) The ganglia are the intrinsic nerves of the heart, controlling its systole and contractions. The investigations of von Bezold and Martius show that abstraction of the influence of the pneumogastric permits of an increase of the heart-beats of only from 120 to 180, while irritation of the sympathetic permits of an increase of beats to 120. The action of the ganglia in increasing the heart-rate is indeterminate. While these experiments explain how in lesions of the pneumogastric or its roots, the pulse may reach 150 to 180, they fail to explain higher rates except by the supposition that the combinations of disturbed action of the cranial and the sympathetic nerves may increase the rate beyond 180, and they also fail to explain the cause of the frequent pulse when there are no nerve lesions manifest. Hoffmann's explanation, it is true, as confirmed by Gerhardt and Hering, would explain the cause of high degrees of the arrhythmic frequent pulse, but not of the rhythmic variety. The subject of these interpolated beats has been treated of extensively by Cushny[4] and by Mackenzie[5] under the name of *"premature pulse."*.....They are *"dwarf" beats* in that they are diminutive as to dimensions and duration, and occur singly or in groups, two or more taking the place of the regular beats—as proved by the sphygmogram.

While, therefore, new facts are being brought forward from time to time, bearing on the cause of the frequent pulse, its pathogenesis is not yet completely understood. The full list of causes, as given by Larcena[6] is as follows:

(1) *Tachycardia in diseases of the heart and blood-vessels.* Under this head is included the increased action of the heart which

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[3] Mackenzie, *The Pulse,* Edinburgh, 1902.
[4] Cushny, *Jour. of Exper. Med.,* 1899, IV. 327.
[5] Loc cit.
[6] Les Tachycardies. Thèse de Paris, 1891.

occurs in overstrain, acute and chronic myocarditis, valvular diseases, pericarditis, angina pectoris, acute and chronic aortitis, arteriosclerosis, and the affections of the heart that occur in Bright's disease.

(2) *Febrile tachycardia.*

(3) *Tachycardia from peripheric compression*—that is, on one or both trunks of the vagus—and from central compression of its nucleus.

(4) *Tachycardia from organic disease of the nervous system.*

(5) *Tachycardia in general diseases:* (*a*) Acute diseases, such as typhoid fever, diphtheria; (*b*) chronic diseases, such as tuberculosis, carcinoma, chlorosis, syphilis, chronic malaria, chronic rheumatism of the joints; (*c*) convalescence and exhaustion.

(6) *Tachycardia:* (*a*) From alcohol, coffee, or tea; and (*b*) from drugs, digitalis, atropine, etc.

(7) *Reflex tachycardia* from the brain, heart, lungs, stomach, liver, intestine, uterus, abdomen, bladder, prostate gland, brachial plexus; and

(8) *Tachycardia in neuroses:* Graves' disease, hysteria, epilepsy, and neurasthenia.

There are three forms of the frequent pulse: the *temporary,* the *paroxysmal,* and the *permanent.* An example of the temporary is the frequent pulse resulting from any violent exertion. Examples of the paroxysmal form are seen in neurasthenic states, in which there is a sudden frequency of the pulse and a sudden decline, lasting hours, days, weeks, or months, and often leaving the patient exhausted and sometimes with a dilated heart. The permanent form is frequently seen in chronic tuberculosis, in which there may be no considerable rise of temperature; or in tertiary syphilis with pulmonary complications. This latter form of frequent pulse continues to the end of life.

In the paroxysmal varieties the pulse is usually small and compressible, due probably to imperfect filling of the vessels. In 30 cases given by Martius it ranged from 80 to 180; the average being from 120 to 140. The pulse frequently, however, may reach 300 and more. Bristowe has recorded a pulse of 308, but this is very exceptional. Such pulses can only be counted with the *sphygmograph.*

The frequent pulse may occur in childhood, middle age, or advanced years. Broadbent has recorded a case at 10 years of age and another at 81.

In the paroxysmally frequent pulse the symptoms vary. Almost all the patients are anxious and complain of lassitude. There is often præcordial oppression. Some patients are cyanotic, others are not. Some attend to business as usual. The lungs are usually free. Among German writers such as Riegel and Martius[7] there have been noted instances of pulmonary emphysema, which appeared with the frequent pulse and disappeared with it. Martius gives three cases. It is well, however, to keep in mind that in Germany clinicians are apt to rely on a low position of the liver in their diagnosis of emphysema, a sign that elsewhere is not regarded of so much importance.

In eight of Martius' cases there was cardiac dilatation, which was a proof that the cases were severe. Among other signs that have been noted are venous thromboses, swelling and pulsation of the veins of the neck, albuminuria, and œdema.

There is much to learn in the matter of treatment. As, however, pressure on the pneumogastric nerve will reduce pulse frequency, it has been tried and with temporary success. Iced water and strong coffee have sometimes been effective; so have Hoffman's anodyne and diffusible and alcoholic stimulants. Dr. W. H. Thomson, of New York, in a record of six cases of tachycardia associated with various forms of neurasthenia, ascribes the disease to gastro-intestinal intoxication, and reports success under the use of the salicylates, especially strontium salicylate in 15-grain doses, weak mercurials, intestinal antiseptics, and a carefully regulated diet, which excluded highly nitrogenous food. On the other hand, when the frequent pulse is reflex, so that we can ascribe it to non-toxic diseases of the stomach, kidneys, ovaries, uterus, or other organ of the abdominal or pelvic cavity, it may be due to arterial hypertension (Huchard), or vasomotor paresis. Clement and Hirsch attribute the pulse to mere excitement of the nervous system. In the permanently afebril frequent pulse it may be impossible to reduce the pulse-rate materially; but we must always keep in mind that it is merely a symptom and not necessarily a distressing one. Nor should we attempt by the use of drugs, such as hellebore, digitalis, and the like, to reduce cardiac frequency; for in so doing we only increase personal discomfort without accomplishing any useful purpose. If, however, the constitutional affection, be it tuberculosis, syphilis, or any exhausting disease, improves, the pulse will fall at a rate corresponding to the improvement. It is the general condition of the patient, therefore, for which we should be so-

[7] *Berl. klin. Woch.*, 1875, No. 31.

licitous, rather than his pulse. A pulse of 120 to 130 does not necessarily distress, and it may not be inconsistent with a fairly active life.

The prognosis is unfavorable in recurrent cases, but a man may possibly live with it to the age of 81 years, and recovery may take place from a pulse-rate of 260.

The following case, taken from my hospital records, is an example of periodic palpitation, of a severe character:

Case LIV. Paroxysmal Afebrile Palpitation; Aortic Endocarditis; Embolism; General Dropsy.—C., a young man, was admitted to hospital in January, 1875. He gave a history of violent exercise, such as running up and down stairs on wagers. He had led a dissipated life. Three months previously he first experienced dyspnœa and palpitation on exercise, so that at length he was unable to go upstairs without losing his breath. The pulse at the wrist was found to be 140, with quick-drop beats, when he was perfectly quiet and composed, but in attacks of palpitation the frequency would reach 180 to 200, and then it caused præcordial pain and dyspnœa. From three to ten attacks would occur at night. His face was pale and swollen, and his legs œdematous. Orthopnœa. The patient died in coma.

At the post-mortem examination the pericardium was found to contain 8 ounces of fluid. The heart was greatly dilated. Tremendous infiltration of aortic valves was present—one fringe-like mass waving to and fro in the blood current. The spleen and the liver were the seat of old and new embolic processes.

I have reported two cases of the temporarily frequent pulse[8] due to fatty heart. In one, a lady of 65, the pulse, which before treatment by carbonated baths and exercises was as high as 104, fell after treatment to an average of 81. In the other, a gentleman weighing 237 pounds, with a pulse of 100, experienced after a similar treatment a drop of 70, while the quality improved, and dyspnœa disappeared. (Cases XXXVII and XXXVIII.)

The following cases, also from my hospital and private records, illustrate some of the more important varieties of the frequent pulse:

Case LV. The Paroxysmally Febrile Frequent Pulse of Uræmia;. Endocarditis.—A young widow, aged 31 years, was admitted to hospital in May, 1884, in a comatose condition and with partial suppression of urine. A systolic murmur was noted at the apex. Two days later uræmic convulsions set in, and continued with few intermissions until death. With the first convulsion the pulse was 118;

[8] *Post-Graduate,* March, 1899.

it rose to 132, with the respirations 34, and the temperature 102° F. Before death the pulse fell to 104, the temperature rose to 103.8° F., and the respiration to 50. At the post-mortem examination endocarditis of the mitral and aortic valves was found, but both valves were sufficient. The valvular difficulty must therefore have played a subordinate rôle. The kidneys showed the usual signs of chronic diffuse nephritis. The frequency of the pulse, it will be seen, was not proportionate to the fever, so that it is fair to assume that the frequent pulse was due to the uræmia.

Case LVI. Afebrile Frequent Pulse; Aortic and Mitral Endocarditis; Uræmia.—C., a widow, aged 48 years, was admitted to hospital June 27, 1885. Three months before admission she was taken ill with cough, expectoration, night sweats, and dyspnœa on exertion. The urine was diminished and the feet were swollen. The heart was found enlarged, and a loud mitral systolic murmur, conveyed to the left, was heard. There was also an aortic direct murmur carried up the great vessels, and the veins of the neck pulsated. The liver was enlarged. The pulse was irregular, 128. The urine contained albumin (25 per cent.), and hyaline and granular casts. At the post-mortem examination the ante-mortem evidences of aortic and mitral disease, enlarged liver and diffuse nephritis, were confirmed. This case was afebrile, so that the frequent pulse was probably due to the uræmia.

In the following two cases it is probable that the paroxysmally frequent pulse, though febrile, was due to pericarditis:

Case LVII. Febrile Frequent Pulse; Acute Articular Rheumatism; Acute Serofibrinous Pericarditis; Acute Diffuse Nephritis.—M., aged 39 years, a painter, was admitted to hospital November 19, 1883. Two weeks before admission he was taken ill with a sharp attack of acute articular rheumatism. On examination the joints were found swollen. There was also a blue line on the gums. The heart's action was feeble. The urine had a specific gravity of 1030 and contained albumin. On November 27 double friction sounds over the apex, not transmitted, were heard. The pulse was 120, the temperature 101° F., and the respiration 44. On November 28 the patient became delirious, the friction sound was not so distinct, and the temperature was 106° F. Until death, November 30, the pulse continued to be rapid and feeble. At the post-mortem examination there was found œdema of the lungs, chronic diffuse nephritis, and a fatty liver. The pericardial sac contained 15 ounces of clear serum. Both parietal and visceral layers were covered with

plastic material. The case was regarded as a typical example of the "hairy heart," and to this acute pericarditis was attributed the frequent pulse.

Case LVIII. Rupture of a Cyst in the Broad Ligament; Peritonitis; Septicæmia; Acute Fibrous Pericarditis; Paroxysmally Frequent Pulse.—B., colored, aged 30 years, was admitted to the hospital April 16, 1878. About three months previously she had noticed a tumor in her abdomen, and since then had suffered from several attacks of peritonitis. There was cough with reddish expectoration. The temperature was 99° F., the pulse 130, and the respiration 40. On April 22 the temperature was 101.6° F., the pulse 150 to 160, and the respiration 32; on April 23 the temperature was 101° F., the pulse 140, and the respiration 40; on April 24 the temperature was 101° F., the pulse 136, and the respiration 28. During the succeeding six days the pulse varied from 136 to 140, and the temperature between 99° and 100° F. The patient died on May 6, and at the post-mortem examination several pints of pus were found in the abdominal cavity, with cysts of the broad ligament, one of which had ruptured. The two confronting layers of the pericardium were covered with a fresh "hairy growth." In this case, as in the preceding, the pulse-rate persisted between 136 and 160, although the temperature fell as low as 97.8° F., and did not reach 103° F.

Intances of the permanently frequent pulse are not rare. We all meet with them in advanced tuberculosis.

Syphilis is prone in its later stages to produce a permanently frequent pulse. Such a case came to me for treatment in 1897, and continued under my care for upward of two years. The pulse at the beginning of treatment averaged between 125 and 130, but fell to an average of 111, under treatment by carbonated baths and resistance exercises. The patient had locomotor ataxia, luetic phthisis, mitral endocarditis, and arterio-sclerosis. But with this complication of lesions he managed at times to attend to a pretty active business. Death was due to an intercurrent affection. (Case XLVII.)

In another case, also one of locomotor ataxia, which I saw at the instance of a well-known specialist, the patient had been ill for 15 years, and 10 years previously had had violent pains in her head with diplopia, followed 2 years later by lightning pains, and after another 2 years by ataxia. Her pulse maintained a pretty uniform rate of 100 when in bed, where she was confined by paresis of the muscles of the lower extremities.

The following case of permanently frequent pulse is particularly interesting, because a post-mortem examination was obtained:

Case LIX. Syphilitic Phthisis and Meningitis; Aortic and Mitral Endocarditis; Permanently Frequent Pulse.—K., aged 28 years, an artist, was admitted to the hospital April 7, 1884. The patient had contracted syphilis and was intemperate. For five years his pulse had been irregular. Five days before admission he had a hemoptysis. The hæmorrhage was controlled by ergot, and the patient improved so much as to gain 20 pounds. But during the last six months of life his pulse rose to 150, and he became dyspnœic. During the last ten days the pulse ranged from 141 to 150, and he was delirious. At the post-mortem examination the lungs and the liver showed gummas. The patient died of syphilitic phthisis and cerebral meningitis, and the former was held to have caused the frequent pulse, which is not an uncommon sign in advanced syphilis.

Chapter XVI.

PULSUS INFREQUENS.*

The prevailing terminology of this affection is unfortunate. I refer to the words Bradycardia[1] and Spanocardia that are in use, and I may say the same of Araiocardia and Oligocardia, that have been suggested.

The use of the termination *cardia*[2] implies that the key to the actions of the arterial current is to be found, from a clinical point of view, in the heart, rather than in the peripheral arteries. And yet a comparison between the readings of recording instruments that register the heart and pulse beats simultaneously, as shown by Mackenzie[3] and others, illustrates that there may be a wide variation as to frequency and rhythm between the two.

To illustrate, a double contraction at the left ventricle may be registered in a *sphygmogram,* as a single one; while a *cardiogram* will show cardiac contractions of which there are no traces in the sphymogram. This latter circumstance, characteristic of the "intermittent pulse" (to be distinguished from the "deficient pulse" where both cardiac contraction and the pulse beat are simultaneously "missed") is, of course, very common. Now though the heart regulates the general circulation, its essential characteristics are better exhibited at the periphery than at the center. And for two reasons. The peripheral arteries are more accessible, and therefore can be more conveniently studied than the heart, and they give us more information because cardiac action studied at a distance can be better appreciated. It is not uncommon to judge of motive forces by their remote effects. In telegraphing or telephoning, the one who transmits the message cannot judge of the quality of the instrument so well as the one who receives it. And so clinically, blood circulation is better estimated by the pulse than by the heart; so that there is a reasonable objection to the use of all terms ending in *cardia.* But, apart from this, the qualifying prefixes are either improper, as in the use of *brady,* or are vague in meaning. True

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[1] Introduced by Grob, *Deutsch. Archiv. für klinische Medicin,* 1888, XLII., p. 574.

[2] Kardios, heart; brados, slow; spanos, deficient; araios, rare; obligos, few.

[3] Mackenzie, *Study of the Pulse,* Edinburgh and London, 1902.

Bradycardia implies that the ventricular contraction is prolonged beyond the usual time, in other words, that it is slow; but so far as I know, slow ventricular contraction occurs only in aortic stenosis or in aneurisms near the aortic orifice, where some obstruction like an atheromatous plate narrows the lumen of the vessels, the contraction of the left ventricle being necessarily prolonged, in order to permit it to force the column of blood through the constricted passage.

This is not, however, the common acceptance of the term, which is simply that the number of pulse beats to the minute is abnormally small. As a matter of fact, pulse beats, in the infrequent pulse, may be quick or slow.

To my mind, of all the terms suggested no one expresses this numerical deficiency so well as the Latin *Pulsus Infrequens,* the infrequent pulse.

The pulse rate, as is well known, varies more or less according to circumstances, such as the age and height of the individual, atmospheric temperature, the time of day, and acquired or inherited peculiarities. In the adult male the standard is set at seventy-two; in the adult female, at seventy-six to eighty. And yet it is by no means rare for a person in apparently good health to have a pulse anywhere in the sixties. By general consent, however, a pulse below sixty is regarded as infrequent.

The *pulsus infrequens* may occur at almost any period of life. Prentiss[4] has reported one instance at sixteen months. It was caused by an injury to the neck, followed by an abscess between the medulla and pons.

Grob, from elaborate study, has found that it may occur as late as ninety, but usually between twenty and forty. In my experience it is more often seen in the middle period of life, or after it.

The infrequent pulse in adults (where the rate falls below 40) is rare, though most practitioners with large experience have probably seen occasional instances at some time or other. If, however, we should adopt sixty as the figure below which all pulses are to be reckoned as infrequent, we would find them comparatively common. According to Grob's experience about one individual in forty[5] has an infrequent pulse. Thus far it has been unfortunate that most observers have failed to note the relation in number per minute between the pulse and heart beats, though the importance of ascertain-

[4] Prentiss, *Transactions of American Physicians,* 1889, p. 120.
[5] Grob, 82 in 3,578 patients.

ing this variation was pointed out by Stokes[6] in 1846, when he told of a patient whose heart beats were thirty-six to the minute while the pulse was twenty-eight. Since that date the importance of this point has been emphasized sufficiently to have merited general attention.

Four different relations may be observed between the action of the pulse and the heart: (1) The heart and pulse may beat simultaneously. (2) The heart beats may not all be communicated to the wrist. (3) The auricles may pulsate more frequently than the ventricles and arteries. (4) Contractions of the several chambers may occur when the heart has been removed from the body. In fact, even when the muscular tissue of the walls of the heart has been cut into small pieces, they may be made to contract by various stimuli,—a fact that has been known since the time of Vesalius.

The first two of these statements have been satisfactorily proved by clinical experience; the last two by physiological experimentation, where electrical currents and other stimuli have been known to produce muscular contractions hours after death.

The infrequent pulse has two principal varieties, the *physiological* and the *pathological*.

Of the first two, we have two well-known instances, the infrequent pulse of inheritance and the pulse of pregnancy.

Prentiss has recorded several instances where persons whose pulses averaged thirty to thirty-two were in apparently sound health, and historians tell us not only that Napoleon's pulse was forty even in the midst of a battle, but that he felt uncomfortable when it rose to sixty. The most remarkable instance, however, is, I think, that recorded by Vigouroux,[7] who had under his observation a laborer whose pulse never exceeded twenty. The man never experienced any illness so far as he knew, except on one occasion when he had a short and slight gastric attack, which was successfully treated. In this connection it is interesting to remember that Czermak[8] could stop the action of his heart for a few beats by pressure on the pneumogastric, and Quincke[9] has verified this experiment. Besides, the heart has been stopped by stopping the respiration.

The infrequent pulse is more common in males than in females, the ratio being about five to one, according to Prentiss's tables.

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[6] Stokes, *On the Heart, etc.*, 1855, p. 329.
[7] Vigouroux, *Gazette des Hopitaux*, 876, p. 788.
[8] Czermak, *Viertel Jahresch. für pract. Heilkunde*, 1863, p. 190.
[9] Quincke, *Berliner klinische Wochenschrift*, 1875, No. 15, p. 190.

Of the pathological we have two subdivisions, the *Paroxysmal, Periodic* or *Temporary,* and the *Chronic* or *Essential.* Under the causes of the former come the infections, typhoid, diphtheria, pneumonia, erysipelas, puerperal affections and influenza, best seen during convalescence; toxæmias from lead, tobacco, tea, coffee, digitalis, uræmia, cholesteræmia and syphilis; functional nervous disturbances; reflex influences from the skin or gastro-intestinal tract; and temporary debility.

Among the causes of the chronic or essential variety are organic diseases of the brain and cord. According to Prentiss the infrequent pulse is due chiefly to organic diseases of the brain or cord, epilepsy and organic heart affections, though in eighty-nine of his cases the cause was stated to be unknown in thirty-five.

Grob in his etiology, based on personal observations, is quite definite on this point. In a series of 100 cases he has put the etiological factors as follows, as to relative frequency:

Physiological	6
Idiopathic	1
Articular rheumatism	24
Circulatory disturbances	1
Digestive disturbances	10
Diseases of the central nervous system	6
Infections and constitutional diseases	9
Convalescence from typhoid especially, but also diphtheria, measles, pneumonia, and erysipelas	43
	100

The paroxysmally infrequent pulse, according to Grob, represented 112 out of 140 of his cases. His experience that the paroxysmally infrequent pulse largely predominates coincides with my own views. The pulse rate in these instances is subject, under pathological conditions, to wide variations in range, while, as we have seen, under physiological conditions it maintains a tolerably steady rate. In the first named conditions very low rates have been recorded. Holbertson has published one instance where the pulse fell on one occasion to seven and one-half in a patient who had attacks of vertigo and loss of consciousness following an accident on the hunting field. At the post-mortem examination it was found that there had been pressure on the medulla and upper

part of the cord, the result of fracture of the occipital bone and upper cervical vertebra. Bony union of the parts had ensued, but with displacement of the fragments. Other instances have been published where the pulse fell as low as to four,[10] and even three.[11] This last case was one of the paroxysmal variety, and the patient rallied from the attack.

The explanation of the infrequent pulse is not simple. We can realize that pressure on the pneumogastric may cause it, as in Czermak's and Quincke's experience, and there are many instances in which there was organic disease at the base of the brain from pressure by bone, as in Holbertson's case.[12] This nerve passes from its root in the medulla down to the cardiac plexus. The augmentor (accelerator) fibres of the sympathetic also pass down from near the same spot to the cardiac plexus, supplying energy to the muscular tissue of the heart, though the precise course of the fibres is unknown. Possibly they pass down through the cord.

The pneumogastric regulates, *i. e.*, moderates or restrains, the energy of the heart, thereby slowing the pulse.

The function of the intrinsic ganglia in the heart substance is not yet understood. They may supply motor energy to the heart, independently of the spinal nerves or the sympathetic. And yet they appear to lose vitality after separation from the peripheral nerve system. Certainly the heart beats in the fœtus, before any trace of nerve fibres can be found. Lastly, it appears that the muscle tissue of the heart may assume the initiative, acting independently of any nervous influence, as recent experiments would seem to prove. The paroxysmal cases may be due to reflex excitations of the pneumogastric, though the stimulus of almost any afferent (sympathetic) nerve, (such, for example, as the abdominal sympathetic), may cause them. For a blow on the abdomen conveys the impulse to the medulla through the pneumogastric, slowing or stopping cardiac action. These attacks may also be due to depression of the augmentors, such as occur in nervous or muscular strain, and in gastro-intestinal irritation. They may also be caused by diminished action of the accelerators. In the permanently infrequent pulse we appear to have these causes in continuous action.

There may or may not be symptoms. In the physiologically

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[10] W. Henry Day, *British Medical Journal*, 1880, Vol. I., p. 113. The pulse beat four times a minute for about four minutes during an attack of unconsciousness.

[11] Prentiss, in case seventy-nine.

[12] Holbertson, *Medico-Chirurgical Transactions*, Vol. XXIV., p. 76.

infrequent pulse, such as the hereditary, or congenital, or pulse of pregnancy, there are no untoward symptoms. In fact, evidence goes to show that in most of them, or certainly in many, an increase in rate begets no disagreeable sensations.

On the other hand, the infrequent pulses of the infections, such as typhoid and the toxæmias, poisoning by tobacco, digitalis, tea or coffee, uræmia and anæmia, cholesteræmia and syphilis, are so wrapped up in the symptomatology of their several affections that a description of their several symptoms would carry us beyond the scope of this chapter.

We have now to consider the remaining forms of the neuroses (the so-called idiopathic varieties), a definition which, from a theoretical point of view, fits them conveniently enough, because they may turn out to be due to reflex excitations, as from gastro-intestinal disturbances, or skin diseases, or to direct excitations as after severe muscular exercises or nervous strain. In these latter cases the symptoms are those of general lassitude, prostration, præcordial oppression, a sense of constriction, choking and dyspnœa, and are apt to be associated with nervous strain.

On the other hand, the results of severe muscular exercise are, according to Huchard, apt to be reflected in the nervous system by attacks of vertigo, convulsions, unconsciousness, of short or long duration, epileptiform or apoplectiform seizures, loss of rhythm, a systole associated with varying rates of infrequency of pulse, *Cheyne-Stokes respiration*, with a synchronous pulsation of auricles and ventricles, which come on without warning or with an ill-defined aura.[13] This latter variety has now been erected into a group under the name of the *Adams-Stokes syndrome*,[14] because these two men were the first to describe it. Cases of this variety may survive for several years.

I saw a case with some of these features many times in consultation in 1901 (Case LX). An old gentleman with arterio-sclerosis, during a prolonged attack of heart failure, with a pulse in the thirties, while the heart beats were not far from normal, developed orthopnœa ascites and Cheyne-Stokes breathing during a period of unconsciousness that last over a week. But recovery took place, and after about two years his physician reported that he was in fairly good health, driving in the park in good weather, and going to the theatre when he felt so inclined. Digitalis was used in small doses

[13] His, Jr., *Deutsch. Archiv. für klinische Medicin.*, Vol. 64, p. 316.
[14] Huchard, *Mal. du Cœur*, Paris, 1893, p. 309.

for the ascites for brief periods only, the treatment otherwise being symptomatic.

As a matter of fact, the Adams-Stokes syndrome is a complex of symptoms, consisting essentially of *loss of consciousness, dyspnœa,* with the *infrequent pulse.* In some cases, as in No. LX, there was Cheyne-Stokes breathing in addition. Many varieties have been described. The disease is usually one of old age, but it is said it may occur at any time of life. The attacks, though alarming, are not necessarily fatal, but they imply that the central nervous system is involved. Adams, in 1827, was the first to describe it, but Stokes put it prominently before the profession. However, it was forgotten until recently, when it was revised by Huchard.

On the other hand, there is a group that seem to be dependent on gastro-intestinal irritation, and its signs are flatulence, acid dyspepsia, nausea and vomiting.

The diagnosis offers no difficulties, but we should never, in these cases, rest satisfied until we have examined the heart, to find whether it beats synchronously with the pulse. The sphygmograph applied first to pulse and then to heart will be instructive in these instances. We should also carefully inquire into the possible causes, finding out first whether it is physiological or pathological, and if the latter, whether it is not merely one of the symptoms of the several diseases that have been enumerated. Of the prognosis but little can be said because the details are very scanty. In the physiological variety it is favorable so far as we know, but the instances are few, and the expectation of life has not yet been worked out.

In the *paroxysmal forms* the prognosis depends upon our success in mastering the disease of which it is a symptom, and each affection gives a different expectation. For example, the infrequent pulse of digitalis or tobacco ceases when these herbs are withdrawn and in the infections it ceases with established convalescence; while in chronic diseases like lithæmia, uræmia, and syphilis a good outlook can be assured only when these diseases are held under firm control. Now if the physiological variety together with the paroxysmal represent, as according to Grob they appear to, more than eighty per cent. of the cases, the prognosis is on the whole favorable. The permanently pathological form has a less favorable prognosis.

In the treatment no greater mistake can be committed than to aim at acceleration of the pulse by medication. Experience has abundantly shown that such treatment invariably has bad results.

The physiological cases require no special treatment, and even the pathological appear to do best when let alone, so far as the frequency of the pulse is concerned.

Especially when the infrequent pulse is associated with a recognized affection or condition like typhoid fever, or lithæmia, syphilis, gastro-intestinal irritation, skin disease or anæmia, the treatment apposite to the underlying affection will affect the pulse favorably, and no special medication for the infrequency is necessary. Even alcoholic or diffusible stimulants should be used with caution. The nitrites and bromides are safer, but it is always better, so far as possible, to treat the underlying disease, without special reference to the infrequency of the pulse.

When, however, we have reason to suspect that the infrequent pulse is due to a functional nervous disturbance, or are in doubt as to its exact cause, sedatives like the monobromate of camphor, asafœtida, valerian and Hoffman's ether are the remedies, par excellence, together with carbonated baths and resistance exercises, carefully regulated diet, and observance of the sound rules of health.

The following cases taken from my pathological records and private practice illustrate the graver forms of the infrequent pulse:

Case LXI. Temporarily Infrequent Pulse Due to Digitalis.—C., age forty-four years. For five years had suffered from rheumatism, and for eighteen months from dyspnœa and palpitation. One week before admission to hospital his feet began to swell, and on admission he was found to be othopnœic. Urine contained 20 per cent. of albumin by bulk. After treatment by digitalis, in half ounce doses of the infusion, his pulse fell one day to 36, when the digitalis was stopped. On the next it had risen to 38, and three days later to 54. Compressed air was then given and pulse reached 84. Subsequently digitalis was given, but the pulse having fallen to 52, it was stopped. About two and a half months later the patient died from rupture of the chordæ tendinæ of the mitral valve (it was thought). The aorta contained extensive atheromatous plates. The heart weighed 32 ounces. The infrequent pulse was ascribed to digitalis.

Case LXII. Temporarily Infrequent Pulse; Mitral Stenosis; Chronic Nephritis; Ascites, treated by Digitalis.—J., age thirty-seven years. Three years before admission to hospital he began to have palpitation, dyspnœa, and præcordial pain, and three months previously, œdema of the feet, the urine being reduced to 20 ounces.

On examination, no radial pulse was found. Extreme dyspnœa. Cough and expectoration. Murmur in mitral area. Under the infusion of digitalis, in half ounce doses, improvement ensued, but after two weeks' use of it the pulse began to be infrequent and the general condition worse. After the digitalis was stopped, the patient's condition improved, but the pulse becoming irregular again it was resumed, carbonate of ammonia and spiritus frumenti being added to each dose. The improvement in the pulse was only temporary, for one month after admission it had fallen to 40. During the summer the patient was absent from the hospital, returning in the autumn with no radial pulse, irregular cardiac action, and urine reduced to 12 ounces per day.

From this second attack the patient did not rally. At the post-mortem examination the heart was found to be fatty, hypertrophied and dilated, weighing 22 ounces. The mitral had a button-hole opening. There was also chronic nephritis.

Case LXIII. Temporarily Infrequent Pulse; Mitral Disease; Chronic Nephritis; Abdominal Dropsy.—A lady from Louisville, Ky., came under my care in the Post-Graduate Hospital recently with pulse 64, temperature 98°, respiration 24. She had been under pretty constant medication by digitalis and opiates. During her stay in the hospital there was temporary suppression and subsequently lobar pneumonia. On four occasions she had attacks of heart failure, the pulse registering once 28, and three times 44. She recovered under the use of nitrites and alcoholics, with occasional use of digitalis for diuretic effects only. At the time of her return to the South the pulse had risen to 76.

Case LXIV. Mitral Stenosis; Phthisis; Infrequent Pulse.—In still another case of mitral disease, seen in consultation, where the patient had suffered from hæmoptysis and had suppression and albuminuria, the radial pulse ranged from 37 to 40, and was intermittent and irregular. Though I have learned that both Cases LXIII and LXIV are now dead, I am not aware that any post-portem examinations were made.

The following cases illustrate lesser degrees of infrequency when the rate falls below the standard, viz., 60:

Case LXV. Infrequent Pulse in General Tuberculosis and Tubercular Meningitis.—S., age nineteen years; stable boy. Six months before admission to hospital he had caught cold, as he expressed it, and later had lost 25 pounds. He complained of pain in head and a stiff neck. Pulse 42, temperature 100°, respi-

ration 20. Dry tongue, scowling brow, abdomen retracted. Albuminuria. After five months' stay in hospital he suddenly became delirious. Pulse weak, 32. A month later, with temperature 99.5° and respiration 24, the pulse had risen to 64; but after another month it had fallen to 52. About a year later the temperature became subnormal (97.5°), respiration 24, pulse 60. At the post-mortem examination tubercular pericarditis was found. In the lungs, liver and kidneys, and at the base of brain miliary tubercles. Cerebral ventricles distended with serum.

Case LXVI. Melancholia; Temporarily Infrequent Pulse.—Z., a young man, listless and feeble, complaining of pain about the head, spine and chest, with a previous history of glycosuria, unable to apply himself to any intellectual work, was under my care in 1898-'99. Lateral curvature with protrusion of right scapula. Misshapen head, marked difference between the lateral halves. Left exophthalmos. Pulse 60 to 64. Under treatment by carbonated baths and exercises the pain in the back disappeared and the pulse became more frequent, so that from an average of 60 it reached 72. After leaving my care for the summer the patient relapsed into his former condition, but after a second course of treatment the pain in the head disappeared and the pulse again improved. The infrequent pulse was attributed to his neurotic condition, the symptoms being those of chronic basilar meningitis.

Case LXVII. Mitral Stenosis; Periodically Infrequent Pulse.—In August, 1899, I had under my care a patient who came to me suffering from præcordial oppression, dyspnœa, and a dilated heart, with feeble impulse, weak and irregular action, rate 52. Presystolic murmurs over the mitral area, not conveyed. Occasional thrill. After treatment by carbonated baths and resistance exercises, together with strychnine, his pulse rose to the normal; his heart nearly regained its normal dimensions, and there was absence of præcordial pain and dyspnœa; and the patient led an active life as the member of a very prominent firm in this city for a number of years. His pulse rate was 60 to 64.

Case LXVIII. Infrequent Pulse in Uræmia in Association with Diabetes and Mitral Disease; Temporarily Infrequent Pulse.—A lady of middle age, the wife of one of our most distinguished practitioners, was placed under my care some years ago. As a child she had suffered from inflammatory rheumatism, and later from sciatica, getting relief from atropine and morphine. When first seen she had albuminuria; sugar in large amount and some suppression. Vertigo,

pain alternating between the occiput and vertex; dyspnœa. Pulse 56, intermittent. Heart's sounds almost inaudible, but after rapid motion about the room a slight murmur, conveyed to the left, was detected.

The uræmia, and with it the infrequent pulse, was in this instance promptly overcome by the use of the muriate of pilocarpine combined with alcoholics. According to my experience, this drug may be given in divided doses up to as much as one or even two grains per day, if guarded by alcoholics and watched by a competent nurse.

In the following case the infrequent pulse has a chronic character:

Case LXIX. Arteriosclerosis; Apoplectiform Attacks; Lithæmia; Infrequent Pulse, at First Periodic and Later Chronic.—A patient in middle life came under my observation in consultation many times in the April of 1901. In the preceding month he had had an apoplectiform atack, followed by stiffness in his knees and hips, and a sense of general lassitude. The usual local applications were made; in addition, he took 20 grains of aspirin four times a day, and in a few days was relieved of his pains. Muscular inability of the left masseter remaining, he took salophen in 20-grain doses every three hours, but was obliged to return to aspirin for relief of pain. At this time the pulse had fallen to 50. When I first saw him he was taking morphine in small quantities, as he had used it previously for rheumatic attacks. I immediately stopped this drug, putting him on one-fortieth grain doses of sulphate of strychnine, together with half-ounce doses of whiskey every four hours, and a liquid diet. Pulse was 40, but full and regular, synchronous with the heart. Respiration 22 to 24. Under this treatment the pulse had risen, on May 5th, to 60. Digestive disturbance causing intermittence of the pulse, strychnine was diminished and caffeine in one-grain doses with asafœtida, three grains, substituted; while occasional doses of nitro-glycerine one one-hundredth of a grain, were added. Under this treatment the pulse continued to rise, so that on May 6th it was 68. As it was deficient in force, however, digitaline (Merck) was given on one occasion four times during the day, nitro-glycerine being substituted for it in doses of one one-hundredth of a grain every fifteen minutes, wherever necessary. During all this time about two and one-half ounces of whiskey were being given daily, and after this nitro-glycerine and strychnine as required, with whiskey, at intervals of about three hours. On May 10th, without ob-

vious reason, the pulse had again fallen to about 42, but as it had force and regularity, no effort was made to increase its frequency. For about five days it remained at 40, being strong and regular. Later, arsenious acid in one one-hundredth grain doses and cactus in the fluid extract form, in one minim doses, were given, the pulse gradually rising under them to 60. On May 17th it had reached 70. The sulphate of quinine in eight-grain doses and morphine in one-eighth grain doses for relief of the pain replaced the arsenic, but the cactus was given without interruption, and with no other remedy for a week, when strychnine was again given. The pulse then returned to its normal frequency and so continued, although on a single occasion it fell to 52. On June 26th the patient was suddenly attacked with pain in his left arm and wrist, which became swollen and the pain recurred every morning for five to six days. The pulse fell to 45. On the morning of July 5th he was awakened with buzzing in his ears and great dizziness; no pulse at the wrist. Aromatic spirits of ammonia, an ounce of whiskey, and a one-hundredth grain dose of nitro-glycerine were taken, and relief was obtained in about half an hour. This attack, however, was so prostrating that the patient was confined to his bed with it until September 6th, the pulse ranging from 35 to 44. The treatment during this time was by strychnine one-thirtieth grain, nitro-glycerine one hundredth, and whiskey one-half ounce, taken every three hours. Suprarenal extract in three-grain doses was given for a week, but did not sustain the pulse, and was therefore abandoned. Some improvement in the pulse, however, was always noted after the use of the static battery. It would rise to about 50 on these occasions. On the 23d of November the pulse reached 60, when all remedies except whiskey were suspended. During 1902 the pulse averaged between 48 to 54, the remedies used being strychnine, nitro-glycerine and suprarenal extract, increased to five grains, three times a day. In March of 1903 I examined him, with the following results: Pulse 36 to 44, of fair force and regular. Heart beats synchronous with the pulse. No enlargement of the heart. No displacement of the apex; no murmurs, but impulse lacking; no thrill; accentuation of the second sound at apex; no palpitation; liver a little enlarged; some bronchitis. Physical condition good; takes no remedies; eats well, sleeps well and walks from one to two miles a day without fatigue or embarrassment of respiration, but he has the facies arterio-sclerotica. At my last examination (1905) his pulse was 48.

This interesting case has now entered upon the chronic stage. By some it would be called an instance of permanent bradycardia. I prefer to call it the chronic form of the infrequent pulse.[15]

In September of 1903 I saw two cases that are worthy of note, though I have alluded to one of them. (Cases LXX—I.) In the first, which I saw in the practice of Dr. John S. Warren of this city, the patient had a pulse of 38, after an apoplectiform seizure. In the other, also already alluded to, the patient, after a similar attack, of an evanescent character, had a pulse as low as 19, with a maximum of 28, and, with his pulse at this rate, took a trip to Europe. He is now attending to a rather active business.

[15] This history was for the most part prepared for me by the patient himself.

Chapter XVII.

GRAVES' DISEASE.[1]

Graves' disease is characterized by a complex of symptoms that are neither constant nor definite, though protrusion of the eye-balls, enlargement of the thyroid, a rapid and often dilated heart, and various neurotic manifestations play the most important rôles. Less conspicuous parts are assumed, with varying degrees of frequency, by a number of disorders whose relation to the disease is not so well defined; the more prominent of these being loss of appetite, emaciation, diarrhœa, muscular weakness, with or without tuberculosis, and alterations in the texture and behavior of the skin.

It is apparent, therefore, that Graves' disease may have different types, each of which, of course, has different phases. The recognition of these facts is especially important in the application of remedies, and it is equally apparent that Graves' disease, being partly a functional and partly an organic disorder, offers great difficulties to the nosologist.

Yet we may safely and correctly speak of an *acute,* a *subacute* and a *chronic* form; or we may distinguish a *temporary* or acute form from an *essential* or *chronic,* though it may not always be possible to draw sharp lines of distinction between them. But they are valuable in discussing the disease from a medical point of view. On the other hand, the surgical aspects of the disease are best subserved by the recognition of a *primary* form, *i. e.,* where there is a tolerably contemporaneous development of symptoms sufficiently distinctive to justify the diagnosis of Graves' disease; and of a *secondary,* where equally characteristic signs are sequels of an old goitre.

As in many other diseases of apparently recent discovery we have only to hunt back in the old literature to find that Graves' disease was described long before the time of Graves or Basedow, or Flajani, whose names have been tacked on to the disease by English, Germans and Italians with a zeal that has spoken much for their patriotic instincts, but less for their knowledge of general medical literature. We have but to turn to that treasure-house of patholog-

[1] If this disease is to be named after an English physician it should be called Parry's disease. At this date, however, a change of name might only add to the present confusion in the nomenclature.

ical material, Morgagni's[2] De Sedibus, to find that as early as 1762 Morgagni made a post-mortem on a woman of 40 (of neurotic type and delicate constitution) who, without apparent cause, was taken with amenorrhœa, loss of appetite and palpitation lasting for six months, followed by a sensation of fulness in the throat, difficulty in swallowing, neuralgias, especially of the arms, occasional spasm of the masseters, and finally œdema of the right lower extremities. At the post-mortem examination he discovered an enlarged thyroid. It will be noted, in this case, that he not only detailed two out of the three cardinal symptoms, viz., the enlarged thyroid and palpitation, but also mentioned a number of lesser signs, the so-called "*formes frustes*" described by Charcot[3] in 1885. He failed, however, to designate it as a new disease. Then, in 1802, Flajani[4] of Rome, wrote of the connection between goitre and continuous palpitation, and gave the histories of three cases; though no mention was made of exophthalmos in any one of them. Still, on the strength of these reports, the Italians sometimes speak of Flajani's disease. When, however, we consider the work of Parry,[5] the claims of all others as to the first satisfactory elucidation of the disease, sink into insignificance.

Parry's first statements were made in 1796, though his book was not published until 1825. He was the first to enumerate the three signs, an enlarged thyroid, hypertrophy or palpitation of the heart, and exophthalmos, known now as the cardinal signs of the affection, and to state that they were distinctive of a special disease, which, as he declared, had not previously been described. His first case (seen in 1786) was of a woman 37 years of age who, while in the puerperal state, had an attack of rheumatic fever, which was followed by palpitation, and three months later by a goitre, exophthalmos, loss of appetite, diarrhœa, night sweats and œdema of the extremities. He detailed in all eight cases, and his descriptions covered nearly all of the symptoms, both major and minor, such as dilatation of the carotids, dyspnœa, the nervous manifestations and œdema. In the last seven there was no exophthalmos, and no allusion was made to tremor, which, however, appears to have been first noticed in the eighties of the last century (1883).

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[2] Morgagni, *Liber* v. 1762.

[3] Charcot, *Des formes frustes de la maladie de Basedow. Gaz. des Hôp.*, 13, 15, 1885.

[4] Flajani, *Collez. d'osserv. e. rifless, chir.*, IV, Roma 1802, p. 270.

[5] Parry, *Collections from Unpublished Med. Writings*, London, 1825, II p. 3.

Graves[6] published his celebrated paper in 1835, but the subject was not elaborated by him until 1849. Meanwhile, others in Great Britain had published similar cases. Graves, however, claimed that the disease was a neurosis of the heart, or of the vessels of the neck; though of the two varieties which he contra-distinguished, one was associated with heart disease and the other was not.

But prior to the final elaboration of this subject by Graves, Von Basedow,[7] of Germany, had written of the same subject, in 1840, and the name Basedow's disease is pretty generally applied to it by Germans.

Since the publication of Von Basedow's article attention has been called by Marié[8] to *tremor* as a sign of the disease, and we have discovered something since then of its hereditary character, its behavior under the galvanic current, and its relation to the sexual apparatus. We have also learned much about the thyroid gland, and especially that it elaborates a chemical substance, the failure to excrete which produces myxœdema.[9] Besides, we have found that in certain primary cases cure follows galvano-puncture; and in some secondary cases, perhaps, extirpation by the knife.

Graves' disease is rare. Flint, with his extensive practice, tells us that he saw only five cases in ten years. And yet it is quite certain that many escape notice, especially when the enlargement of the thyroid is slight. Probably there are conditions of climate, locality and habits of life that determine it; but this matter is not clear at the present time. In twelve cases of which I have notes there was but one male. It may occur at almost any age, and is rather more common among Jews than among other races. In men it comes in the third, fourth and fifth decades by preference; in women it usually occurs under the age of 40. Of 980 cases collected by Buschan (*Die Basedowsche Krankheit,* Wien and Leipsig, 1894), 805, or 82 per cent., were women. It is very apt to develop in persons of a neurotic type and may be hereditary. It is not at all uncommon to find that it has been produced by some physical or mental strain, usually the latter. In one of my cases it occurred after the first parturition.

Case LXXII. Graves' Disease, Tuberculosis.—M., married,

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[6] Graves, A newly observed affection; *London Med. and Sur. Journal,* May 23, 1835.

[7] v. Basedow, Exophthalmos. Caspar's *Wochenschrift f. d. Ges. Heilkde,* Nos. 13 and 14.

[8] Marié, *Contributions, etc. Thèse de Paris,* 1883.

[9] Mœbius, *Die Erkrankungen der Schildrüse.* Wien, 1896.

23, a resident of this city came to my clinic at the Post-Graduate, complaining that she had been losing flesh, had pain in the right chest, cough, difficult respiration, headache and loss of appetite. The eyes protruded slightly; the thyroid was somewhat enlarged. The heart's action, however, was normal, and there were no special nervous manifestations. The patient had noticed these symptoms of Graves' disease for about three years, and they seemed to date from the first parturition. This patient was found to have pulmonary phthisis in the second stage.

From time to time views have been advanced that the cause of the disease lay in the sympathetic, and efforts were made to discover special lesions. It is true that degeneration of sympathetic fibres, atrophy of ganglion cells, and pigmentation of both fibres and cells have been found by some observers; but, on the other hand, others have failed to find them. Moreover, we have learned that in normal nerve tissues these degenerative changes are going on more or less continuously. Nor have researches in the brain and cord in these cases been any more successful.

In one case, however, Filehne (*Zur. Path. der Bas. Krankheit; Sitz. Ber. der Phys. Med. Soc. zu Erlangen*, July 14, 1879) relates that he produced an exophthalmos, rapid action of the heart, and enlargement of the thyroid by division of the anterior fibres of the restiform bodies. But after all, emotional causes may equally well produce all these symptoms. Evidently, there is one constant condition, however, and this is a vasomotor paresis—and while it may have an organic base, it is just as likely to be a functional disorder, certainly at first.

Buschan, in his prize monograph, accordingly, divides Graves' disease into two varieties, (1) where there is a genuine neurosis, with marked psychic and vasomotor symptoms, and (2) where it is symptomatic, *i. e.*, called into action by peripheral lesions of the sympathetic, as in intestinal disorders, operations on the nose and teeth, or by reflex influences from the brain. It has also been maintained that thyroid hypertrophy is superinduced by an excess of certain toxins which the alimentary canal generates in health, but which normally are neutralized by the action of the gland. Being in excess, they superinduce a thyroid hypertrophy. Yet it seems quite as reasonable to believe that the diseased thyroid may permit of the development of the toxins by withholding certain matters necessary to their destruction.

Graves' disease is slow in developing. It is said that the onset

is accompanied with tremors. Of this I have no personal knowledge. Tremors are often noticeable, however, after the disease is recognizable in the thyroid. They are like those of paralysis agitans or chorea, but more rapid. Headaches are common, and sweating may be profuse during the acute stage, even when there is no tuberculosis, which later is common, in my experience. The polyuria or glycosuria, discoloration of the skin, artificial urticaria, exaggerated reflexes, affections of phonation, numbness of the limbs, sleeplessness, irritability, confusion of ideas, melancholy, or even more serious diseases, are further evidences of the neurotic nature of this disease. Epilepsy has sometimes been observed, but it must be remembered that some of these nervous manifestations may be fruits of the same neurotic tree—associated affections and yet not parts and parcels of the disease.

In one of my cases there was facial spasm, but whether an intrinsic symptom or an associated one, I do not know. It appeared long before there was any noteworthy sign of thyroid enlargement.

Case LXXIII. Graves' Disease; Thyroid Enlargement effectually Treated by Galvano-puncture.—F., 44, unmarried, a neurotic subject, has been under my observation more or less for a period of 27 years, during all of which time she has had cardiac disease. Twenty-five years ago she began to have attacks of heart failure, and has had them at intervals ever since. Mitral stenosis, cardiac dilatation. Has also had enlarged venous radicles in lower extremities, with at times ecchymoses. During all this time has had hemifacial spasm and atrophy.

Exophthalmos and enlarged thyroid developed at a time she was not under my care, but without tremor, and without tachycardia. The gland was practically reduced to the normal size by galvano-puncture (Professor Emmet). Since that time the exophthalmos has disappeared, and under aconite, suprarenal extract and nitroglycerine taken for occasional attacks of heart failure, she has done fairly well. For the past eighteen months, I understand, she has had no medical treatment whatever.

Hyperæsthesia, hemianæsthesia and paræsthesia have all been noted. Among the trophic symptoms come pigmentation of the skin, especially bronze discoloration about the eyes, or on scattered parts of the body. Sometimes they look like spots of sunburn, in other cases the skin is as dark as in a mulatto.

The following case is an example of this discoloration:

Case LXXIV. Graves' Disease; Epileptiform Seizures; Uni-

lateral Pigmentation of the Skin; Malarial Fever; Cardial Dilatation; Tremor.—F., 23, unmarried, consulted me in March, 1901. For a year she had been complaining of præcordial pain, palpitation and headache, with dysmenorrhœa, irritable bladder, migraine and constipation. From childhood had attacks of unconsciousness, when her face was suffused, feet and hands were cold, and she was rigid. Her attacks lasted about half an hour. The next day she was very stupid and prostrated. She also had occasional attacks of malarial fever. Upon examination the apex was found to be under the left nipple. The pulse was inclined to be rapid and irregular. Unilateral pigmentation of the skin of the right side of the face. Membranous vaginitis, tremor, moderate enlargement of the thyroid. No exophthalmos. After treatment applicable to her malaria and constipation, her neurotic condition, malaria and dysmenorrhœa, she gradually improved, so that on October 28th of the same year her mother reported that she was "quite well." Hysteria in most of its manifestations may add a further complication, and probably did in this instance. She had also at time the sudden "giving way of the legs," the para-paresis described by Charcot. She would at times feel a gradual diminution of power, and then her lower limbs would suddenly give way altogether.

The *skin* may be red or pale, abnormally dry or abnormally wet. There is a form of erythema that is common on the face, usually in the form of spots, though pigmentation is more common. The dark areas may be diffuse or in spots, as around about the eyes or the nipples, on the neck, in the arm pits, on the abdomen, or in any location there is pressure, as under the garters. The mucous membrane is rarely discolored. The nails are free. The face is most affected. The patients also flush easily, and mechanical friction causes a dilatation of the capillaries which persists for a considerable time.

One of the first signs to which the attention is called is palpitation. The arteries, veins and capillaries become gradually soft and dilated, especially those of the neck; there may also be throbbing of the abdominal aorta. The signs of cardiac dilatation are not evident at first, but they set in early. The murmurs, in the absence of valvular disease, are usually heard at the base, and the cause may be the prevailing anæmia. When dilatation is established, the apex beat will be diffuse. As the disease advances there may be some irregularity in its action. In severe cases, or in the final stage, there may even be delirium cordis. I have seen one

case (Case LXXV) where the pulse was so rapid that it could not be counted, but complete recovery took place.

Case LXXV. Acute Graves' Disease; Frequent Pulse; Recovery.—Mrs. X. was seen by me in consultation with two other physicians in February, 1897. She had previously exhausted about all known remedies, and had consulted many eminent medical men. She was confined to bed, and was in a highly hysterical condition, crying, wringing her hands and complaining of a "flutter" or "twitching" about her heart. Skin bathed in sweat. Pulse too rapid to count. Stomach distended with gas, which she belched at intervals. Nausea and at times vomiting. Great præcordial distress, but respiration normal. No exophthalmos, but enlarged thyroid and tachycardia. Epigastric pulsation. Pseudo-angina. Attacks of this kind she was subject to, and they had reduced her 75 pounds weight in two years, although she was still corpulent. No cardiac murmurs, but apex 5¾ inches from the median line. No cyanosis. Under treatment by diet, laxatives, stomachics and resistance exercises there was general improvement, the pulse falling to 66. Eventually there was a complete recovery, I was told, by one of her physicians.

The thyroid may be uniformly enlarged, or only in one lobe. In a small number of cases, which some have estimated at about 8 per cent., it will not be involved at all. On this point there is a pretty general agreement. As a rule, enlargement of the gland follows the palpitation, but the swelling may come and go. Usually the dilatation of the veins is well seen in those coursing over the gland. There may even be heard a blowing murmur over the gland from its dilated vessels. Various changes may take place in it. At first it is apt to be soft and yielding; later induration may set in, or it may become cystic. The enlargement may get to be so great, especially when cysts develop, that deglutition, phonation and respiration are interfered with.

The diagnosis of implication of the thyroid is made with unerring accuracy, as we all know, by noting that the tumor goes up and down with every attempt to swallow.

As a rule, in point of time the exophthalmos follows the goitre. It varies in degree, and usually involves both eyes. In extreme cases the protrusion of the eyes may be so great as to prevent closure of the lids and make glasses necessary as a protection for the eye-balls. In these cases there may be the *Von Graefe symptom,* where there is inability of the lid to follow the downward

movement of the eye, or the *Stellwag symptom*, of actual retraction of the upper lid. A moderate swelling of the lymphatic glands has been noticed, also an enlargement of the spleen.

A symptom which should not be omitted is the *anæmia* which is quite frequent. In some cases independent of renal diseases which are late occurrences, it may lead to local œdema. In one of my patients œdema of the upper lids was a very marked symptom, and lasted for years.

In women menstruation is usually normal. But sometimes atrophy of the sexual organs, bilateral or unilateral, has been observed, with falling out of hair and atrophy of the mammæ. On the other hand, there may be unusual development of the breasts. There is usually no implication of the kidneys, bones or joints. Diarrhœa is not uncommon. It comes on without apparent cause, and is usually painless. In other cases there is constipation.

One form of the glycosuria has been called alimentary, because it will disappear when the carbohydrates are eliminated. On the other hand, there is a form of glycosuria that is not affected by the diet. In any event it is not a prominent symptom.

So far as the diagnosis is concerned there is no difficulty when any two of the three cardinal signs, palpitation, goitre and exophthalmos are present; but in the absence of two of them, when the minor manifestations have to be taken into consideration, there may be great difficulty in reaching a satisfactory diagnosis. Besides these atypical forms, we have to reckon with abortive cases. When, however, we find a patient of neurotic disposition that has one of the cardinal signs, associated with symptoms of tremor, alterations in the character and behavior of the skin, hyperæsthesia or paræsthesia, the diagnosis can be made with a reasonable amount of safety.

The *course of the disease* is variable, but is usually slow. Acute cases have been known to get well in a couple of weeks, and yet death may occur in six weeks. The average duration of chronic cases is five to ten years. Cases which take long to develop are less dangerous to life; but if the strength fails rapidly, vomiting is persistent, and the heart action becomes irregular, the outlook is unfavorable. The chief points bearing on the prognosis are the action of the heart and the emaciation. If the frequency of the pulse diminishes and the patient puts on flesh, the outlook is encouraging. Violent pulsation, vomiting, diarrhœa, diseases of

the cornea, paralysis, mental disturbances, are of bad omen (Buschan). The disease is more violent in men than in women. The prognosis is not affected by age except that in advanced life there is less capacity for resistance. If with failing heart there is renal inadequacy with albuminuria and dropsy, the prognosis is bad; so also in cases complicated by phthisis.

According to V. Graefe,[10] complete cure takes place in 25 per cent.; considerable improvement in about 50 per cent. About 12 per cent. die of the disease. There is hope, however, even in the worst cases. When the exophthalmos and goitre are pronounced, the prognosis is naturally more unfavorable. Though I know of no spontaneous cures, they are, I think, within the bounds of possibility.

Failure to treat this disease successfully has been largely due to misconceptions of its nature, and consequently to the use of the most inappropriate remedies. There is no standard treatment suitable for every case. Each must be a law unto itself, remedies being so applied as to control the particular elements that are conspicuously offensive. Therefore, while iodine, iron, arsenic and various other drugs, hydrotherapy, electricity, electro-puncture and operative procedures all have their uses, they are only to be employed in distinct types or phases of the disease. There can be no doubt that on account of the recognition of this principle we are more successful than formerly in our treatment.

Assuming that we are called upon to treat one of the primary or genuine types, where the neurotic signs, the cardiac disturbance, the enlarged thyroid, and the exophthalmos are in evidence, and there is nothing apparent to account for it, rest should be enjoined, with separation from the family, and all sources of excitement or nerve strain prohibited. It may perhaps be imperative to send the patient at once to some quiet resort, where there can be no worry or cause of excitement. Under such circumstances some form of the Weir-Mitchell treatment is useful. If the pulse happens to be very rapid, the patient should be kept in bed; but under no circumstances should drugs like digitalis or its congeners, or veratrine, be given to lower the action of the heart. The heart, on the contrary, needs to be soothed, and bromides, camphor, valerian or asafœtida are indicated.

Coffee, tea and tobacco must be interdicted. As soon as the

[10] v. Graefe, *Deutsch. Klinik.*, 1864, s. 158.

pulse has fallen measurably, hydrotherapy is important and carbonated baths are useful. Acute cases, however, should certainly not be sent to the baths of Nauheim, Homburg, Franzensbad, or Pyrmont. The artificial method as practiced in this city is more appropriate. After such a course, the patient may make a tour to the mountains, Saratoga or the Adirondacks, or if abroad, to the Carpathians, Tyrol, Switzerland, or Scotland, not going to an altitude of more than fifteen hundred feet. In acute cases cold applications to the heart may give relief. In the subacute stage electricity, galvanism, faradism or the static current are helpful. Such applications appear sometimes to cause a fall in the pulse and control the tremor.

In using the galvanic or faradic current, one pole should be applied to the angle of the jaw and the other to the episternal notch, in order to reach the sympathetic of the neck. For the spine, one pole should be applied over the fifth dorsal vertebra and the other over the gland. The patient should understand that he must not expect improvement until months have elapsed. The current should be weak, and only applied daily, or two or three times a week, for one minute.

The drugs that have been used are numerous. It is safe to say that so far as reducing the number of pulsations are concerned, drugs are practically inert, and that veratrine especially is inadvisable. But if the palpitation and frequent pulse are to be reduced by drugs, first in order come the bromides, with camphor, aconite and nux vomica. Belladonna is useful, especially in the form of plasters. Where there is chlorosis, iron and arsenic are useful. Iodine is also of value. Theoretically, so far as its action on the vessels is concerned, it is harmful, as it causes vasodilatation and so accentuates the disease. In syphilitic cases, however, it may be of considerable service. Nitrites, so far as they are vasodilators, are not indicated, but ergot, strychnine and cactus, being vasoconstrictors, may be useful.

So far as the use of thyroid extract is concerned, recovery took place in one of my cases, which I saw in the acute stage, under the use of this agent, the thyroid extract, given in five grain doses three times a day, but it took about two years. Another case, however, seen by my colleague, a surgeon, who saw the first case with me, was not benefited by the treatment and died within a year. My patient had such a large goitre that it produced dyspnœa, the palpitation was marked, and there were nervous symptoms as well.

Surgical interference seemed to be imminent, and yet under the thyroid treatment he was entirely cured.

The following are the notes of this case:

Case LXXVI. Acute Graves' Disease; Treatment by Thyroid Extract; Recovery.—In the summer of 1896 I was asked to see the following case with Dr. Heffinger, of Portsmouth, New Hampshire: J. F. R., 32, a mason, after some family misfortune that weighed heavily on him, found that he had so large a thyroid that it interfered with his swallowing. When seen by me he was intensely nervous, and had a frequent pulse. Between the skin and the thyroid was also a smaller tumor, about the size of a robin's egg, which was plainly cystic, and entirely disconnected with the thyroid. I advised aspiration of the smaller tumor, the internal use of the iodide of potassium, external use of lead ointment, injections of Lugol's solution, and, these failing, thyroid extract. The man then passed from under my observation, but two years later presented himself to me. The thyroid enlargement had disappeared, and with it the frequent pulse, and his other nervous symptoms; but the little cystic tumor still remained, apparently of about the same size. It appeared that, under the directions of his physician, he took the thyroid extract in 5 grain doses three times a day, but there was no positive improvement until he had persevered for about six months. Then he noticed a diminution in the gland. The thyroid extract was accordingly continued. Symptoms of thyroidism would occasionally be manifested, when the remedy would be intermitted, to be resumed again when these symptoms had disappeared.

Since 1898 he has had no occasion to take the extract, and I learned in 1903 that he regarded himself as well. Dr. R. Abrahams of this city has called my attention to a record of three cases treated by him, one by mercurial inunction and two by saturated solutions of the iodides (*Phil. Med. Jour.*, Feb. 9, 1901). Apparently all of the three cases developed after syphilitic infection, and were cured by anti-syphilitic remedies, in periods ranging from six to ten weeks. This is another illustration of what medicines may do in such conditions.

On the other hand, Moebius and others claim to have had success by feeding their patients exclusively on the milk of goats, whose thyroids had been removed. The theory of this treatment was that these animals *might* develop in their systems a substance

that would neutralize the toxins of the patients. It is said that the results were encouraging.

Personally, I have tried injections of iodine and alcohol without avail. And in one of my cases where partial extirpation had been practiced in Vienna, the general symptoms were not relieved, though the gland was diminished. The following is a case treated by me in the earlier years of my practice, and without success:

Case LXXVII. Chronic Graves' Disease, with Special Neurotic Manifestations.—Mrs. S. consulted me on February 8, 1876. There was marked exophthalmos; great enlargement of the thyroid, palpitation and nervous excitement. Great difficulty in breathing and articulating, a sensation as if "the throat were swollen on the inside," loss of power and numbness in hands and arms, confusion of ideas. On May 29th of the same year the tumor had diminished a little, but on February 21, 1877, the condition had not improved. On September 9, 1878, when I last heard from her, she expressed herself as no better. Palpitation had continued. She also had "pounding" in her head at night, and had lost weight. Digitalis had been given her (but not by me, of course) without any good effect.

In the twelve cases of which I have notes, it is noteworthy that five of them exhibited signs of pulmonary tuberculosis, and two œdema of the extremities.

Relief follows surgical treatment also. Extirpation, partial or complete, is justified in extreme cases, without doubt, when other methods have failed, and there is no cachexia or cardiac weakness. Heydenreich[11] has reported on 61 cases of removal of the gland.

There were 50 cures or improvements and 4 deaths and 5 failures. Tetanus developed twice. Buschan, in 116 cases, reported as results 23 deaths (22 per cent.), 45 improved, 3 unchanged, 45 uncertain. In Heydenreich's cases, however, it has been claimed that most of them were old goitres to which the other symptoms had attached themselves later, *i. e.*, they were cases of secondary exophthalmic goitre. In primary cases it may be a success, but how much of a success is not certain. In these, too, there may be failure and death, the result of the operation. This operation is always a serions one.

Jonnesco (*Internat. Clinics,* Vol. 1, 1903), of Bucharest, has claimed that bilateral sympathectomy of the cervical sympa-

[11] Heydenreich, *Semaine Med.,* XV. 32, 1895.

thetic will relieve the symptoms in a majority of the cases. In a demonstration made by him at his clinic in Bucharest he showed three cases that had been operated on during the previous five years and claimed that the relief was lasting. In all, the size of the goitre had notably diminished and the frequent pulse, the tremor and exophthalmos; and he thought the effect produced was better than in ablation of the thyroid gland, partial or complete, an operation that he regarded as both dangerous and not to be depended on. In his operation he extirpated the lowest ganglion of the cervical sympathetic. From his report, however, the results were far from satisfactory. The symptoms had diminished, but had not disappeared.

Chapter XVIII.

ANGINA PECTORIS.[1]

Looking at the matter broadly, angina pectoris is for the present the most convenient name for a small group of affections in which the predominant feature is an intense pain over the præcordial region, occurring in paroxysmal attacks, never prolonged so as to become chronic, and in some severe instances associated with the subjective symptoms of impending death. The pain may radiate from the præcordial region in various directions and to various distances, often to the left arm. Sometimes it will originate at a distant part and fly to the heart.

Affections of the coronary system are conspicuous among the pathological findings, while degenerative changes in the ascending aorta, aortic endocarditis, myocardial disease and general arteriosclerosis are found with varying degrees of frequency. No one of the lesions, however, is absolutely constant.

The term angina pectoris depicts a characteristic symptom of these affections, has served us well for over a hundred years, and involves no unproved theories. We retain it as we have retained the term epilepsy, which does not describe the essence of the disease, but merely a symptom. It is sometimes better to "bear those ills we have, than fly to others that we know not of."

Fortunately, the name does not lead us astray. It includes a perfectly well known class of cases for which we institute remedial measures, in the way of prophylaxis and treatment, that relieve and often cure.

Angina pectoris was certainly known as early as the time of Morgagni,[2] who gave the clinical history of a case occurring in his individual experience and the post-mortem features, which included atheroma of the aorta, disease of the aortic valves, coronary arteries, myocardium, and general arteriosclerosis.

A few years later Lorry saw a similar case in a captain of cavalry at Besancon, and the letter describing it, according to Peter,[3] was dated February 23, 1768. On the 21st of July in the same year Heberden, in a communication to the Royal College of

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[1] Known variously as stenocardia, sternalgia, neuralgia cordis, asthma dolorificum, syncope dolorosa, or anginosa.

[2] Morgagni. *De Sedibus*, II. Epist., XXVI., 31 *et seq.*

[3] Peter, *Mal. du Cœur*, Paris, 1883, p. 662.

Physicians in London, gave it the name *angina pectoris.* The picture he drew was so accurate that he is justly recognized as having been the first to contradistinguish it from cardiac asthma,[4] a distinction which, unfortunately, is not always kept in view at the present day.

In 1772, we are told by Parry (*Angina Pectoris,* London, 1799) that Jenner first saw calcification of the coronary arteries in a specimen dissected by John Hunter, and in the year 1788 Parry recognized a coincidence between sclerosis of the coronary vessels and angina pectoris.

It remained, however, for Walshe[5] to separate it into two principal varieties, the *true* and the *false,* a valuable distinction from a clinical point of view. The false variety, sometimes called *angina hysterica,* is exceedingly common in nervous women, without any associated anatomical changes in the heart or arteries.

In place of *false angina,* the name *angina pectoris-motoria* was, I believe, first suggested by Landois,[6] who held that angina partook of an exaggerated vasomotor disturbance,[7] and found it in chlorotic or anæmic girls undenr emotional or cerebrospinal disturbances, which caused either increased arterial tension or vasomotor paresis. Under this same name sub-varieties have been described, notably one by Nothnagel[8] in persons whose limbs had been stiffened by exposure to cold, where relief was obtained by external heat. Somewhat analogous cases have been described by Bamberger.

Others have preferred to divide angina pectoris into the *mild* and *severe* forms, and there is some reason for this plan. Occasionally the false or pseudo angina will produce more excruciating pain than the true variety. I have known such instances.

Inasmuch as coronary disease is known to be the special pathological concomitant of true angina, while affections of the great vessels and the heart are of less frequent occurrence, some regard the former as *primary* and the latter as *secondary* causes.

True angina is certainly a very rare disease, though the laity talk glibly about it, as an everyday occurrence. The paroxysmal attacks that eventually carried off the great John Hunter and the

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[4] Heberden, *Commentaries on the History and Cure of Disease,* 1782, Chapter LXX.

[5] Walshe, *Dis. of the Heart,* 1873, 5th 4th edition, p. 209.

[6] Landois ,*Correspondenz-blatt der Deutsch. Gesellsch. für Psychiatrie,* January, 1866.

[7] Niemeyer held this view.

[8] Nothnagel, *Special. Path.,* XV.-2, s. 592.

bony hardness of his coronary arteries, left a deep impress on the minds of the laity and medical men in England, and its effects are still seen. But how many of the prominent men supposed to have died of angina really died of ordinary heart diseases or apoplexy, we shall never know. Certainly it is a rare disease in hospitals.

In a series of 823 cases, fairly complete as to clinical details and autopsical findings, in two hospitals, extending over a term of ten years in one and fifteen in the other, I have no recorded mention of a single instance of true angina pectoris. And yet coronary diseases were common enough.

In a series of 2,300 consecutive cases observed by Dr. Abrahams in the clinic of my colleague, Professor Wainwright, at the Post-Graduate Hospital there was not a single case of true angina pectoris, though false angina was of frequent occurrence. Professor Burt, while in the outdoor department of Bellevue Hospital, in the division for heart and lungs, with an average of 600 new cases a month, has said he did not remember to have seen a single instance of it. False angina, however, was common in women who drank strong, rank tea.[9]

The most conspicuous special lesion is obstruction of the coronaries by atheroma, or deformative arteritis, with or without embolism or thrombosis. In John Hunter's case, where the clinical signs had been most pronounced, the coronary arteries were so hard that it was difficult to divide them with a knife, and their transverse sections did not collapse, while the aortic[10] and mitral valves showed "ossification," as calcification was then called.

In Gautier and Huchard's[11] 70 cases, however, there was coronary disease in only 38, or 53 per cent. The following in detail were their findings in angina pectoris:

Lesions of the coronary arteries alone, or with other cardiac or arterial lesions	38
Aortic insufficiency	12
Aneurism of the arch	5
Fatty degeneration of the heart	4
Hypertrophy and dilatation	4
Pericarditis	3
Suppuration into mediastinum	1
Negative	3

[9] *The Post-Graduate*, Dec., 1904, p. 1216.
[10] Adam, *Life of Hunter*, 1817, p. 203-4.
[11] Tacchi, *Gaz. Med. di Rom.*, 1890, XVI., p. 97

In a later communication, however, Huchard[12] has given a different result. In the 145 cases he collected he found the coronaries affected in 128, or 88 per cent., and as follows:

In 64 both coronaries.

In 37 the left coronary.

In 15 the right coronary.

In 12 not specified.

In all of the 128 there was obstruction or stenosis, 121 times by atheromatous stricture or thrombosis, five times by embolism, twice by compression. The other cardiac lesions were regarded as of minor importance.

And yet, it should be remembered that he appears to have been able to collect only 128 cases of coronary disease associated with angina, while thousands of cases of coronary disease unconnected with angina could be equally well collected, if any one were to make a search for them. In my own experience I should be quite willing to say that I have seen hundreds of cases of coronary disease without a single manifestation of angina pectoris.

It may also be said, in explanation of Huchard's statistics, that during the period of life when angina is most prevalent, viz., between 50 and 60, degeneration of the coronary arteries is the rule rather than the exception. To put it even stronger, most persons after middle life have arterial disease, and many at a much earlier period of life.

In order to realize the diversity of opinion that has prevailed as to the cause of the pain, we have but to consider the views held by prominent men.

Heberden thought it was due to the contraction of a hollow organ (the heart), and he compared it with the spasm of hollow viscera, such as the intestines and the uterus, contractions of which, we know, produce violent pain. This theory would appear to necessitate some alteration in the pulse, which, as a matter of fact, is not apt to be altered in rhythm, though there may be often and perhaps usually is, I think, some hypertension.

Some have regarded it as a paralysis of the heart. Undoubtedly, if angina should result in death, there would be paralysis of the left ventricle. Others have held that it is a manifestation of gout; but often there are no lithæmic manifestations. The neurotic theory was early advanced. Laennec[12] and Lartigue[13] thought it originated in the pneumogastric, and Lancereaux, Peter, and Bazy[14] found, in several autopsies, apparent infiltrations of the car-

[12] Laennec, *Dis. of the Chest*, 1834, p. 65.
[13] Lartigue *Gaz. Med. de Paris*, 1847, p. 775.
[14] Peter, *Mal. du Cœur*. p. 703.

diac plexus; but on the other hand, infiltration of this plexus by exudations or new growths does not always produce pain, and if this latter view is correct, it should be permanent. But the pains of angina are only temporary.

Friedreich[15] and Romberg[16] thought it a functional affair,—a neurosis. Trousseau[17] compared it to the nerve explosion of epilepsy. Bouillaud[18] ascribed the pain to phrenic and intercostal irritation. Piorry[19] called it a brachio-thoracic neuralgia. Allan Burns[20] located it in the cardiac vessels, which by their stiffness resist the blood-pressure, whenever the other systemic vessels are distended.

Deprivation of blood as in embolism will, it is known, cause severe pain sometimes, but not always. Lauder-Brunton[21] has held that the pain is due to rise in tension of the peripheral, *i. e.,* coronary arteries, and experiments made by him in 1867 suggested the use of the nitrite of amyl, a vasodilator, which has been successful. This is strong proof, certainly, that the pain is due to hypertension of the vessels. There are some difficulties, however, in this theory, viz., that in such instances as Hunter's the coronaries were too stiff to contract. And yet hypertension may have taken place in the arterioles and capillaries. Indeed, we cannot imagine that nutrition of the organ could have been maintained, without a sufficiently healthy tone in a goodly portion of the vessels, to allow of their functioning.

Somewhat akin to this is the view of Nothnagel that the pain is due to a spasm of the vasomotor nerves of the heart, causing great increase in blood-pressure. Albutt[22] has claimed that the pain is not cardiac.

Another view is that there is a circumscribed ischæmia from deficient blood supply to the heart walls, and the effect is like that produced by pressure of a distended stomach on the heart. In this connection it is well to know that the coronaries often terminate in comparatively large trunks, from which capillaries are given off,

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[15] Friedreich, *Traite des Mal. du Cœur,* Paris, 1873, p. 531.
[16] Romberg, *Dis. of the Nervous System,* London, 1853, Vol. I., p. 125.
[17] Trousseau, *Clin. Med.,* London, 1868, Vol. I., p. 592.
[18] Bouillaud, *Mal. du Cœur,* Paris, 1841, Vol. II, p. 613.
[19] Piorry, *Traite de Med. Prat.,* Paris, 1842, Vol. VIII., p. 143.
[20] Burns, *Dis. of the Heart,* Edinburgh, 1809, p. 136.
[21] Lauder Brunton, *Pharm. Ther. and Mat. Med.,* 1885, p. 666.
[22] Allbutt, *Phil. Med. Journal,* June 16, 1900.

and that these unite to form reservoirs between the muscle fibres while the capillaries freely anastomose, so that interference with the circulation in a cardiac artery means interference with a large amount of capillary tissue. This anatomical peculiarity indicates that there is a peculiar vascular network in the walls of the heart, and that contractions of these vessels and reservoirs may cause painful sensations, unlike any others in the body.

Out of these facts and theories, diverse as they are, a few important data can be elicited, which are: The most predominating lesions that have thus far been observed have been coronary diseases; and with a great deal less frequency, atheroma of the ascending portion of the arch, aortic endocarditis, or some form of heart disease, or perhaps pericarditis. With the angina there is sometimes high blood-pressure in the left ventricle, and apparently any cause that will produce high arterial tension will in certain persons precipitate an attack. The pneumogastric is probably implicated, as indicated by the spasmodic action of the stomach, which expels gas in great quantities, at the close of an attack. On the other hand, the passage of a large amount of a pale colored urine implies that the sympathetic is also involved, and this view is strengthened by the fact that various forms of excitation in remote parts will also precipitate an angina. Inasmuch, however, as these attacks partake at one time of neuralgia in one nerve and then of another, the spinal cord must be the medium of transmission of the nerve impulses. And as fright will bring on an attack, the cerebrum at times is involved. Furthermore, it may have a toxic origin. For example, it may be due to an excess in tobacco, tea or coffee. Huchard claims that tobacco causes a functional spasm of the coronaries, and his contention may be correct.

Angina has a hereditary character; but then, the various underlying conditions, such as nervous excitability, coronary disease, and arteriosclerosis are also, in a large measure, hereditary.

The exciting causes are known to be unusual muscular activity and sudden emotions or reflexes, especially from the abdominal organs.

The pathogenesis of angina must therefore, at times certainly, be referred to the central, peripheral, motor, or sensory filaments of the entire nervous system.

True angina has been chiefly observed in males. Pye-Smith found that the proportion was 7 to 1; others have made the ratio even greater. In Huchard's 237 cases of true angina his propor-

tion was 195 men to 42 women (about 5—1); in his 141 pseudo cases 98 were women and 43 men (about 3—1). True angina rarely occurs before 40; usually between 50 and 60. In Forbes'[23] cases 72 out of 84 were over 50.

When an attack of true angina seizes a patient, the pain is usually referred to the sternum, about its middle. It is apt also to radiate down the left arm, less often down the right arm or even leg, sometimes to various parts of the abdomen or pelvis. An *angina sine dolore* has also been described. It reminds one of the play of Hamlet, with the part of Hamlet omitted. However, it has been spoken of as a dull ache, replacing the regular attack with all the concomitant symptoms except the excruciating pain.

In a well marked instance of true or severe angina, the face will ordinarily be pallid, and the forehead covered with sweat, while the respiration is not affected; this condition contrasting sharply, therefore, with the cardiac asthma of valvular or myocardial disease. The heart's action may be increased or diminished, or may be unaffected. Unless death is imminent, the pulse is usually regular and of ordinary frequency. As the attack passes off, a sensation of weakness is felt. Sometimes there is formication or numbness, afterwards usually belching of wind and the discharge of urine of low specific gravity and pale color; all of which betray the essentially neurotic character of the disease.

The pain is a distinctive feature of true angina, but it is in no sense a pressure pain. It is the pain of an intense neuralgia, so excruciating that the patient feels that he must keep absolutely still until it has passed. In my experience the "sense of impending death" has not been a prominent feature, but on several occasions the patients have expressed themselves as feeling that the chest was being compressed, as if in a vise. In one instance the patient said she felt as if a "house were resting on her chest." But there are degrees in the amount of pain felt. The attack is usually brief, more especially after the first attack, for then the patient's experience has taught him or her how to manage the seizure. Usually of only a few minutes' duration, it may last several hours, and in one of my cases an intense angina continued for several days.

The immediate cause of an attack may be due to several causes, as I have already mentioned. In one of my cases it was produced by sexual intercourse, in another by the smell of fresh paint, and in a third by prolonged conversation. A common cause is undue

[23] Walshe, p. 203.

hurry, or walking in the face of a sharp wind. In one of my patients it seemed as if the gastro-intestinal distention was the cause of the attack; certainly, it was the forerunner of it.

Death has been ascribed to poisoning of the heart muscle by the arrested metabolism, but this process is too slow to explain it. The causes are various. It may be due to paralysis from deficient innervation, or the pressure of abdominal organs, such as the stomach, which as a rule is overdilated.

Reflex Angina Pectoris, the angina vasomotoria of Landois, is associated with visceral or peripheral disturbances, without known organic lesions. Peripheral neuralgias are supposed to be exciting causes. It will be noted, however, that such peripheral neuralgias are alleged to have excited true angina. In the hysterical variety there should be the characteristics of hysteria present. Pains in other localities, with hyperæsthetic areas, should be associated. While the distinction between true and false angina is theoretically easy, it may be very difficult.

All the circumstances of the case must be considered in making a differential diagnosis. If in the male sex, after 50, and in association with general arteriosclerosis and some form of heart disease, of the aortic valve particularly, the diagnosis of true angina may be made with a considerable degree of confidence.

On the other hand, in young people, women especially, of neurotic history and lowered vitality, in the absence of arteriosclerosis or any form of heart disease, the diagnosis of pseudo-angina may be made with an equal degree of confidence.

In angina from poisoning by tobacco, tea or coffee there is the history of indulgence in these luxuries, with their associated cardiac and neurotic symptoms, that cease when the cause is removed. As regards the differential diagnosis from cardiac asthma, we see actual dyspnœa in the latter. In pseudo-angina there is never any actual dyspnœa, for the patient can draw a long breath if he makes the attempt. In cardiac asthma there is engorgement of internal organs, and externally we see the physical signs of venous congestion. There may, however, be instances in which it is very difficult to distinguish between the true and false forms, as I have already said,

Nitrite of amyl is often a specific in the treatment of true angina. A few drops, three to five, according to the severity of the attack, placed on a handkerchief relieve the patient. Nitroglycerine taken by the mouth in doses of 1/100 or 1/50 of a grain will

sometimes give relief in less than a minute. I use for this purpose a capsule containing nitroglycerine gr. 1/100, menthol gr. 1/50, amyl nitrite gr. 1/4, with oleo resin of capsicum, gr 1/100. Attacks occurring in my office or in my presence are thus easily relieved. But the nitrite of amyl alone is not always sufficient; in chronic cases it sometimes fails to give relief.

In cases where these remedies are not at hand, morphine should be used hypodermically, and then followed up by inhalation of chloroform or ether, without waiting for the morphine to act.[24] The patient may pour a few teaspoonfuls of ether into a saucer and inhale the fumes. Balfour's[25] plan is to put a sponge soaked in chloroform into a wide-mouthed bottle, and then allow the patient to inhale the fumes until he gets relief. Heat applied to the chest by a hot water bottle or bag, replaced by mustard leaves or poultices, will often give relief. Cold applications, however, are sometimes quite as useful.

If there is any sign of heart failure, brandy, whiskey or carbonate of ammonium are indicated, the latter being almost universally applicable. Digitalis is too slow to be useful. In a case I saw recently I found the aromatic spirit of ammonia of the greatest assistance. It also promoted the expulsion of gas by the rectum. After the paroxysm has passed, aconite will be found useful in two minim doses t.i.d.; also, later, arsenious acid in 1/100 grain doses. If there is arteriosclerosis, arsenic should be kept up for a while and then replaced by iodide of potassium, sodium, or strontium, in from one to five or ten grain doses.

In the pseudo cases Hoffman's anodyne is indicated, or the monobromate of camphor in grain doses, asafœtida in three to ten grain doses, and finally musk in five to eight grain doses.[26] The cretægus oxycantha or hawthorn has been used with benefit. The dose is five to twenty drops of the tinct. t.i.d. I have used it, but not sufficiently to speak of it with positiveness. Some prefer the nitrites. They are used extensively in England and France. The nitrite of sodium is a remedy I sometimes use in doses of from one to three grains three times a day. It may be used as a preventive, and, as such, my friend, Dr. Abrahams, uses it.

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[24] The tendency to formation of an opium habit in this disease must be kept in mind.

[25] Balfour's *Dis. of the Heart*, London, 1898, p. 331.

[26] I have recently been using the iodo-nucloids made in tablet form by a Chicago house. Each tablet contains a half grain of iodine, and I give as much as 2 to 2½ grains three times a day. The tablets are well borne by the stomach.

In one case of what I then believed was true angina, in a young married woman, where the pain was agonizing, I gave complete relief by the continuous current; certainly the pain did not recur during the weeks she was under my care.

As to the prognosis in true angina of the primary variety, meaning by this where there is no associated disease of aorta or aortic or other forms of organic heart disease, I do not know that we are prepared to form an opinion. Coronary disease itself is quite compatible with a fairly long life, judging from the frequency with which it is found at post-mortems; but in the secondary form the forecast is certainly worse. And yet it is uncertain. There is apt to be an interval of a year or so, at any rate, between the attacks; and the disease may continue for years. It has been known to exist in some instances from 30 to 40 years. And yet a single attack may be fatal at any time, we may well believe; for it only requires sudden paralysis of the left ventricle, or the larger part of it. And yet the patient may, in exceptional instances, entirely recover.

If he can live where he is not exposed to cold or to excitement and avoids hurry and digestive and other excesses, his chances are vastly improved, for it is just these conditions that often bring on an attack.

Case LXXVIII. True Angina Pectoris; Aortic Regurgitation; Lithæmia.—B., 54, liquor dealer, came to my clinic at the Post-Graduate School, passing a high-colored urine loaded with uric acid, but without a previous history of rheumatism. No syphilis. Five years previously he had first experienced palpitation and dyspnœa, with "soreness" in the region of the heart. Three years later he developed what was classed as angina pectoris, pains of a "darting" character located in the heart and running down the left arm, and accompanied by a "sense of impending death."

On physical examination, the impulse was found to be much diffused and weak. Aortic reguurgitant murmur. Water-hammer pulse in all the arteries. After protracted treatment, lasting over six months, by alkalies, hepatics and iodides, it is noted in the record kept by my clinical assistants, that "he was much improved."

Case LXXIX. Angina Pectoris; Arteriosclerosis; Glycosuria; Albuminuria; Apoplexy.—Mr. H., a retired merchant, 74 years old, of full habit and rather obese, was sent to me in September, 1899, by Dr. H. V. Barclay. He had been complaining of dysphagia and obscure gastric attacks coming on at night. On phys-

ical examination his pulse was found to be 80 and feeble, with hard radials, rolling under the fingers. Respiration 24. Impulse at apex hard to detect. Apex one-half inch outside the nipple and one-half inch below the intermammillary line. Heart sounds indistinct. One and one-fourth per cent. sugar in urine. Traces of albumin; specific gravity 1015. He was put on carbonated baths and resistance exercises. Owing to his feeble condition the baths and exercises were stopped on the 13th day of treatment. On this day he had a gastric crisis at night, and the next morning his head was confused. On October 23 a musical murmur was heard with systole at the apex. He was now put on potassium iodide. Two or three attacks of angina had been brought on by the smell of cooking, and others by gastric or intestinal distention. On October 27 an attack was induced in my office by a smell of fresh paint. Relief was obtained by capsules of nitrite of amyl with nitroglycerine, etc., the formula of which I have given. Patient nervous and hysterical. For three weeks he was free from attacks, using the capsules only to ward off attacks, but his diet was restricted. Under the iodide of sodium increased to nine minims of the saturated solution, three times a day, the patient continued to improve, although he had occasional attacks of dizziness, and was troubled by a sound like that of running water in his right ear, indicating the gravity of the disease in his cerebral arteries. Like many old men with arterio-sclerosis, however, he became restless under treatment, and in December passed from under my care. In the following February he died of an apoplectic attack.

In cases of advanced arteriosclerosis like this one, the prognosis must be bad. No remedy has yet been found that will cure calcification of a vessel. The following case may or may not have been one of true angina:

Case LXXX. Angina Pectoris; Direct Aortic Murmur; Cyanosis; Flatulent Dyspepsia; Superficial Oedema.—A young married woman, of fine physique, weighing 140 pounds, was referred to me by her physician. She had been ill for five or six years, and for the previous eight months had been under active treatment for her heart affection. Notwithstanding this, she grew gradually worse. Previous to her illness she had been given to athletic sports, such as riding, swimming, etc. The attacks of angina were regarded by her physician as of the *true* variety. The præcordial pain was described as of "a house resting on the chest." Flatulent dyspepsia,

dyspnœa and transient cyanosis were other symptoms given. Attacks were brought on by nervous strain of any kind, particularly by prolonged conversation. They were temporarily relieved by nitroglycerine.

On physical examination the pulse was found to be 80, soft and weak, but without intermission. No impulse at the apex, but burning pain. No thrill; præcordium and sternum sensitive to the touch and swollen; cording of lymphatics under left breast; apex under sixth rib ⅛ inch inside of mammilla and 2⅜ inches below intermammillary line; at apex slight systolic murmur. At base systolic murmur carried well up into the great vessels.

Potassium citrate, nitroglycerine, strophanthus and strychnine had been given. I ordered all medicine stopped, and gave camphor monobromate in two grain doses, carbonated baths, and resistance exercises, with massage. After four days of this treatment the swelling of the breasts had disappeared, and in fifteen days she was sufficiently improved to return home. In the following September she was enjoying life, camping in the Maine woods, and feeling, as she expressed it, "restored to health."

This patient reports to me from time to time and has had no recurrence of her angina since the above treatment, which was in 1900.

The following is an example of the false form:

Case LXXXI. Cardiac Dilatation; Mitral Regurgitation; Irritable Heart.—Mrs. R., a young married woman, was referred to me in January of 1899. She was slight in build and a neurotic subject, addicted to hysterical seizures; had morbid fears, pseudo angina, insomnia and gastric disturbances, with constipation. Her medical treatment, prior to my taking her under my care, had consisted of stomachics, laxatives and sedatives. On examination February 1 her apex was found in the fifth space, in the line of the nipple. At the apex harsh and distinct systolic murmur, carried round to the left. At the base a soft systolic murmur, confined to the aortic area. Heart sounds feeble. Pulse intermittent. Urine normal. After a preliminary rest and attenion to her general condition she was ordered a limited number of movements, each lasting fifteen minutes. These were gradually increased in number for eight consecutive weeks, given daily with constantly increasing force, with the result that the heart sounds became more distinct, the basic murmur disappeared, the menstrual flow increased, and the attacks became gradually

milder, while there was marked general improvement. During the six weeks following the end of the treatment she had but one attack. The apex had moved towards the median line 1⅝ inches. The contraction of the heart in this case of dilatation goes far, in my mind, towards proving that the pain of angina is due to dilatation. I believe, however, that it is often more than dilatation. There may also be spasm, as Heberden held. *Unequal tension* expresses this double condition, according to my view. But how explain the unequal tension on the theory that coronary disease is an important factor? In this way, if you choose: Coronary disease produces fatty degeneration of the heart, because the supply of blood to the organ is insufficient, the result being weak spots corresponding to the areas that are imperfectly nourished. The heart muscle, therefore, cannot contract evenly, and the very unequal tension causes the greater pain. Charcot held the view that it was due to local spasm, similar to the local spasms of the intestine in influenza, the fibrillary contractions of facial muscles in cerebral disease or the spasm of the muscles of the extremity in the "intermittent claudication" of Bouley, the veterinarian, who first saw it in horses.

In the last of my cases, that of false angina, I would explain the pain as caused by spasm of the cardiac muscular tissue of the heart, which in this case was essentially normal. The contractions pressed on the nerves and ganglia, causing the pain, and each spasm left the nerve elements more irritable and more predisposed to another attack.

This theory will, therefore, explain the cause of the pain in both the true and false forms. In fact, the closer we look at the two, the less essential are the differences between them.

Chapter XIX.

THE GENERAL MANAGEMENT OF HEART DISEASES.

As distinguished from the special treatment applicable to the several varieties of heart disease, their general management is to be considered.

First of all, we should hold in mind the particular variety and stage of the disease we are treating, and at the same time the natural limitations of all remedies, so far as their therapeutic actions are concerned. This statement is obviously a truism, and it will be so admitted, in the abstract; but in the concrete, that is, in actual practice, its force is not fully recognized. Take, for example, digitalis. It is too apt to be prescribed in all forms of heart disease. This is certainly wrong. While the drug is admirably suited for broken compensation, with renal inadequacy, it is as positively contra-indicated in cardiac neuroses, like the "tobacco heart," and is absolutely dangerous, if there is a tendency to apoplexy. Again, while the salicylates have a specific action in acute rheumatic endocarditis, they are only prophylactics in the chronic forms. Furthermore, as I shall endeavor to show, each cardiac drug has a specific and limited sphere of utility. These facts do not always have their proper weight, when cardiac drugs are ordered by physicians.

Though this chapter is devoted to treatment rather than prophylaxis, it may be said that, inasmuch as endocarditis is usually a secondary phenomenon in constitutional affections, such as lithæmia or the exanthems, or in some septic poisons, as in the malignant forms of cardiac disease, it follows naturally that successful treatment of the primary disease should favorably influence its secondary manifestations. When, however, endocarditis has supervened, the constitutional disease should be treated, as well as the special cardiac manifestations. Of course, in the acute stage *rest* in a supine position is indicated; also, *local applications* of a cooling nature; perhaps opiates; possibly counterirritants to the præcordium. But thus far I have not seen the necessity for adopting Caton's[1] plan of giving mercurials and iodides during this stage, though it goes without saying that they are positively necessary in some of the chronic forms.

In malignant endocarditis I have been sceptical, until recently,

[1] *British Medical Journal*, Feb. 9, 1901.

of a cure under any circumstances. So far as my experience has gone, these cases have proved to be pyæmic in character; so that the chances of recovery can hardly be much better than in pyæmia. But I have seen recovery in pyæmia, and I do not close my eyes to the records of other successful cases. The *sulpho-carbolate of sodium* in 2-5 grain doses, or other intestinal antiseptics at frequent intervals, injections of *streptococcus serum; colloidal silver* (Argentum Credé) by inunction in 10 per cent. strength, for adults, or in 1/6 grain doses 2-3 times a day, in pill form, offer at least some hope of relief, where without antiseptic treatment, in some form or other, the forecast is very gloomy.

In the treatment of valvular diseases we recognize three stages. First, a gradual evolution of the affection with the production of dilatation and hypertrophy. Second, a stationary period where dilatation and hypertrophy counterbalance the valvular changes. Third, a period in which the equilibrium of the cardiac mechanism is lost. The first period includes and outlasts the acute exacerbation, but continuing until compensation is established, it may have a duration of months, or even years. So far as the treatment of this stage is concerned, after the febrile period is passed, an effort should be made to arrest the constitutional disorder. Attention should be given to each morbid symptom; the diet should be so regulated as to conform to individual idiosyncracies; errors of digestion should be corrected; overexertion of every kind, and exposure to cold or sharp winds, should be avoided. The patient should also be warned against great altitudes; also the superheating of the body in a damp, hot atmosphere, as in the Turkish or Russian bath.

If he is an intelligent adult, it is best, at the outset, to explain to him the nature of his malady, so as to secure his coöperation. I know of no disease where it is so important for the patient to thoroughly understand his physical limitations. Concealment of them is a fundamental error, that many of us have committed in times gone by. If the patient is properly informed, however, the chances are that with comparatively little medication he will pass successfully through this stage. In fact, a couple of grains of the monobromate of camphor for his palpitation, or a minim or two of the tincture of aconite every few hours, will usually so modify this symptom, as to make it bearable, if it is not arrested; while glonoin in 1/100 grain doses, or a nitrite, in one grain doses, every hour, even in threatened heart failure, will often be sufficient.

In the second or stationary period, the patient has reached a

plane of tolerable safety. Alarming cardiac symptoms due to lack of compensation have disappeared. Theoretically, it is true, that embolism is now to be feared, from the washing off of granulations or débris from about the valves; but even if this accident occurs, its effects are apt to be transient. For embolism of some sort is a common feature of valvular disease, though rarely fatal. And while embolism of the lungs may lead to temporary embarrassment of breathing or even to hæmorrhage, it is seldom fatal, except in one of the malignant forms; or when the vascular occlusion occurs at a nerve centre; and even in this last instance, the lesion is not absolutely irremediable. So that when the patient has reached the second stage and satisfactory compensation has been established, he needs no treatment, no matter how loud his murmur. Subject to the limitations of his bodily condition, and his individual capacity for work, he may resume his former methods of life, guarding himself against danger by the rules already laid down. And by rigid adherence to them he may live an average life.

Patients come to me not infrequently in this stage to take a course of carbonated baths and exercises, but I invariably decline to give them, simply because they are unnecessary.

If, however, compensation breaks, introducing the third stage, prompt treatment is important, if life is to be saved. Such early symptoms as shortness of breath on overexertion, difficulty of sleeping in the prone position, palpitation and arrythmia indicate that danger is at hand. And yet rest in bed, freedom from care, a laxative and restricted diet, with perhaps an anti-fermentative and digestive tablet or powder, will usually give relief, certainly for a time.

These incidents being noted, however, it is better to institute treatment by baths and exercises, and not wait for cyanosis and serous effusions. Surely a plan that will avoid appealing to *venesection, hydrogogues,* and *opiates,* those "three firm friends more sure than day and night" to some of our profession, merits very serious consideration. Besides, of all agencies at this time, baths and exercises are the most efficient; indeed, they enable us to dispense with all the "three horrid sisters," as I call them; and also with drugs that are improperly used to stimulate the heart. In addition to the active and passive movements to be employed, the patient should be instructed to walk daily in the open air, gradually increasing the distances; but never permitted to hurry. In fact, no form of physical strain should be allowed. There is a neu-

rotic element in almost all cases of broken compensation; in fact, in most instances of dilatation.

Either kind of strain may cause heart failure and death. There is also another danger, especially in aged persons. It is their tendency to bolt down unmasticated food, or eat too freely when suffering from over-fatigue, or if the digestion is weak. Acute indigestion is the result, with gaseous distention of the gastro-intestinal tract. The extra-cardiac pressure produced in these cases may, and often does, kill, by interference with the action of the heart. The locality is also to be considered. The elevation should not exceed 1,500 to 2,000 feet, and the prevailing climate should be mild, equable and dry, so that exercise can be taken daily in the open air. There are few contraindications to the use of baths and exercises; but patients with parenchymatous nephritis, or arterio-sclerosis, where apoplexy is threatened; diabetics when the amount of sugar is large; and phthisical subjects are not likely to be benefited, and are therefore improper subjects for this treatment.

In the subacute dilatation of neurotic or anæmic young people, especially those who lead sedentary lives, or have long hours and poor food, where carbonated baths and exercises are not available, nutrients like malt, iron, quinine, arsenic, and the alkaloids of nux vomica, help to check dilatation and restore the heart's tone. In general, *strychnine* in 1/60 to 1/30 grain doses, or the milder *brucine* in larger doses, are good nerve tonics; but contracting both heart and arterioles, they are undesirable for continuous use. Besides, I hold them to be cumulative in their action, occasionally producing digestive disturbances. In the debility of old persons I use *arsenic* in 1/100 grain doses, or the arseniate of iron in 1/60 grain doses. I have also used, in the fat heart, the *cratægus-oxyacantha*, or hawthorn berry, as a substitute for strychnine, and have found it of some utility. The American berry appears to be the best. The dose is 5 to 10 minims of the tincture. It is important to get a good article. Under the same category falls the *cactus*, or cereus grandiflorus (night-blooming cereus) of Mexico. It appears to regulate by stimulating the vasomotor centers. One to two minims[2] of the alcoholic tincture is useful in myocardial diseases. It appears to have no cumulative effect. Somewhat similar in action is the *suprarenal extract* given in doses of 1 to 3 grains of the powder, in gelatine capsules, three times a day. It is prompt

[2] Dr. Green, of Chicago, uses only 1-5 of a drop of the alcoholic tincture. *Am. Med.*, Nov. 2, 1901.

and energetic in its action, which is sometimes felt in a few seconds, may last three hours or more, and has no unpleasant after-effects. It is essentially a heart stimulant. Among cardiac sedatives I still use, to a limited extent, *valerian*, but prefer the *bromide of zinc*, given in 1/10 grain doses. Sometimes, as already indicated, I rely on the tincture of *aconite* in 2 minim doses every 3 or 4 hours. While aconite is a vasomotor dilator, it is also a diuretic, to some extent, and of course a mild diaphoretic. It is useful in cases where digitalis and drugs of this group have been used to excess. It quiets the tumultuous heart; in the mildly neurotic heart I prefer to use *camphor monobromate*, especially in the fluttering heart of emotional women. It is also useful in nocturnal palpitation, such as is so frequently seen in diabetics and in aortic disease. Another drug of limited use in *belladonna*. It is helpful in 5 minim doses of the tincture, given in anginoid attacks and arrhythmia. I use it chiefly, however, as an external application. In severe attacks of dyspnœa I am apt to give *nitroglycerine* in doses of from 1/100 to 1/25 of a grain, sometimes giving the latter dose at intervals of ten minutes, until relief is obtained. While nitroglycerine contracts the heart, it is a vaso-dilator, and diminishes arterial resistance, so that it relieves the laboring heart. As the action of the nitrites lasts about an hour, they should be given at hourly intervals, in threatened heart failure. In very severe cases I give them every five minutes until relief is obtained. Nitroglycerine and the nitrites are efficient and safe remedies, and I have never seen any lasting bad effects from them, even when I have given very large doses. In sudden and violent attacks I give capsules each containing 1/100 of a grain of nitroglycerine, ¼ of a grain of amyl nitrite and 1/50 of a grain of menthol, with 1/100 of a grain of the oleoresin of capsicum. These ingredients are suspended in 10 minims of oleum Ricini. Relief is usually obtained in less than a minute, often in a few seconds. The capsule may then be given safely at intervals of four hours for days together, to prevent subsequent attacks.

This combination, which is my modification of an English formula, has met with a favorable reception, but it is not very stable. In fact, it is unreliable in action, unless freshly prepared. I never use any preparation of opium in chronic cases. One of the most unfortunate results of the improper administration of digitalis and its congeners is, that they sometimes produce such distressing results, that patients take relief in opiates and alcoholics. Nor is it

much of an improvement on the opiates to give chloral or its analogues, chloralamid, urethran and ameline hydrate. They are primarily hypnotics. If, however, a *hypnotic* drug is absolutely essential, I use *paraldehyde* or *trional.* As a rule, however, if a patient has been addicted to these drugs, if one substitutes for them a course of baths and exercises, the result will be a revelation; for it will often be found that they can be dispensed with, especially if the carbonated bath is taken just before bedtime.

Laxatives are essential in the treatment of chronic heart diseases. They should be carried to the point of producing at least two liquid, or semi-solid, movements a day. For this purpose I have been in the habit of using waters that contain the sulphate of sodium. In place of them I often use a preparation which has the effect of a saline aperient, a hepatic and a diuretic. It consists of equal parts of sulphate of sodium, phosphate of sodium, and the neutral tartrate of sodium. The dose is 1 to 2 drachms dissolved in hot water, and should be taken one hour before breakfast. I also use a hepatic consisting of the resin of podophyllin, ½ grain; extract of colocynth, 1 grain; extract of hyoscyamus, 1 grain; and extract nucis vom., 1/6 grain; one pill to be taken every other night. To get the best effects of this combination, however, it should be freshly made.

In some instances, especially after middle life, I give small doses of *calomel* as often as twice a week, to get the laxative, sedative and tonic effects of the mercurial. In arterio-sclerosis the iodides are indispensable, and in the syphilitic form mercury is equally essential. Some of my patients take the *iodides,* with intermissions, year in and year out. I find them especially suited for aortic disease. I usually begin with 5 minims of the saturated solution of the iodide of sodium, increasing the strength gradually, but rarely surpass 10 minims at a dose. Sometimes I find 3 or 4 minims enough. Iodine in various other forms may also be used.

But satisfactory treatment is almost always incomplete without some medication directed to the gastro-intestinal tract, and here is a field for the whole range of digestives and anti-fermentatives. Unfortunately there is more empiricism necessary to meet the indications here, than in any other phase of cardiac disease, and much ingenuity is often required to devise the precise formula required; but success will be the reward of patience. In case of overweight, great help is obtained from a strict *dietary.* I have devised the following one, and have used it with success.

8 A.M., breakfast, 3 ounces cold meat without fat; 1 ounce gluten or whole wheat bread; a cup of postum with milk, or lemonade.

10:30 A.M., a cup of beef tea.

1 P.M., 5 ounces of lean meat or fish; salad with French dressing.

3:30 P.M., 8 ounces of milk and vichy (half and half).

6 P.M., 3 ounces of rare meat or fish, with pickles and salad; 1 ounce Graham bread; stewed fruit.

8:30 P.M., 8 ounces of milk and vichy water (half and half). Vichy or plain water may be taken as desired during the day.

This is not a severe dietary, as compared with some that are used.

As a rule, I use *digitalis* only when special complications arise—as, for example, when I am confronted with urinary suppression, its attendants or sequelæ, and where prompt effects are important. The more I see of heart disease, the more I am convinced that ordinary digitalis is not only of uncertain composition, but unreliable, and therefore a dangerous remedy; especially if it is to be used indiscriminately. It may produce spasms of both heart and vessels. In fatty hearts, I hold it to be particularly dangerous, and even in urinary suppression, in our attempts to get its diuretic effect, we may cause fatal cardiac contraction. Digitalis and its derivatives are slow, requiring some hours before their physiological action is obtained; therefore, we cannot depend upon them in emergencies. If given as a diuretic, digitalis may be advantageously combined with 20 grains of a bitartrate, and given at intervals of four hours. Digitoxine is now generally admitted to be more uncertain in action than digitalis, and I avoid it. Merck's digitaline in my hands, however, has always proved satisfactory.

Digitalis, however, has a distinct field in mitral diseases where the pulse is irregular, and where there is renal insufficiency with or without dropsy. It steadies the pulse, slows it, makes it firmer; and stimulates the capillary circulation, which is the ultimate object to be attained. Favorable action is heralded by increase in the urine. If it acts badly, the signs of warning are nausea or vomiting, a small and irregular pulse, embarrassed respiration, and diminution of urine.

When a standardized preparation of digitalis[3] cannot be obtained, it is best to use powdered leaves in doses of 2 to 5 grains

[3] Such as has been made by Parke, Davis & Co.

several times a day. A small number of persons can take digitalis with intermissions for long periods of time with benefit. But these cases are exceptional, and do not disprove the rule. The African *strophanthus* is, in a measure, a fair substitute for digitalis, though the two have somewhat dissimilar physiological effects. As a heart compressor, I hold that strophanthus acts more powerfully than digitalis, but as an arterial compressor it has less power. It slows the heart beat, shortens systole, and lengthens diastole; unlike digitalis, it is not much of a diuretic and is not cumulative; unfortunately it is apt to produce diarrhœa and nervous irritability. It is well to begin with small doses, say 3 minims of the tincture, gradually increasing them and noting the effect of the drug. It should not be long continued, however, and must always be administered cautiously. It acts more promptly than digitalis, and herein is its great advantage.

Convallaria majalis, the lily of the valley, also belongs to the digitalis group. It slows the heart, and is a diuretic without cumulative action, but in small doses it is an emetic and purges actively, like jalap or scammony. In mild forms of cardiac disease it has no place, but when cardiac stimulation, with mild diuresis and catharsis, is needed, convallaria is applicable. There are comparatively few instances, however, in which it is indicated. The standardized drug should be used exclusively. In adonis, from the *adonis vernalis,* or false hellebore, we have a drug whose glucoside adonidine is allied to digitaline. It slows the pulse, increases its force, and is a diuretic without cumulative action. It is useless in functional disorders, but is a fair substitute for digitalis, as a heart stimulant. The initial dose of 3 minims may be gradually increased. The *adonis vernalis* is sometimes confounded, as it should not be, with the *adonis æstivalis,* which appears to have no value as a heart stimulant, and whose dose is three or four times that of the *adonis vernalis.* Too much reliance should not be put on *caffeine,* which is not always made from coffee—sometimes from ordinary tea, guarana or the kola nut; the citrate, a favorite form, contains only 50 per cent. of caffeine. Primarily, caffeine produces cardiac contraction, leading, in large doses, and with some persons, to spasm and even paralysis. Secondarily, its effects are depressing; while it has proved an uncertain diuretic in my hands.

Sparteine from the tops of the scoparius, or broom, increases blood pressure, slows the heart, but is unreliable. Its action is chiefly upon the heart muscle. I have found it a fairly good diu-

retic and heart stimulant for cardiac dropsy, in doses of 1/20 to 1/10 of a grain. *Diuretine,* the sodium salicylate of theobromine, is something of a diuretic. I have used it in 15 grain doses. *Apocynum,* the Canadian hemp, I have found of some value in dropsy, as it is a diuretic and a cathartic. It should be distinguished from Indian hemp or cannabis indica. Half a grain of powdered apocynum may be given at dose. In cardiac œdema I have been most favorably impressed by the *muriate of pylocarpine,* which I have given in divided doses to the extent of nearly 2 grains in a day, carefully watching its effects and guarding them by stimulants; and I have had no unfavorable experience with it.

As a rule, it is best to use single remedies, rather than combinations. Otherwise, if the combination has no good effects, we are forced to discontinue it, and to throw discredit on all the ingredients while one of them may be the very one we need.

If œdema of the lower extremities is not relieved by these remedies, massage may be effectual. If it fails, the skin of the legs may be punctured with an ordinary *cambric* or *triangular needle.* In uncomplicated cases no further operative procedures are desirable.

From the foregoing recital, it is seen that we have a large number of simple drugs that are available in heart diseases. By a judicious selection or combination of them, aided by baths, exercises, diet and other agencies, we can usually treat our patients successfully without resorting to any of the digitalis group; or at least we need not call upon them except under very special conditions. Digitalis has powerful defenders, such as Huchard and Broadbent. But the digitalis we buy in this country is apt to be of uncertain composition. If druggists substitute the seeds for the leaves, light is thrown on the matter. Then different preparations have different effects. For example, according to Porter,[4]

[4] W. H. Porter.

the infusion has a different effect from the tincture, because in the infusion the active principles, digitoxine and digitaline, are barely soluble in water; while digitaleine and digitonine, two others of the five active principles which are soluble in water, antagonize one another. Hence the infusion is comparatively weak. But in the tincture both the digitoxine and digitaline are soluble in alcohol, as also the digitonine; while the digitaleine (nearly as soluble) is also present. Hence the tincture is comparatively strong.

In Germany this difficulty is met by the use of the powdered leaves, as my friend, Dr. Groedel, of Nauheim, advises. He begins with four, or even eight, grains of the powdered leaves,

given in divided doses for a single day. If no harm ensues, he gives still larger doses on the third day, and continues the treatment for several days. And he is able by this plan to give it, in many instances, where other methods, such as those by baths and exercises, are unavailable. Even if he finds there is at first a bad effect, he returns to the digitalis, after an interval, and gives it a further trial. Some of his patients take digitalis continuously for months at a time. I have never had occasion to prescribe digitalis continuously, however, using usually the nitrites instead. As a matter of fact, in ordinary cardiac failure I am disposed to treat everything but the heart. If one treats the associated conditions, he will not be so often called on to use heart stimulants. Many mild chronic cases of broken compensation, or heart weakness, improve at once after a restricted diet and moderate exercise.

In persons of middle life it is too much the habit to "overeat." The rule, "always to rise from the table with an appetite," is peculiarly important for these persons; for with them acute attacks of indigestion are serious matters.

It is a little difficult to know just what *exercise* to prescribe for people with well-ordered compensation. Usually horseback exercise is permissible; occasionally it is not. Most people may ride a wheel "on the flat," but they should not attempt high hills. Walking is good. Usually I make my patients walk at least two miles a day. Another point is of importance. In ordinary cases of broken compensation, very small doses at comparatively short intervals are more effective than larger doses at longer intervals. Furthermore, when it is necessary to give large doses of any drug, in attacks of heart failure, it is important to gradually reduce the dose, and lengthen the interval, just as soon as there is a proper response to the action of the drug.

Chapter XX.

NAUHEIM METHODS IN CHRONIC HEART DISEASES WITH AMERICAN ADAPTATIONS.[1]

Successful practice of the Nauheim system in chronic heart diseases includes the use of carbonated brine baths, resistance exercises and massage; while the diet and general health of the patient have to be regulated. It follows, therefore, that this method requires the immediate supervision of a physician, and that no masseur or operator, however skilled in his branch, can be competent to employ it, independently of a physician, even if permitted to do so by our State laws.

While the advantages of carbonic acid gas as a therapeutic agent are generally known, a method of using the *dry gas* has been brought before the profession in this country by Dr. A. Rose, of New York, and successes with it have been reported in neuralgias, pareses, rheumatism, amenorrhœa, indolent ulcers, catarrh, etc.

Though it has been used in many of the prominent bathing resorts for a long time, the application of it in chronic heart diseases has been a more recent affair. In fact, so far as we know at present, it appears to have originated with Beneke, who at one time practiced medicine in Nauheim. But his claims were modest, for in his time the waters were chiefly used for gout and rheumatism. Later Oertel, a physician who also practiced in Nauheim, elaborated a system that combined baths, massage and graduated hill-climbing. This method came to be known as the *Terrain Cur.* After Oertel's death, however, his method fell into comparative disfavor. Finally, August Schott, while treating neurasthenics by the Ling system of resistance movements, found that they stimulated the heart, and with it the general circulation. Hence the origin of the *Schott system.*

To-day the methods employed at Nauheim are mainly the elaboration of the work of these three men, though the present physicians of this locality, such as Groedel, Theodor Schott and others, have added much to the details of the methods, while their large experience has qualified them to define its limitations.

For particulars as to further details of the methods employed

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[1] Originally published in *Internat. Clinics,* Vol. I, ser. 13. Feb. 1904.

at Nauheim, the reader is referred to the numerous articles that have been written on this subject, and especially to the papers of Dr. W. C. Rives (*New York Med. Jour.*, 1896, LXIV, pp. 471-479) and Dr. Victor Neesen (*New York Med. Jour.*, March 10,1900). Both of these physicians have enjoyed the unusual advantages of assisting in the treatment of heart diseases at Nauheim, and later, of practicing these methods in this country.

In the year 1897,[2] I began to apply the Nauheim system in chronic heart diseases, using liquid gas and following out Nauheim methods as well as I could, but I found it impossible to transplant the system bodily to this country. Modifications to suit our altered conditions were necessary, and I purpose in the following pages to detail the adaptations made. In the first place, after a few months' trial of liquid gas, I abandoned it for reasons that need not be detailed here, and began generating the gas by the combined use of an acid sodium sulphate and the bicarbonate. This was merely an adaptation of the Hamburg method, known as Sandow's. I have generated gas in these two ways for over eight years, and thus far without any accidents or bad results. During this period carbonated brine baths have come to be used extensively in typhoid fever, also in neurasthenic states, lithæmia and various other affections,[3] and the sphere of their utility is being gradually extended.[4] My experiences, however, have enabled me to define in my own mind with tolerable accuracy both the scope and the limitations of the system, so far as heart diseases are concerned. I cannot preface what I am about to write with anything better than the following sentences: Successful application of the Nauheim system requires the closest attention to details. Disregard of them means failure. The largest success comes from the most careful attention to each apparently insignificant feature.

My methods are as follows: I use mainly, as I have always done, resistance exercises; by which I mean exercises where the patient makes voluntary movements that are resisted by the operator. These movements are quite gentle, and there are intermissions between them. If there should be any weariness on the part of the patient, or if his breathing becomes rapid or the pulse rises, a pro-

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[2] On Jan. 28, 1897, I read a paper on this subject before the Med. Soc. of the State of New York.

[3] See Reports by Putnam and Fitz, *Boston Medical and Surgical Journal*, March 13, 1902; and S. Baruch, *Medical Record*, January 10, 1901.

[4] The experiments of numerous observers have demonstrated not only that contraction of the heart usually follows the baths, but that the cardiac action is made less frequent, more regular and stronger. (See Camac in *Johns Hopkins Bulletin*, Feb., 1904.) I have verified these facts many times by the use of Cook's modification of the Riva Rocci sphygmanometer.

longed intermission is given, until the equilibrium of respiration or pulse is regained. There should always be, however, an intermission of at least one minute between each exercise of the series. The operator uses both hands, one supporting the part being exercised, whatever it may be, the other resisting the movement gently, but firmly. But the operator's hands in resisting should grasp the limb or trunk or head with only sufficient force to steady it during the required resistance. At the end of each movement the part is restored to its natural position. The resistance should, in every case, be graduated to the patient's strength, and it is here that the experience of the operator comes into play—for his trained hands easily recognize the amount of the patient's strength, as soon as the latter begins the execution of a movement.

During the entire seance the patient should breathe naturally. As a rule, each muscle is made to do its work with a gradually increasing force, from the beginning to the end of the course.

The movements themselves are flexion, extension, adduction, abduction, and rotation of the limbs, neck and trunk. The object of the exercises is to improve the action of the heart and general circulation. The following is the general outline for the first two weeks of an ordinary course: I begin with the *passive*, that is, unresisted movements, such as I introduced in 1898. They are useful in all cases, but especially in any form of embarrassed respiration, and are, in effect, the movements of artificial respiration.

In one variety adapted for women and stout persons the patient lies on the back with the head a little elevated (Fig. 25). The operator then stands at the patient's side and, passing his two hands beneath the chest, raises it slowly and gently, as far as it will go without lifting the patient off the couch or bed. The patient should aid the operator as far as possible by making his respiration coincide with the movements of the operator. One or two natural respiratory efforts should take place after each artificial movement. From 8 to 16 are the limits in the two minutes devoted to this exercise. Then the chest is allowed gradually to return to its normal position. With very obese persons two operators may be required. With a folded sheet passed under the back, and each operator grasping an end, the procedure can be employed with ease. It is called "*chest raising*," in distinction from the next-named exercise.

The second respiratory movement, which is suited for children and persons of slight build, is shown in Fig. 26. It is called

FIG. 25.—Chest raising. Operator indicated by an X.

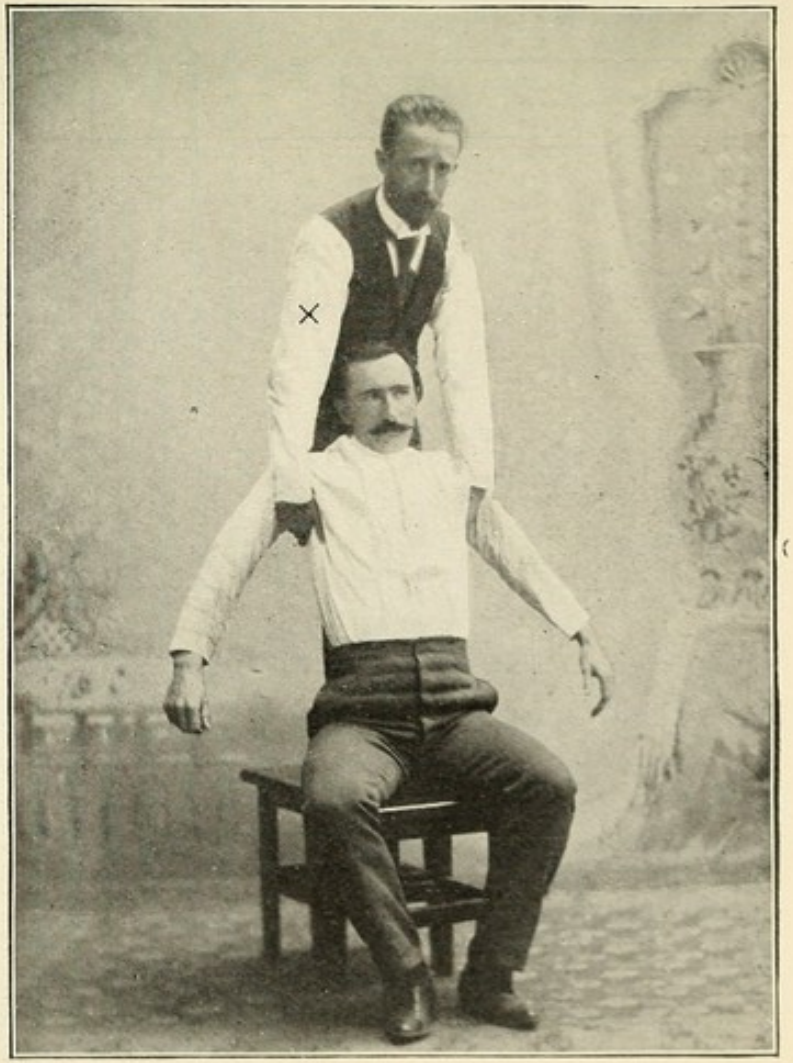

FIG. 26.—Shoulder raising. Operator indicated by an X.

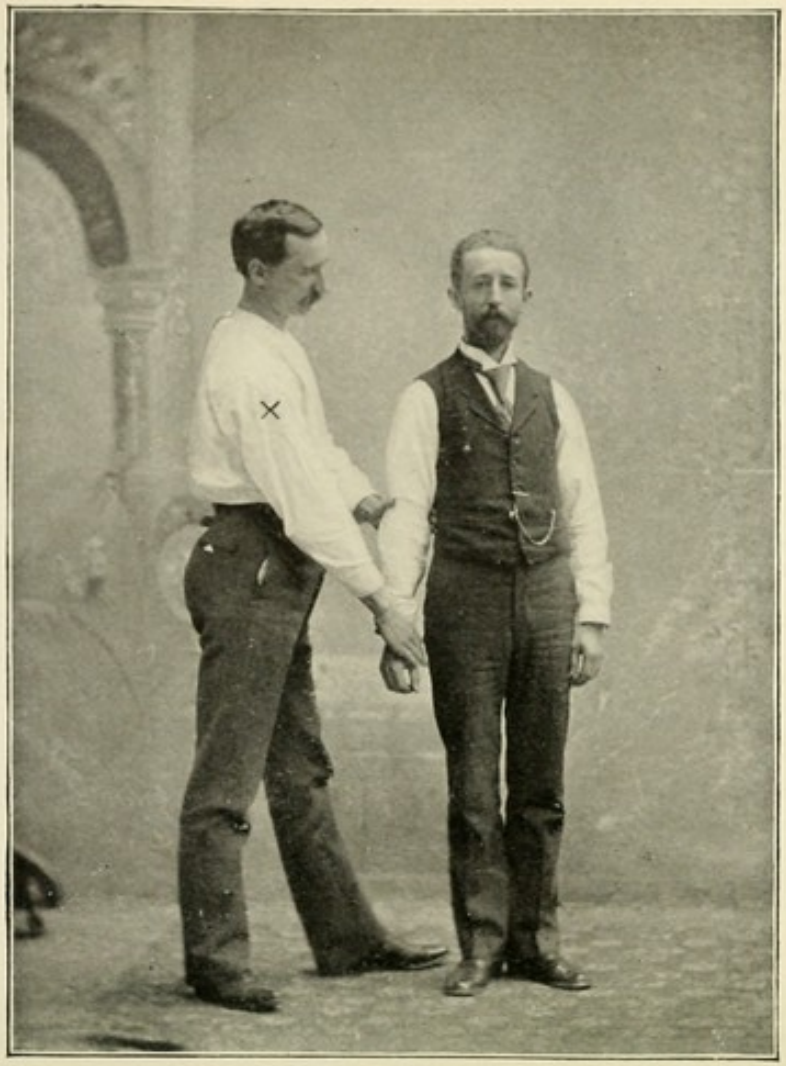

FIG. 27.—Forearm flexion and extension. Operator indicated by an X.

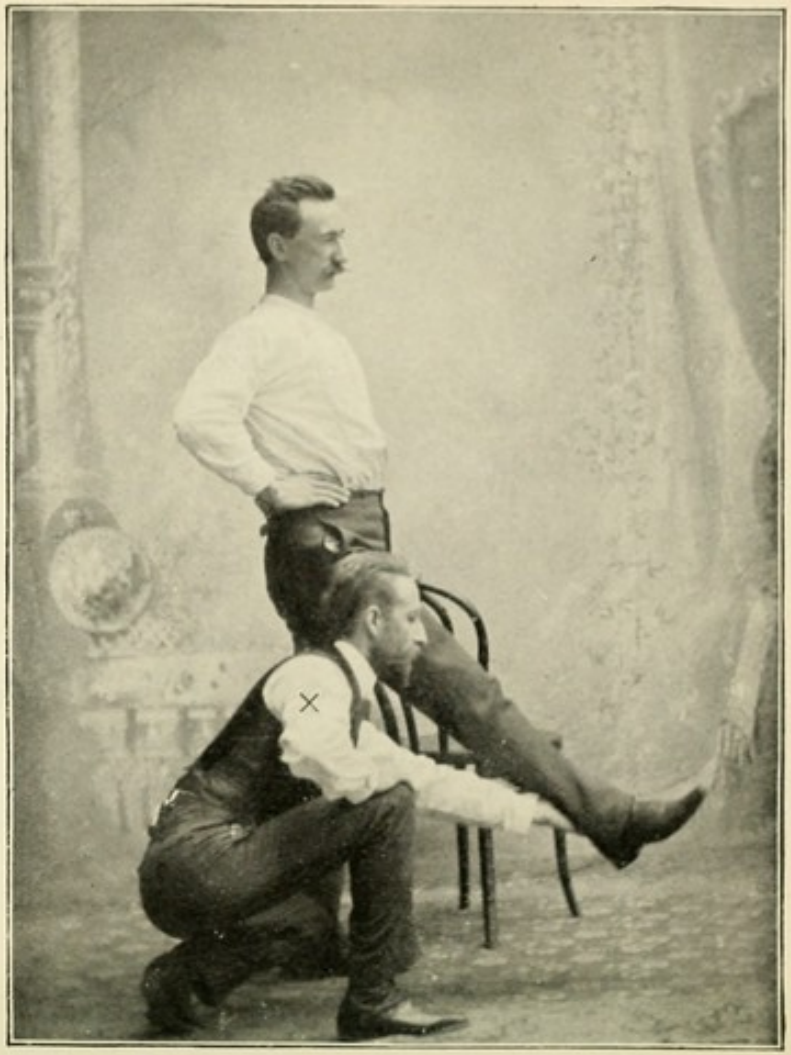

FIG. 28.—Leg and thigh flexion and extension—Extension. Operator indicated by an X.

"*shoulder raising.*" The patient sits on a stool while the operator stands on another behind him. The operator passes his hands over the patient's shoulders in front and grasps him by his armpits; he then lifts the patient's shoulders upwards as far as they will go without his being raised from the stool, the patient offering no resistance. As in "chest raising," the patient should aid the operator slightly by making his respiration coincide with the artificial movements of the operator. One or even two natural respirations should be made after each artificial movement. From 8 to 16 should be the limit of the artificial movements, in the two minutes devoted to this exercise. After each movement, whether passive or resisted, massage, percussion, or vibration is given first to the extremities and then to the trunk, the foot and leg being first massaged; later the hand and forearm.

The first resistance movement following, after an interval of a minute is *forearm flexion* and *extension* (Fig. 27). The arm of the patient is supported by one hand of the operator applied above the elbow, while the other rests lightly on the patient's hand. The patient now flexes the forearm on the arm to the extreme limit, the operator gently resisting. Then the arm is returned lightly to its place by the operator.

Extension is begun when the forearm is flexed, after the customary interval. The operator still supporting the arm above the elbow, grasps the wrists lightly and resists, while the patient extends the arm, carrying his hand down to the naturally dependent position. Then follow hand and forearm massage, which should be light, and not prolonged more than two minutes. The next resistance movements are *leg and thigh flexion* and *extension.* (Figs. 28 and 29). The leg is first extended forward by the patient and then returned to its original position, and after the intermission carried backward. Resistance is given in each case by the operator pressing the open hand against the lower part of the limb. Slight massage of the shoulder and arm, for two minutes, follows.

The next resistance movements are *thigh flexion and extension* (Figs. 30-31). To give this movement properly the patient should sit on a chair or lounge, with his neck firmly supported in some way. This position is not only comfortable for the patient, but it enables the operator to use a little more resistance, than would otherwise be deemed advisable, without disturbing the heart's action. During any exercise the patient should, if possible, support himself by resting his hand on a chair or some other firm object.

Then follows light chest percussion or flagellation with the tip of the fingers. This movement is particularly useful in chronic heart disease. It appears to aid in contracting the heart[5]—stimulating the respiratory centers through the cutaneous nerves.

The next movements are *trunk flexion* and *extension* (Figs. 31 and 32). The patient, placed with his back against the wall and his feet standing out some seven or eight inches, bends his body forward, the operator resisting with one hand over each shoulder-joint. When the body is bent over to form an angle of about forty-five degrees with the wall, the patient erects or extends himself, the operator now resisting with his hands over the scapulæ.

The course is concluded by massage of the thigh and back. To each of the exercises in this schedule two minutes are allowed, with intervals of one minute each, so that the length of the entire seance is under thirty minutes. This scheme is applicable for the first two weeks in ordinary cases, and is known in my system as Scheme No. 1. The following is a brief outline of it:

Scheme No. 1.

1. Chest lifting—lying or sitting	2	minutes.
Intermission	1	"
2. Foot and leg massage	2	"
Intermission	1	"
3. Forearm flexion and extension	2	"
Intermission	1	"
4. Hand and forearm massage	2	"
Intermission	1	"
5. Leg and thigh flexion and extension	2	"
Intermission	1	"
6. Arm and shoulder massage	2	"
Intermission	1	"
7. Thigh flexion and extension	2	"
Intermission	1	"
8. Chest percussion	2	"
Intermission	1	"
9. Trunk flexion and extension	2	"
Intermission	1	"
10. Thigh and back massage	2	"
Total length of seance	29	minutes.

During the second and third weeks the number of exercises remains the same, but a new group of muscles is exercised, and the duration of each exercise is lengthened.

The course begins with chest-lifting, foot and leg massage, forearm flexion and extension, and hand and forearm massage, but in place of leg extension there is *leg abduction* and *adduction*—more difficult movements (Figs. 33 and 34). The illustrations show how each movement is made by the patient and how it is

[5]Abrams, *Med. Rec.*, Jan. 5, 1901.

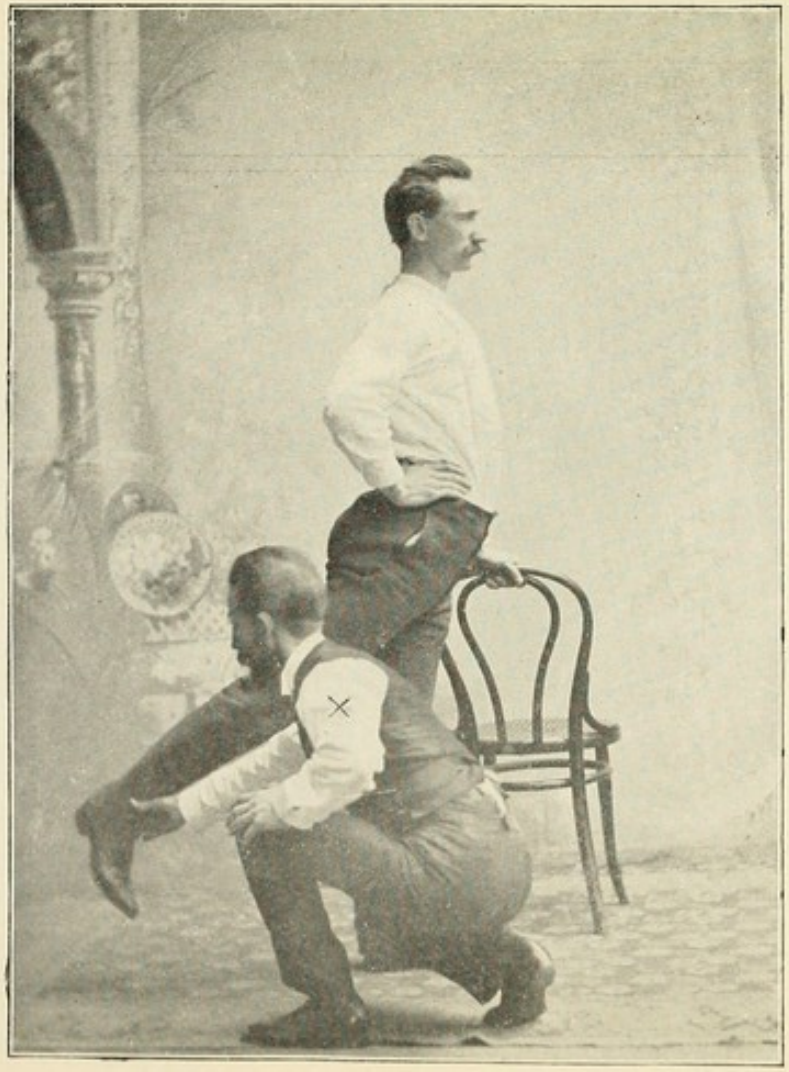

FIG. 29.—Leg and thigh flexion and extension—Flexion. Operator indicated by an X.

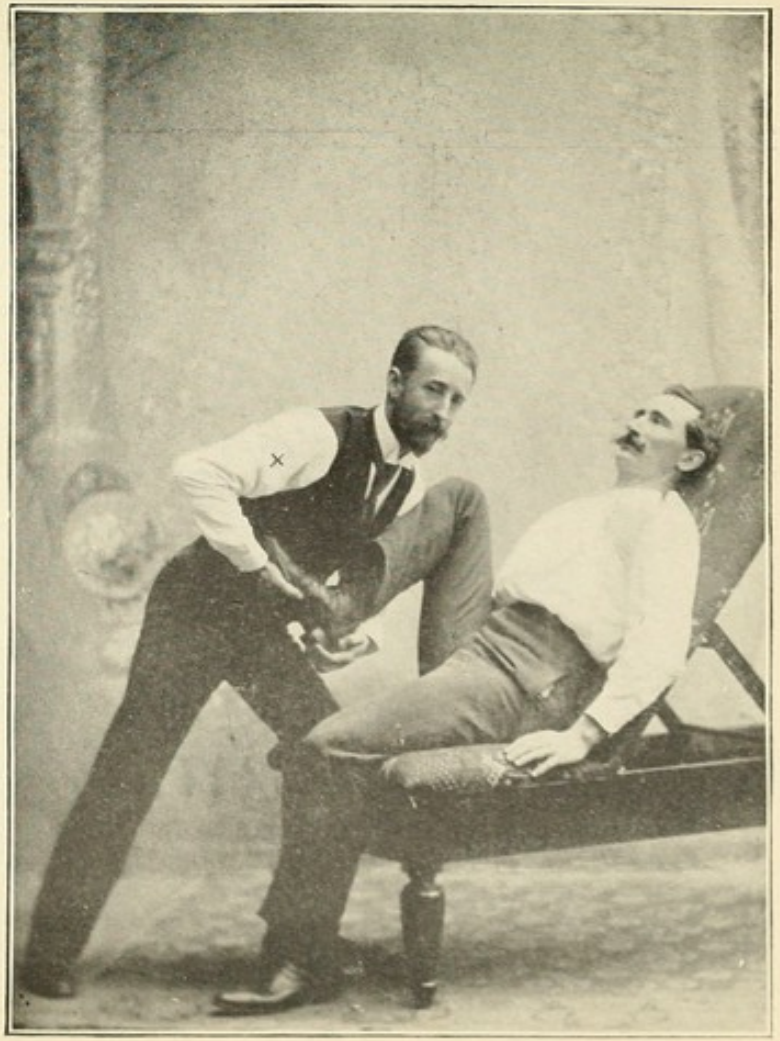

FIG. 30.—Thigh flexion and extension. Operator indicated by an X.

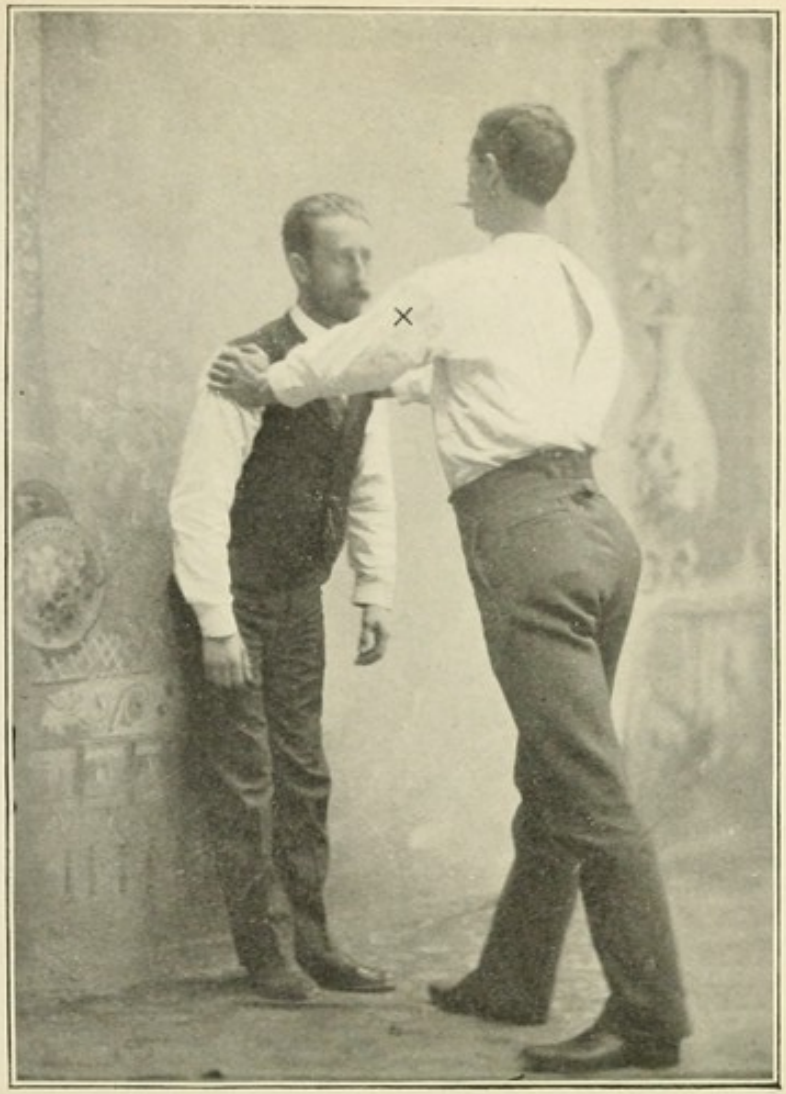

FIG. 31.—Trunk flexion. Operator indicated by an X.

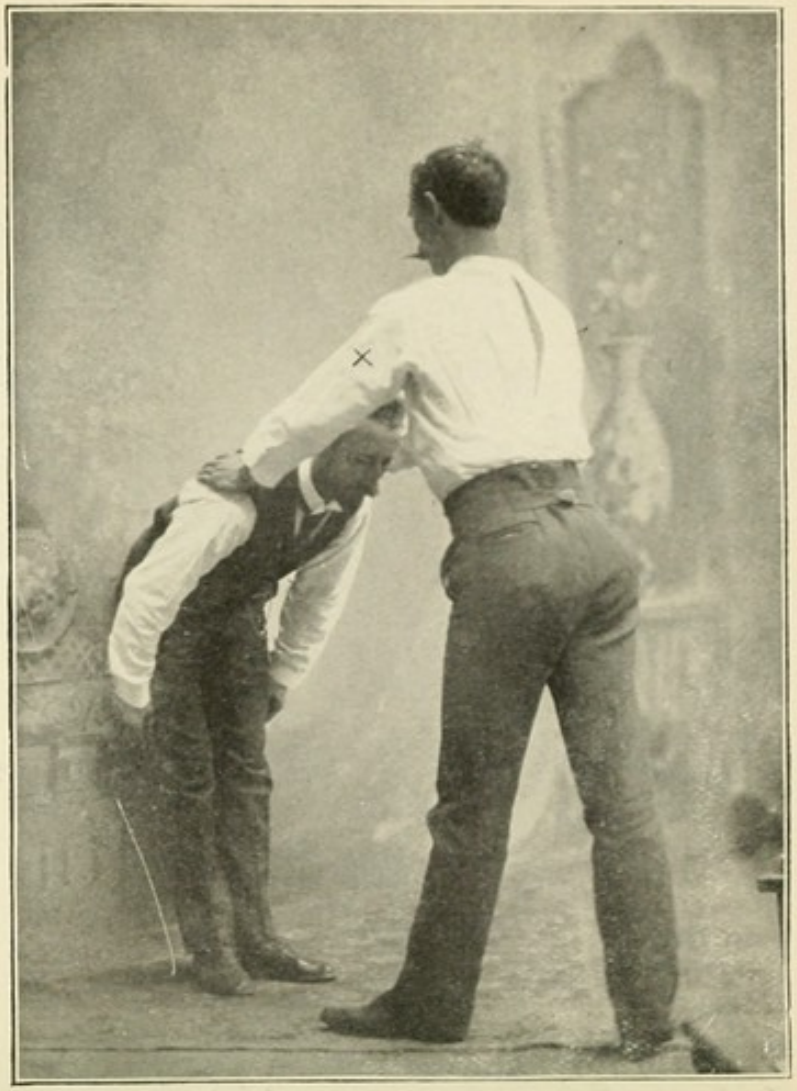

FIG. 32.—Trunk extension. Operator indicated by an X.

resisted by the operator, who, in each case, resists with the open hand pressed against the lower part of the leg.

Then after arm and shoulder massage, comes, in place of the trunk flexion of Scheme No. 1, *trunk rotation.* The method is as follows:

The operator, standing close to the patient, whose feet are firmly fixed, grasps one shoulder with each hand and assists the patient to turn his body about, on its vertical axis, as far as possible (Fig. 35). Then the patient untwists and retwists himself until he has turned around equally far in the opposite direction. Then the patient slowly assumes his natural attitude.

Chest percussion is then followed by *arm separation,* "the swimming exercise" (Figs. 36 and 37). The patient, holding his wrists parallel, carries them backward until in line, the operator resisting, after which the latter returns them easily to their original position. It is not desirable to carry the arm forward with resistance, because such a movement would fix the chest, through the action of the pectoral muscles, thus interfering with natural respiration. During this exercise the patient may sit, stand, or lie down. If he stands, he should brace himself slightly by putting one foot forward. This series ends, as in scheme No. 1, with *thigh and back massage.*

The following is a brief epitome of

SCHEME NO. 2.

1. Chest lifting—lying or sitting	2	minutes.
Intermission	1	"
2. Foot and leg massage	2	"
Intermission	1	"
3. Forearm flexion and extension	2	"
Intermission	1	"
4. Hand and forearm massage	2	"
Intermission	1	"
5. Leg abduction and adduction	3	"
Intermission	1	"
6. Arm and shoulder massage	2	"
Intermission	1	"
7. Trunk rotation	3	"
Intermission	1	"
8. Chest percussion	2	"
Intermission	1	"
9. Arm separation	3	"
Intermission	1	"
10. Thigh and back massage	3	"
Total duration of seance	33	minutes.

In Scheme No. 2 each of the resistance exercises consumes 2 to 3 minutes and some newer groups of muscles are involved.

In Scheme No. 3, which is applicable for the fifth and sixth weeks, a further change in the series is made, so far as the resistance

movements are concerned, and some new ones are introduced that might not be well borne in the beginning of the course—two particularly, *"quarter-circling"* and *"head rotation."*

Scheme No. 3 is as follows: After the chest lifting, and foot and leg massage comes "quarter-circling" (Fig. 38). The patient sits on a stool, the operator behind him with one hand on the patient's shoulder in order to fix it, the fingers of the other hand lightly grasping the wrist. The patient's arm being extended downward, it is carried first upward and forward, then downward and backward over a quarter of a circle, the operator resisting. Then, after the regulation hand and forearm massage, comes rotation of the head on the neck, or, if this cannot be well borne, simple flexion of the head on the neck. Then follow arm and shoulder massage, trunk twisting, and chest percussion, the last resistance exercise being *lateral flexion of the trunk* (Fig. 39). The trunk is simply bent on the pelvis laterally, the operator standing in front of the patient and facing him, resisting him as he bends his body well over to one side. One hand of the operator rests on the ribs beneath the axilla, the other on the opposite hip. The trunk having thus been placed in a position of extreme flexion the patient attempts to straighten himself and carry his body over to the other side in extreme lateral flexion. This whole movement from extreme flexion on one side to extreme flexion on the other, is resisted by the operator. By reversing this exercise, there is another movement, the operator of course reversing the position of his hands. This series ends as usual with thigh and back massage. In brief, it is as follows:

SCHEME No. 3.

1. Chest lifting—lying or sitting	2	minutes.
Intermission	1	"
2. Foot and leg massage	2	"
Intermission	1	"
3. Quarter-circling forward and backward	4	"
Intermission	1	"
4. Hand and forearm massage	2	"
Intermission	1	"
5. Head rotation or flexion	3	"
Intermission	1	"
6. Arm and shoulder massage	2	"
Intermission	1	"
7. Trunk Twisting	4	"
Intermission	1	"
8. Chest percussion	2	"
Intermission	1	"
9. Trunk flexion (laterally)	4	"
Intermission	1	"
10. Thigh and back massage	3	"
Total duration of seance	37	minutes.

Fig. 33.—Leg abduction. Operator indicated by an X.

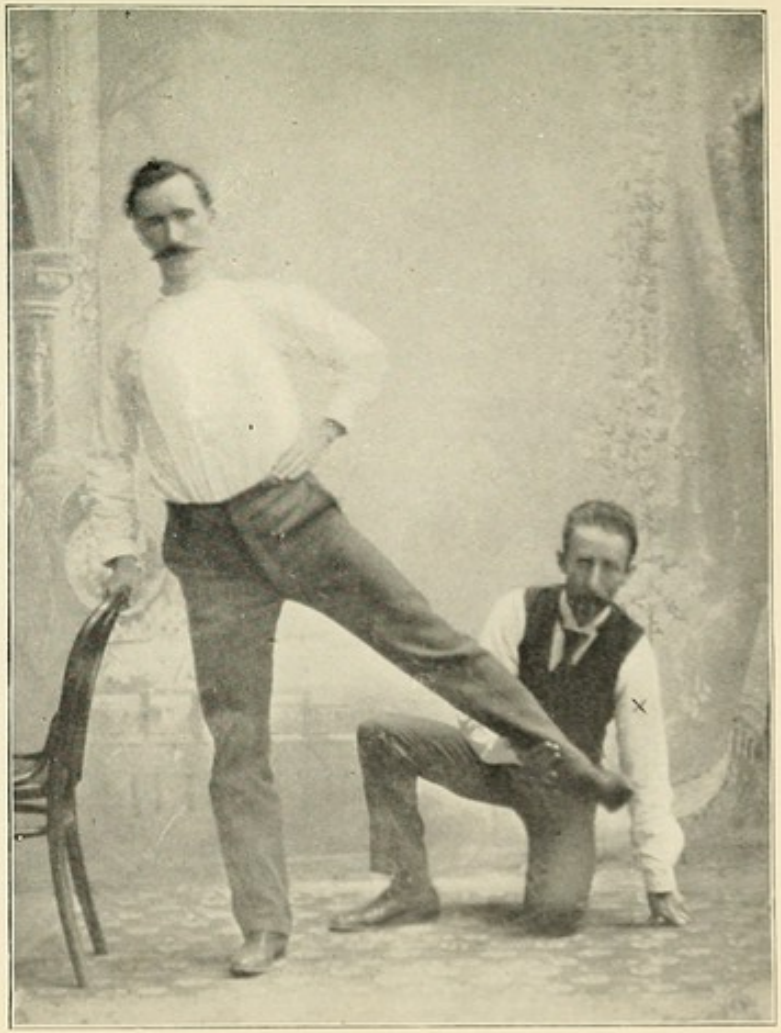

Fig. 34—Leg adduction. Operator indicated by an X.

Fig. 35.—Trunk rotation. Operator indixated by an X.

Fig. 36.—Arm separation. Operator indicated by an X.

It will be noticed that in the final series the exercises involve newer and larger groups of muscles, and that all of the great muscles of the body have been exercised. The total duration of the seance is also longer and it is intended that more force should be applied.

By adopting a course such as I have outlined, success will be attained in appropriate cases. Failure in carrying out this part of the treatment is usually due to carelessness in the selection of operators, for on them success is dependent. This topic has been well placed before the profession by Dr. Victor Neesen.[6] Indeed, unless operators have had special training in this branch of their work, success is hardly to be expected, for the method is a peculiar one. Following the Swedish system, many operators are too rapid in their movements. Others fail to observe the required intermissions. Some are over-persuaded by their patients, and allow them to exert undue force. Others fail to make the patient breathe naturally. Others blindly follow cast-iron rules, even where it is plainly apparent that from some idosyncrasy, or temporary disability, the patient cannot follow the rules, in every particular. Perhaps the most common and worst fault is that undue force is used in massage; or the operator disregards the rule that heart cases should have no abdominal massage, unless it is expressly ordered by the physician. It has therefore been a public benefit that the attempts of masseurs to practise the Nauheim treatment, without medical supervision, has been a failure, in this city at least.

Fundamental rules for the operator are:

1. Perform each movement slowly, steadily, and gently.

2. Each single movement is to be followed by a distinct intermission, in which there is entire relaxation of the patient's muscles. It is a good plan to have the patient rest himself in a chair after each movement or series of movements. Double movements should be followed by longer intermissions. In the intermission between the single or double movements, the operator should take the pulse and respirations and record them on a slip prepared for the purpose.

3. Movements of the arm should alternate with those of the leg or body.

4. No part of the body should be held so tightly as to compress the blood vessels.

5. The patient should be made to breathe naturally during the seance.

6. The operator should be on the watch for irregular breathing,

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[6] *New York Medical Journal*, May 18, 1901.

pallor, or blueness of the lips or face, or any sign of personal discomfort or disturbance on the part of the patient. Upon the advent of any of these signs the exercises must be suspended, for they indicate that there has been too much resistance, or that the movements have been too rapid, or the intermissions too short.

7. In the matter of exercises and massage, always err on the side of light and delicate treatment.

There are many movements besides these that have been enumerated. In my first article I described 38, but physicians now usually limit themselves to a much smaller number. To the present list of 15 double or single movements I sometimes add *flexion of the wrist* and *ankle*, making a total of 17 movements. But I now omit all exercises in which the hands are raised above the head, because the increased resistance thus opposed to the column of blood throws more work on the heart.

A word about *massage, percussion,* and *vibration:* They are not only useful in the treatment of chronic heart diseases, but should form a part of any system; though they are less valuable than the exercises and baths. They are especially useful in lithæmic and neurotic patients.

Abdominal massage, however, is prohibited, because in dilated hearts, for which this treatment is mainly applied, the liver, spleen, and other chylopoetic viscera are apt to be both swollen and tender, and therefore liable to injury. Besides, as shown by physiological experiments, abdominal massage tends to inhibit the heart's action. Massage or percussion or vibration is given in the intervals between the voluntary or resistance exercises, largely because it puts a bar upon undue muscular efforts on the part of the patient or operator.[7]

THE ARTIFICIAL BATH.

The course of treatment, which usually last from four to six weeks, is best preceded by a short period of rest—from two to three days at least, during which the patient's various functions are investigated. At first the baths should be moderately warm and saline; later cooler and more saline; still later, mildly effervescent; finally chloride of calcium is added, and the salines and carbonic acid gas increased, while the temperature is decreased.

The duration of an immersion should be from four to fourteen

[7] That these exercises improve the rhythm and force of the heart and diminish its frequency I have demonstrated, latterly in the Manhattan State Hospital West at clinics held there.

FIG. 37.—Arm opposition. Operator indicated by an X.

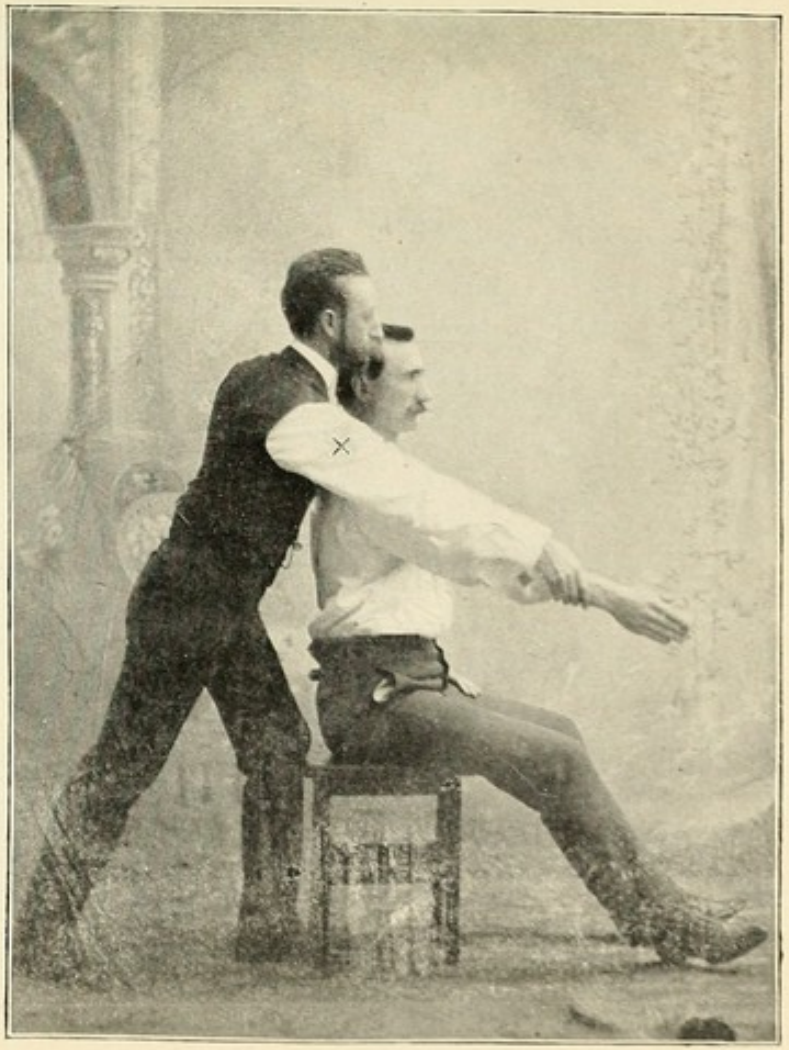

FIG. 38.—Quarter-circling. Operator indicated by an X.

FIG. 39.—Trunk flexion laterally. Operator indicated by an X.

minutes, but no longer. The temperature should not exceed 98° F., nor fall very far below it. I rarely let the temperature fall below 90° F. Sometimes I do not decrease it more than one degree during the entire course. At first the baths are given with an intermission every third day, later every fourth day, still later, every fifth day. On the day of intermission the physical condition of the patient should be thoroughly looked into. The effect of carbonic acid gas on the skin is that of an irritant (Rose); with the salines stimulating the vasodilators (Baruch).

An artificial Nauheim bath can be improvised in many different ways. Any one who can get sea salt, rock salt, or ordinary bathing salt, calcium chloride, sodium bicarbonate, and muriatic acid, and has at his disposal a wooden, porcelain, or enamelled tub, can so imitate the original Nauheim method as, I believe, to retain its essential therapeutic qualities. But in practice it has been found better to use an acid sodium sulphate in place of hydrochloric acid, and this has been done by the manufacture of solid discs of the acid sulphate, which may thus be readily handled without harm to one's person or clothes. The manufacturers supply the carbonating ingredients in a wooden box about 8x6x4 inches containing in dry compact form eight discs of the acid sodium sulphate wrapped in heavy lead foil, and four packages of sodium bicarbonate.[8]

In preparing the baths, break the discs with a hammer, put them on a plate, and powder them over with the bicarbonate. In about one minute after the ingredients are put in the bath, the gas will be liberated.

Both the bicarbonate and the acid sulphate are almost chemically pure. All the water of crystallization has been eliminated from the discs, which contain about forty-two per cent. of free acid. They are hard and do not deliquesce at any temperature, which is important. They keep for an indefinite time, but, being hard, dissolve slowly. By this method the evolution of carbonic acid gas is continuous until the tablets are dissolved, and as much of the gas is given off, as there is in an ordinary carbonated bath at Nauheim.

For bathing salt I often prefer to begin with an ordinary American sea salt, as by so doing, I get the benefit of the iodides and bromides it contains, but if this is not important, I use a good quality of dairy salt, such as the Genesee. At the end of the first

[8]Manufactured by the Cassebeer Pharmacal Company, 108 Fulton Street, New York City.

week, I carbonate the baths. Then when a large amount of salt is to be used, I substitute ordinary bathing salt for the sea salt, increasing at the same time the amount of carbonic acid gas. When the gas exists in a fair percentage in the bath, its presence is shown by minute bubbles attaching themselves to the body of the bather. At the middle of the course I fortify my ordinary bathing salt with the imported Nauheim Mutterlauge (the so-called concentrated brine salts). By so doing I get the requisite quantity of the calcium chloride for the stronger bath. These imported Nauheim concentrated salts (Mutterlauge salts), contain about seventy-five per cent. of calcium chloride. If for any reason it is desirable to have a bath in which all the ingredients are thoroughly dissolved, pure calcium chloride is preferable to the concentrated brine salts.

An ordinary Nauheim bath contains from two to three and one-half per cent. of salt, of which eighty-two per cent. is sodium chloride and ten per cent. calcium chloride.

As therefore the imported concentrated salts are rich in calcium chloride, by using the proper proportion of them, we can get at the same time the requisite quantity of calcium.

My scheme for the ordinary Six Weeks' Course is as follows:

First Week. (No. 1 Bath).—One-half per cent. plain warm salt bath. (Two pounds of bathing salt to 50 gallons of warm water.) Temperature, 98° F. Duration four minutes. Intermission on the third and sixth days of this week.

Second Week (No. 2 Bath).—Three-quarters per cent. warm salt bath. (Three pounds of bathing salt to 50 gallons.) One-quarter per cent. carbonic acid gas (two discs to one package). Temperature, 97° F. Duration, six minutes. Intermission on the fourth day of this week.

Third Week (No. 3 Bath).—One per cent. warm salt bath (Four pounds of bathing salt to 50 gallons.) Temperature, 96° F. Carbonated acid gas one-half per cent. (four discs to two packages). Duration, eight minutes. Intermission on the fourth day.

Fourth Week (No. 4 Bath).—One and a quarter per cent. salt bath. Five pounds of bathing salt to 50 gallons.) Nauheim concentrated brine salts, 8 ounces, i. e., a half pint of the liquid salts, or Mutterlauge; or 6 oz. chloride of calcium. Carbonic acid gas, three-quarters per cent. (Six discs to three packages.) Temperature, 95° F. Duration, ten minutes. Intermission on the fourth day.

Fifth Week (No. 5 Bath).—One and a half per cent. salt bath. (Seven pounds of bathing salt to 50 gallons.) Concentrated

brine (Mutterlauge salts), three-quarters of a pint (12 ounces), or 9 ounces chloride of calcium. Carbonic acid gas, three-quarters per cent. (Six discs to three packages.) Temperature, 94° F. Duration, 12 minutes. Intermission on the fifth day.

Sixth Week (No. 6 Bath).—Two per cent. salt bath. (Ten pounds of bathing salt to 50 gallons.) Concentrated wet brine salts (Mutterlauge), 16 ounces, i. e., one pint; or 12 ounces of the dry chloride of calcium. Carbonic acid gas, one per cent. (Eight discs to four packages.) Temperature, 93° F. Duration, 14 minutes. Intermission on the fifth day.

It will be noticed that the amount of carbonic acid gas in the bath varies from one-quarter to one per cent.

The number of baths in a full course is 35, but sometimes a less number is sufficient. Twenty-four is a good average number. With robust people it may be well to commence at once with the carbonated brine bath. Sometimes, especially with delicate persons, the Number 4 bath will be as strong as is desirable.

The method of preparing the bath is as follows: Fill the bath-tub—preferably of porcelain or enamelled iron—with 50 gallons of water at about 105° F.; then add the ingredients as follows: first add the required quantity of bath salt, then place the discs of the acid sulphate (broken or pulverized), intermixed with the sodium bicarbonate, on a couple of saucers or on a single plate on the bottom of the tub. An evolution of gas takes place in a minute and lasts 20 or more minutes. The patient should enter the bath when it has reached 98° F. or lower, according to the directions, and remain in it the prescribed length of time. As soon as the gas is evolved it will be seen attaching itself to the trunk and limbs in the form of minute bubbles.

The advantages of the artificial bath may be briefly summarized as follows:

It contains the chief natural constituents of the Nauheim waters, and, by the scheme that has been given, is applicable in most chronic heart diseases. The carbonic acid gas is held in better solution, by the artificial bath, than by the natural waters of Nauheim. The artificial bath is cleaner than the natural, which contains substances of no obvious utility. The artificial bath is taken at home rather than in a public resort and just before retiring, so that there is little or no danger of catching cold. An artificial bath costs less in this country than in Germany, and the total cost of the treatment brings it within the reach of a moderate purse. There is no danger

from free carbonic acid gas, as it is held in closer combination than in the Nauheim waters. These baths can be given at any time of the year. The discomforts of a foreign trip are avoided.

On the other hand, there are certain advantages in the Nauheim natural baths. Whatever of virtue there is in the natural water is retained. The patient has the choice of numerous physicians of repute in the treatment of chronic heart disease. The patient, being away from home, is for the time entirely freed from many of the annoyances of business, or the cares of private life, in the comparative seclusion of a small watering place,—matters of value in the treatment. He may find the Nauheim course an agreeable feature of a summer jaunt abroad.

Whether it is better to take a course at home or abroad is for each individual to decide after comparing the advantages of one with the other, as seen from his special point of view.

So far as the actual advantages of the treatment are concerned, it is certainly quite as well given in this country as in Europe, and with more comfort to the patient. In fact, one disadvantage, and a serious one, of the European baths, is that they are not given in the late autumn, winter, or early spring; only in the late spring, summer, and early fall.

The above method of treatment is at present the best for general use, and can be practiced in the home.

There is, however, another method of preparing the bath, known as the Erlinger system, which is in use by the Harveian Society of New York City.[9] Liquid carbonic acid gas from a drum is allowed to pass into a metal chamber containing the brine solution. Paddles rotated by machinery mix the gas so intimately with the water that it is retained for a long time in the water. The temperature of the bath is regulated by the addition of the required amount of hot water, and the strength of the bath is made to conform to the physicians prescription. In place of the concentrated brine salts, chloride of calcium is used, in the requisite quantities.

[9] At 135 West 45th Street, New York City.

Chapter XXI.

PROGNOSIS IN HEART DISEASE.

The forecast in heart diseases involves many considerations, and is therefore beset with uncertainties. Age, sex, station in life, habits and occupation, the variety of the disease, its benign, malignant, functional or organic character, its location, extent and duration, its complications and accidents, the constitutional diseases with which it is associated, the presence or absence of compensation, and management, intelligent or otherwise, are governing factors in the expectation of life.

In infants under one year the outlook is particularly unfavorable. During childhood it is worse than in adult life. Children with serious valvular diseases before eight, seldom reach adult life. As they approach puberty, heart weakness is apt to set in. Holt found that in 225 cases of congenital heart disease, 60 per cent. were fatal before the end of the fifth year, and one-half during the first two months; 16 per cent. of the cases, however, lived over 16 years, and 8 per cent. over 30 years. Serious valvular lesions in children progress more quickly than in adults. Owing to their rapid growth, and the demands made on their strength, cardiac dilatation occurs early. Acute diseases still further increase the danger to life. (*Dis. of Infancy*, New York, 1902.)

But the danger is not immediate. In fact, both acute and chronic endocarditis in children are rarely of themselves fatal, death being due to associated conditions, such as pneumonia or pleurisy.

We see then how it is that the first years of life are especially dangerous for children with heart disease. However, according to Koplik (*Dis. of Infancy*, N. Y., 1902), in mild forms of heart disease in children, recovery is to be expected, though the outlook in rheumatic patients is especially unfavorable, for they are apt to succumb within a few hours after the first attack.

Women have a better expectation of life than men, because their lives are, on the whole, less strenuous and more orderly, though during pregnancy and parturition they run great risks; in the one case from the upward pressure of the abdominal organs, and in the other from the severe efforts of expulsion, with resulting cardiac strain, due to the suddenly altered circulation. Heart failure may

cause death under any of these circumstances. In pregnant women, however, the greatest distension and pressure is forwards rather than upwards. Though instrumental interference under anæsthetics is frequently resorted to, the treatment of heart complications is much better understood than formerly. According to Edgar, if the disease is valvular, and there is compensation, the danger to life is small, and need not be taken seriously, though the induction of artificial labor may sometimes be necessary. Mitral stenosis is most dreaded by obstetricians. If death does not result, there may be placental apoplexy or abortion. Nitrous oxide or ether are the preferable anæsthetics. During pregnancy and after child-birth, strychnine is sometimes given, digitalis rarely. Uncompensated endocarditis is unfavorable for mother and child.

For men whose occupations call for continuous or violent physical strain, exposure to inclement weather, especially if they take stimulants in the place of proper food, the prognosis is, as a rule, unfavorable. Yet among men or women who live regular lives, free from its ordinary vicissitudes, a fright or mental strain, overeating, arrested digestion, the immoderate use of tobacco, or, indeed, any sudden tax on the system, may cause a fatal result. In very old people a rather common cause of sudden death from heart disease is the eating of indigestible food, or more food than can be digested.

It is true that from the gradually accumulating facts about heart diseases, we are getting more definite views as to the expectation of life, and from the increasing care with which post-mortem examinations are made in our public institutions, we may expect still greater definiteness in the future. But still we are very deficient in data for making accurate estimates, so that our forecasts are necessarily vague. Still a certain number of facts governing the expectation of life are known.

It is generally admitted that functional diseases of the heart do not much affect longevity, though they certainly make the organ more susceptible to disease, while the malignant forms, whether due to new growths or infective endocarditis or carditis are pretty generally fatal. Certainly two-thirds of the recorded cases of malignant endocarditis have proved fatal.

On the whole, perhaps, myocardial diseases are less dangerous to life than the endocardial. Certainly myocardial degenerations due to the continued fevers, like typhoid and diphtheria, will, in the great majority of cases, disappear after convalescence, and, reason-

ing by analogy, degenerative changes due to other causes should yield under appropriate treatment; it is certain that the fat heart of corpulence will greatly improve under the intelligent application of reduction methods. At the same time *sudden death* occurs more frequently in myocardial disease than is generally supposed. Persons with very fatty hearts will sometimes die as if from an apoplectic stroke. The cause may be acute dilatation, or embolism due to thrombi detached from the chambers of the heart; in some cases rupture; or they may die more gradually from extrinsic causes, such as pneumonia, or gastro-intestinal distention; more generally, however, from affections of internal organs (in which the lungs, kidneys, liver play the most important roles), and which, indeed, are the terminal affections.

In myocardial diseases where there is arteriosclerosis, or in degeneration of the heart walls, where there is a persistently feeble pulse, especially with difference in the radials, the danger line is always near at hand. Myocardial syphilis must also be reckoned with as a cause of death.

Mitral obstruction is more serious than insufficiency. Mitral obstruction, if severe, means that the mitral disease is well established. Moreover, embolism may occur as often as in 20 per cent. in cases of mitral obstruction, in my experience. In Hayden's 15 cases death was at an average age of 29.26 years. In Broadbent's 53 cases of mitral stenosis it was 33 years for males and 37.38 for females. Samways (*Brit. Med. Jour.*, Feb. 5, 1898), in 196 cases taken from the records of Guy's Hospital, found the average age at death for males was 38 1-3 years, and for females the same. In severe stenosis the average age was 33.6 years, in the milder, 43.6. In 42 fatal cases by Fagge, the average age was 37.83. (Hayden, *Dis. of the Heart*, Dublin, 1875.) In one-third of them, however, there was complication with other valves. According to my personal statistics, in one series of 19 cases death most frequently took place between 37 and 38, but the extremes in these cases varied from 23 to 70, and the mean age was found to be 39.2. By reference, however, to page 60 of this manual, the average age at death in Dyce Duckworth's cases, 264 in number (not all, however, supported by post-mortem evidence), was 35. In a first series of 100 collected cases, I found the average age 35, in a second series 33. Sansom found it 32.7. In another series of 57 fatal cases, ranging from 13

to 67, 26 or 45.6 per cent. fell between the ages of 30 and 40 inclusive. The expectation of death is therefore in the fourth decade, and so far as we know at present at about the age of 34. I have, however, under observation a patient of 73, with typical signs of mitral stenosis, including the purring thrill.

In mitral stenosis sudden death is as rare, comparatively, as it is frequent in aortic insufficiency. In five of my cases it was said that the disease had lasted anywhere from three to thirty years.

Aortic insufficiency is certainly one of the most dangerous of valvular diseases, but at the same time it is less so in the absence or arteriosclerosis or angina, or where there is little displacement, and the contour of the heart is not much altered. Whether it is more grave than aortic stenosis has been questioned. In 50 of my cases the averages of age at death was 40 against 41.7 for aortic stenosis, but the stenosis was, for the most part, complicated with other valvular affections.

Pure aortic stenosis has a more favorable outlook. Comparatively few cases have been recorded, however. In 8 of which I have records, the average age at death was 56, two reaching the age of 70 and one 90.

In Hayden's 26 aortic cases, 50 per cent. were combined with insufficiency. I make it 65 per cent. in my cases. Fagge says, and I think with truth, that in combined aortic stenosis and insufficiency the prognosis is governed by the insufficiency. Judging from my figures, aortic obstruction in complicated cases is almost as serious as incompetence, but much less so in uncomplicated cases. In aortic obstruction, however, arteriosclerosis must always be regarded as an unfavorable sign. But the prognosis depends largely on the grade of obstruction. If little, the prognosis is not bad, comparatively speaking. Broadbent found the average age at death 40 years, while his oldest case was 53. He thinks aortic stenosis less serious than aortic insufficiency or mitral stenosis, in which I agree with him on the whole. However, *sudden death* in my experience is as common in aortic stenosis as in incompetence. In both I found it 20 per cent. Hayden made it 18 per cent. in all forms of aortic disease.

Mitral insufficiency is compatible with a long life. This is now the accepted opinion. Indeed, in uncomplicated mitral insufficiency the outlook is better than in any other form of valvular disease. The relative form is quite common after physical exercise, in recovery

from fevers, and in neurotic disturbances. Most athletes suffer from it at some time or other, but it usually disappears when the strain is past. On the other hand, it may be a fixture in some, as in laboring men, if there is continuous and hard strain. Still, simple mitral insufficiency acquired in early life appears to yield more easily to compensatory hypertrophy than any other form of valve diseases.

In the organic variety it is apt to be complicated with other valvular diseases. My figures show this complication in 86 per cent. of cases. Moreover, I believe that mitral stenosis is preceded by mitral insufficiency. I am not sure that it ever causes death of itself. In fact, in a record of 102 fatal cases of heart disease, I have not found a single instance of organic uncomplicated mitral insufficiency that caused death.

To my mind, therefore, the comparatively harmless character of uncomplicated mitral insufficiency is clearly established. The great danger, however, is that in practice mitral stenosis, one of the most dangerous of the common valve lesions, will be mistaken for mitral insufficiency, the least dangerous. This error has often been made, as my hospital records show.

In congenital pulmonary insufficiency the prognosis is never good, but in acquired disease, I have known of one instance in which the patient reached the age of 70.

In congenital pulmonary obstruction (stenosis), the majority die before the fourth year, and of tuberculosis, but the age of 40 has been reached. Acquired obstruction has a more favorable outlook. Owing to the position of the valve it is apt to be compressed by external influences, such as adhesions. Of my four cases one lived to be 56, and in 15 of my collected cases 42.8 was the average of age at death. One patient (Schwalbe's) lived to 84. These cases, however, are too few for statistical purposes. Pulmonary obstruction is the point about which all congenital cardiac anomalies center. Independent of the pulmonary lesion, they are apt to be incompatible with a long life.

Tricuspid obstruction is for the most part an acquired disease. It is very rarely congenital, and when so, is soon fatal; it is, in fact, the most dangerous of all valvular diseases. Usually it occurs in the train of organic valvular diseases; and is the latest of them. Patients seldom reach 40, but one of Leudet's cases lived to be 64. Exceptionally old age may be reached. In one case that I have reported, the patient lived to be 70. Samways, in 196 cases of valvular

diseases, found 32 of tricuspid stenosis, so that it is not extremely rare. The prognosis depends largely upon the condition in life. It is more dangerous in women than in men; and in laboring classes than in those whose circumstances are easy.

In tricuspid insufficiency the prognosis is bad, if it is secondary to lung disease or some other valvular affection. It is apt then to be a terminal affection. It may, however, be a temporary affair, and of little account. Yet if it persists, the significance is very grave. The order of gravity, as given by Wilks, Peacock, Bristowe, Fagge, and Pye-Smith, is:

1. Aortic regurgitation.
2. Mitral regurgitation.
3. Mitral stenosis.
4. Aortic obstruction.

And as given by Green (*Medical Examination for Life Ins.*, 1900), it is:

1. Aortic regurgitation.
2. Mitral stenosis.
3. Aortic stenosis.
4. Mitral regurgitation.

While Walshe made the order:

1. Tricuspid regurgitation.
2. Mitral regurgitation.
3. Mitral stenosis.
4. Aortic incompetence.
5. Pulmonary obstruction.
6. Aortic obstruction.

On the other hand, Broadbent and Leyden have it:

1. Aortic incompetence.
2. Mitral stenosis.
3. Aortic stenosis.
4. Mitral regurgitation.

In the statements and figures just given, it is apparent that if we are to judge of the gravity of the disease by the average age at death, the order should be as follows in the four conditions named:

1. Mitral obstruction (stenosis).
2. Aortic insufficiency.
3. Aortic obstruction (stenosis).
4. Mitral insufficiency.

This order of gravity often obtains, according to Broadbent, in

childhood and early adolescence. My statistics, however, are drawn almost entirely from adult cases, and, I believe, from a larger series of post-mortem cases than have, as yet, been utilized in the solution of cardiac problems.

Certain other matters bear on the prognosis. For example, it is to be remembered that the intensity of a murmur does not indicate the gravity or extent of the inflammatory process. Well defined murmurs that come and go are apt to belong to loose vegetations and are therefore alarming. Musical murmurs, attended with fever, always give an unfavorable prognosis. They are likely to be caused by rupture or ulceration of a valve. Loud murmurs suggest a large opening. The change from a loud to a soft murmur, however, is apt to indicate commencing heart failure. Diastolic murmurs are the most dangerous. The Cheyne-Stokes or Adams-Stokes signs are significant of cerebral implication, and though they may disappear, in rare cases, point to eventual danger from cerebral disease. Recurring attacks of inflammatory rheumatism make the outlook unfavorable. In any form of endocarditis, a sharp attack of pneumonia, or even any prolonged illness that wastes the vital forces, may cause heart failure.

In the main, however, in valvular disease, the prognosis hangs on efficient or non-efficient compensation. If the apex beat is in about the normal position, the rhythm regular, the action of the heart good, and there is no considerable increase in the transverse dulness, auscultatory phenomena are of subordinate importance.

In fact, so long as compensation is maintained in these cases there is no immediate danger of *sudden death*, unless from embolism, which, however, may occur at any time. Still the embolus may not reach a point of vital importance. It may lodge in the liver or lungs, without doing much harm; if in the medulla or pons, it will probably cause immediate death. I have seen a case where a clot lodged in the pons produced this result. If, however, it is lodged in a "silent center," it may do no harm. Diseases of the right side of the heart are more immediately dangerous because they produce more venous congestion, which leads in turn to a greater exudation of fluid and its attendant consequences. It is commonly believed that the dropsical effusions seen in the last stages of cardiac diseases are due to congestion of the kidneys. This seems to be true in most cases, but the liver and spleen are *almost* as often involved. Indeed, the hobnail liver is quite common in old cases of heart disease.

In compensatory hypertrophies the right ventricle fails, as a rule, earlier than the left. Hypertrophy compensates for a variable period, dependent chiefly on the extent of the disease in the valve or valves affected. It was at one time computed that the expectation of life in ordinary cases is from two to four years after compensation has failed. The expectation of life in cardiac diseases has been greatly improved, however, by a closer attention to diagnosis, a more discriminating use of drugs, and a recognition of the success obtained by mechanical methods. Whether or not the claim made by the advocates of the Nauheim system that it adds an average of ten years to life, is true, it is an interesting problem and one worthy of close statistical inquiry.

Chapter XXII.

TRUE ANEURISMS OF THE LARGER VESSELS AND THEIR BRANCHES.

Aneurisms more often affect the thoracic aorta than any other artery of the body. Sometimes they are *sacculated,* that is, formed by a local bulging of the vessel's walls. But general or non-saccular dilatations are just as truly aneurisms, for the Greek word "aneurysma" means a dilatation, and in the ascending portions of the arch these dilatations are frequent enough, under the generic name of *fusiform aneurisms,* because the dilated portion of the aorta is spindle-shaped. Other varieties are much rarer, such, for example, as the arterio-venous, where there is a communication between an artery and vein, such as results from a penetrating wound. There are still other kinds, but the present chapter is mainly concerned with sacculated aneurisms.

In passing, it may be said of fusiform aneurisms that they stand apart from the sacculated. Confined mostly to the ascending part of the arch, they are seldom large enough to produce any pressure symptoms, and rarely have the thrill or double murmur of the sacculated variety. In fact, they have a life history of their own, which places them in a special category. They are discussed elsewhere.

Sacculated, otherwise known as *true* thoracic or abdominal aneurisms, may reach the size of a child's head, but seldom come to our notice under the size of a pullet's egg. Though occasionally due to mechanical injury, they are generally the result of arterial disease or weakness.

Syphilis, gout, alcoholism, and severe manual work, are the reputed causal factors, and the imputation is probably just, because each of these agencies will cause abnormal thickness of the arteries. I am inclined to think that syphilis is the most potent cause, though it is not easy to prove it from statistics. Malmsten made it the cause in 80 per cent., 5 per cent. being due to gout and 5 per cent. to alcoholism. My limited experience also indicates the predominating influence of syphilis.

Males are certainly more liable to aneurism than females. In fact, the proportions have been put at 8 to 1. In my 11 cases here recorded, but 1 was a female. Naturally it occurs most frequently

in the middle decades of life, when degenerative changes are most common, such as the third, fourth and fifth, especially the latter.

Aneurisms may persist for years, or at any time cause death from internal hemorrhage, or in some indirect manner, as by strangulation, pressure on the trachea, pneumonia, heart failure, etc., as the cases given later show. Spontaneous cure is seen in small aneurisms, rarely in larger ones. In the development of aneurisms, much depends on the environment of the individual. The growth is most rapid in those who lead a life of activity, excitement, or indifference to the laws of health. When a cure takes place, it is because the sac has been filled with fibrin, and its walls impregnated with the salts of lime, both shrinking so as to change the tumor into a solid ball; or it may be, that some peripheral inflammation has enveloped the sac and contracted it, as in Case No. LXXXII.

In the differential diagnosis of these aneurisms there are no really pathognomonic signs. In fact, reliance must be placed both on physical and subjective phenomena, and yet the one or the other, or both, may be absent. This statement may help to explain why my experience tells me that in thoracic and abdominal aneurisms the diagnosis is made only in a little more than 25 per cent. of the cases.

In the diagnosis of a thoracic or abdominal aneurism we first think of a pulsating tumor. If the orifice leading into the sac were large, the walls thin, and the contents only liquid blood, we should more often feel the abnormal pulsation. The opening, however, may be small and the contents largely composed of clotted or laminated blood. Perhaps the sac is so deep down in the tissues as not be reached by any kind of palpation. In such instances, we recognize neither pulsation nor heaving impulse. If, however, the sac, in a thoracic aneurism, for example, reaches the level of the intercostal cartilages, the episternal notch or the ribs, pulsation may be felt. When the sac is large, it may be mapped out by *percussion*. or by the *X-ray*. I successfully accomplished this by the latter method in a thoracic aneurism brought to my notice by Dr. Katzenbach of this city as early as 1897. In this instance the aneurism was probably filled with more or less laminated fibrin, which gave the shadow.

Aneurisms are most frequently found in the arch of the aorta, and I think it best to consider this class together, whether they proceed from the ascending, transverse or descending portion, because it is practically impossible *intra vitam* to distinguish just where they originated. The symptoms, being due to pressure, can not be

referred to the vessel, but to the sac, and the latter may assume any position in the thorax, that is, it may project to the right or left, upwards or downwards, forwards or backwards. Still it is true, taking the sum total of signs in a number of these aneurisms, in the several divisions of the arch, there will be certain symptoms more apt to be specially connected with each division, as will be shown.

In general, if the sac compress the superior vena cava, there will be enlargement of the veins of the head and neck; if the inferior vena cava, œdema of the lower extremities. If the sac press on the trachea, it may produce cough and dyspnœa, and even suffocation, as in Case No. LXXXV; if on the right pulmonary artery, embolism may follow. If the recurrent laryngeal is pressed on, there may be hoarseness or difficulty in phonation. Compression of the œsophagus will cause dysphagia; pressure on the vertebra, erosion of the spine. Rupture may take place into the œsophagus, peritoneum, pericardium, superior vena cava, trachea or bronchi, etc. Very occasionally an aneurism bursts externally. If there is a large thoracic tumor, there will generally be displacement of the heart. A small aneurism may give rise to a diastolic or systolic murmur, or both, and yet, at times, it may be impossible to distinguish these murmurs from the valvular. However, the murmurs are usually louder than the latter. The sternum may, in advanced cases, be eroded and pushed forward, making a tender and painful tumor. The hand pressed on it may detect a thrill, or perhaps a pulsation. Separation of the fingers placed over the pulsating area may show its expansile character. Usually the pulsation is to the right of the sternum. If the sac has solid or semi-solid contents, there will, of course, be no expansion. The sac may also press on the thoracic duct. The innominate or left carotid, or sub-clavian, may be involved in the tumor. There may be pain from pressure on the cardiac plexus, on the nerves of the pleura, pericardium or skin. Pressure on the branches of the sympathetic causes *inequality of the pupils,* as seen in Case No. LXXXIII, pressure on the pneumo-gastric, *spasm of the œsophagus* and vomiting. If there is pressure on a bronchus there may be catarrh of the mucous membranes, with retention of the secretions and broncho-pneumonia that will cause death. Pressure on the innominate or sub-clavian artery will necessarily produce some effect on the pulse of the affected side, and this is a valuable sign. Pressure on the innominate vein may produce venous congestion of the side of the head. There may be compression, obstruction and obliteration of the pulmonary artery. An important

sign of aneurism in this location is thought by some to be "*tracheal tugging.*" This is produced in the following way: let the patient stand and raise his chin to the farthest extent, then seize the cricoid cartilage between finger and thumb and raise it gently; if there is an aneurism of the arch, the pulsation of the aorta may possibly be transmitted through the trachea to the fingers.[1] This should be an early sign, but it does not appear to be very reliable. Grimshaw found it in 16 per cent. of persons who had no aneurism. There may be or may not be cardiac hypertrophy. A laryngeal examination is important, as it may reveal paresis of a vocal chord. If there is any weakness of the chord, it will be indicated in uttering the exclamation "Ah." Paroxysmal attacks of dyspnœa are attributable to paresis of the pneumo-gastric. Bronchorrhœa and suppuration of the lungs may also be due to paresis of this nerve. These signs may occur when there is even a small aneurism of the aorta, and such aneurisms are often fatal. The *pain* may be very intense. Small aneurisms of the transverse portion, where the tumor is no larger than a hen's egg, are common, and the diagnosis may sometimes be made by an experienced clinician, from a due consideration of all the signs physical and rational, as Case LXXXIII. shows.

When the tumor is the size of one's fist, percussion should indicate its locality, but I have known a thoracic aneurism ten inches in diameter to escape the notice of a careful practitioner. A loud murmur indicates a small opening, and vice versa. In one of my cases the sound resembled the strokes of a locomotive piston. In such cases we may distinguish these murmurs from those of the heart. According to Douglas Powell, however, in about one-half the cases there is no murmur in thoracic aneurism. In seven of my cases murmurs occurred in but two. The bruit may perhaps be best heard by opening the mouth and introducing the stethoscope between the teeth. The heart is pretty sure to be hypertrophied and sometimes displaced, while valvular lesions will often be a complication and the diagnosis difficult.

The following cases are taken from my records during the period when I was pathologist to the St. Luke's and Presbyterian Hospitals of this city:

Case LXXXII. Aortic Disease; Aneurism of the Ascending Portion of the Arch; Syphilis; Death from Heart Failure. Tendency to Spontaneous Cure.—M., 38, a painter, was admitted to hospital January 10, 1883, with a history of syphilis, and complain-

[1] Oliver.

ing of soreness over the lower part of his chest. On physical examination an aortic regurgitant murmur was recognized. The patient died after a short stay in hospital, during a sudden attack of dyspnœa lasting only 25 minutes.

At the post-mortem examination there was found to be both aortic regurgitation and stenosis, and the cause of death was attributed to heart failure; but there was also a sacculated aneurism about the size of a hen's egg above the aortic valves. The sac lay partly under the root of the right lung, where it had caused much irritation, leading to deposits of fibrin, which had prevented rupture. Gummy tumors were found in the liver. This case is a good illustration of the fact that a thoracic aneurism in this locality up to the size of a hen's egg is seldom diagnosticated, even by the best clinicians.

In aneurism of the transverse portion of the arch, there is a wide range of pressure symptoms, but, as a rule, they are not so marked as in aneurism of the ascending portion.

Case LXXXIII. Broncho-Pneumonia; Chronic Diffuse Nephritis; Aneurism of the Transverse Part of the Arch; Death Due to Pneumonia and Chronic Diffuse Nephritis.—R., 38, painter, was admitted to hospital September 28, 1885, with a previous history of gonorrhœa and alcoholism. He complained of pain beneath the sternum, cough and expectoration, dyspnœa and vomiting. Pulse 120 and weak.

On examination, signs of broncho-pneumonia were noted in both lungs, with enlargement of superficial veins over anterior portion of the chest. Stridor and orthopnœa supervened, with uræmia. Dilatation of left pupil. An aneurism of the arch was diagnosticated. The patient was put on the iodides, but succumbed in less than ten days, during an attack of dyspnœa. At the post-mortem examination a sacculated aneurism the size of a hen's egg was found projecting from the posterior portion of the transverse arch, just beneath the origin of the innominate, the opening into the sac being only three-eighths of an inch in diameter. The sac had pressed on the trachea, the right side more especially, eroding its rings. Both lungs had deposits that looked like gummas. The sac had not ruptured. The cause of death was ascribed to the broncho-pneumonia, which may have been due to retained bronchial secretion, to paresis of the pneumo-gastric, or to chronic diffuse nephritis, of which there was ample post-mortem evidence. This case is remarkable in that, with so few signs, this small aneurism was made out during life. The

diagnosis was based on the tracheal implication, broncho-pneumonia, and uneven dilation of pupils, backed by a syphilitic history.

Case LXXXIV. Aneurism of the Transverse Portion of the Arch, Rupture of the Sac; Internal Hemorrhage.—J., 40, France, agent, was admitted to hospital July 31, 1883, with symptoms of dysphagia, dyspnœa and difficulty in retaining food. Suspicious looking ulcer on legs. Pulse, 132, temp. 98, resp. 32. Patient died four days after admission in a suffocative attack, which lasted about an hour and a half. At the post-mortem examination it was found that an aneurismal sac had developed from the superior and posterior aspect of the transverse part of the arch, the innominate and left common carotid being involved. The sac had pressed on the trachea and œsophagus, and there was in it an orifice 1x½ inches in diameter leading from the sac. The stomach contained about 40 ounces of blood with clots. In this instance no diagnosis was made during life. Death was due to internal hemorrhage.

Case LXXXV. Aneurism of the Descending Part of the Arch; Death by Strangulation from Pressure on the Trachea.—A male of 27, saddler by occupation, entered the hospital Nov. 2, 1882.

He had a cough, dyspnœa, paroxysmal asthma, and tracheal obstruction, with cyanosis. Respiration stridulous, rough and prolonged. A prominent sternum complicated the diagnosis, and led to the suspicion of enlarged mediastinal glands. The only relief obtained was from the inhalation of oxygen.

At the post-mortem examination there was found an aneurismal sac the size of a pullet's egg, given off from the upper and posterior part of the descending aorta, the left common carotid and left subclavian being also involved. The lower margin of the sac had compressed the trachea, causing the embarrassed respiration. The aneurism was not recognized during life. Cause of death, strangulation.

In aneurism of the descending arch, pain is a most prominent symptom. The pressure signs are less noteworthy. There is sometimes a severe burning or aching pain near the spine in the entire scapular region. There may also be intercostal neuralgia. The sac may press on the left bronchus, which may obliterate the pulse in the abdominal aorta, or delay pulsation in the arteries of the lower extremities.

The sac may erode and destroy portions of the ribs and eat away the bodies of several dorsal vertebræ. One of the most con-

stant signs in disease of this part of the arch is severe pain, striking through from the third left interspace or fourth rib to the body of the scapula. Other signs are cough, paroxysmal asthma, dulness on percussion, evidences of pressure on the trachea, and possibly pain in the epigastrium. There is usually a fixed pain, occasionally a pulsation in the scapular or epigastric region. The left chest wall may be expanded.

CASE LXXXVI. Aneurism of the Descending Part of the Arch; Death due to Uræmia.—A laborer, 34, entered the hospital June 8, 1878, complaining of a pain in the back, that had lasted a year. He also had epigastric pain, tenderness in the lumbar region, tumidity of the abdomen, and mucous evacuations. He died of uræmia.

At the post-mortem examination a large aneurismal sac five inches in greatest diameter, and extending upwards six inches, was found behind the descending arch. It was adherent to the body of the fourth dorsal vertebra, and had destroyed the bodies of the tenth and eleventh dorsals. The necks of the ninth, tenth and eleventh ribs were eroded. No positive diagnosis appears to have been made during life. The aneurismal sac was pretty well filled with laminated fibrine and had not ruptured. Even in this case of a large aneurism the tendency to spontaneous cure was noteworthy.

Case LXXXVII. Aneurism of the Descending Arch; Rupture into the Trachea; Death from Internal Hemorrhage.—A male of 46 was admitted to the hospital on September 19, 1872, with the following symptoms and physical signs: Cough, expectoration, pulmonary emphysema, flatness over left side down to fourth rib. Below fourth rib, resonance with loud blowing sound. Bronchial breathing. Heart hypertrophied and beating tumultuously. No valvular murmurs. No emaciation. These physical signs were attributed to phthisis.

The patient died of internal hemorrhage and at the post-mortem examination the following facts were disclosed. The upper part of the left pleural cavity was occupied by an aneurismal sac the size of one's closed fist. It sprang from the inner and descending part of the arch and grew backwards. Rupture of the sac into the trachea had taken place. The left lung was crowded down by the sac, and that portion nearest the latter contained puriform liquid. Diagnosis not made.

Sometimes aneurisms of the thoracic aorta may develop below

the arch. They are apt to be of large size, and erode dorsal vertebræ, as the following case indicates.

Case LXXXVIII. Aneurism of the Descending Thoracic Aorta; Death from Internal Hemorrhage.—A glass cutter, aged 41, was admitted to the hospital May 1, 1879. He stated that a pain about his heart had kept him from work. There was also pain to the inside of the left scapula.. Insomnia. Patient unable to lie on left side. Lower part of chest expanded.

On examination, a pulsation was seen and felt at the lower and inner margin of the left scapula. At this point there was a double murmur. The patient died of internal hemorrhage.

At the post-mortem examination it was found that a large aneurismal sac had developed from the upper part of the descending aorta and had ruptured at the lower and posterior portion. It contained 90 ounces of serum and clots, and had eroded the bodies of the fifth, sixth, seventh and eighth dorsal vertebræ; also the fifth, sixth and seventh ribs, cutting the latter in two.

Aneurisms of the abdominal aorta are usually just below the diaphragm, near the cœliac axis, and above the superior mesenteric artery. The sac may project in any direction. If it push forward, pulsation may be felt below the ensiform cartilage, usually to the left of the median line. Palpation will detect it if there is any expansile pulsation. In these aneurisms, however, such pulsation is not always present. Or if there is pulsation, it may come from a tumor beneath the vessel or above it. Besides, pulsation of the abdominal aorta is very common in nervous people with vaso-motor disturbances.

If the tumor projects upwards it may attain considerable size without being detected. The chief symptoms are subjective. Pains of an annoying character referred to the spine may radiate into the chest or down through the abdomen, but they may be intermittent. The sac may displace the internal organs, such as the kidneys. It may press on the common bile duct, or on the cæliac axis and its branches. Small aneurisms sometimes form on the branches of the abdominal aorta, the splenic, superior mesenteric and hepatic arteries. Sometimes the superior mesenteric is obstructed by embolism or thrombosis, and hemorrhage and infarction of the intestines may be the result. The chief symptom is pain or numbness. It may be referred to the back or front. Uusually there is a fixed pain in the epigastric region.

Inspection shows a rounded tumor that may have an *expansile*

pulsation and *thrill.* Auscultation gives a diastolic or systolic murmur, or both. The femoral and radial pulses may be delayed. Pulsating tumors of the stomach, liver or pancreas are mobile, and the pulsation disappears to a considerable extent when the patient is put into the knee-elbow position.

Case LXXXIX. Aneurism of the Abdominal Aorta; Rupture into the Retro-Peritoneal Region.—A male of 25, addicted to alcohol, with a probable history of syphilis, was admitted to hospital October 4, 1876, with the following symptoms: Dulness in the small of the back, a sense of heaviness on both sides. Flying pains about the body. Pain in the left thigh extending to ankle. Pulsation in left lumbar region, and in the middle of the abdominal cavity. Thrill and bruit below the ensiform cartilage, and over the lower dorsal spines. The diagnosis was correctly made.

At the post-mortem examination it was found that the aneurismal sac extended from the last dorsal to the last lumbar vertebra. Its dimensions were 7x4½ inches, and it had an hour glass constriction at the center. The opening from the aorta was just below the cæliac axis. The body of the last dorsal vertebra was eroded. The escaped blood formed a tumor the size of a child's head behind the peritoneum. Possibly a spiculum from the eroded vertebra tore open the sac.

Case XC. Aneurism of Abdominal Aorta; Rupture into the Peritoneum. Death from Internal Hæmorrhage.—A male of 40 entered the hospital December 1, 1882, complaining of pain in the epigastrium, right hyperchondrium, both groins and stomach, for which he was taking opiates. His urine and stools were dark colored. On examination, a pulsating tumor was found in the epigastrium. Death occurred from rupture of the aneurismal sac into the peritoneum. The sac itself was only two inches in diameter. It sprang from the front of the aorta and involved the cæliac axis and superior mesenteric artery. The rupture took place just above the level of the pancreas. Four ounces of blood-clot, and four of bloody serum, were found in the peritoneal cavity. As the sac was small, the symptoms prior to rupture were not pronounced.

Case XCI. Aneurism of the Abdominal Aorta. Death from Internal Hæmorrhage.—A male of 47, car driver, was admitted to hospital January 27, 1887, with a bubo and gonorrhœa. He described himself as a moderate drinker. Shooting pains down the thighs, legs and in the abdominal cavity were ascribed by him to a severe cold bought on by exposure to cold and wet. The pain in

the lower extremities was found to be confined to the left sciatic and crural nerves. The patient died of internal hemorrhage, and at the post-mortem it was found that a small sacculated aneurism, rising from the anterior surface of the abdominal aorta, just below the cœliac axis, had eroded the underlying vertebra and that a spiculum of bone had lacerated the sac, causing internal hemorrhage and death. Nodules resembling gummas were found in the lungs. There were also copper-colored scars on the legs.

Aneurisms of the pulmonary artery are comparatively rare, and in half the cases, are probably associated with congenital disease. On the other hand, aneurism of the branches of the artery are common, if we include under this name the minute aneurisms found in tuberculosis phthisis. Rupture of these aneurisms in phthisis often causes death from hemorrhage. The aneurism begins as a periarteritis and endarteritis. This matter does not interest us at present. Generally, dilatations of the pulmonary artery are due to obstruction of the branches of this vessel, or constriction at their orifices. This latter is very rare. The following is an example of the latent variety.

Case XCII. Aneurism of the Pulmonary Artery. Death from Embolism.—C. A., widow of 50, entered the hospital August 4, 1881, with a history of rheumatism, anæmia and cyanosis. She was also found to have dyspnœa and œdema of the extremities. Pulse weak, irregular, intermitting every fourth beat. Heart enlarged. Loud blowing murmur at the base, and diffused over nearly all the precordial space, but most distinct between nipple and sternum. First sound rough. The patient died suddenly. At the post-mortem examination the pulmonary artery, with its right and left branches, was found considerably dilated. On the right pulmonary was an aneurismal sac the size of a duck's egg, with solid contents. On the left pulmonary, at a corresponding point, was another aneurism of about the same size. The cause of death was supposed to have been embolism in one of the pulmonary arteries, by which the blood was rapidly dammed back on the heart.

In thoracic and abdominal aneurisms I am disposed to think, from my experience, that surgery has been unsuccessful, and that distal pressure, ligation, introduction of foreign bodies like wire, injection of fluids, as the perchloride of iron, application of electricity, or compresses, achieve but temporary, if any, improvement, while medical remedies offer far better hopes for the unfortunate patient.

Of all considerations to my mind, the first is *rest;* the second is

restriction in food and drink; the third, a *cardiac sedative.* Our object is to coagulate the blood, remembering that this is the remedy adopted by nature in curing aneurisms, which, as Case No. LXXV. shows, may be cured spontaneously if the tumors are small. First in importance is rest in the recumbent position. It causes relaxation of the sac, and favors slow coagulation, which may lead to solidification of the fibrine in layers. Second in importance is diet. The *Tufnell method* is perfectly legitimate. It is a modification of Valsalva's, who treated his patient by frequent bleedings, and of *Bellingham's,* whose was a mild starvation. Tufnell's method allows the patient, for breakfast, two ounces of milk or cream, and two ounces of wheat bread with butter. For dinner, three ounces of meat, three ounces of potatoes or bread, four ounces of water or claret. For supper, four ounces of bread and two ounces of milk or tea. In all, twelve ounces of solids and eight ounces of liquids are given. Tufnell reported ten cases successfully treated in this way. His object was to reduce the volume of blood, while the fibrine was increased. If improvement is attained, it should appear in from four to six weeks. Iodide of potassium or sodium are drugs that may be used. Commence with five grains three times a day and increase to fifteen or twenty; or iodine in other forms may be used. Stop only when there are symptoms of iodism. The treatment may be continued for months and years. When there is a visible tumor, cold may be applied by means of *Leiter's tubes,* part of the bag resting on the aneurism. Apply at intervals, at first, and then continuously; cold tends to relieve the pain and reduce the sac. After a thorough course of two months, the patient may gradually resume his work, if the symptoms warrant it. In the case of a cure, the tumor will remain, but it will shrink into a hard ball. Among the remedies that are useful is aconite in from one to two minims of the tincture, increased gradually to four or five minims every four hours. Digitalis is dangerous; it may burst an aneurism.

Broadbent has reported a case of aortic aneurism that lived ten years, and Hayden a case of abdominal aneurism that lived eleven years. There is, therefore, some encouragement in the medical treatment of aneurism, though aneurisms of large size do not afford us much ground for hope of a cure.

The following is a summary of the important points to be remembered:

In the diagnosis of thoracic and abdominal aneurisms, no signs

are pathognomonic, but both subjective and objective phenomena are to be considered.

Success in the treatment of these aneurisms depends on their early recognition, as large aneurisms are pretty surely fatal, either from rupture or pressure.

Syphilis, gout and alcoholism are causal factors, so that the importance of combating them cannot be overestimated. Unfortunately, physicians do not sufficiently recognize the agency of these conditions, and are apt to be especially unsuccessful in detecting syphilis.

The larger aneurisms, whether thoracic or abdominal, have thus far baffled the best efforts of modern surgeons, while medical treatment has at least insured a longer lease of life and offers greater chances of cure, if treatment is undertaken early.

Chapter XXIII.

AORTITIS.

Most persons have aortic disease after middle life, and many at an earlier period. This much was probably known centuries ago; for wherever post-mortem examinations were allowed and practised, aortic lesions must have been readily recognized by the naked eye. It is not improbable that they were known before the Christian era. Certainly Galen's presumed contemporary, Aretaeus,[1] the Cappadocian, alluded to them, and he is supposed to have flourished in the second century. Not to mention other medical writers, Morgagni[2] described some phases of them. But up to the time of Scarpa[3] they were so little understood that the most characteristic type of chronic aortitis, the cylindrical or fusiform (false aneurism), had not been differentiated from the sacculated or true aneurism.

There are two principal division of aortitis, the *acute* and *chronic* (known also as the *subacute*), for there is no pathological or clinical line of distinction that can be profitably drawn between the latter two.

So far as etiology and pathology are concerned, these matters are tolerably well understood, but the clinical features of aortitis have elicited sharp discussion since Portal's time (1803), and as regards the acute form, without making much progress. For many, indeed most, clinicians do not believe that it can be recognized, in any locality, even under the most favorable circumstances *intra vitam*, independent of traumatism. On the other hand, in some instances, the chronic form, as seen in the ascending arch of the aorta in the variety known as Hodgson's Disease, can sometimes be differentiated with little difficulty, as is shown in Case XCVI. As simple dilatation of the aorta may not be inflammatory, it is considered separately.

There are two varieties of acute aortitis, the *acute primary*, where it constitutes the sole inflammatory lesion, and the *acute secondary*,[4] which is invariably associated with the chronic form, as exemplified by such chronic manifestations as fibroid deposits, fatty

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[1] Aretaeus, On the Causes and Symptoms of Acute Disease, London, 1856, Book II, p. 445. (Trans.)

[2] Morgagni, On the Seat and Causes of Disease, Vol. I, p. 389. (Trans.)

[3] Scarpa, Réflections et Observations Anatomico-chirurgicales, Paris, 1809, p. 1.

[4] Huchard.

degeneration, arteriosclerosis, atheroma, or a combination of them.

On the other hand, the primary acute form includes infiltration of the walls of a vessel with serum, fibrin or lymphoid corpuscles; or suppuration, ulceration, formation of granulation tissue, or even gangrene; making it easy to separate it from the chronic form, on a purely pathological basis.

The acute secondary form is found only in association with chronic processes, to which it bears the same relation as the acute manifestations of typical rheumatic gout bear to the chronic enlargement of the joints, of which they are part; for the two processes work together simultaneously within the affected area.

Acute aortitis is also occasionally caused by tuberculosis or syphilis, both of which are known to have special affinities for arteries. It is also due to infections, such as malignant endocarditis and puerperal fever; possibly to eruptive fevers, and probably to acute gout or other diatheses or intoxications. It may originate in the walls of the vessel, or be a metastatic deposit, or an extension from adjacent parts, as in pericarditis, possibly in pleuritis or pneumonia. Case XCIII appears from prima facie evidence to be an example of the latter character, though it is not altogether conclusive to my mind. I give it because out of 915 post-mortem examinations of which I have clinical notes, it is the only one I have found.

Case XCIII. Articular Rheumatism; Malignant Endocarditis; Ulcer of the Aorta.—R. 56, male, was admitted to hospital May 21, 1881. Four weeks previously he had his first attack of articular rheumatism, which involved the large joints and left him feeble and scarcely able to stand. Convalescence began in three weeks, but dyspepsia, headache, epigastric distress and irregular chills, followed by fever and sweating, supervened. There was never at any time œdema, but the urine was scanty and contained a trace of albumin.

On examination a double mitral, and later a double aortic murmur were detected. Pulse irregular. Ten days after admission pneumonia set in and caused death on the same day.

A diagnosis of probable ulcerative endocarditis had been made. At the autopsy, which unfortunately was for some reason delayed and was confined to but a few organs, blood was found in the pericardium. For an ulcer had eaten through the substance of the heart from the aortic to the mitral valves, and blood was extravasated beneath the endocardium. There was also an ulcer in the aorta. As old infarcts and puckerings were found in the spleen, it is probable that there had been a benign endocarditis at some time, and that the

malignant or ulcerative character was a superadded manifestation. This case is obviously incomplete in many ways, but it appears none the less to fall into the category with Andral's single case[5] and the four of Lebert,[6], Spengler,[7] Schutzenberger,[8] and Leudet,[9] respectively.

In a case of aortic and mitral disease Spengler found a small abscess, the size of a hazel nut, just above the semi-lunar valves.

Schutzenberger also found in his case an abscess the size of a nut at the origin of the aorta, and between the middle and internal coats. There was an associated pericarditis.

In Leudet's case, likewise, there was an abscess (the size of a filbert) between the internal and middle coats, communicating by an orifice the diameter of a small pea with the lumen of the aorta. In all these instances there were the clinical evidences of a purulent infection.

In tuberculosis the aorta may also be the seat of acute or chronic tubercular deposits. A number of such instances have been described by Blumer.[10] Syphilis of the aorta has also been recognized by numerous clinicians.

So far as the prognosis is concerned, there is always a bare probability that an acute attack may end in recovery. That such an event is possible is shown in Case XCIV, where more or less cicatrization was found in the aorta; but inasmuch as the causes, so far as we know with certainty, are apt to be chronic affections like syphilis, tuberculosis and gout, the disease is most likely to assume the chronic form. Even if the acute variety is not purulent, but is represented by an infiltration with fresh plastic matter (the so-called *gelatiniform plaques*), degeneration will in time occur, ulcers may develop, or plates of atheroma of a bony hardness, so that the acute will pass by slow transition into the subacute or chronic form. Of course, in cases associated with malignant endocarditis, or a purulent infection, a fatal termination is to be expected.

Acute aortitis was first brought prominently to the notice of the profession by Portal[11] in 1803, but, as already stated, widely divergent ideas as to its clinical aspects have prevailed about it since his day.

[5] Andral, *Path. Anat.*, Vol. II, p. 243.
[6] Lebert, *Krankheiten der Arterien. Virchow's Path. and Thera.*, 1867, Vol. II, s. 347.
[7] Spengler, *Virchow's Archiv.*, IX, 1852, s. 166.
[8] Schutzenberger, *Archives Generales*, 1861, II, p. 581.
[9] Leudet, *Archiv. Gen. de Med.*, Ser. 5, 18, p. 581.

In 1824 Bertin and Bouillaud[12] reported sixteen cases, but the changes seen at the autopsies were not typical, and apparently the reddened condition of the aortas, on which they laid so much stress, was due to post-mortem inhibition. Indeed Huchard,[13] a firm believer in acute aortitis as a disease capable of being recognized at the bedside, has accepted only one of these cases as conclusive.

In 1837 Bizot[14] published three cases, claiming that the signs were anasarca, intense fever, prostration and delirium, while, as a matter of fact, œdema and delirium have no necessary connection with acute aortitis. His first case was probably one of pericarditis, and the last two of Bright's Disease, which was unknown at that time.

These misconceptions, however, were in due time pointed out by Laennec,[15] who showed that inflammation of a vessel was to be distinguished from post-mortem inhibition, and that among its characteristics were swelling, thickening and a development of new vessels.

In detail the pathological changes are as follows: The signs of inflammation are, as a rule, seen first in the internal coat, which becomes reddened, and later takes on a pellucid grayish or grayish-yellow color; then the inflamed part swells and is raised up above the level of the surrounding tissue. These elevated plaques are soft and have a translucent appearance—whence they are known as *gelatiniform plaques*—and they may either remain of a gray or yellowish-gray tint or become rose colored. They are infiltrated with serum, fibrine or lymphoid corpuscles, perhaps an admixture of two or more of them; which accounts for the variations in their color. After the plaques soften, they may fungate and leave erosions. The outer coat is eventually involved, the middle coat, which contains elastic plates and fibres and smooth muscle tissue, suffering least.

Acute inflammation is in a measure produced by injuries causing hyperdistension, as in the arch of the aorta, where the full force of the systole is felt; but even in acute aortitis, as it is seen in the arch, when intense pressure has been brought to bear on the walls of the vessel by violent physical exercise, there is apt to be, and

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[10] Blumer, *Am. Journal of the Med. Soc.*, 1899, n. s. cxvii, pp. 19-25.
[11] Portal, *Cours d'Anat. Medicale*, Vol. III, 1803, p. 144.
[12] Bertin and Bouillaud, *Treatice des Maladies du Cœur, et des gros vaisseaux*, 1824, p. 4.
[13] Huchard, *Maladies du Cœur*, etc., 1893, p. 445, *et seq.*
[14] Bizot, *Soc. Med. d'Observ.*, 1837, p. 332.

perhaps always is, some vice in the circulatory fluid which transforms a simple dilatation into an aortitis with dilatation.

While, therefore, there is no doubt that aortitis occurs as a pathological fact, clinicians have, as stated, been disposed to give it a wide berth. Even Jaccoud[16] has differed from many of his confrères of the French school, and stated that there are no symptoms by which the acute can be separated from the chronic form; while Von Schroetter[17] denies that it is an independent disease, with distinctive signs and typical course.

According to Peter,[18] the clinical diagnosis turns on palpitation, difficult breathing, local pain, a burning sensation under the sternum and active pulsation of the aorta, but he is unable to distinguish it from angina pectoris. Quain[19] has made the signs acute substernal pain, with oppression, palpitation, quick and feeble pulse, elevation of temperature and a harsh systolic murmur at the seat of inflammation and transmitted up the vessel. Huchard[20] says that if the patient has the characteristic dyspnœa, with a substernal sense of burning or tearing, one thinks of aortitis, and if, in addition, the heart is hypertrophied, without much valvular accentuation, and there is a double-bellows murmur, more or less rude in quality, the diagnosis is easy. To him, however, the acute primary form has three special signs (1) labored breathing, (2) pain, and (3) syncopal attacks. Unfortunately, he gives no illustrative case that is satisfactory. Certainly in the vast majority of cases the acute stage does not reveal itself by any pathognomonic signs.[20] In the absence of proof, therefore, that acute aortitis is capable of demonstration during life, it is useless to discuss its treatment.

Chronic aortitis is a peculiar affection, combining, as already stated, acute manifestations (the acute secondary of Huchard) and the chronic; both of them progressing side by side. The causes are diatheses such as gout, or intoxications like lead and alcohol, while hypertension from over-work, perhaps from tobacco, together with senility, are contributing factors. The chronic may supervene on the acute process, itself induced by infections like typhoid or the eruptive fevers.

Whatever causes the inflammation, the net result is that the vessel wall gets to be thickened, less elastic and less vascularized. The process may involve the orifices of the branches, which are narrowed and stiffened, and if the disease extends become obliterated. This sometimes occurs to the orifices of the coronary arteries. The loss of elasticity in the aorta, infiltration of its wall and narrow-

ing of the calibre of its branches, inevitably lead to dilatation, unless it is checked by the calcifying process (atheroma).

Joseph Hodgson, in his prize essay (*Diseases of the Arteries and Veins,* London, 1815), appears to have been the first to call attention to the chronic aortitis especially associated with the peculiar dilatation of the ascending portion of the arch of the aorta which goes by his name. It is also known as the cylindrical or fusiform aneurism.

Hodgson's description of it holds good to-day. He says it is most frequently found in the ascending part of the aorta, the vessel forming a huge pouch, usually commencing just above the semilunar valves, but there is no loss of continuity in the vessel; in fact, it may occupy only one side of the vessel. His subjective signs were dyspnœa, palpitation, syncopal attacks, and copious expectoration of mucus. As an objective sign was the small and intermittent pulse. There are, however, many others. Inasmuch as the disease begins with an endarteritis, there may be a bellows murmur, pain if there is pressure from dilatation, prolongation of systole, and cardiac dilatation. So soon as the aorta is dilated it is also elongated, and the right subclavian is pushed up. If the patient lies on the back and the shoulder is elevated, the dilatation can be felt.

By percussion it may be found that the vessel is broadened; it is normally 1½ to 2 inches wide in adult males; 1 to 1½ inches in adult females. In dilatation the right margin of the vessel may be as much as 5½ inches from the median line. The following is an illustrative case:

Case XCIV. Chronic Aortitis of the Ascending Aorta; (Hodgson's Disease), Stenosis and Insufficiency of the Aortic Valves; Chronic Nephritis, and Meningitis.—W., 64, male, was admitted to hospital July 8, 1882, with a previous history of alcoholism, rheumatism and privation.

He complained in particular of a sense of constriction beneath the sternum, and of cough. His extremities were œdematous. Urine was found to be scanty and albuminous, containing casts of various kinds. Chronic nephritis was a constant feature of the case. On auscultation a double murmur was heard at the base of the heart, which, from the post-mortem findings, may have been

[15] Laennec, *Dis. of the Chest,* p. 743, 1838.
[16] Jaccond, *Leçons de Clinique,* 1884-5, s. 117.
[17] von Schroetter, *Nothnagel's spec. Path. u. Ther.,* XV, Theil. II.
[18] Peter, *Mal. du Cœur,* 1883, p. 776.
[19] Quain, *Dict. of Medicine,* 1883.
[20] According to Douglas Powell, in Pepper's System, Vol. III, p. 800.

due to endocarditis of the semilunar valves. Apex in the fifth space and one-half inch outside of the nipple. Among other noteworthy symptoms during his illness, which lasted fourteen weeks, were irregular pulse, syncopal attacks at night, dyspnœa, delirium alternating with hebetude, and at the last, coma.

At the post-mortem examination there was found an adherent pericardium, and aortic stenosis with insufficiency due to aortic endocarditis; while the aorta represented a great variety of changes, from simple inflammation to fatty degeneration, or atheroma, or destruction of tissue with some attempt at repair. The whole arch was notably dilated and constituted a true fusiform or cylindrical aneurism (*Hodgson's Disease*). Both kidneys were examples of chronic diffuse nephritis, one being much contracted. There was thought to be slight cirrhosis of the liver. Ascites. Chronic cerebral meningitis.

This case presented several features of interest from the point of view of the aortitis. It was of the chronic form, illustrating the acute manifestations often seen in that affection. The cicatrization also demonstrated that nature can and does effect some sort of cure. But there was no evidence that any of the signs, subjective or objective, belonged to the aortitis, which contributed little if anything to the fatal result. This was due to uræmia, conjoined with meningitis, though the cardiac difficulty may have been a predisposing factor. Even the substernal constriction was relieved by stimulants. It should have increased, if the pain had been due to aortitis.

It is not unlikely, however, that the diagnosis could have been made, if special attention had been directed to the matter. Percussion would in all probability have disclosed an increased area of dulnss over the aorta; it would have been found that the aortic direct murmur was carried more to the right than usual, while the aortic endocarditis and the elongated heart would have made the dilatation probable or possible in a man sixty-four years of age. Nowadays the X-ray would have shown the dilatation, as it did for me in the following case:

Case XCV. Chronic Aortitis; Aortic Endocarditis; Albuminuria; Glycosuria; Infrequent Pulse.—R., a retired man of business, 64 years of age, was referred to me in May, 1899, by his physician, on account of a troublesome epistaxis, ascribed to arterial disease. The subjective symptoms were shortness of breath, occasional fainting attacks; at times pain over the heart, sometimes at the apex, sometimes in the neck or joints. Gouty deposits in the

finger joints. Pulse 80, intermittent. Respiration 16. Enlargement of the superficial veins of the chest, especially on the right side. Facies arterio-sclerotica. Double bruit over the aorta. Loud systolic murmur with first sound, propagated up the great vessels and with second sound radiating to the right and also down the sternum. No mitral murmur. Heaving impulse.

The apex was found just within the nipple and two inches below the intermammillary line. The loud murmur with the first sound at base about corresponded with an area 5 inches in diameter, ex-

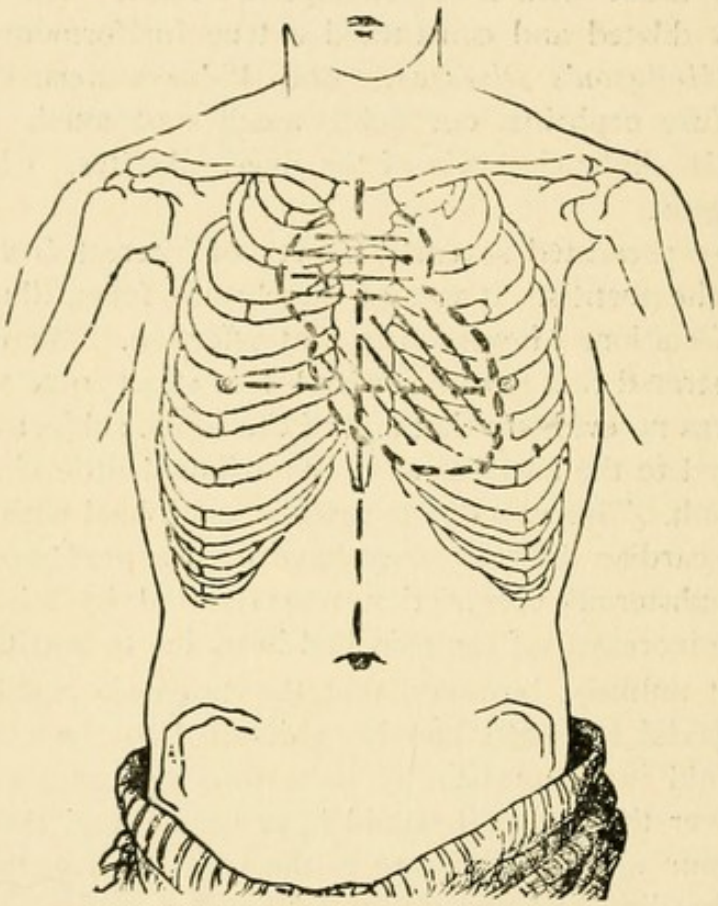

FIG. 40.

tending 3½ inches to the right of the median line and 2 inches to the left of it. By *fluoroscopy*[21] this area threw a somewhat dark

[21] Prof. Samuel Lloyd assisted me in making the X-ray picture, which I drew on the fluorescent screen, and on the same day, in sending a copy of my fluorograph to his physician, I wrote: "There appears to be above the heart a tolerably well-defined shadow which corresponds to the aorta, while at this point I find a double bruit. I therefore think there is a dilatation of the aorta, probably with extensive atheroma, and that this condition causes the aortic double sounds primarily." In the year following this condition was depicted by Cabot (*Physical Diagnosis*, N. Y., 1900, p. 172). Cabot believes that dilatation of the ascending portion of the arch occurs in almost every case of aortic regurgitation.

shadow, and to my mind indicated a fusiform dilatation of the aorta (ascending portion of the arch), although (see Fig. 40) the shadow was not as dark as I have seen it in sacculated aneurism. Examination of the blood showed 95 per cent. of hæmoglobin. Daily amount of urine, 24 ounces; specific gravity, 1025; traces of albumin; sugar, 2 per cent.; hyaline casts; urea, 6 grains per ounce. The patient took a course of baths and resistance exercises at Nauheim. On his return he was no longer short-winded, but had occasional shooting pains over the heart. Pulse 72, one intermission instead of four per minute. Respiration 16. Superficial veins of chest not so prominent. Murmur the same, but cardiac enlargement diminished. As the patient contracted a rheumatic attack on his return voyage, he was put by me on the hot-air treatment and the iodides were given.

On January 2, 1901, the examination of the urine was as follows: Specific gravity, 1015; amount, 33 ounces; no albumin; no sugar; traces of indican. On March 23, 1901, the patient expressed himself as free from all disagreeable symptoms, although the auscultatory signs were little changed.

The X-ray examination was made for the purpose of determining between a true or sacculated aneurism and a dilated aorta. I did not elicit any increased dulness over the dilatation, probably because in percussing, I failed to place the patient in the position with the chest bent downwards. There were no mitral murmurs, but the evidences of aortic stenosis and insufficiency were recognized, as is seen by my notes, and they are confirmed by my colleague, Prof. S. S. Burt. A feature of the case, however, was paroxysms of the infrequent pulse, which varied from 48 to 58 during a great part of the three years and more he was under my care. In the summer of 1903 he passed from under my observation, and I have no subsequent notes of his case. He died in the December following.

Percussion may be made directly on the sternum, and if the breadth is found to be greater than the normal we have dilatation. If there is pericarditis it may involve the pericardium, and we may have the signs of dry pericarditis. Pressure on the brachial plexus may also cause atrophy of the corresponding limb, or dysphagia may result from pressure on the œsophagus. Again, it is said, the pouch of the aorta may press forward and cause pulsation, seen and felt through the skin in the second right space.

A most noteworthy sign is aortic insufficiency, and with it the

"long heart," two conditions that are very apt to be associated with chronic aortitis.

Gueneau de Mussy[22] thinks that an important sign is a second cardiac sound heard over the course of the vessels, and metallic or clanging in character, the *"chant du crapaud"* or *"bruit tympanique,"* so-called from its resemblance to the croaking of a frog or the clanging of a drum. According to Paul (*Dis. of the Heart,* N. Y., 1884) the signs of Hodgson's Disease are:

Dyspnœa on effort; vertigo due to cerebral anæma; hard radials; sinuous arteries. As the aorta does not extend beyond the sternum in health, and is 15 to 20 centimeters below the episternal notch, if the dulness extends beyond these points we have dilatation. Bruit in aorta and large vessels, and over surrounding organs. Unequal pulse due to obstructive changes in vessels. Dulness over upper part of the sternum and right lung.

The bruit may be as much as 4 centimeters from the right border of the sternum. The aorta may be perceptible above the aortic notch. The bruit, which lasts through the entire ventricular systole, may be double when there is a sharp projecting plate, which gives a murmur both in systole and when the blood flows back to close the semilunar valves. As the roughness in the aorta increases, there is more harshness in the murmurs. The heart now hypertrophies, and the apex may be in the sixth left space. There may be compression of the trachea.

Laboulbène (*Anat. Path.,* Paris, 1879, p. 640) gives an additional sign of Hodgson's Disease, namely, the projection upwards of the subclavian as a means of determining the dilatation of the aorta.

Sansom (*Twentieth Century Med.,* Vol. IV.) gives the following signs: Difficulty of breathing with a sense of weight or constriction of the chest, the peculiar respiration being that of a long and painful inspiration with a short expiration. Orthopnœa. Pain of a severe character in the mid-sternum, or radiating to the neck and down the right arm. (Albutt[23] finds this pain may be absent, and he refers it to the root of the aorta.) Other signs given are insomnia, coldness of the extremities, vertigo, dyspepsia. Angina pectoris is apt to be associated.

If there is decided præcordial pain, the patient should rest in bed, while cold applications may be made to the chest. Chloralamid in 20 to 30-grain doses may be given to promote sleep, perhaps an

[22] Gueneau, de Mussy, *Clinique Medicale,* Vol. IV, p. 470 *et seq.*
[23] Albutt, *Lancet,* July 18, 1903.

opiate for a similar reason, or to relieve pain. The treatment in general is that of arteriosclerosis.

Simple dilatation of the aorta, independently of disease of the coats, may occur as a result of obstruction of the vessel in advance of the dilatation, or of violent cardiac action, and is common, according to Quincke, among men whose work consists of violent muscular effort, such as stokers and sledgers. It is due to the resistance offered to the systemic circulation by muscular contraction; and also to the backward pressure in the distended veins.

There is only one place where the dilatation of the aorta can be felt by the fingers, and that is in its abdominal portion, where it may also be seen.

Such dilatations may be permanent, but more commonly are of a temporary character, and due to paresis or paralysis of the vasoconstrictors.

Chapter XXIV.

ARTERIOSCLEROSIS.

The meaning of the word arteriosclerosis, originally applied to proliferative and degenerative changes in the aorta and larger vessels, has been gradually expanded in accordance with our increasing knowledge, so that now, while it is strictly limited, etymologically speaking, to changes in the arteries, it is intended to be descriptive of systemic processes that embrace not only arteries, capillaries, and veins, but lymphatics also. Plainly, then, Thoma was correct in contending that *arteriosclerosis* is no longer adequate to express this condition of the vessels, which is more properly an *angiosclerosis,* if we look at the subject from the broad point of view taken by pathologists. At the same time, there is no reason to abandon the word arteriosclerosis, because both from a pathological and clinical point of view, the changes in the arteries are the most conspicuous incidents of the affection. To be sure, it may not have been demonstrated that angiosclerosis can affect every organ and tissue of the body, but it is certainly true of the capillaries and small arteries of the pia, retina, kidneys, heart, lungs, spleen, stomach, brain and spinal cord, so that it is probable that no organ, tissue or vessel escapes. In the capillaries and smaller vessels, where the disease appears to originate, the changes consist of infiltration, fatty, calcareous and hyaline changes, with pigmentation, followed by dilatation, then contraction, perhaps embolism, thrombosis, or even rupture. These changes are less conspicuous in the veins, though they, too, undergo hypertrophy, atrophy and degenerative alteration, certainly in such systemic diseases as gout, syphilis and tuberculosis; and all vessels are in a measure similarly though not equally affected. In syphilis and gout, however, the arteries suffer most from the poison. So far as the larger arteries are concerned, the following anatomical facts should be borne in mind. A layer of flattened endothelium cells lines the vessels. External to them is the tunica intima, or true internal coat, composed of networks of elastic tissue arranged longitudinally. These two coats go to make up the internal coat of the older writers. More externally, the middle coat is made up of muscular fibres arranged transversely, together with elastic fibres. Still more external is the outer coat, made up of connective tissues.

In arteriosclerosis, using the term in its restricted sense, as applied to arteries only, and as the condition is seen by the naked eye, there are *three stages* into which it can be conveniently divided. Exposing the interior of the vessel, the inner surface is marked here and there, in a somewhat irregular way, by greyish-white or pellucid patches, which appear to be actually adherent to the lining membrane. As a matter of fact, they lie between the tunica intima and media. This is the first stage. The material of which the deposit is made up is a firm but elastic substance, the result of inflammation, and developed from the deeper cells of the tunica intima. The condition is therefore an *endarteritis,* and Virchow's contention, that it is developed from the tunica intima, is correct. But this newly formed tissue is not destined to produce healthy tissue in every instance. Like all the results of inflammatory action, it is deficient in vitality, and is soon apt to fall a prey to fatty change; so that the original whitish or pellucid substance may become yellow, and its consistence pasty. In other words, we have *atheroma*—the second stage. Now, this atheromatous process once started may continue to extend. At first the endothelial coat resists, while the internal coat offers less resistance; but in the end the former gives way, leaving the inner surface of the vessel rough and worm-eaten in appearance—the *atheromatous ulcer.*

On the other hand, while the patch may disintegrate, it may not discharge its contents into the vessels, but may simply dry up and calcify. This is the third stage. The appearances are now distinctive. If in the aorta, plates or spicula of bony hardness, possibly an inch or more in length, may project into the lumen of the vessel. In the larger arteries these plates may take the form of bands encircling the vessels. Arteriosclerosis in the second and third stages is quite common in the aorta. Indeed, after middle life there is usually more or less of atheroma or *calcareous substance* in it, and at this period each of the three stages can often be seen in the aorta of a single person; the pearly deposits of the first and the bright yellow of the fatty and calcareous deposits of the second and third stages being outlined against the reddened tissue of the normal portion of the vessel. In syphilitics this process may be seen in young persons who have not undergone appropriate treatment. Sclerosis, however, is not evenly distributed in the arteries, so that the discovery of stiff radials does not imply a similar condition in every vessel of the body. There appears to be some determining cause that relegates the disease to a special part, and in point of

frequency, so far as our present information goes, we may safely say that the distribution is about in the order following: first, in the aorta, then in the cerebral vessels, coronaries, and renal vessels, and finally in arteries of the extremities.

There are various dangers to which the arteriosclerotic individual is exposed. Owing to the retardation of the blood current by obstruction, and the loss of elasticity in the vessels, there will be failure in the nutrition of the various parts. In the brain this may cause cerebral softening, independently of embolism or thrombosis. Where there is an atheromatous ulcer in a vessel, the blood may penetrate between the coats and cause a dissecting aneurism, or the yielding of the external coat may cause a saccular aneurism. Sometimes the vessel bursts; this is liable to occur in cerebral vessels that have very thin walls. Occasionally a diseased coronary bursts; or an artery may be more or less completely blocked by fibrine lodged on a calcareous spicule or a roughened surface. This may cause senile gangrene, and it is observed in the extremities. *Embolism* in distant parts is also another incident. Owing to the rigidity of the arteries, the left ventricle is forced to hypertrophy in order to do more work. Dropsical effusions are also common, not so much from the dilated arteries as from the dilated veins, capillaries and lymphatics.

In the capillaries the disease appears to begin with some alteration in the contents of the endothelial cells about the nuclei; the whole body of the cell being infiltrated, subsequently, so as to become turbid in appearance. In the arteries the several coats are successively invaded, until the walls of the vessels become infiltrated with a material that is at first fibroid, later fatty, and finally calcareous, if the process proceeds sufficiently far. As for the veins, it is probable that the disease commences in the same way—from the endothelium of the inner coats. Those who maintain this theory believe that the special irritation causing the inflammation is due to poisonous bacteria, or their toxins, coursing in the blood; or, as in lead poisoning, to the absorption of metallic substances. But others believe that the poisons are absorbed by the vaso-vasorum of the vessels, and penetrate from without inwards. This latter explanation would hold good for arteries and veins only, and not for the capillaries, for they have no vaso-vasorum. On the whole, if we are to accept a single theory, the former harmonizes best with known facts.

Arteriosclerosis is also a senile change, due, perhaps, to paralysis

or atony of the muscular coats of the vessels. There are two forms of arteriosclerosis, the nodular and the diffuse. The nodular chiefly affects the larger vessels of the body and extremities and those at the base of the brain. In this form the process, microscopically speaking, seems to be as follows: The deeper layers of the intima are thickened by the irritation of bacteria, or by the morbid constituents of the flowing blood. These poisons cause proliferation of the cells, which eventually develop into fibroid tissue. Later the muscle cells become swollen and undergo hyaline change. Eventually the adventitia, or external coat, is so involved that, as already said, there gets to be general infiltration of the entire vessel. In the diffuse form the change is more uniformly distributed. Both intima and media undergo the same hyaline change, while the elastic coat disappears. When the smaller arteries are involved, their lumens are apt to become contracted; though infiltration of a vessel may take place without appreciable narrowing of the lumen. However, in a certain number of cases the contraction may be so great as to lead to obstruction, partial or complete, and so to thrombosis and embolism. Occasionally an artery may be entirely obliterated by this process of infiltration and contraction—*obliterating arteritis.*

A probable chain of events, so far as the heart and arteries are concerned, appears to be: first, an increased tension of the arteries, due to obstruction in the capillaries, followed by a loss of elasticity in the arteries and slowing of the blood stream, so that the vessels no longer aid in propelling the blood. Influenced by these conditions, the left ventricle becomes hypertrophied, and later dilated. Increased tension, however, leads to infiltration, and this in turn to degenerative changes, by which in the final stage the artery may be converted into a rigid tube. To a certain extent the infiltration of the walls of the vessels is a compensatory process, for by it the dilated vessels are strengthened; but fortunately this new tissue is apt to suffer from innutrition, and degenerates in consequence, so that an aneurism will form, or even rupture may take place. In syphilis we see examples of the one, and in senile change, of the other. On the other hand, the new tissue may become tolerably well organized, and, contracting, aid in restoring a dilated vessel, more or less, to its former size. Unfortunately, the beneficial results of the cicatrical contractions are apt to be few, and the degenerative changes many, so that the vessel is unevenly distended.

In some cases, the heart is not found hypertrophied; it has then probably been at some time hypertrophied, but has afterwards con-

tracted. This would explain, in part, why the senile heart in arteriosclerosis is sometimes small. Another cause of contraction may lie in the shrinking of the heart walls from the fibroid changes. But the disease extends beyond the vessels, for in every organ affected the parenchyma is sure to suffer, because its nutrition is affected. In the brain, as we have seen, there may be softening; in the kidneys, contraction; in the heart, atrophy or degeneration of the muscle fibres, with interstitial fibroid deposits originating in the sheaths of the vessels and penetrating between the muscle bundles. When the pulmonary or bronchial arteries are affected, the lung parenchyma also suffers. There are other conditions, namely, *acute aortitis* and *acute arteritis,* which should be alluded to here, because they have a bearing on the chronic disease. There is no doubt that acute aorititis exists, and its occurrence has been associated with a sense of oppression or substernal pain, dyspnœa, some irregularity of the pulse, and fever. But we have not yet, I think, reached the point where it can be differentiated from acute endocarditis.

Acute arteritis, however, offers less difficulties. It may occur in acute or chronic infective diseases, such as typhoid and influenza, acute rheumatism and diphtheria. It appears to elect by preference the arteries of the lower limbs. In fact, the theory that inflammation of the smaller arteries plays an important role in the causation of the exanthems of eruptive fevers, due possibly to the toxins of the causative bacteria, is plausible. In diseases where the strain falls on the left heart, the left ventricle is more prone to this connective tissue infiltration and muscular degeneration; but where the burden falls on the right heart, the right ventricle is most often affected, as in tubercular and syphilitic phthisis. The occurrence of hæmorrhage in arteriosclerosis is thus explained.

The greatest pressure on the arterial wall is just in front of the thickened part. Here it begins to yield, little by little, becoming thinned and stretched and finally somewhat sacculated. When the sac gives way, hemorrhage takes place. In arteriosclerosis there is an increased tension in the vessels. The radial pulse is hard and firm; in advanced cases, rigid. The heart is usually hypertrophied, though in senile cases it may be of normal size or even atrophied, owing to causes that have been described. The heart usually beats with vehemence. Stiffness and tortuosity of the radials is another important sign. In determining the character of the peripheral arteries, the pulse especially, three fingers are useful. If, when moderate pressure is made with the finger nearest the heart, you can

still feel the pulse beat with the other fingers, there is a moderate amount of arteriosclerosis. If by moderate pressure you do not affect the pulse beat on the distal side, there is great arteriosclerosis. Other symptoms are vague tingling or numbness, especially in the left arm and fingers when these parts are at rest, and coldness of the limbs and feet. The muscles are soft and there is a tendency to corpulency. The pulse is also apt to be less frequent than normal. Precordial pain is often developed by exercise or unusual exertion. After middle life a ringing second sound over the aorta is thought to indicate arteriosclerosis; if coupled with high arterial tension it may be regarded as pretty good evidence of coronary sclerosis. Angina pectoris has been held to indicate implication of the coronaries; but the connection is not constant. In fact, angina is rather rare in coronary sclerosis; and yet coronary sclerosis is very common in angina.

Among other prominent signs that have been thought worthy of note is the sallow, pale face; its color, however, may be red from the abnormal development of the arterioles, the *facies arteriosclerotica*. Sometimes pecular wreaths or festoons formed of dilated venous radicles are seen as a girdle about the lower part of the chest. I have several such cases under my observation now, but they are not pathognomonic of ordinary arteriosclerosis; at least they are to be seen sometimes in the phthisis of young people.

If the heart gives way, the pulse will become weak, intermittent or deficient; attacks of gastralgia may occur with or without angina, with palpitation and cardiac asthma. Sometimes there will be vertigo, or the Adams-Stokes syndrome.

There are various types of the disease. In the first place there is the *cardiovascular*, such as is seen in the gangrene of the extremities in diabetics and in senility.

The following case is illustrative:

Case XCVI. Gangrene of Feet; Arteriosclerosis, Aortic and Mitral Endocarditis.—Some years ago I made a post-mortem examination in the practice of one of our late surgeons. An abstract of the case is as follows: A gentleman, 49 years of age, was seen in consultation on August 15, 1877. Some 20 years previously there had been a rheumatic attack and subsequently others, attended with cardiac symptoms. In the previous spring the patient, while in bed, became suddenly unconscious and remained so for three hours. In the latter part of June he began to suffer from pain in the lower extremities, chiefly in the calves, and particularly in that of the left

leg. A week before, he first noticed a violet discoloration in the fourth toe of the left foot. When examined by the attending physician, no pulsation was found in the femorals below Scarpa's space. Four days after the beginning of the gangrene in the left foot, a similar process, in a similar place, appeared on the right foot. No cardiac murmurs were detected. If present, it was thought they had been masked by the feeble action of the heart. The question of amputation was entertained, but this procedure was not advised. The patient lingered several months, to die with symptoms of pneumonia. At the post-mortem examination the heart was found large. Both ventricles were dilated, the left considerably hypertrophied. Aortic orifice contracted and calcified, also the mitral (button hole opening.) In the upper lobe of the left lung there were hæmorrhagic infarctions of various sizes, with surrounding pneumonia. Kidneys also contained infarctions; surfaces granular; Spleen small, contracted and containing calcific deposits and cicatrices. Abdominal aorta marked by atheromatous patches throughout its whole extent. The right common iliac thickened by calcific deposits and at points ulcerated. Occlusion of the left femoral just below the point where the profunda was given off. From this point downward the vessel was filled with a firm plug. In the right femoral the plugging occurred in the lower part of the vessel, just above the popliteal.

In another case (Case XCVII), occurring in the hospital practice of the late Dr. Alfred C. Post, where gangrene had involved nearly all of the left hand, the patient was a man 58 years of age, who entered the hospital suffering from great debility, emaciation, nausea and vomiting. At the post-mortem examination all the arteries of one extremity were found to be thickened and atheromatous, the radial being occluded by a thrombus. In these instances of arteriosclerosis in peripheral arteries, there are apt, of course, to be associated, as in Case XCVI, symptoms of vascular disease in other parts. But there may be also the purely *gastrointestinal* type, due to interference with the circulation and leading to congestion of internal viscera, as indicated by signs of acute or chronic dyspepsia, marked gastralgia and flatulency, perhaps nausea and vomiting. On the other hand, these same symptoms may be due to interstitial thickening of the chylopoetic viscera. The glycosuria so common in aged arteriosclerotics, however, may be explained by the sclerotic condition of the pancreatic arteries. On the other hand, there may be localized ischæmia of the stomach, causing functional inadequacy.

The theory that many gastric or duodenal ulcers are due to cir-

culatory disturbances is based on sound theoretical principles, in so far as such disturbances are competent to produce embolism, thrombosis, or even hæmorrhage, anywhere in the body; but as a matter of fact, it does not appear that the stomach or intestines suffer much from this condition of the circulation, probably on account of their excellent collateral circulation. Still Berthold,[1] of Berlin, has reported that of two hundred and ninety-four cases of gastric ulcer, in one hundred and seventy (or 58 per cent.), there were disorders of circulation, chiefly endocarditis and atheroma, and Steiner[2] found an even larger percentage (71 out of 110, or about 64 per cent.).

The following is an illustrative instance of this type:

Case XCVIII. Gastric Ulcer; Arteriosclerosis.—Mrs. D., 67, English, an alcoholic subject, was first seen by her physician September 1, 1883. She then complained of pain in the right side, loss of strength, sleeplessness and gastric distress, associated with acid eructations and vomiting. Constipation and colicky pains, with movements that were at times dark, and even black and offensive. The patient had been dropsical for years and had suffered from asthma. Weight, about 200 lbs.

On examination there was found a diffuse heart beat, no murmurs, but sounds muffled. Heart's action irregular. Pulse 80-100. Small, rigid radials. Indications of pneumonia and cirrhosis. General œdema. Urine scanty, high colored and albuminous. Patient peevish, with signs of hebetude, but no special brain symptoms. She died in coma. At the post-mortem examination the heart was found enlarged and the aorta dilated, but the valves were free. In the lower lobes of both lungs small areas of pneumonia. Kidney granular. Liver cirrhotic. The stomach contained a small ulcer, but it was thought to be so insignificant, that it could have given no signs. Its cause, however, may have been the rupture of a minute artery in the organ.

The following case of arteriosclerosis is interesting because the cause of death, while ascribed to some affection of the stomach or duodenum, is still obscure.

Case XCIX. Hæmorrhage, Probably from Duodenal Ulcers Due to syphilitic arterisclerosis.—A laborer, between 40 and 50, had been complaining of a pain in the cardiac end of the stomach for several weeks before admission to the hospital. While at his work, in which

[1] Berthold quoted by Welch, *Amer. System of Med.*, Vol. II.
[2] Steiner, *Pepper's System*, Vol. II, p. 487.

he was not exposed to special strain, he began bringing up blood by the mouth and later passed it per anum. He died a few hours after admission, with all the signs of internal hemorrhage. At the post-mortem examination there were found clearly marked copper-colored spots on the left leg. Left heart atrophied. All valves free and sufficient. In the aorta, atheromatous patches and calcareous plates. Aortic arch dilated throughout. At the junction of the first and second portions of the arch on the convex side, a small sacculated aneurism. In the remaining portions of the artery, endarteritis. Colon and lower portions of small intestines filled with blood. In the stomach, which contained blood, the rugæ were prominent and deeply stained with blood. No ulceration of the stomach, but in the duodenum, just below the pylorus, were several suspicious-looking spots in the mucous membrane, with apparent loss of substance, possibly from the rupture of miliary aneurisms. The liver was larger than normal, but not cirrhotic. The evidences therefore pointed to death caused by minute ulcers of the duodenum, due to syphilitic arteriosclerosis.

In *chronic renal disease* with induration, there is an early disturbance of renal functions, though the urine at first shows nothing pathological. There are then no positive subjective symptoms. But when there is a low specific gravity and an occasional trace of albumin with casts, renal sclerosis is at hand. In fact, a diagnosis based on these points is apt to be correct; especially if there are hyaline casts with the granular, and the amount of urine is abnormally large. Even here, however, there is often a general hypertrophy of the cardio-vascular system more or less pronounced, from the one extremity to the other. The following is an illustrative case:

Case C. Lead Poisoning; General Arteriosclerosis.—X, a male, painter, aged 79, was treated by his physician before admission to the hospital for wrist-drop from chronic lead poisoning, and weak heart. Under the use of the iodide of potassium he partially recovered the use of his hand. Shortly before admission, while at work, he had an attack of aphasia, became unconscious, developed mania, and later had a right hemiplegia, which lasted until death. At the autopsy, February 6, 1885, the heart was found enlarged, but the valves were free. The aorta, however, was a mass of atheromatous patches. In the muscular tissue of the heart the striations were indistinct, and showed evidence of brown pigmentation. A diagnosis of brown atrophy was made.

Kidneys small and contained cysts. Surfaces granular. Liver an example of red atrophy. Dura mater thick and adherent. These changes may be taken to have implied vascular thickening.

The following is a more marked instance:

Case CI. Arteriocapillary Fibrosis of Kidney; Uræmia.—A patient of 67 was attacked with continuous nausea, vomiting and some dyspnœa. Varying quantities of a pale urine. Sp. gr. 1010 to 1018. Albumin 2 to 20 per cent. All kinds of casts present. Obstinate constipation. Slight muscular twitchings, relieved by morphia. Pupils contracted. Patient died semi-comatose. At the post-mortem examination the heart was found hypertrophied, the liver fatty and pigmented. Aorta dilated and thinned. In lungs fibroid induration. Spleen hard and indurated. Chronic diffuse nephritis (the small contracted kidney). The left kidney weighed only three, and the right four ounces. Stomach small and contracted. Fibroid induration of the pylorus, and a constriction about a foot above the sigmoid flexure. In this case, where the clinical symptoms indicated an arterio-capillary fibrosis (Gull and Sutton), there was some fibroid induration of internal organs, while the aorta and peripheral arteries apparently showed no thickening. Then there is the *cerebral type,* associated with cerebral anæmia, hæmorrhages, miliary aneurisms, and softening, as well exhibited in the retina and choroid, with many psychic disturbances, such as loss of memory, or senile dementia, vertigo, pain in head and neck, involuntary tremors, and diplopia, with specks or flashes before the eyes.

Case CII. Cerebral Hæmorrhage; Arteriosclerosis.—A man 84 years of age was found locked in a room and unconscious, with some right facial paralysis and right hemiplegia. Superficial reflexes absent on both sides, and deep reflexes on the right side. Died of pulmonary œdema a few hours after admission to the hospital. At the post-mortem examination the heart was found but slightly hypertrophied, but the muscular substance was soft, pale and flabby. Valves free. In the aorta some fatty changes, but no atheroma. Oedema of both lungs. Kidneys small and granular. Chronic diffuse nephritis. Liver small and apparently cirrhotic. In the brain a clot the size of a hen's egg involved the anterior and external portion of the lenticular nucleus and external capsule, encroaching on the convolutions of the Island of Reil. About the clot the cerebral substance was soft.

Liver and kidneys instances of arteriosclerosis; also a cerebral hemorrhage, due, presumably, to the bursting of a *miliary aneurism.*

In the *spinal type,* well exemplified in the locomotor ataxia of syhpilis, we have a disease that presumably originates in the vessel of the cord, as does also, probably, the general paralysis of the insane.

In the *pulmonary type* evidences of endarteritis and periarteritis have frequently been found in the lungs and bronchi in cases of emphysema and spasmodic asthma, leading to the conclusion that these vascular changes may sometimes be due to gout. Similar changes are seen in pulmonary tuberculosis and syphilis.

The following case is an example of arteriosclerosis associated with syphilitic phthisis.

Case CIII. Syphilitis and Tubercular Arteriosclerosis. Death from Pulmonary Hæmorrhage.—J., 30, stone cutter, of tubercular ancestry, had suffered from cough and pain in the right side of the chest for six months before admission to the hospital. During this time he had lost 15 pounds of flesh. For the last three months had suffered from dyspnœa, and for the last four weeks from night sweats. He died of pulmonary hemorrhage. At the post-mortem examination the heart was found enlarged, the left side more especially. Valves free. At the apex of the left lung was a large cavity surounded by smaller cavities, but the whole lung was interspersed with fibroid tissue, separating the small lobules in a peculiar manner. There were also numerous nodules from the size of a pin's head to that of a pea. Most of them could be shelled out of the capsules, leaving cavities with a smooth lining. There were still other nodules that looked like miliary formations. The hemorrhage had taken place from one of the cavities lined by a smooth wall. The lung was thought to be the seat of both syphilis and tuberculosis, but a diligent search for the bacilli of tuberculosis both in the lung tissue and in the sputum, was unsuccessful. The right lung was in a similar condition. The kidneys were the seat of chronic parenchymatous disease. While no special examination of the vessels was made, it is a known fact that the vessels in syphilis are usually, if not always, involved, so that in the absence of the bacillus tuberculosis after diligent search had been made for it, the conclusion seemed to be justified that the cause of death was internal hemorrhage due to the rupture of a syphilitic vessel. Though arteriosclerosis is eminently a disease of advanced life, it may occur earlier, especially in syphilis and in chronic nephritis.

Inasmuch as the longer we delay treatment in these cases, the

less are the chances of success, it behooves us to make an early diagnosis and immediately set about a regular plan of treatment. Of course, prophylaxis aside, we should endeavor as soon as possible to remove the cause of the disease, though this may not be always possible. Age and heredity are and ever will be the bars to success; and it may not be possible for a painter, for instance, to give up his profession. But over-indulgence in food and drink, and the "over-strenuous life," can be combated, while the danger of gout and syphilis can be minimized, for they are not altogether intractable. In fact, we may say that both of them, as well as tuberculosis, can, in many instances, be held in check.

Apart from these considerations, the patient should lead a quiet life, free from its turmoils. And with good fortune and little treatment, an arteriosclerotic may live to a good old age. All exercise should be moderate, but at the same time a certain amount of exercise is necessary. Baths and resistance exercises are as useful in the milder cases, as they are useless or dangerous in advanced cases. If hot baths cause excitement, as they are apt to do in these cases, or resistance exercises cause pain or any other untoward symptoms, they should be stopped and not resumed. If there are any suspicions of apoplexy, both baths and resistance exercises should be sternly interdicted. In arteriosclerosis the medicine that is indicated before all other is *iodine*. It should be used year in and year out, if we are to expect good results. Iodide of sodium, iodide of potassium and hydriodic acid may be followed by the iodides of arsenic, iron and strontium, or free iodine. Iodine appears to lower the blood-pressure without diminishing the force of the cardiac contractions. Aconite is also useful, and camphor, but digitalis and convallaria are harmful. The nitrites, however, are always of service. Together with the iodides, they are at present our sheet anchors.

Chapter XXV.

SURGERY OF THE HEART.

It has long been known that wounds of the heart may not be immediately fatal, but when Fischer[1] published his series of 452 cases in 1867 he showed that persons could live for several days with heart wounds, and that spontaneous recovery followed in from 7 to 10 per cent.

There are three classes of cases. In the first the puncture is very small, such as might be made by a knitting-needle, and the muscular tissue about the wound closes it, so that little if any blood escapes into the pericardial cavity. In the second class, the wound does not reach any of the chambers of the heart, so that unless the foreign body happens to open a good-sized vessel, the only result is oozing of a small amount of blood into the pericardium. In the third class the wound is "valvular," that is, a chamber of the heart is opened, but by so oblique an incision that it is closed by every cardiac contraction, and no very large quantity of blood escapes.

It is only within the last ten years that operations on the heart have been successful.

The experiment of *heart suture* on a dog by Del Veichio[2] in 1895 first showed the possibility of a successful operation on the human kind, which was accomplished by Rehn[3] in 1897. Up to Oct. 1, 1904, acording to the *Lancet,* there had been 60 cases of cardiac suture with a recovery of 22, or 38 per cent. Accordingly, a preceding mortality of at least 90 per cent. has by skilful surgery been converted into a mortality of only 62 per cent.

According to the *Lancet:* "The symptoms of wound of the heart vary. If the pleura is wounded and blood escapes into the pleural cavity there are anæmia and the signs of pneumo-hæmothorax. A splashing sound indicates pneumo-hæmopericardium; in some cases a friction sound is heard. If there is external hæmorrhage the stream may be continuous or in jets. If the blood is confined to the pericardium the pulse is very feeble and death may result from pressure on the heart. The diagnosis of wound of the heart may be difficult or impossible. The position of the external wound is not

[1] Fischer, *Arch. f. Klin. Chir.,* Band IX, 1867-8.
[2] *Del Veichio, Reforma Med.,* 1895, Vol. II, p. 38 *et seq.*
[3] Rehn, *Verhand. d. Deutsche. Gesellschaft. f. Chir.,* Berlin, 1897, XXVI.
[4] *Lancet,* Oct. 1, 1904.

a safe guide. The rule in doubtful cases should be to enlarge the wound, to ascertain if it penetrates the chest wall, and if there be symptoms of hæmorrhage or of pressure on the heart to operate."

In the history of a case of successful suture of the heart reported by Hill[5] in a negro stabbed with a penknife, the results in 37 cases were reviewed and the following conclusions drawn:

The operation for heart suture is entitled to a permanent place in surgery. Every heart wound should be operated on immediately. Even if there is only a suspicion of it, an exploratory incision should be made. Chloroform is the preferable anæsthetic. The wound should not be probed. Rotter's[6] operation renders the access to the heart easy. Steady the heart before attempting to suture it, either by placing the hand under the organ, and lifting it up; if the hole is large enough, introduce the little finger. Catgut sutures should be used, as wounds of the heart heal promptly. The sutures should not involve the endocardium, should be interrupted and tied during diastole. As few as possible should be used. The pericardium should be cleansed, but no fluid poured into the sac. The wound of the pericardium should be closed. If symptoms of compression ensue, reopen the wound and drain.

In a bullet wound case recently reported to the Paris Society of Surgery (Tuffier, *Bull. et Mem. de la Societé de Chir*, 29, 1903, p. 957), where the radiograph disclosed a foreign body in the left wall of the heart, and moving with that organ, a portion of the second left costal cartilage was removed. The finger passed under the border of the lung located the bullet. The overlying tissue was then laid bare and the ball extracted. The patient recovered.

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[5] Hill, *Me.d Rec.*, Nov. 29, 1902.

[6] Rotter, *Verhandl. d. Gesell. Deutsch. Naturfor. und Aerzte*, 1899, II, s. 541.

APPENDIX.

I. CONGENITAL HEART AFFECTIONS.

Malformations of the heart are numerous. They may be serious or trivial. In either case they rarely attract much clinical interest, because radical treatment is impossible. At best it can only be palliative.

Most of the defects are due to arrested development in the valves, septa, or large vessels. Of these the most common are either patency of the foramen ovale in the inter-auricular septum, or improper closure of the interventricular septum. Among other instances of arrested development is the heart with only one auricle and one ventricle, or two auricles and one ventricle, the bi-locular and tri-locular hearts respectively.

Valvular anomalies are also comparatively common. The leaflets may be more or less than the usual number. In some cases antenatal inflammation may have glued the segments together, as in inflammatory rheumatism of the mother during pregnancy. Among the anomalies of the larger vessels, the aorta and pulmonary may originate jointly; or the aorta may be displaced to the right, or even arise from the right ventricle. Then the ductus anteriosus, or Botalli, which in fœtal life unites the aorta with the pulmonary artery, may remain open after birth.

But if there are defects in the pulmonary artery or aorta, this patency of the ductus arteriosus may be salutary. In fact, if there is a stricture of the pulmonary orifice, a supply of blood to the lungs through the ductus arteriosus may be the means of maintaining life. Without doubt, many of the minor anomalies are co-related to the major forms, one in one place compensating for one or more in others. In some instances, however, the malformations are not sufficiently compensated for, and the circulation is profoundly disturbed.

Others are of little consequence. Minute orifices in the auricular septum or in the valves may evoke no symptoms; in fact, the foramen ovale may remain partly open, without deranging the circulation. Even in conspicuous malformations, life may be maintained for years.

Of 181 cases collected by Peacock, 119 came in this category; 155 or 86 per cent. living beyond the 12th year.

As for symptoms, the chief is cyanosis, which appears within the first week of life, and is seen in the fingers, toes, lips and ears. The fingers and toes are usually clubbed. This condition is called *morbus caruleus* (blue disease), and is due to imperfect æration of the blood. As additional symptoms there are dyspnœa and cough, and the patients are lethargic.

There is another condition called *cardiac hypoplasia,* where the heart is small from birth, while the rest of the body develops in proper proportions. Virchow believed that this condition was closely related to chlorosis or hæmophilia. The volume of the heart may be reduced one-third. The arteries of the aorta are also small. After a time the increased work of the heart may cause hypertrophy. In some instances there is a defective development of the front wall of the chest. Again, the pericardium may be absent.

II. TUMORS OF THE HEART.

Tumors of the heart are very rare and are usually secondary. The varieties are *sarcomas, myomas, myxomas, fibromas, gummas, angeiomas, lipomas, carcinomas,* and *cysts.* Secondary carcinoma is the most frequent of all neoplasms, though still comparately rare. It has no preference as to site. Often it is merely an extension from a contiguous disease.

Sarcomas are rarer than carcinomas. They occur, however, at any age, and are of several varieties. Myxomas have been found in 9 instances. (Whittaker.) Rarer still are myomas and the remaining four of the connective tissue group.

Tuberculosis is not uncommon in the heart; it is apt to involve the pericardium.

III. ANEURISM OF THE HEART.

This term is applied to a partial dilatation of the heart wall or septum. A weak spot develops in either, from some process of softening or rupture, and then the weakened tissue is "ballooned out." The clotted blood collected in the sac may then cause sudden death by discharging into the heart cavity, causing embolism; or the sac may burst, causing death as the result of shock, from the sudden hæmorrhage.

Syphilis is probably the most frequent cause of cardiac aneurism, either by myocardial softening due to gummatous tumors or infiltrations; or from the arteriosclerosis of a vessel in the substance of the heart.

There are no characteristic symptoms, and it is not probable that a positive diagnosis has ever been made, even where cardiac aneurism has been suspected. The prognosis is unfavorable, as the disease does not tend to self-limitation. The treatment is symptomatic. In a case that came to my knowledge, however, a man in attempting to lift a heavy weight was very suddenly taken with heart failure, as shown by his rapid and irregular pulse. A cure came with rest. Possibly, and I think probably, in this instance some fibres in the wall of the heart ruptured an dthere was a temporary acute aneurism, from which he recovered when the ruptured fibres had healed.

IV. PARASITES OF THE HEART.

The Cysticercus and the Ecchinococcus may be found in the heart. They are comparatively rare, and are usually met with in the walls of the heart, but occasionally involve the valves, rendering them incompetent. Hydatids vary in size from a pin's head to an orange. If the sac ruptures into a heart chamber, death may at once ensue. This accident has happened on several occasions. Deposits of actinomycosis have also been found in the walls of the heart. The diagnosis cannot be made with certainty, but it may be suspected, if deposits of the parasites have been found elsewhere in the body; in such instances embolisms frequently occur. If a tumor can be located in the walls of the heart, aspiration may possibly reveal the parasitic character of the tumor; or if a distal vessel is plugged, the embolism may be found to be parasitic. Surgical treatment offers the only ground for hope of life.

V. TREATMENT OF CHRONIC HEART DISEASE AT FRANZENSBAD.

The success that has attended the management of heart diseases in Nauheim, Germany, has led to the introduction of the same line of treatment in other European Spas, notably in Franzensbad, which lies in the northwesterly corner of Austria, where it touches Saxony and Bavaria.

Situated on a broad and well-cultivated plain, through which the beautiful river Eger and its tributaries flow, and at an elevation of more than 1,500 feet above the sea, it is walled about by mountains and forests that protect it from the cold winds and storms, while the air is necessarily cool, pure and light, even in the middle of summer.

The town itself is attractive, quiet and restful. Shady walks are numerous. Picturesque drives extend in every direction. Good

hotels and apartments are numerous. Excellent music and a good theatre are other attractions. All the bath houses and springs are under the control of the municipality, whose officers are intelligent, progressive and efficient.

Franzensbad, like Carlsbad and Marienbad, occupies a central position in Europe, and is conveniently reached by the best express, sleeping car, and de Luxe services from Dover, Paris, Berlin, Frankfort and Vienna.

The springs are a dozen in number, all charged with carbonic acid gas and other chemical ingredients, in which they only differ as to relative proportions.

According to published analyses, some of them are more strongly charged with carbonic acid gas than any other springs in Europe. The most prominent ingredients are the carbonate of iron, Glauber's salt, common salt, and the alkaline carbonates, together with the free carbonic acid. The waters contain but small percentage of the salts of lime, silicic acid and earthy matters. The springs in which iron predominates are used internally for improving the character of the blood. Those that contain alkalies and Glauber's salt constitute a feature of the treatment. Those having the largest amounts of carbonic acid and iron are used for the baths.

The temperature of the waters varies from 50 to 55° F. Those used for bathing purposes are heated by different methods. By the *Pfriems system,* steam is turned into the tub, so as to bring the water up to the desired temperature. The water is by this system heated *directly.* This, in common parlance, is called the *Mineral* bath. The proportions of gas and iron are small. It is suited for weak, delicate or nervous persons. It is the initial bath for those who take the cure. Stronger baths are prepared by the *Schwarz* or *Reinerz systems,* in which the tub has a double bottom, the intermediate space being fitted with steam coils, so that the water may be heated to the desired temperature without being diluted. Such a bath is called a *Steel bath,* also an arbitrary word, but meaning that the water is heated *indirectly.* These Steel baths are suited for robust men or women, or for patients who require a stronger bath than the *Mineral.* Lastly, there is the *Flowing bath,* where the carbonated water, which has been previously heated to the required temperature, flows through the tub in which the patient is immersed. These last are taken at the close of a course in heart diseases.

The appliances at the baths are such that any degree of strength

can be obtained, and brine, the chloride of calcium or other ingredients may be added in any proportion that may be necessary.

Each of the three kinds of baths, whether *Mineral, Steel* or *Flowing,* has three grades, the strength of the bath constantly increasing with each successive grade. In point of strength the order is as follows:

Mineral,	1		Steel,	1	
	2	The weakest.		2	The medium.
	3			3	
Flowing,	A				
	B	The strongest.			
	C				

In the regular heart disease course, the patient takes his carbonic acid gas treatment in these nine different grades of strength. The weakest baths contain no brine.

If the duration of the bath is not too long, the effect is very stimulating, but if too long, irritability and fatigue will be the results. To prevent inhalation of the gas the water should not be allowed to reach more than to the middle of the chest, or the tub may be covered by a linen sheet. Inhalation of the gas will, of course, cause some headache, dyspnœa or palpitation; but as a matter of fact such an occurrence is very rare, because the gas rises only a few inches above the level of the water. At the strong *flowing baths,* however, where the agitation of the water causes the gas to rise higher than in the other baths, there is always an attendant in the room. Usually the sensations after the bath are very agreeable; the large and airy bathrooms, that are features of the Franzensbad bath-houses, obviating the likelihood of any possibly unpleasant effects from inspiring the gas. For further particulars, the reader is referred to the book entitled *Franzensbad; Its Mineral Waters and Baths, by Dr. L. Fellner, 1904.*

Fellner[1] agrees with Winternitz of Halle that the carbonic acid gas is absorbed by the skin, and that it has a reflex action on the vaso-motor centers, causing dilatation of the arteries and capillaries, which produce a sensation of warmth. Through its influence on the pneumo-gastric it lessens the frequency of the pulse, and deepens inspiration. Dilatation of the vessels permits them, of course, to be

[1] Fellner, *Verhand. der Chr. Herzkrankheiten,* 1904.

better filled. Diminution in cardiac frequency favors diastole, so that the ventricles are also better filled, and discharging more blood, take up more blood and so relieve venous congestion. Deep breathing also increases the siphon-action of the heart. In these ways there is a tendency to restore the balance of circulation between the arterial and venous systems. The heart itself is likewise improved, for the coronary arteries are better filled, and the organ is supplied with more and better nourishment.[2]

According to Fellner, resistance exercises also lessen the frequency of the pulse and increase the blood pressure, because they contract the muscles, causing them to press on the delicate walls of the veins and lymphatics, facilitating the flow of their contents towards the heart. The organ, therefore, is better filled, and the left ventricle has more blood to distribute throughout the systemic circulation, and so the tendency to congestion of the veins and lymphatics, so common in chronic heart disease, is overcome.

VII. REGIMEN IN CHRONIC HEART DISEASE.

Rules to Be Observed by the Patient.

Liquids. Do not drink more than half a glass, to a glass (four to eight ounces of water), at meal time. Water may be taken freely two hours after eating; on rising in the morning; and at bed time.

In case stimulants are necessary, pure Moselle wine can be taken at lunch or dinner with an equal quantity of water, preferably Highland water. Aerated water should be avoided. A small quantity (half an ounce) of good whiskey can be taken in place of the Moselle. Use no fermented liquor, and take alcoholics only as above directed. May take Zoolak or Vichy. Avoid coffee or tea, unless the latter is weak. Take Postum in place of coffee. Use milk only if it agrees.

Foods. Eat no white or rye bread. Use Graham, whole wheat bread, unsweetened Zwieback or toast.

Fish. Avoid salmon and white fish. Eat broiled, baked, or boiled fish, but without dressings or sauces. May eat oysters and clams.

Meats. May eat roast beef, mutton, lamb, veal, venison, sweetbread, fowl or game. Avoid ducks and geese. Avoid, however, gravies, stuffed and breaded meats. May eat plain omelettes or scrambled eggs.

Vegetables. May eat peas and Lima beans (in moderation),

[2] Many walks in Franzensbad are laid out with reference to the *Terrain cur.* As the distances are indicated by signboards, patients can follow the directions of the physician in walking the exact distance he prescribes.

string beans, oyster plant, tomatoes and spinach, koli-rabi, young carrots and beets (stewed), egg plant, rice and hominy.

Dessert. May eat plain rice and hominy, or custard, or apple pudding. Wine jellies, if not sweetened; stewed fruits. May eat, occasionally, ripe peaches, pears, and grape fruit. May eat stewed cherries, pears, peaches, and apples. Avoid all forms of cheese.

Tobacco. Avoid all forms of tobacco.

Eat slowly and at regular hours; masticate thoroughly.

Do not partake of a great variety of dishes at any one time, nor eat large quantities of anything very hot or cold.

Under-cooked vegetables, overdone meats, hard-boiled eggs, are to be avoided.

A moderate quantity of food for adults should average daily about ten ounces of animal food, thirty ounces of vegetable food, and fifty to eighty ounces of liquids, principally water.

VII. THE MODIFIED RIVA ROCCI SPHYGMOMANOMETER.

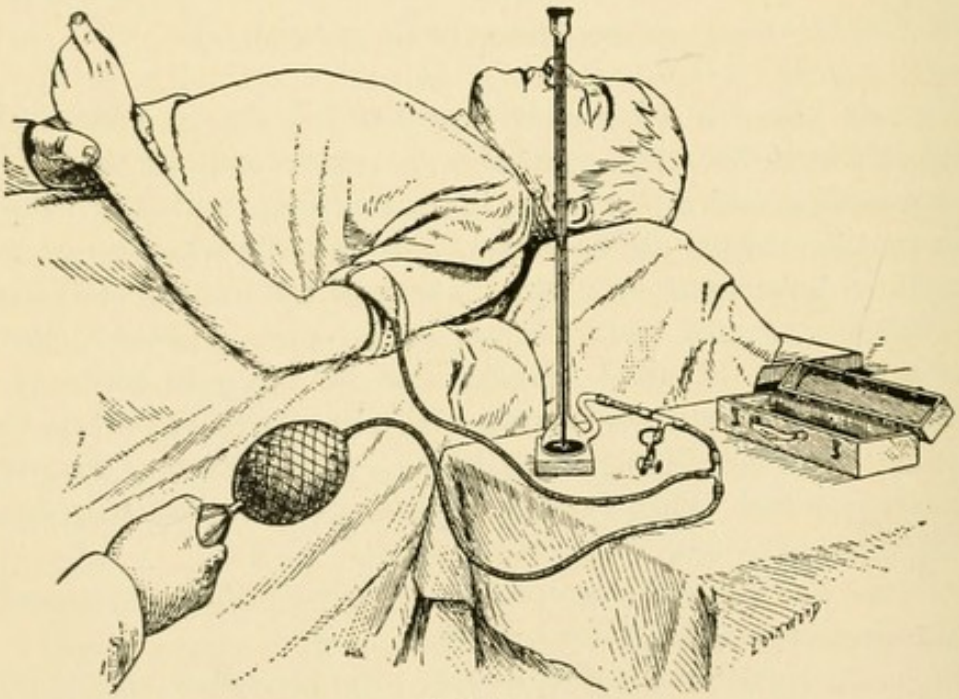

Fig. 41.

This instrument is thus described by the makers: It is an apparatus for estimating artificial blood pressure or pulse force, modified by Dr. Henry Wireman Cook, of the Johns Hopkins Hospital, Baltimore, Md.

The most valuable indication derived from palpation of the pulse

is the indication of *arterial tension.* The value of such estimates is in direct proportion to their accuracy.

A knowledge of actual, definitely determined pulse tension is of special value in the diagnosis of many morbid conditions where variations from the normal are characteristic, as, for example, in apoplexy, traumatic brain compression, surgical or traumatic shock, nephritis, cardiac diseases, aneurism, lead poisoning, hæmorrhages, uræmia, etc.; also in the treatment of conditions where correction of an existing pulse tension is aimed at, under which are included all the conditions just mentioned above, and in addition, the larger class of toxic cases in which depression of the vaso motor system is a prominent feature and calls for stimulation. In these cases it is specially important to be able to follow variations in pulse-force accurately, and to meet such variations with proper therapeutic measures.

The apparatus consists of a system of closed tubes connected with a rubber bulb held by the operator, a hollow rubber band (placed around the arm or leg of the patient), and a mercury manometer. By the law of the diffusion of gases, equal pressure is transmitted to every point throughout this closed air system. When the pressure is raised by the operator to such a point that the pulse of the patient distal to the constricting band is obliterated, the height of the mercury column in the manometer is held to be equivalent to the maximum arterial blood pressure.

The arm-piece is placed around the patient's upper arm, midway between the elbow and shoulder, and adjusted to fit. The operator, with one hand, increases the pressure by squeezing the hand bulb, and, with the other hand, palpates the patient's radial at the waist. When the pressure just obliterates the pulse at the wrist, the height of the mercury column is noted, and it is then allowed to drop slowly until the pulse returns. This manœuvre is repeated without letting the air out, and by merely squeezing and releasing the reservoir bulb. The point above which the pulse is obliterated and below which it returns is the reading of maximum arterial blood pressure. A determination within two or three millimeters should be considered satisfactory.

A reading of mean arterial blood pressure may be made with this instrument, as described by Prof. Gumprecht, by finding the point when the greatest excursion of the mercury column occurs during cardiac systole after clamping off the tube leading to the reservoir bulb.

The normal maximum blood pressure averages, when lying at rest:

For children of 1 to 3 years..... 85 to 95 mm.
For children over 3 years..... 95 to 110 mm.
For adult females .....115 to 125 mm.
For adult males .....125 to 135 mm.

The mean arterial pressure is about three-quarters of the maximum. Any one at all trained in pulse palpation can make an accurate reading at the first trial. An estimation takes from fifteen to thirty seconds.

Sold by the Kny-Scheerer Co., 225 to 233 Fourth Ave., New York City, or Eimer & Amend, 18th St. and 3rd Ave., New York City.

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