

PART III.

DISEASES OF THE CIRCULATORY SYSTEM.

I. DISEASE OF THE PERICARDIUM.

PERICARDITIS.

Definition.—An inflammation of the pericardium or serous covering of the heart.

Varieties.—Acute, Plastic or Fibrinous, Subacute or Sero-fibrinous, which includes the Purulent and Hemorrhagic; Adhesive or Chronic Pericarditis.

ACUTE PLASTIC PERICARDITIS.

Synonyms.—Fibrinous Pericarditis ; Dry Pericarditis.

Etiology.—This form of pericarditis occurs more frequently in the young and middle-aged than at any other period of life, and occurs in males far more frequently than in females. It may be divided into primary and secondary forms.

The primary form occurs very rarely, though bruises or injuries of various kinds may result in so great an irritation and determination of blood as to give rise to inflammation. The old idea, once so prevalent, that cold was the exciting cause, is rapidly giving way before more careful observation and experimentation, and nearly all writers are now agreed that pericarditis is a secondary affection. Metchnikoff goes so far as to declare that there can be no such a condition as idiopathic pericarditis.

First in importance as a causal factor may be classed rheumatism; Bouillard declaring that in every case of rheumatism there will be some lesion of the heart; and while we regard this as a very extravagant statement, we may be quite conservative and yet place rheumatism as the cause in at least fifty per cent of all cases of pericarditis.

Chronic nephritis and tuberculosis may give rise to the acute form, but

is more common in the subacute variety. Toxins from the infectious diseases seem to influence the pericardium quite early, resulting in inflammation. Scarlet fever, measles, diphtheria, influenza, and typhoid fever in particular, give rise to it, though any infectious fever may have its influence in the same direction.

The extension of the inflammatory condition from neighboring organs, especially pleurisy and pneumonia, is a frequent cause, more of this form than of the other varieties. Carcinoma by poisoning the blood and encroaching upon neighboring tissues may give rise to this form.

Pathology.—The changes that occur may be general or local, usually the latter, and are similar to those which occur in pleurisy and peritonitis. At first the membrane is red, smooth, injected, and swollen, but soon becomes rough and thickened by the deposit of a fibrinous exudate. As a result of the friction of the surfaces, the membrane becomes roughened or wrinkled, resembling tripe in appearance, and when the exudate is thick, this friction results in giving the membrane a jagged-looking surface, giving rise to the shaggy or hairy heart of the older writers.

In this variety there is but little serous fluid, the natural secretion being arrested or greatly diminished. The myocardium may also be affected; in fact, there can scarcely ever be a severe pericarditis without involving more or less the heart itself. Where the disease has continued for some time, the heart is apt to be flabby and dilated, with more or less fatty degeneration.

If adhesions of the two surfaces have taken place, there is apt to be hypertrophy of the heart. At other times the nutrition of the heart is so impaired as to give rise to atrophy.

Symptoms.—*Primary Form.*—If the disease is primary, which is very rare, and if the patient be a young subject, there will be a chill, followed by a febrile reaction, a dry skin, scanty secretion of urine, constipation, and the general symptoms of an inflammation.

There is pain in the precordial region that varies from a dull, aching sensation to one of an intense, sharp, lancinating character, which extends from the nipple to the back and down the left arm. There is a sense of great anxiety, and though there may be but little pain. the

patient has an anxious expression that can not be disguised. The pulse varies from one hundred to one hundred and thirty per minute, and in the early stage is full and strong.

If the pleura is involved, there will be embarrassed respiration.

Secondary Form.—Since the very large per cent of cases of pericarditis is preceded by some other disease, the symptoms relating to the heart are more or less obscured, and often the disease is entirely overlooked and the discovery made post-mortem. In confirmation of this is a statement made by an ex-intern of our city hospital, that of five post-mortems that came under his observation where pericarditis was found, not a single case had been diagnosed during life.

If the inflammation be very acute, pain of greater or less intensity will be felt in the precordial region, extending to the left arm, with more or less constriction in the precordial -region. Dyspnea is often present, but not a constant feature. The pulse is increased in frequency, and, though full and bounding in the early stage, becomes more feeble in long protracted cases. The fever that attends is rarely severe in character, the temperature not often exceeding 102°.

Physical Signs.—*Inspection*, if the patient be spare, may reveal increased force of the apex-beat. In severe cases the veins of the neck are swollen, and the pulsation of the jugulars are visible.

Palpation.—Palpation may reveal friction fremitus, which is due to the rubbing of the changed pericardial surfaces one upon the other, and is most intense to the left of the sternum. During the early and later stages it is more readily detected, there being but little effusion at these stages.

Percussion.—But little, if any, information is gained by percussion.

Auscultation.—The most positive information, and we might say pathognomonic signs, are obtained by auscultation. The pericardial friction rub is due partly to the exudate and partly to the dry condition of the membrane. This sound is usually double, and corresponds to both diastole and systole, though it may be triple and sometimes quadruple.

The sound is generally more pronounced than endocardial murmurs,

and is harsh or creaking, resembling the sound of bending new leather, the sounds becoming more smooth and diffuse as the effusion increases.

The maximum sound is heard between the fourth and fifth interspace near the sternum. The sound is intensified by changing the pressure of the stethoscope, moderately firm pressure giving the maximum sound, while very firm pressure causes it to entirely disappear.

The intensity of the sound is also influenced by the respiration, being usually louder on inspiration, though occasionally louder on expiration. Change of position will also influence and modify the sounds; thus the sitting position gives greater intensity to the sounds than when the patient is lying down.

Diagnosis.—In some cases the diagnosis is very readily made, while in others it is quite difficult, and in some cases impossible. The most positive sign is the characteristic friction rub, and to the skilled and practiced ear, the harsh rubbing or creaking sound near the ear is readily distinguished from the blowing and more distant sound of endocarditis.

We would recognize it from valvular lesions by the more constant and long-continued adventitious sounds of the latter, and also by the fact that change of position from the sitting position to that of lying down does not effect so marked a change in the latter as in the former disease; also, the modified sounds, by changing the degree of pressure of the stethoscope.

Pleural sounds are magnified during respiration; in fact, are suspended if the patient be requested to hold his breath; while in pericarditis the suspension of respiration does not necessarily impair the sounds.

Prognosis.—The prognosis is favorable, so far as life is concerned, the disease rarely terminating fatally; however, there is great danger of more or less adhesions, that leave the heart subject to more serious wrongs in later years. At times it assumes the chronic form.

In rare cases resolution is complete, the disease lasting but a few weeks. Where death occurs, it is usually the result of an intense primary disease, such as a severe croupous pneumonia, or severe chronic nephritis, or severe valvular disease.

Treatment.—The patient should be made acquainted with his true condition in order that he may the more readily acquiesce in the quietude which he will be compelled to assume to get the best results from treatment. He should occupy the recumbent position the greater part of the time, and should refrain from all conversation or reading that would tend to undue excitement.

The diet should be light, one that is readily appropriated, and given in concentrated form. Very little fluid should be allowed, no more than is absolutely necessary, and hot drinks should be entirely prohibited.

During the acute stage, to control the fever, we use the special sedatives. Aconite for the small frequent pulse, five drops to a half a glass of water, of which a teaspoonful will be given every hour. Occasionally we find excessive heart power in the early stage with a full bounding pulse; in this case veratrum ten to thirty drops to a half a glass of water, and a teaspoonful every one, two, or three hours.

Where there is a sense of weight and oppression, not due to effusion, give lobelia a half dram to a half a glass of water, teaspoonful every hour.

Bryonia.—Where there is pain of a sharp, lancinating character, bryonia is a remedy of great merit; being an anti-rheumatic and at the same time an agent whose specific action is upon serous membranes, it is doubly indicated.

Asclepias combines nicely with bryonia, especially if the skin be dry and harsh.

Macrotys will be the better remedy where there is muscular soreness; a dram to a half glass of water will give better results than the small dose. The early Eclectics accomplished better results from a decoction of the fresh root, but this is not readily obtained by the majority of physicians, and we will have to depend upon the less efficient tincture.

After the more active symptoms subside, to establish secretion from the kidneys and promote absorption of the exudate, potassium acetate, well diluted, will be a good treatment.

Digitalis, strychnia, and nitroglycerin must not be used too early, or we will overstimulate and exhaust the power of the heart, and only when the soft pulse indicates the failing power of the heart are they permissible. Cactus and crataegus, however, may be used at any stage.

When he have the history of rheumatism, the anti-rheumatics, given according to their indicated use, will prove beneficial. If uric acid be present, potassium acetate, or lithiate, should be given till it disappears. Where the patient is able to travel, change of air and climate will often prove highly beneficial, though a very high altitude should be avoided.

PERICARDITIS WITH EFFUSION.

Synonyms.—Sero-fibrinous Pericarditis; Hemorrhagic Pericarditis; Purulent Pericarditis.

Etiology.—This form is frequently preceded by the acute form just described, or, like the former, it follows or is accompanied by rheumatism. Bright's disease is not an infrequent forerunner of it. Tuberculosis is also a common cause, especially of the purulent and hemorrhagic variety. Septicemia and the eruptive fevers are also diseases that should be considered in the purulent and hemorrhagic forms.

In children, the disease may come on so insidiously that quite a pericardial effusion may have taken place before the physician discovers it. In such cases there is generally a tubercular taint.

Pathology.—This variety is frequently, if not always, preceded for a few days by plastic pericarditis, and is attended by the same anatomic changes; namely, a smooth, swollen, and injected membrane, in the early stage, followed soon after by a plastic exudate, usually more pronounced than in the acute form.

The pericardial layers being covered with a sticky exudate, gives the membrane a roughened appearance. This is soon followed, however, by an effusion of variable character and quantity, serum largely predominating.

If the result of rheumatism, the effusion will be serous in character; but

if caused by Bright's disease, tuberculosis, cancer, the eruptive fevers, septicemia, or pyemia, the effusion will be of a lower grade, pus largely predominating, or there will be a mixture of pus and blood.

The quantity varies from a few ounces to two or three pints. With the absorption of the more fluid portion of the exudate, the lymph becomes organized, and adhesions take place, sometimes so firmly as to almost, if not entirely, obliterate the peri-cardial sac.

If the exudate is composed largely of pus, the myocardium presents a roughened and eroded appearance, and, being softened by the presence of pus, degeneration of its walls takes place, or there will be dilatation and thinning of its walls. Endocarditis is also a frequent result.

Symptoms.—If a primary lesion, a rare case, the symptoms common to all inflammations are present; namely, the chill, followed by fever, accompanied by a dry tongue, arrest of the secretions, increased temperature, and increased frequency of the pulse. There is nausea and sometimes vomiting. Pain of a dull, aching character is felt in the precordial region, or, if the pleura is involved, it will be of a sharp, lancinating character, extending to the back and down the left arm.

As soon as effusion takes place, dyspnea becomes the most distressing feature. If large in quantity, the left lung is burdened by pressure, and the breathing is quite labored. The right ventricle is also pressed upon, obstructing the cardio-pulmonary circulation. Although the pulse may be full and strong during the early stage, it is now small and weak, owing to pressure by the effused material.

When the disease is secondary, the primary lesion may so overshadow it that it will be entirely overlooked, especially if it follow pleurisy or pneumonia, and often the disease is not recognized till the dyspnea becomes marked, or there is effacement of the intercostal spaces; even then the disease has been overlooked and pronounced pleurisy.

In tubercular children, the disease may come on insidiously, the child growing anemic; he is of a waxy or transparent color, with a gradually increasing dyspnea, till the pericardium becomes distended with the effused fluid.

Physical Signs.—*Inspection.*—The skin and mucous surfaces are pale

and cyanotic in appearance, and the veins in the neck are usually more distended and prominent than in health.

In the young, there will be, if much effusion, effacement, or even bulging of the intercostal spaces.

The breathing will be labored, and the patient will have an anxious expression peculiar to heart affections. The position, if lying, will be dorsal, though some will experience greater relief by lying upon the left side, thus giving greater relief to the right heart.

As the exudation increases, the upright position is assumed, with the head and shoulders thrown forward.

In the early stage, the apical beat is increased and is quite perceptible, but disappears with the presence of the effusion.

Palpation.—During the early stage, the apical beat is increased and felt in the normal position, but as the exudate appears, it becomes more feeble and is felt at a higher point and to the left, finally disappearing with the increase of the effused material.

Oppolzer taught that the apical beat changed with the position of the patient; thus if the beat had disappeared, changing the patient to the left side, or bending the body forward, would cause its return. Gerhardt, however, well says that this is not peculiar to pericarditis, as the apex beat is changed even in health by change of position.

If myocarditis accompanies the disease, the systole is greatly enfeebled, and the apex beat disappears quite early. In case of hypertrophy, or where there are old adhesions, the apex beat may be retained throughout, notwithstanding the presence of a large quantity of effused fluid.

Percussion.—The increase in dullness depends upon the amount of effusion, the dullness assuming a triangle, the base being dependent. The dullness may extend, in extreme cases, from a half inch to the right of the sternum, to the right nipple line, and as far to the left as the axillary line, and as high as the second, or, in extreme cases, to the first interspace to the left of the sternum.

Should there be old pleural adhesions confining the lungs to the anterior chest-wall, the pericardium, with its fluid, will be carried backward, and percussion in this case would give resonance, the area of dullness being diminished rather than increased.

Auscultation.—The friction sound, already described, is heard during the initial stage, but disappears with the presence of the effusion, to return again with its absorption. The heart-beat, at first strong, becomes gradually weaker as the disease progresses, and is not due, as has generally been regarded, to the increased distance from the chest-wall by the intervening fluid, but to the weakened condition of the muscular walls of the heart, due to more or less disease of the myocardium as a complication. This view is held by Shrotter, who gives, as proof, that the fetal heart-sounds are heard through a much larger quantity of amniotic fluid than ever occurs in pericarditis.

Where the fluid is small in quantity, we may hear the murmurs due to endocarditis, when this complication exists.

Diagnosis.—This disease is often overlooked, owing to the primary lesion; but if a careful examination is made, it can be recognized by the characteristic friction rub in the early stage, and the triangular area of dullness, extending in severe cases to the first interspace.

The pericardial sound is a rough, grating noise near the ear. The endocardial sound is blowing, and distant from the ear.

We recognize it from pleurisy by the absence of the sharp, lancinating, stablike pain characteristic of pleurisy, and also by the cessation of the friction sound during a momentary suspension of respiration, the friction sound continuing in pericarditis without regard to respiration; from cardiac dilatation, by the history of rheumatism, of the former, also septic or infectious diseases, and the presence of pain in the cardiac region. In cardiac dilatation there will be a history of heart disease, an absence of fever and pain, and there will be no friction sound in the latter.

Prognosis.—The prognosis in this form must be guarded; for while some cases are so mild as to pass unnoticed, others are so severe as to prove fatal in a few days. In mild cases, the disease may terminate favorably within a week or two, all evidence of inflammation

disappearing, and the effusion of serous material be entirely absorbed in the course of two or three weeks. When the disease is the result of scurvy or pyemia, death may occur in forty-eight or seventy-two hours.

The condition of the heart must also be taken into consideration in the prognosis. If the heart be in good condition—that is, if there be no structural change—the prognosis will be favorable, but just in proportion as degenerative changes take place' will the outcome be unfavorable.

If endocarditis complicate the disease, valvular lesions are almost sure to exist, and this always renders the disease more grave, as does dilatation of the heart.

The cause giving rise to the disease must also be taken into consideration in making a prognosis; thus septicemia, scurvy, Bright's disease, and the infectious fevers give rise to a more grave form than rheumatism.

The character of the effusion also determines to a great extent the gravity of the ca'se. Thus, if serum alone is the product, the case may be hopeful; but if it be purulent or hemorrhagic, the outlook will be unfavorable.

Treatment.—In the early stage the treatment will be the same as for the plastic form; namely, absolute quiet, and the avoidance of everything that would irritate or excite the patient. The indicated sedative, and bryonia, asclepias, macrotys, or lobelia will be given, as the case may require. Spigelia will be useful during this stage, where there is a sharp, stabbing pain, accompanied by great oppression and undue anxiety. As the disease progresses and the effusion becomes more pronounced, we rely upon such remedies as give tone to the overburdened heart and stimulate the absorbents to carry off the accumulated fluid. Strophanthus influences the heart favorably when given in the small dose, and at the same time excites the kidneys to greater secretion. To a half glass of water add ten or twenty drops. of the tincture, and give a teaspoonful every one, two, or three hours.

Apocynum.—Of the many remedies recommended for cardiac troubles, however, I know of no remedy in the materia medica equal to that of apocynum, especially with effusion in the pericardium. The action is

similar to that of digitalis, but it is not cumulative. The cardiac impulse grows stronger, the dyspnea becomes less, palpitation disappears, and, through its influence on the kidneys, diuresis is greatly increased. If given in large doses, it produces copious watery stools, and, where the patient is not feeble, this action is not undesirable. If the specific tincture be used, add from ten to thirty drops to a half glass of water, and give a teaspoonful every hour. If you do not secure satisfactory results from this, then use the decoction, made from the fresh root. To two ounces of the crushed root, add ten ounces of water, and reduce one-half; two hours or more should be occupied in its preparation. Strain and add sufficient alcohol or glycerin to prevent fermentation; of this, commence with five drops, gradually increasing the dose as the stomach will tolerate it, till you reach the maximum dose, one teaspoonful, which may be given every four or five hours. Some patients can not take over ten drops at a dose, the remedy being exceedingly bitter and somewhat nauseating. In dropsies of the heart it has no superior, and I very much question if it has its equal.

Apocynum is also an anti-rheumatic, and where there is edema of the joints it is doubly indicated.

Digitalis.—Where the heart's action is very rapid and feeble, digitalis in the small dose will give good results.

Where rheumatism has been a marked feature in the case, and if there be muscular soreness, macrotys should be used. The early Eclectics obtained great results from the decoction, and where the fresh root can be secured it will often give better results than the tincture, though in most cases the tincture will not disappoint. The dose of the latter will be twenty to thirty drops in a half a glass of water, a teaspoonful every two, three, or four hours.

Cactus.—Where the heart's action is feeble, cactus should be given. This may be alternated with any of the above-mentioned remedies. Where the effusion is very great, causing great dyspnea, and the patient takes on a cyanotic appearance, para-centesis should at once be performed. The fourth interspace near the sternum is the point to be selected. If the patient be very feeble, not more than two or three ounces should be removed at the first operation, more being withdrawn a few days later.

If the effused fluid be of a purulent character, echinacea, baptisia, the

chlorates or mineral acids, would be used according to special conditions as expressed by the tongue. Should the effused material be especially offensive, free incision has been recommended and a free drainage established.

The patient should be given nourishing food. though fluids must be restricted to as small amount as is compatible with health. During convalescence, the patient should avoid anything of an exciting nature, and be careful not to do anything that will overtax the heart.

ADHESIVE-PERICARDITIS.

Synonym.—Chronic Pericarditis.

Etiology.—The disease may follow an acute attack, the effused material not being completely absorbed, and the sticky, gluey residue becomes organized, and adhesions follow. Again the disease comes on so insidiously that it is never suspected, even though extensive adhesions have taken place, and they are only revealed during an autopsy. In the latter case, no history of an acute attack can be discovered, and the disease becomes chronic from repeated irritations, probably due to rheumatic attacks, or it' may be the result of tuberculosis or malignant growths.

Pathology.—The amount of adhesions varies, depending largely upon the character of the inflammation. In some it is very slight, while other cases present a total obliteration of the cavity, and between these extremes are every gradation of organization.

When tuberculosis or malignant growths involve the heart, the thickening is quite extreme. In the rarer case the pericardium undergoes calcification, or even ossification. This follows more often when the effusion has been purulent in character.

Drummond reported a case of extreme calcification of the heart in a sailor aged forty-three, who was able to perform his work up to a few weeks before his death. On autopsy, it was found that the pericardial sac was nearly obliterated, and the pericardial layers were extensively calcified.

The process of calcification had extended so as to involve the heart muscles, in which had developed great bonelike plates, which had to be sawn through, and which had a thickness, in spots, of an inch. The whole posterior surface of the right ventricle was composed of a triangular chalk-plate three inches high and three and one-half inches broad at the base. A thick, bone-like mass ran across the whole left ventricle, penetrating the entire wall of the heart like a wedge, and reaching into the cavity of the left ventricle. ("Twentieth Century Practice.")

Symptoms.—The symptoms are obscure in many cases, and not sufficiently pronounced to attract attention, the disease not being suspected during life, and only revealed post-mortem while searching for other conditions. Where there is extensive thickening or calcification of the pericardium, the circulation is more or less obstructed and attended by precordial oppression and a sense of constriction and dyspnea. The pulse is rapid, feeble, and irregular, and of low tension, known as the *pulsus paradoxus*.

The free movement of the heart is prevented where the adhesions are marked; hence hypertrophic dilatation of its chambers is a frequent sequence.

Physical Signs.—Since the general symptoms in many cases are insufficient to draw the attention of the physician to the patient's true condition, the attendant should make a physical examination of all of his chronic cases, especially those with a history of rheumatism, for quite definite knowledge may be thus gained.

Inspection.—As a result of the adherent pericardium, there will be a sunken or depressed condition in the intercostal spaces and over the precordial region. One of the most common as well as most valuable signs is "the systolic tug," which occurs with each pulsation and may be seen near the sternum, between the seventh and eighth interspace.

On examining the back of the patient a visible retraction of the chest will be observed between the eleventh and twelfth ribs, during each systole. This is known as Broadbent's sign.

If dilatation has not taken place, the apex-beat is visible over a much larger area than normal; but after dilatation, owing to its enfeebled

condition, the apex beat can not be seen.

Palpation.—The apex beat remains in a fixed area notwithstanding a change of position. One of the most reliable physical signs is the diastolic rebound or shock following the drawing in of the anterior chest-wall during each systole. This rapid rebounding of the chest-wall may suddenly empty the jugular veins, giving rise to the diastolic collapse, or Friedreich's sign; this, however, may also occur in cardiac dilatation without adhesions.

Percussion.—In a majority of cases of pericarditis there are adhesions between the pleura and pericardium, thus preventing an overlapping of the heart by the lung; this accounts for the increased area of dullness upward and to the left; this, however, is not pathognomonic, as we have a large area of dullness where the pleura is adhered to the chest-wall, and no cardiac lesion.

Auscultation.—The sounds heard on auscultation are variable, and, on the whole, not very reliable, since similar murmurs are heard in other cardiac affections. Before dilatation takes place, no murmurs are to be heard, but with the gradual dilatation the murmurs begin, increasing in intensity with the progressive increase of the cavities.

Diagnosis.—We are to distinguish this from pericarditis with effusion, and also from simple hypertrophic dilatation. We recognize it from the former by the fixed apex beat not being influenced by change of position, by the concave or depressed precordial region, while in the latter there is convexity or bulging of the intercostal spaces. The diastolic shock or rebound is absent where there is effusion. In simple hypertrophic dilatation the murmurs are almost identical, but the apex beat is not so circumscribed and there is no depression of the interspaces.

Prognosis.—The compensatory changes that take place in hypertrophy of the heart establish a harmonious balance of forces, and the patient, if not overtaxed, is comparatively comfortable, and may live for years after there are pronounced adhesions. In course of time, however, myocardial degeneration takes place, and if this be accompanied by dilatation, a sudden termination need cause no surprise. The disease is very chronic in its character.

Treatment.—The physician is rarely ever consulted in the early stages of the disease, or the diagnosis is not made till such organic changes have taken place that a radical cure is almost out of the question, and the best the physician may hope to accomplish, is to stay further organic changes, relieve such unpleasant complications as may arise, and render the patient as comfortable as possible.

Where the patient is able to profit by such advice, we would recommend for residence a climate where the air is dry, pure, and where there is abundant sunshine and equable temperature; where the patient can live in the open air the greater part of his time with the least expenditure of vital force. He should avoid all excitement and such exercise as would prove exhausting or overtax the heart. The diet should be nutritious and easily digested.

Cactus, digitalis, cratsegius, strophanthus, and like remedies, will be administered as they may be needed. Cactus, especially, will be a good remedy to continue indefinitely, three or four doses per day. Iodide of arsenic, 2x, may be given, with the possible hope that it may assist in the absorption of fibroid deposits, but too much dependence should not be placed in drugs to accomplish this end.

After dilatation becomes marked, cactus, digitalis, and strychnia will be used to support the heart's action, and the patient must be kept very quiet.

HYDROPERICARDIUM.

Synonym.—Dropsy of the Pericardium.

Definition.—Hydropericardium is a non-inflammatory condition of the pericardium, attended by an accumulation of sero-albuminous fluid.

Etiology.—Hydropericardium is generally the result of a retarded or interrupted circulation, and is usually an accompaniment of ascites or general dropsy, which is frequently seen in the last stages of chronic nephritis, or old valvular troubles. It also follows scarlatina, nephritis, tuberculosis, cancer, diabetes, degenerative changes in spleen and liver, Addison's disease, and all conditions leading to marasmus.

It may be due to local causes obstructing the circulation of the coronary vessels, to mediastinal tumors, to aneurism, or to thrombus of the cardiac veins. In fact, any disease that gives rise to the formation of fluid in the cavities may be attended by dropsy of the pericardium.

Pathology.—Hydropericardium is not a disease of itself, but is always secondary; hence a varied condition is presented. It may be, there will be structural disease of the kidney, spleen, liver, or heart itself; frequent valvular lesions are found. The pericardium itself remains unchanged.

The accumulated fluid is usually clear, of an amber color, though it may become turbid by the presence of fibrin or red blood corpuscles. It is alkaline in reaction.

Symptoms.—Being a secondary lesion, and coming on insidiously, we find no pronounced or characteristic symptoms other than dyspnea. If the transudation commences in the extremities, gradually invades the peritoneum and the pleura, dropsy of the heart undoubtedly prevails, and the dyspnea is the symptom that confirms the condition. There may be a sympathetic cough due to pressure from the distended pericardium. The physical signs, excepting the friction sound, are the same as in pericarditis.

Inspection.—As a rule, inspection fails to throw any light upon the case: in fact, there are no very reliable physical signs, other than those of pericarditis. The pulse depends upon the condition of the heart, though it is generally small and frequent. If there is excessive distention, the patient will have attacks of dizziness or vertigo. The secretion from the kidneys is scanty, turbid, and presents heavy deposits.

Diagnosis.—The distinction between pericarditis with effusion and hydropericardium is not always easy. If we remember, however, that pericarditis is more of an acute disease, is attended with more or less pain, has the characteristic friction sound, and has a history of being preceded by some one of the infectious fevers, rheumatism, pleurisy, and tuberculosis, the diagnosis becomes more easy.

Prognosis.—The prognosis depends almost altogether upon the disease that gives rise to it. If the heart is in good condition, there being no structural change in its valves, orifices, or muscular tissues, the prognosis is quite favorable. If, however, the process of degeneration

has taken place in kidney, liver, lung, or spleen, and these are followed by dropsy, the outlook will be unfavorable, even though the heart be in fair condition. Some very severe cases, however, even when the transudation is excessive and is found in all the tissues, make happy and permanent recoveries.

Treatment.—To one who has never used apocynum in hydropericardium, a pleasant surprise awaits him. It is the remedy par excellence. Under its administration the secretion from the kidneys is largely increased, the stools become watery, and the fluid from all the tissues rapidly subsides, the dyspnea is relieved, and the heart's action improves.

Where the remedy can not be retained, the compound officinal infusion of digitalis is a good remedy. Convallaria may be used in combination or alternation, and will greatly add to its efficiency. Cactus adds tone to the heart, and should not be overlooked in this disease.

Where there is severe structural change in any of the important viscera, the treatment will simply consist in using such remedies as will aid the heart and at the same time, as far as possible, carry off the fluid. Where the accumulation is extreme and the dyspnea great, we should perform paracentesis as recommended in pericarditis with effusion.

HEMOPERICARDIUM.

Definition.—Hemopericardium is an infiltration of blood into the pericardium.

Etiology.—This is entirely distinct from the hemorrhagic effusion that occurs in the course of tubercular, cancerous, and cachectic pericarditis, and is most frequently the result of a rupture of an aneurism of the aorta or coronary arteries, and in very rare cases from rupture of the heart. It may also arise from injuries such as bullet wounds, fracture of the ribs, sternum, etc.

Symptoms.—The symptoms vary, and depend altogether upon the exciting cause. Where hemopericardium is the result of a rupture of the myocardium or an aneurism of the aorta, the patient is suddenly seized with excruciating pain, a deathly pallor or dusky hue overspreads the

face, and the patient dies quite suddenly. When due to rupture of a small aneurism of the coronary artery or one of its branches, and but a small quantity of blood finds its way into the pericardium, the results are not so serious, though it is attended by dyspnea and a sense of fullness in the pericardial region. If the infiltration continues, the distention becomes extreme, the dyspnea painful to observe, the pulse weak, and the patient dies of exhaustion. The physical signs are the same as in hydropericardium.

Diagnosis.—The diagnosis is often made only during an autopsy. When a person is known to have had an aortic aneurism, or a myocarditis, and is suddenly seized with excruciating pain, becomes pallid, followed by collapse and death, hemopericardium may be suspected. When the patient has suffered violence by a knife-stab, bullet wound, etc., the diagnosis is not so difficult.

Prognosis.—The prognosis is almost always unfavorable, though aspiration, followed by judicious treatment, has resulted favorably in a few cases.

Treatment.—Stimulants, such as strychnia, nitroglycerine, and camphor and ether, hypodermically to support the heart, will be used, and absolute quiet and rest must be enjoined. If the distention is great, aspiration should not be delayed. Eichhorst reports a case where he averted death by this measure. Should the patient survive the first few days, the treatment would be symptomatic, the remedies used in hydropericardium and pericarditis being chiefly used.

Aspiration might be followed by the introduction of a pint of normal saline solution directly into the circulation, with the hope that its effects would prove of permanent value.

PNEUMO-PERICARDIUM.

Definition.—Pneumo-pericardium is an accumulation of air in the pericardium.

Etiology.—The presence of air or gas in the pericardium is a rare disease, and occurs by the establishment of communication with the air, either through diseased processes, such as cancerous or tubercular

ulceration or through injuries; thus a ruptured pulmonary cavity might result in this condition, or the perforation of the esophagus by malignant processes would give rise to this lesion. Sometimes pus in the pericardium will generate gas, and consequently be a causal factor.

Pathology.—Pneumo-pericardium, pure and simple, seldom, if ever, occurs, and is a combination of air and pus, air and serum, or air and blood, and should properly be termed pyo-pneumo-pericardium, seropneumo-pericardium, and hemo-pneumo-pericardium. Not infrequently pericarditis is set up as a complication.

Symptoms.—The symptoms of this affection are similar to those of pericarditis with effusion, and can only be distinguished from the latter by the physical signs. The pericardium is distended, and percussion reveals dullness and resonance according to the amount of air and fluid present. These sounds change with change of position.

Auscultation reveals a splashing or metallic sound, due to the movements of the heart in the fluid and gaseous contents of the pericardium. These are quite pronounced, and sometimes may be heard without placing the ear to the chest.

If pericarditis exists, the friction sound may also be heard. The pulse is weak and the dyspnea is a marked feature. The disease is usually of short duration, terminating generally in death.

Diagnosis.—By careful attention to the physical signs already mentioned, the condition can usually be recognized.

Prognosis.—The prognosis is nearly always unfavorable, owing to the nature of the disease giving rise to it.

Treatment.—The treatment will be about the same as that recommended for pericarditis and hemopericardium, though but little hope may be entertained in this disease. The pericardium should be punctured with an aspirating needle or small trocar, which will give temporary relief.

CHYLO-PERICARDIUM.

Cases have been reported where, owing to a rupture of the lacteal vessels, an accumulation of chyle takes place in the pericardium. The physical signs show an increased dullness, but the diagnosis could only be made post-mortem.

II. DISEASES OF THE HEART.

ENDOCARDITIS.

Definition.—Endocarditis is an inflammation of the lining membrane of the heart, and is generally confined to the valves, though other parts may be affected.

Varieties.—There are two varieties of endocarditis, acute and chronic; the former being again divided into acute proper, and ulcerative or malignant.

SIMPLE ACUTE ENDOCARDITIS.

Synonym.—Endocarditis Verrucosa.

Definition.—Simple acute endocarditis is an inflammation of the endocardium, characterized by the formation of small, bead-like excrescences on the margin of the valves.

Etiology.—Endocarditis is rarely, if ever, a primary affection, and where there is apparently no antecedent lesion to account for it, there is in all probability an acid or toxin that has not given rise to any marked lesion, yet has existed in latent form.

Rheumatism stands first as a causative factor. Pepper says it is the cause in from 60 to 85 per cent of cases examined.

Pneumonia is also a fruitful source of this affection; so is Bright's disease and the infectious fevers, scarlatina especially; but in measles, diphtheria, and typhoid fever, endocarditis is rare. Chorea and tonsillitis, when of a severe type, have also been found to precede this

affection.

Stengler and Wegheim each records gonorrhoea as an important factor in producing endocarditis, though this and the septic fevers are more likely to result in the ulcerative or malignant forms. Syphilis may also give rise to endocarditis.

Pathology.—The morbid changes are, first, a reddened and injected appearance of the endothelium, which soon becomes opaque and swollen from congestion of the small blood-vessels. This swelling or thickening of the membrane furnishes a favorable resting-place for deposits of fibrin, and we have small, beady deposits from the size of a pin-point to that of a pea, or even larger. These small, beady excrescences may become detached, and, floating off in the general current, give rise to embolism in distant parts; viz., the brain, kidneys, or spleen; and, as a result of this, we may have hemorrhagic infarction of these organs.

The inflammation is mostly confined to the valves, the mitral being far more frequently involved, the aortic following next.

Osier gives an estimate of the frequency with which, in one hundred and eighty-seven cases, different parts of the heart were affected, as follows: Aortic valves, 53; mitral valves, 77; tri-cuspid valves, 19; the pulmonary valves, 15; and the heart-walls, 33. The left heart is most constantly affected in the adult; the right in fetal endocarditis; the reason, as explained by Anders, is that before birth the right side, and after birth the left side, are the most active, and that this increased activity accounts for the location of the inflammation. The lesion may not be confined to the valves, but include the endothelial lining of the cavities, and also the chordae tendineae.

When resolution takes place, the excrescences are gradually absorbed, though there is apt to remain some thickening of the tissue. As a result of the inflammatory process, there is nearly always more or less myocarditis, and in severer cases the pericardium will share in the general ravages.

Symptoms.—Perhaps in the whole range of heart affections there are few as well-defined subjective symptoms present as in endocarditis. The disease comes on so insidiously that its presence is confirmed before it is

recognized, or possibly never is known unless determined by an autopsy.

The symptoms commonly given—pain in the precordial region extending from the left nipple to the back and down the left arm, palpitation, and dyspnea—may occur in pericarditis or myocarditis, or they may be entirely absent. However, if the patient is suffering from rheumatism, and there is an increase in fever, rapid pulse, increase of temperature, pain in the region of the heart, with dyspnea, a careful examination must be at once made for the characteristic bellows murmur. In the more aggravated cases, the patient will lie on the back, or incline to the left side. There will be distention of the veins of the neck, with marked cyanosis.

Physical Signs.—*Inspection.*—The patient is found lying on his back, or inclined to the left side. In severe forms there will be fullness of the cervical veins, with a general cyanotic appearance. The apex beat may be visibly increased, though usually not perceptible.

Palpation.—The results of inspection are confirmed by palpation. The impulse, if weak, suggests myocarditis as a complication. In some cases a systolic thrill may be recognized.

Percussion.—Percussion gives negative results in a large per cent of cases; but if complicated by myocarditis with dilatation, the area of dullness will be increased, especially in the transverse diameter.

Auscultation.—Auscultation gives us the most positive information in the blowing systolic murmur, telling us of mitral insufficiency. There may be aortic murmurs accompanying this, or a double systolic murmur over the tricuspid valves. If the endocarditis arises as a complication of chronic valvular disease, the sounds of the latter are but little, if any, changed, hence are but of little diagnostic value. We are to remember, however, that these adventitious sounds may be heard in other affections of the heart, or they may be so feeble as not to be recognizable at all.

Diagnosis.—This is a disease that is very apt to be overlooked, unless the more pronounced symptoms are present; namely, rapidity and irregularity of the heart-beat, distress in the precordial region, and dyspnea with mitral murmur. It is important, therefore, in all cases of

acute rheumatism and the infectious diseases, to make a thorough physical examination of the chest daily. If the murmur is soft and over the base of the heart, it is most likely due to anemia or to functional derangements; but if it be over the apex, and is the mitral systolic murmur, the diagnosis is quite conclusive.

To distinguish the ulcerative or malignant from the acute is often impossible, though the aggravated symptoms attending the latter enable us to recognize it from myocarditis.

Prognosis.—If no complications exist, endocarditis rarely proves fatal at the time, though it is often the beginning of permanent lesions of the valves. If the primary lesion is grave, the prognosis must be guarded, or if complicated with myocarditis or pericarditis, it will result unfavorably.

Treatment.—The prevention of endocarditis can be accomplished in many cases, if the proper anti-rheumatics are used in the primary disease. If the result of infectious fevers, rest in bed, precaution against taking cold, and the proper antiseptics, will give the minimum cases of endocarditis.

In the management of this affection, great care must be taken to secure rest and quiet. The patient should be placed between blankets, and all company, or anything that would tend to excite the patient, must be forbidden. For the excitation of the heart in the early stage, we use the direct sedative,—aconite for the small, frequent pulse, or veratrum if the pulse be full and strong. For the dyspnea, lobelia is one of our best remedies; ten to twenty drops to water four ounces.

For the pain, if there is muscular soreness, use macrotys one-half dram, to four ounces of water. If the pain is sharp and lancinating, simulating pleurisy, bryonia is the better agent: ten drops, to water four ounces. If there is puffiness of the face, swelling of the joints, with pericardial effusion, apocynum is to be given. When the heart becomes weak, cactus, digitalis, convallaria, or strophanthus may be given. Where there is great oppression or a sense of constriction of the chest, with sharp, stabbing pains, give spigelia.

Alcoholic stimulants, nitroglycerin, and strychnia are to be freely given when the heart flags. Iodide of potassium has long been given for its supposed influence in producing absorption of the vegetative growths,

but its beneficial effects have been largely magnified.

The diet should be generous, though easily digested, and a sparing use of fluids should be advised. The convalescent period should be watched very carefully, to prevent taking cold, and also to avoid any and all exercise or excitement that would produce a strain upon the weakened valves. A climate where the temperature is equable and there is plenty of sunshine, and not of too high altitude, will be the most beneficial.

ULCERATIVE ENDOCARDITIS.

Synonyms.—Malignant Endocarditis; Infectious Endocarditis; Mycotic Endocarditis; Diphtheritic Endocarditis.

Definition.—A form of endocarditis developed during some severe infectious or septic disease, and usually characterized by ulceration or suppuration of the valves. It seldom occurs as a primary affection, though a few cases have been reported.

Etiology.—One of the most frequent diseases to be followed by ulceration is pneumonia. Of two hundred and nine cases reported by Osier, twenty-five per cent were due to pneumonia; in fact, endocarditis with pulmonary lesions is very apt to be malignant in character. Rheumatism precedes this form of the disease much less frequently than in the simple form. Septicemia, puerperal fever, and the infectious fevers generally, may act as a primary cause. Tuberculosis, typhoid fever, and diphtheria are seldom accompanied or followed by this form.

Gonorrhoea more frequently precedes this form of the disease than the acute variety. Simple acute endocarditis occasionally terminates in the ulcerative form, some septic process of unknown origin having developed. Old valvular lesions are frequent factors in causing endocarditis. It is mostly a disease of middle life, few cases being found in the extremes of life.

Pathology.—As in the simple form, it is the valves that are first affected and upon which the ulcerative and suppurative process expends itself, though there is a tendency to extend to greater areas of the endocardium. The sites most frequently selected are the ventricular surface of the aortic, and the auricular face of the mitral valves, these

surfaces being subjected to the greatest friction. The relative frequency of the different valves may be seen from a report of 209 cases examined. Aortic and mitral valves together, 41; aortic valves alone, 53; mitral valves, 77; tricuspid in 19, pulmonary valves in 15, and the heart-wall in 33 instances. In 9 cases the right heart alone was involved.

The vegetative excrescences are the seat of the ulcerations. They become yellow, soft, and finally may break down, forming abscesses. The ulceration may pass deeper than the membrane, even to suppuration and sometimes perforation. As a result of the partial destruction of the valves, an acute valvular aneurism may occur, though this is rare. As a result of direct extension, purulent myocarditis or pericarditis sometimes takes place.

The secondary or distant lesions of ulcerative or malignant endocarditis are due to septic intoxication or to embolism. When due to the former, we find the spleen, liver, and kidneys enlarged and undergoing degeneration. In case of the latter the softened vegetative deposits may be washed into the blood-current as in the acute cases, with the same result.

If the mitral and aortic valves are the ones involved, the systemic circulation is poisoned and the emboli are lodged in the spleen, kidneys, brain, or cutaneous vessels, while if the tricuspid or pulmonary valves are the seat of ulceration, the lungs are the seat of infarction. Where the brain is involved in this way, meningeal lesions are found, or the deeper structures may be involved, paralysis and softening of the brain following. Various micro-organisms are found at the points of ulceration, the pus-forming kind predominating.

Symptoms.—The symptoms embrace the widest range, including all that are observed in the simple form, with the addition of all those due to intoxication and embolic complication, or they may be so obscure as not to arouse even a suspicion of heart lesion; in fact, quite a large per cent of endocardial cases are only recognized post-mortem. This being the case it is exceedingly difficult to give a satisfactory description of the symptoms of this lesion.

There may be an aggravation of all the symptoms of the primary disease, plus an irregular and frequent pulse, slight pain, and more or less dyspnea. If the patient is suffering with acute rheumatism, and

there is a sudden rise of temperature, an irregular pulse, and oppression in the precordial region, our attention should be turned to the endocardium; or if there are no local symptoms pointing to the heart, and yet a sudden rise of temperature takes place, with irregular pulse, even though there is no aggravation of the joint affection, endocarditis should be suspected.

Should there be emboli, the symptoms would depend upon their location. For example, if located in the kidney, there would be scanty secretion of urine, containing more or less blood. Where the infarcts are in the spleen, there would be severe pain in the left hypochondrium, great tenderness, and more or less peritonitis. If the meningitis develop or hemiplegia suddenly occur, followed by coma, we think of cerebral emboli; while gangrene of the lung with the accompanying pulmonary symptoms, would leave no doubt as to the cause.

Some cases resemble a remittent fever, with irregular pulse and dyspnea, as additional symptoms, directing our attention to the heart. Sometimes in chronic valvular disease, fever suddenly develops, the temperature rises rapidly, there is an anxious expression of countenance, with a sense of oppression in the cardiac region with or without pain; in such cases endocarditis is present. Aside from these varied and irregular forms of endocarditis, two special types have been recognized,—the typhoid and the septic, or pyemic.

Typhoid Type.—Should the disease come on gradually with the customary prodromal symptoms, malaise, headache, etc., the physician is very apt to mistake it for typhoid fever, especially when the above is followed by high temperature, with daily remissions, tympanites, diarrhea, and an eruption somewhat similar to that of typhoid, a delirium not unlike it, and followed by coma and picking at the bedclothes.

The tongue is dry and brown, with sordes on the teeth and lips. In these cases, the cardiac symptoms are completely overlooked, and, even if suspected, a careful examination may fail to reveal the true condition.

The *Septic Type* is apt to follow suppurative processes, like necrosis of bone, puerperal septicemia, and similar lesions. The invasion is usually sudden, and announced by a chill or rigor. The fever is of a remittent type, the temperature frequently being very high. In some cases there is

an intermittent fever, the chill being a characteristic feature. The patient takes on a cachectic appearance, has night-sweats, the tongue becomes dry and brown, the breath is foul, prostration is great, and emaciation rapid. The pulse is rapid, feeble, and sometimes irregular. When dyspnea is marked, the disease is recognized where it otherwise would be overlooked.

The disease runs a varied course, though usually a few weeks is sufficient time for a fatal termination.

Pericarditis and myocarditis are grave complications, and always add to the gravity of the disease. Pneumonia and pleurisy may complicate endocarditis, but are more apt to precede it. The physical signs differ but little from the acute form.

Diagnosis.—The diagnosis is many times extremely difficult, especially where the local symptoms are not pronounced. It may be taken for septicemia or typhoid fever, especially where the forming stage is of long duration. Usually, however, the onset is more sudden than that of typhoid, there is less engorgement of the spleen, and the rash is not the characteristic eruption of typhoid; coupled with this are the frequent chills, copious sweats, and great prostration. In nearly all cases, however, if the pulse is carefully studied, the breathing noted, and the chest carefully examined, the disease will be recognized.

Prognosis.—The prognosis is usually unfavorable, always so when of a severe type. Mild cases may recover, though such cases, when reported, may have been due to a mistaken diagnosis.

Treatment.—The treatment will be supportive and antiseptic. In the ulcerative or malignant form, the cause of the sepsis *must not be overlooked*, for if the source of the stream be poisoned, the body to which it flows must certainly partake of its character. The heart may be poisoned by an old metritis or gonorrhoea, or a foul ulcerative condition of the rectum, or there may be sepsis from some bone lesions, or tuberculosis in some one of its many forms. These wrongs *must be corrected*, for to overlook them is to court defeat.

The source of infection must be removed. The various anti-zymotics will then be indicated. Echinacea one dram, to water four ounces, or baptisia, or it may be the mineral acids, will be called for; if the tongue

be red and dry, the latter would be specific. The chlorates would take the place of acids if there be a coated tongue or fetid breath, or the sulphites, if the tongue be coated with a moist, dirty coating.

The diet should be nutritious and easily digested. The secretions from the skin, kidneys, and bowels are to be carefully looked after, in the hope of removing the waste of the tissues, and preventing the toxins from further infecting the system. During convalescence, and for a long time after, the patient should exercise great care against taking cold. Recurring endocarditis is of frequent occurrence.

Cactus to assist the heart's action and relieve some of the unpleasant features, must not be overlooked. Stimulants will be used freely when the heart flags.

CHRONIC ENDOCARDITIS.

Synonyms.—Chronic Interstitial Endocarditis; Sclerotic Endocarditis; Fibroid Endocarditis.

Definition.—A chronic inflammation of the endocardium resulting in degenerative changes in the valves and orifices.

Etiology.—In studying the causes of this condition, two classes are to be recognized, the one following acute endocarditis, the other beginning as a chronic inflammation. The great majority of valvular changes follow an attack of acute endocarditis, and all writers and observers are a unit in declaring that over 50 per cent of all such cases can be traced to acute rheumatism; especially is this true where the disease occurs in children and young adults.

A certain number of cases of acute endocarditis are found where the rheumatism was of a vague character, and not recognized as such during- life. Where there is no history of rheumatism. the acute endocarditis may have followed scarlet fever, diphtheria, measles, or pneumonia. Chorea is also responsible for the acute form in children, and which in turn is followed by chronic endocarditis. It will thus be seen that the disease, no matter what the exciting cause, precedes the chronic form in the great majority of cases.

In the second class, the organic changes are frequently the result of syphilis, malaria, chronic rheumatism, gout, and alcoholism, which is most likely due to the presence of toxins setting up an irritation which develops the disease.

Severe physical exertion, by increasing the arterial tension, provokes an irritation that is followed by endocarditis. This accounts for the number of cases of valvular heart disease found in athletes and laborers whose work necessitates unusual physical exertion, such as molders, boiler-makers, draymen, and soldiers who make long marches.

Arterial sclerosis and Bright's disease give rise to the disease in the same way; namely, by increasing the arterial tension to the point of constant irritation. Traumatism, following a severe blow or crushing injury, has been known to give rise to valvular changes.

Predisposing Causes.—*Heredity* is generally regarded as predisposing to chronic endocarditis, and the number of heart affections found in some families can hardly be ascribed to mere coincidence, but is in all probability due to the weakened constitution bequeathed to the offspring.

Age determines largely the seat of the interstitial changes. Thus fetal endocarditis affects the right side of the heart, the tricuspid valves being the seat of the disease. In children and early adult life the mitral valve suffers most, while the aortic valve is found affected most frequently in advanced age, though it may be found in early manhood if great physical exertion has been practiced.

Sex.—According to the statistics of F. J. Smith, mitral stenosis occurs more often in women than in men, the ratio being four to one. The reason given for this greater frequency is, that girls and women are more subject to chorea and rheumatism.

Pathology.—The pathological changes are generally confined to the valves, though the entire endocardium may share in the tissue change. The membrane becomes dull, opaque, and covered with an exudate, the membrane losing its elasticity. There is a proliferation of connective tissue-cells in the endothelium, and an infiltration of round cells in the sub-endothelial tissue. These products of inflammation become organized, and give rise to thickening, induration, adhesion, and

contraction; and in the advanced stage of the disease, calcification sometimes takes place. As the result of these organic changes the valves and orifices are variously affected.

The tissue changes begin on the surface, where there is the greatest pressure; thus, when the semilunar valves are affected, the primary lesion begins on the ventricular face, the Aurantian body being involved. When the auriculo-ventricular valves are involved, the auricular side is the first impressed.

As progressive changes take place peculiar results follow. At first the base of the valve is involved, to be followed by partial agglutination of the segments. As they contract, they imperfectly close the orifice and we have valvular insufficiency.

The curling of the valves in some cases is so pronounced as to leave mere stubs. These thickened valves offer obstruction to the free flow of blood, and by their failure to completely close the orifice, permit a regurgitation of blood.

When the mitral valve is the one involved, the edges sometimes become adherent, and as the thickened chordae tendineae contract, the valves are drawn into the ventricles, giving it a funnel-shaped appearance.

The more extensive the adhesions, the smaller becomes the opening, and in some cases but a small narrow slit is observed, and is known as the buttonhole slit. Similar changes may take place in the aortic valves, with very similar results. The curling and consequent shortening of the thickened segments permit regurgitation of blood, and where the lining of the orifice becomes involved, a thickened ring lessens the size of the orifice, giving rise to more or less stenosis.

The inflammation may extend a short distance into the aorta, producing sclerosis of its walls. Less rarely we find these same processes taking place at the orifice of the pulmonary artery, the semilunar valves undergoing the same change in varying degrees as the semilunar valves of the aorta.

The tricuspid valves may share in the destructive changes with varying gradation, and give rise to insufficiency of the valves, obstruction to the free flow of blood by their thickened surfaces, and permit regurgitation

of blood. These sclerotic valves may finally undergo degeneration and necrosis, following which, atheromatous ulcers may form, which in turn still undergo further change by calcification. These distorted valves are then recognized as cartilaginous or ossified valves.

The diagnosis of these changed conditions, as well as their altered function, will be studied separately under the head of Valvular Lesions.

Endocarditis, separate from valvular lesions, while very rare, may sometimes exist. This does not necessarily imply a uniform thickening, but shows a varied condition. The tissue changes may extend deep into the myocardium, to be followed by necrosis and ulceration.

III. CHRONIC VALVULAR DISEASES.

AORTIC INCOMPETENCY.

Synonyms.—Aortic Insufficiency; Aortic Regurgitation.

Definition.—Inability of the aortic valves to properly close an abnormally large aortic opening, or a change in the segments whereby they are shortened by curling of the leaflets, or by calcification.

Etiology.—The predisposing causes of aortic insufficiency are age and sex.

Age.—This is a disease largely of middle or advanced life, though an occasional case is found in early life.

Sex.—Aortic lesions prevail far more frequently in males than females, due to greater exposure, greater dissipation, and greater physical strain or exertion.

Exciting Causes.—*Congenital insufficiency* is a rare condition, and is due to malformation of the valves, owing to a fusion of two segments, most commonly those behind which the coronary arteries are given off; even this may not, for some time, give rise to incompetency; but the malformed segments invite sclerotic changes, and unless the individual leads a very quiet and careful life, sclerosis of the valve is almost sure to develop.

Acute Endocarditis.—That acute endocarditis occasionally gives rise to aortic lesions, can not be denied, though this lesion usually expends its force upon the mitral valves. When it does occur it is generally the result of necrosis followed by ulceration, and this in turn by early death.

In the young, it may be caused by rheumatic endocarditis, though more often this lesion gives rise to auriculo-ventricular changes. Acute affections, however, give the smaller per cent of cases of aortic lesions, long continued irritation from various sources being the common cause.

Strain.—The frequency with which aortic incompetency is found in strong, able-bodied men, whose occupation entailed long-continued physical exertion, called the attention of the profession to the increased tension the segments sustained during the ventricular diastole. So frequently has this lesion been found in those devoted to athletics, that it is often termed the “athlete's heart.” This probably explains the frequency with which a runner falls in a dead faint in the last spurt of a long race.

Syphilis.—That Nemesis which pursues its victim to the grave, and which saps and poisons the vitality of its victim, works its destructive influence upon the aortic valve, and though this is frequently associated with other causes, may of itself produce the lesion. Syphilis is found to a great extent among sailors and soldiers, and the frequency with which this class of men suffer from valvular troubles can not be said to be merely a coincidence.

Alcohol.—Alcohol, by raising the tension in the aortic system, plays no small part as a causal factor, by inducing sclerotic changes in the valve segments.

Uric Acid.—That uric acid has its influence in giving rise to sclerotic changes can not be doubted, since it is a well-known product of various forms of gout and chronic rheumatism, and the etiological bearing of these diseases to valvular changes, is, most likely, largely due to the presence of uric acid. In this way lead poisoning should be considered a factor in producing interstitial changes in the valves, as it favors the accumulation of uric acid.

Aortic incompetency may in rare cases be the result of dilatation of the

arch of the aorta near the valves, owing to arterial sclerosis.

An aneurism above the aortic ring would give rise to the same condition. In this case the insufficiency would be relative.

Pathology.—The change of structure in some cases is entirely in the orifice, the valves being normal, but fail to properly close the abnormally large orifice. This is known as relative insufficiency, and is generally caused by an aneurism or arteriosclerosis: according to Burke, there is a gradual enlargement of the aortic orifice from birth, when it is 20 mm. to the age of twenty-one, when it has reached 60 mm., and after remaining quiescent for twenty years, it again undergoes progressive enlargement for another thirty or forty years; yet, notwithstanding these changes, aortic insufficiency from this source is quite rare.

The valvular lesions are quite varied, and as they are progressive in character, other parts of the heart become involved, which may ultimately involve the entire organ. These changes may be noted somewhat in detail, and are as follows: The lesion may be confined to one or two of the semilunar segments, though usually all three are involved. There may be simply thickening of the valves, which render them stiff and less expansive, interfering with their rhythmic closure, and thus permitting slight regurgitation of blood.

More frequently, the segments are contracted and curled, thus imperfectly closing the orifice, the regurgitation being marked. At other times a segment will become adherent to the intima of the aorta, and the diseased segments in rare cases show laceration following a severe strain. The valves may become rigid from calcareous deposits, and appear petrified, the so-called ossification of the valves.

arteriosclerosis of the arch, or atheromatous deposits, may so obstruct the circulation of the coronary arteries that the nutrition of the heart will suffer, followed by degeneration.

Further changes in the heart are slow and progressive and due to regurgitation. The failure of the valves to properly close the orifice permits the blood to flow back into the left ventricle, and there meets the blood coming from the left auricle; as a result of this overdistention, dilatation of the left ventricle follows. The increased volume of blood in the ventricle calls for increased contractile power to expel its contents,

and this over-exertion results in compensatory hypertrophy. The hypertrophy in some cases reaches an enormous size, and is known as the cor-bovinum—beef-heart.

Dulles records a case where the heart weighed forty-eight ounces. As a result of this dilatation and hypertrophy of the left ventricle, the trabecule and papillary muscles become flattened by the intracardial pressure, the auriculo-ventricular opening becomes enlarged, and as a result the mitral valve fails to properly close the opening, and mitral insufficiency is the result. This leads to dilatation and hypertrophy of the left auricle. The result of this causes engorgement of the lungs, and finally involvement of the right heart. These compensatory changes take place very gradually and with but little effect on the general health, and if no undue or excessive work is required, the patient may live for years with but little inconvenience.

Finally, however, the changes become so marked that the heart fails to properly propel and empty its contents, and engorgement of the lungs follows, cyanosis and dropsy speedily develop, and the case terminates fatally.

Symptoms.—The symptoms include a very wide, range of phenomena, from the mildest to the most pronounced and characteristic. The disease in some cases comes on so insidiously, and the compensatory changes so gradually develop, that the heart is able, if no undue expenditure is called for, to properly perform its function, and the disease is only discovered shortly before death, or in some cases during an autopsy. Most frequently, however, local symptoms develop that draw our attention to this organ, and a careful examination reveals the lesion.

After the hypertrophy becomes marked, great mental excitement or unusual physical exertion is followed by cerebral disturbances, and the patient complains of dizziness, headache, ringing in the ears, flashes of heat, and disturbed vision.

The face may become dusky, and there is throbbing of the arteries. As a result of the cardiac disturbance, there is a sense of oppression, and dyspnea becomes more or less distressing. Pain is a frequent distressing feature, and may be intense, extending down the left arm to the fingertips, or it may be located under the shoulder-blades or in the joints, especially if rheumatism has preceded the disease.

True angina pectoris occurs more frequently in this than in any other form of valvular disease. In some cases there is marked dilatation of the peripheral vessels, which is accompanied by hot flashes and profuse and exhausting sweats, especially where there has been much disturbance of the pulmonary circulation attended by cough and hemorrhage. These cases have been in some instances mistaken for phthisis.

As the disease progresses and there is failure of compensation, all the symptoms already named are aggravated, and new phases develop. The pulmonary circulation is retarded, engorgement of the lungs follows, respiration is difficult, and cough follows with frequent hemorrhage.

The dyspnea is now a marked feature, and is quite distressing on slight exertion. As night approaches, the breathing seems to be more labored, and the patient, partly through fear and partly from a sense of suffocation, is compelled to pass the night in a chair, not being able to lie down. If there is any mitral disturbance, cyanosis becomes marked, the dyspnea becomes more severe, and dropsy develops, especially of the extremities.

The patient becomes quite anemic, which may partly account for the edema of the extremities. To add to the gravity of the case, a recurring endocarditis not infrequently develops, and would be recognized by the prostration and irregular fever developed.

Symptoms of cerebral, renal, or splenic embolism may arise, and would be recognized by paralysis, renal hemorrhage, and pain in the spleen, with more or less enlargement of the organ.

There seems to be a close relation between mental disturbances and cardiac lesions. These patients not infrequently become nervous and irritable, or melancholic and depressed. Insanity may develop towards the final termination of the disease, with suicidal tendencies, and the patient should be constantly watched after the first appearance of mental disorder.

Physical Signs.—*Inspection* will reveal a wide and extreme apex beat. This is most marked between the sixth and seventh interspace, and may extend to the anterior axillary line. In children there may be bulging of

the precordia. Throbbing of the carotids may be noticed where the pulmonary circulation is disturbed ; in fact, the temporal, brachial, and superficial vessels generally, may be seen to pulsate.

Palpation.—If the dilatation is not extensive, a heaving, forcible impulse is felt; but should the dilatation be extreme or the stage of compensation be passed, the impulse is weak and uncertain.

The pulse is characteristic, and was first described by Corrigan, and is often called the “Corrigan” pulse, the water-hammer pulse, the collapsing pulse. It is quick or jerking, and strikes the finger with force, but immediately recedes or collapses. The capillary pulse is frequently seen in aortic insufficiency, and may be noticed by brisk friction on the forehead, after which the hyperemic spot alternately blushes and turns pale.

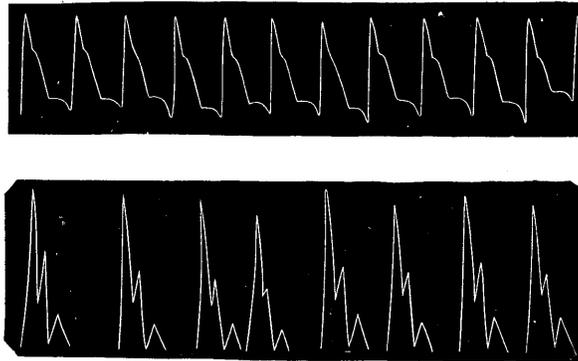


FIGURE 19. TRACINGS OF PULSE AORTIC REGURGITATION.—(Tyson.)

Percussion.—There is a greater extent of dullness in this than in any other valvular lesion, and extends as low as the eighth interspace, and to the left as far as the anterior axillary line. When the left auricle is hypertrophied, it extends upward and to the left of the sternum. If the right heart has shared in the changes, the dullness extends to the right of the sternum.

Auscultation.—The most characteristic murmur of aortic insufficiency is a prolonged, soft, and somewhat musical or loud murmur, occurring during diastole, and is heard most distinctly in the intercostal space to the left of the sternum or beneath the lower portion of the sternum, extending to the third interspace on the left. It is due to regurgitation of blood from the aorta back into the left ventricle, and is heard with the second heart-sound.

It is heard over a greater distance than any other murmur, and may extend from the lower portion of the sternum to the spinal column. In extreme cases, this murmur may be heard in the carotids, and even in the radials. In many cases there is associated with this bruit a systolic murmur, heard over the aorta, but is shorter and more harsh, and is transmitted upwards to the neck. This sound is due to the roughened condition of the sclerotic change in the aorta, and is heard during the first heart-sound. It is more pronounced over the apex, though it may be transmitted to the arteries of the neck.

In addition to these murmurs, where the mitral orifice is dilated and there is relative insufficiency of the valves, there is a second murmur at the apex, which is most likely produced at the mitral orifice, and is known as the Flint murmur, Austin Flint having called attention to it. This somewhat rumbling sound is usually presystolic, and is no doubt due to the inability of the mitral valve to close the orifice, and as a result, the valve vibrates irregularly between the cross current of blood caused by the backward flow from the ventricle, meeting the forward rush from the auricle. Sometimes a double murmur may be heard in the carotids and subclavians, and occurs at the second sound.

Diagnosis.—That many cases are only recognized during an autopsy might lead one to believe that a diagnosis of the true condition is quite difficult; however, if proper care be made in the physical examination, and the progressive changes that take place be kept in mind, the diagnosis is usually not difficult. In no other valvular derangement do we find so great a hypertrophy of the left ventricle, and such extensive dullness downwards and to the left.

Then the diastolic regurgitant murmur, prolonged, soft, and somewhat musical, is the most characteristic and appreciable of all valvular murmurs, and is heard during the second sound. The characteristic water-hammer, or Corrigan pulse, together with the throbbing carotids and temporal arteries, and the alternate blushing and pallor of the capillaries when the cutaneous surface is rubbed,—all these make the diagnosis comparatively easy. When the lesion is unattended by a bruit, and when other valvular lesions prevail, the diagnosis becomes more difficult, and sometimes impossible.

Prognosis.—Aortic insufficiency does not necessarily mean an early death, and though we may not be able to effect a radical cure, the

patient may be made to enjoy life. When the disease comes on insidiously, the compensatory changes enable the heart to do its work satisfactorily, and as long- as hypertrophy equals the valvular derangement, the health is maintained and the patient may even follow quite an active life. With proper instructions as to habits of life and the avoidance of severe physical exertion, the patient may live the allotted time of life. When due to aortic sclerosis, and when atheromatous deposits have taken place, the prognosis is not favorable.

AORTIC STENOSIS.

Definition.—Aortic stenosis is an obstruction at the aortic orifice, due to changes in the segments of the semilunar valves, or arteriosclerosis, or atheromatous deposits. Simple aortic stenosis is very rare, and is nearly always associated with more or less insufficiency.

Etiology.—The stenosis is usually due to a gradual sclerosis of the aortic valves, and this in turn by calcification. These sclerotic changes generally begin in the arch of the aorta and descend to the valves. Occasionally the thickening of the segments may be due to endocarditis, induced by rheumatism. This occurs more frequently in young subjects, older people being more prone to sclerotic changes.

The aorta may undergo these same changes, the leaflets remaining unaffected, though usually both are involved. The rigid and sometimes calcified leaflets narrow the opening, giving rise to stenosis of various grades and forms. Thus adhesions of the edges of the valves may form a funnel-shaped opening or “buttonhole” slit. The condition is sometimes congenital.

Pathology.—In addition to the changed condition of the valves and orifices, certain compensatory changes take place. To impel the volume of blood received from the auricle through the constricted aortic opening, requires increased power from the left ventricle, and, as a result of this increased work, a gradual hypertrophy follows. As long as compensation is maintained— and in some cases it is to the end—there is no further change in the heart. Finally, however, owing to the great ventricular tension, sclerotic changes occur in the mitral valve, and, compensation giving way, dilatation follows.

Sclerotic changes having already occurred in the mitral valve, mitral insufficiency follows; as a result, there are auricular dilatation, obstructed pulmonary circulation, and increased work is required, with its consequent changes of the right heart.

There are not the marked arterial changes in stenosis as in insufficiency, for the arterial walls do not receive so strong a volume after each systole; in fact, if the stenosis is extreme, a smaller amount than normal flows through the arteries. If compensation is well maintained, however, the pulse will remain normal.

Symptoms.—Since the hypertrophy of the left ventricle keeps pace with the stenosis, there may be few or no subjective symptoms for years, and the patient dies of some other affection, the heart lesion being discovered during an autopsy. As soon as compensation ceases, however, the lesion shows some of the characteristic symptoms. The left ventricle, unable to throw the proper volume of blood, anemia of the brain and peripheral parts of the body occur. This is announced by dizziness, headache, marked pallor, and sometimes fainting.

These symptoms appear upon slight exertion or undue mental disturbance. With the loss of compensation also comes a disturbance of the pulmonary circulation, which is attended by cough, embarrassed respiration, and sometimes by hemoptysis. With the general systemic circulation impaired, dropsy is frequently seen, though usually confined to the extremities.

In some cases warty or cauliflower excrescences are deposited on the valves, and these, becoming loose, float off into the bloodstream, and are conveyed to the brain, kidneys, spleen, and other organs, and give rise to embolism of these organs.

Local symptoms would suggest the organ or organs affected. The pulse is usually small, easily compressed, and does not correspond to the ventricular impulse. In some cases it is irregular or intermittent.

Physical Signs.—*Inspection.*—The information gained by inspection depends upon the stage of the disease. Thus, before compensation fails, the apex beat is gradually displaced downwards, and is heaving and forceful, not nearly so pronounced, however, as in aortic insufficiency. After compensation fails, the impulse is feeble. In old people, where the

chest-walls are firm and unresisting, the apex beat is not seen at any stage.

Palpation.—Unless emphysema be present, palpation reveals a characteristic systolic thrill more pronounced than in any other cardiac lesion, and is felt at the base of the heart and at the second intercostal space. The cardiac impulse is usually strong and heaving, though the apex beat may be imperceptible if pulmonary emphysema be present.

Percussion.—The condition of the lungs determines largely the extent of dullness; for though there is marked hypertrophy of the left ventricle, if emphysema be present, but little increase in dullness will be noticed. If not present, the dullness will be increased downwards and to the left. When compensation fails, and the rest of the heart suffers changes, the dullness is materially increased.



FIGURE 20. PULSE-TRACING OF AORTIC STENOSIS.—
ANACROTIC CURVE.—(Tyson.)

Auscultation.—A pronounced harsh, systolic murmur, sometimes musical in character, is heard with greatest intensity over the aortic cartilage, the second right intercostal space, and is transmitted to the great vessels of the neck. We are to remember, however, that the roughened aortic valves, and the sclerotic changes of the intima of the aorta, may give rise to the same sound.

If compensation has failed, and there is marked dilatation, the murmur becomes soft and indistinct. The second sound is weak, and may not be recognizable, owing to diminished blood-pressure in the aorta and the inability of the thickened valves to quickly close the orifice. As there is nearly always present aortic insufficiency, a diastolic or regurgitant murmur is associated with the aortic, giving rise to a double bruit.

Dickenson speaks of a musical murmur heard with greatest intensity in the apex region, and due most likely to regurgitation, through the altered mitral valves.

Diagnosis.—If the patient be advanced in years, and a loud, rough, or musical systolic murmur be heard at the aortic cartilage, the second right intercostal, and transmitted to the large vessels of the neck, and if

there be evidence of hypertrophy of the left ventricle, and a systolic thrill most marked at the base, with a small, quick, and sometimes irregular pulse, the diagnosis of aortic stenosis would most likely be correct.

The loud, harsh, roughened sound, not musical, that accompanies sclerotic valves, however, may be mistaken for aortic stenosis, though this murmur is not so likely to obscure the second cardiac sound as where there is stenosis. In some cases of chronic Bright's disease with ventricular hypertrophy due to aortic sclerosis, a murmur may be developed whose maximum intensity is heard over the base; the intensification of the second sound, however, together with the characteristic urinary symptoms, should enable the examiner to make a proper differentiation. We have basic murmurs in anemia, but here the systolic thrill is absent, and also hypertrophy of the left ventricle.

MITRAL INCOMPETENCY.

Synonyms.—Mitral Regurgitation; Mitral Insufficiency.

Definition.—Mitral incompetency is an imperfect closure of the auriculo-ventricular opening, permitting a regurgitation of blood during the contraction of the left ventricle, and due to an abnormal condition of the leaflets or to an enlarged opening.

Etiology.—The most frequent cause leading to change in the mitral valves, whereby the leaflets become adherent, thickened, or curled, is rheumatic endocarditis, though these same changes may occur from endocarditis due to other causes. Disease of the chordae tendineae, whereby they are forcibly contracted, or become weakened, permitting them to dip into the orifice, may give rise to insufficiency.

Alcohol and syphilis are not to be overlooked as factors producing sclerotic changes in the mitral valve. Aortic valvular changes may give rise to mitral insufficiency, through increased tension of the blood in the left ventricle.

Dilatation of the left ventricle may give rise to enlargement of the mitral orifice, the leaflets remaining whole; the insufficiency here being relative. Incompetency also may be due to ulcerative endocarditis from

the infectious fevers, in which case there may be perforation of the leaflets.

Degeneration of the muscular walls of the ventricle, either by causing such extensive dilatation as to prevent a closure of the orifice, or to so affect the muscular substance as to prevent a proper coaptation of the leaflets during the systole, will give rise to muscular incompetency. Long continued, severe physical training or extraordinary physical exertion may give rise to it.

Pathology.—The changes that are found in this, the most frequent of all valvular lesions, are varied. There will be thickening in some cases, with curling of the leaflets, while in others there will be adhesions of the segments; in another, nodulation or perforation may be seen. In some there is a change in the chordae tendineae, such as undue contraction; or the opposite condition prevails, and there is relaxation. In rare cases there is a rupture of the chordae tendineae.

Adhesions of one or both segments to the wall of the ventricle have also been found. As a result of the inability of the mitral "valves to effectually close the mitral opening following the auricular systole, there is a regurgitation of blood into the left auricle, which meets the stream coming from the pulmonary veins; as a result, there is an increased amount of blood in the auricle, which causes its dilatation. In order to expel this abnormal amount, increased work is thrown upon it, which causes hypertrophy as well. This increased volume of blood in the auricle impairs the pulmonary circulation, and the lungs become engorged.

With each contraction of the left auricle an increased quantity of blood is emptied into the left ventricle, causing dilatation and hypertrophy of the same. As a result of the pulmonary congestion, the right ventricle is not able to completely empty its contents: hence dilatation, followed in turn by hypertrophy of the right heart, takes place. Finally the right auricle passes through the same compensatory changes of dilatation and hypertrophy.

These compensatory changes take place so gradually that the patient may enjoy good health for years; for the hypertrophied heart throws the normal amount of blood into the aorta and general circulation. Finally, however, the incompetency becomes extreme, or compensation fails, and

the left ventricle is unable to properly empty itself, and, as a result, the auricle becomes greatly distended, the pulmonary circulation engorged, the right heart embarrassed, and the systemic veins congested. The result of this pulmonary engorgement causes dilatation of both arteries, and degeneration and atheromatous deposits are not uncommon.

After all this disturbance, the right ventricle heroically does its increased work, and the patient may live for months with embarrassed respiration, cough, and evidences of respiratory lesions; but ultimately the right ventricle is unequal to the task, there is aggravated insufficiency of the tricuspid valves, and general systemic congestion.

The portal circulation is affected, cerebral engorgement follows, cyanosis becomes marked, and the extremities become edematous. Cyanotic induration of liver, spleen, and other viscera takes place.

Symptoms.—Nature comes to the patient's rescue, and, by gradual and progressive changes, so fortifies the cardiac structure that a patient may live for years without the knowledge of a valvular lesion. As long as the hypertrophied ventricles are able to meet the demands made upon them, the patient suffers no inconvenience, save an embarrassed respiration after active exertion, such as going up stairs, climbing a hill, running for a car, or the many seeming necessities of every-day life.

Finally the earlier and minor symptoms develop, even while compensation remains good, and are prophetic of later changes. The face becomes slightly flushed, the lips and ears become blue, and the veins of the cheeks become slightly enlarged. Slight exertion now gives rise to dyspnea and cough, the expectoration often being tinged with blood; palpitation of the heart, with pre-cordial pain, is also often present. The disturbed respiration, the cough, the bloody, frothy sputum, may deceive both patient and physician unless a physical examination be made.

As the disease progresses, there finally comes a period when compensation is disturbed and more pronounced symptoms appear. Venous engorgement is the first evidence of this disturbed condition, and is shown in the increased cyanosis. The skin is not only cyanotic, but also jaundiced, induced by engorgement of the liver.

The dyspnea now becomes marked, owing to pulmonary congestion, and

the patient's distress is apparent. This is especially so when the patient attempts to sleep, as he suddenly awakens with a sense of suffocation and a feeling as though the heart was going to stop its work. Cough is now a prominent symptom, and the patient expectorates a frothy, bloody serum. Dropsical effusion begins first in the feet, gradually extending up the limbs, finally resulting in general anasarca.

The position of the patient is now upright, and he occupies his chair day and night. The liver and spleen become engorged; there is often gastric irritation, with catarrh of the stomach and intestines. The urine is very scanty, highly colored, albuminous, and contains tube casts; sometimes but four or six ounces of chalky, bloody urine is passed in twenty-four hours.

The patient does not often suffer acute pain, the distress mostly arising from dyspnea and his inability to lie down and obtain a good night's rest. Sudden death is a rare termination of this form of heart disease.

Physical Signs.—*Inspection.*—The apex beat is seen to be displaced downward and to the left, depending upon the extent of enlargement of the left ventricle. It may be seen as low as the sixth interspace near the axillary line. In children the precordia is prominent, even bulging, the area of the apex beat is enlarged, and, in the latter stages, is diffuse and waving.

After the right ventricle becomes dilated, we often notice epigastric pulsation. Wavy pulsations in the cervical veins also follow extreme dilatation of the right heart. As compensation fails, the lips and ears become dark and general cyanosis appears.

Palpation.—By placing the hand flat over the precordium, a systolic thrill may be felt over the apex, though this is by no means constant. It is synchronous with the first sound. The apex beat is displaced downwards and to the left, and is full and strong during the period of compensation, but: with its disturbance becomes irregular, and, later, waving and feeble.

By placing the hand over the epigastrium, after extreme dilatation of the right ventricle, epigastric pulsation is observed. The pulse is but little, if any, changed during the period of compensation, though exertion may give rise to some irregularity. When compensation fails, it

loses its impulse and becomes irregular.

Percussion.—Owing to hypertrophy of both left and right ventricles, there is a greater area of dullness in a transverse direction in mitral lesions than in any other valvular disease. It may extend from an inch or more, to the right anterior axillary line. A slight increase in dullness upward along the border of the sternum is due to hypertrophy of the left auricle.

Auscultation.—The most constant and characteristic sign of regurgitation heard on auscultation is the mitral or systolic murmur, which is heard over the apex and partly, or entirely, replaces the first sound. It is of a blowing character, sometimes terminating in a musical tone, and is transmitted to the axilla, and may be heard at the angle of the scapula; in fact, if the contraction be strong, it may be heard all over the chest.

If the contraction be weak, the murmur may be heard over the base of the heart when inaudible elsewhere. Sometimes, by changing from the erect to the recumbent position, the murmur may be heard. At times there may be heard a soft, blowing, presystolic murmur.



FIGURE 21. TRACING OF PULSE OF MITRAL INSUFFICIENCY.—(Tyson.)

Another very important sign is due to hypertrophy of the right ventricle, which causes an increased tension in the pulmonary vessels, that gives rise to the accentuated pulmonic second sound, and is heard over the third left costal cartilage.

Where there is extreme dilatation, and especially if dropsy be present, there can be heard a soft, low-pitched, systolic murmur at the ensiform cartilage and at the lower sternal region, and is due to tricuspid insufficiency. Combined murmurs may be heard of a rough or harsh character, though they are not constant.

Diagnosis.—The diagnosis of mitral insufficiency is generally comparatively easy, if we bear in mind the three characteristic signs:

First, a systolic murmur obliterating the first sound. It is of a blowing character, terminating in a musical note and heard with maximum intensity over the apex, but also transmitted to the axilla and back.

Second, accentuation of the pulmonary second sound, heard over the *third* costal cartilage.

Third, the increased *transverse* dullness extending from an inch or more to the right of the sternum to the left axillary line.

Add to these, blueness of the ears, nose, and lips, dyspnea, and more or less cough, and the picture is complete.

There are two other forms of valvular disease that may be mistaken for mitral insufficiency, since each is accompanied by a systolic murmur—*aortic stenosis* and *tricuspid incompetency*. In aortic stenosis the sound is harsh, and heard best over the base of the heart, while in mitral insufficiency it is heard at the apex.

In aortic stenosis, the area of dullness is but little increased, and that to the left, while the area of dullness in mitral insufficiency is more extensive than any other lesion. The tricuspid systolic murmur is soft and low, and heard with greatest intensity at the *base* of the ensiform cartilage.

MITRAL STENOSIS.

Definition.—Mitral stenosis is a constriction of the left auriculo-ventricular orifice, usually due to valvular endocarditis, though it may be congenital.

Etiology.—Any cause that will give rise to endocarditis should be considered a producing factor of mitral stenosis; sub-acute rheumatism being responsible for the largest per cent of cases, though chorea and chlorosis are responsible for many, and most likely explains the greater frequency of the disease in females—about four to one.

The infectious fevers, especially measles, scarlet fever, and diphtheria, are also causal factors, while whooping-cough, no doubt, gives rise to

valvular lesions from the straining of the valves induced by severe paroxysms of coughing.

It is a disease of early and middle life, rarely occurring after the age of fifty. Stenosis may be due to infiltration of the valvular ring, the valves remaining normal, though this is very rare.

Congenital stenosis has been noted, but is of very rare occurrence.

Pathology.—The morbid changes that take place in the valves, give rise, in most cases, to narrowing of the orifice. These changes consist in thickening and rigidity of the segments, and chordae tendineae, and frequently a fusion of the segments at their edges; not infrequently there are calcareous deposits. These changes convert the valves into a funnel, the base extending into the ventricle.

The degree of stenosis and shape varies. In one it will be circular, and so small as to scarcely admit a goose-quill, while in another it will be flattened, giving rise to a small, narrow slit— the “buttonhole” mitral. The funnel-shaped opening is usually found in young subjects, and rarely in old patients, while the buttonhole slit is rarely found in the young, and nearly always in elderly patients.

Sometimes there will be vegetations upon the valves, obstructing the flow of blood, and in this way the size of the opening is reduced. In nearly all of these cases there will be more or less insufficiency as well as stenosis.

The changes in the heart are as follows: Owing to the diminished opening, less blood is forced into the left ventricle, and, as a result, the ventricle atrophies, the atrophic changes at times extending into the aorta. As a result of the stenosis, the auricle is not completely emptied, hence dilatation, and, later, hypertrophy follows, thus enabling the auricle to expel the blood through the small orifice; later, there is thinning of the walls.

This dilatation and hypertrophy necessarily call for increased work from the pulmonary vessels, which, in turn, become more or less thickened. The lungs become congested, and this constant tension in the lesser circulation is followed by induration of lung-tissue, sclerosis of the vessels, and sometimes by hemorrhagic infarcts. Pepper speaks of a case

where atheromatous deposits in the pulmonary vessels were found to their remotest branches, the left lung being almost solid and airless.

The hypertrophy of the left auricle, for a time, compensates for the great resistance at the mitral orifice, and the patient suffers but little. Sooner or later, however, the auricle can not maintain this equilibrium, congestion of the lungs becomes more marked, and the right heart comes to the relief by taking on dilatation and hypertrophy, which aids the parts already enlarged for compensatory efforts, and the patient's life is still further prolonged; finally, however, the dilatation increases, the tricuspid valves are unable to effect a perfect closure, and tricuspid insufficiency is the result, with congestion of the general circulation and that cyanotic condition prophetic of a fatal issue.

When the stenosis is only moderate and there is mitral incompetency as well, there will be slight hypertrophy of the left ventricle. The apex of the heart is made up almost entirely of the hypertrophied right ventricle.

Symptoms.—The subjective symptoms during the stage of compensation are few and unreliable and may be entirely absent; when present, they are due to emotional excitement or physical exertion, such as going upstairs or performing some unusual muscular effort. The symptoms developed are the result of pulmonary congestion, and consist of dyspnea and cough, attended, at first, by expectoration of frothy mucus, which becomes bloodstained later on.

In the early stage, before compensation gives way, there is evidence of a defective blood-supply to the brain, which is seen in the pallor of the face and mucous membranes. Owing to the small amount of blood thrown into the left ventricle, the pulse becomes small, frequent, and irregular. A sharp stitchlike pain is frequently present in the apex region, and undue exertion may be attended by hemoptysis.

After compensation gives way, the symptoms become quite pronounced, and are almost identical with those of mitral insufficiency, for in a large number of cases the lesion is a combined one. Owing to pulmonary obstruction, the dyspnea becomes constant, and is greatly exaggerated on exertion. The face now becomes dusky, and the veins of the neck are distended and pulsation is visible. The liver becomes swollen, with thickening of the bile-ducts, causing jaundice, more or less pronounced.

The urine becomes scanty, high-colored, and contains albumen. Dropsical effusion begins in the feet, rapidly extending up the limbs to the body, general dropsy resulting.

If vegetative deposits become loosened, they may float off into the general current, and give rise to embolism of the brain, kidney, or spleen, in which case local symptoms would determine the seat of disturbance.

Physical Signs.—*Inspection.*—If there be exaggerated hypertrophy of the right ventricle, there will be undue prominence, especially in children, over the lower half of the sternum and the fifth and sixth costal cartilages, otherwise the chest remains normal. The apex beat may be seen in the normal position, though chiefly over the lower sternum and adjacent cartilage.

Increased tension of the pulmonary artery will frequently be shown by a visible pulsation in the second, and sometimes the third and fourth, intercostal spaces. Epigastric pulsation is seen when there is great congestion of the liver. When compensation fails, the impulse of the heart becomes weak, and sometimes can not be seen, though pulsation may be seen in the enlarged veins of the neck.

Palpation.—A presystolic thrill, harsh or grating in character, and terminating in a sudden shock, synchronous with the pulse, is pathognomonic of mitral stenosis. It is best heard during expiration, and over the third and fourth interspaces. When absent, it may be made to appear by rapidly clapping the hands over the head.

The apex beat is felt more forcibly over the lower sternum and over the third and fourth interspaces. Epigastric pulsation is often pronounced, especially when there is enlargement of the liver. The pulse, owing to the weak impulse of the left ventricle, is small though regular during the period of compensation. When compensation fails, the pulse becomes irregular.

Percussion.—Where there is excessive hypertrophy of the right ventricle dullness may extend to the right nipple line. If the left auricle be greatly dilated, there will be extension of dullness to the left of the sternum as high as the second rib. Where there is marked pulmonary engorgement, there may be dullness over the entire left lung, and, if care be not

exercised, it may be mistaken for consolidation of phthisis. Increase in transverse dullness will suggest an associated hypertrophy of the left ventricle.

Auscultation.—In well-marked cases a presystolic murmur, prolonged and of a harsh, rumbling character, may be heard within and above the normal apex beat. The area is quite limited, and, when compensation fails, may disappear entirely. If palpation be practiced at the same time, the murmur and thrill will be found to be synchronous. The murmur terminates suddenly with a distinct shock.

The first sound, sharp and distinct, following immediately after the rumbling sound, is another characteristic symptom, and is probably due to the sudden closure of the tricuspid valve, induced by hypertrophy of the right ventricle. This sound may be present when the murmur has not been heard, or when it has disappeared. The second sound at the pulmonic cartilage is of a sharp, ringing character. These adventitious sounds are more distinct and readily heard before failure of compensation, and are of great value in determining the valvular lesion.

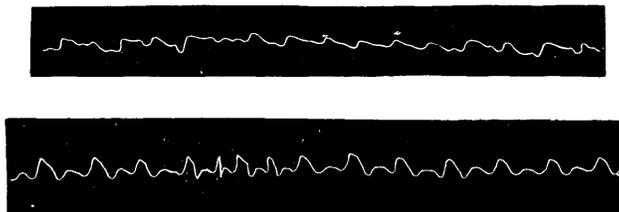


FIGURE 22. TRACINGS OF PULSE OF MITRAL STENOSIS.—(Tyson.)

After compensation fails, there is such a confusion of sounds that but little knowledge is obtained from them.

Diagnosis.—The characteristic features that point to mitral stenosis are, first, a presystolic murmur, prolonged, harsh, and rumbling, heard above yet near the apex; second, a presystolic thrill at the apex, synchronous with the murmur; third, the sharp, ringing character of the pulmonic sound; fourth, the increased dullness extending to the right of the sternum; fifth, the small, regular pulse before compensation, followed by a small, irregular pulse after compensation; sixth, wavy pulsations of the veins of the neck.

TRICUSPID INCOMPETENCY.

Synonym.—Tricuspid Regurgitation.

Definition.—Tricuspid incompetency is an imperfect closure of the tricuspid valves, due to dilatation of the right ventricle or to disease of the valves.

Etiology.—While tricuspid insufficiency may be the result of organic valvular lesion, it occurs far more frequently as the result of dilatation and is relative. Actual disease of the valves may result from fetal endocarditis. Notwithstanding the rarity of sclerosis in the valve segments, Bramwell's statistics show that fifty per cent of all cases of endocarditis are attended by tricuspid insufficiency.

It is far more common in children, and decreases with advancing years. It is generally due to mitral disease. Obstruction to the pulmonary circulation, due to chronic bronchitis, when associated with emphysema and tuberculosis of the lung or fibroid pneumonia, is a frequent cause.

Pathology.—As a result of the failure of the tricuspid valves to close the auriculo-ventricular opening, there is regurgitation into the auricle, which, meeting the great mass of blood from the venae cavae, causes congestion and enlargement of the entire venous system. Having no assistance from a fellow-member, as the left heart has, in its many efforts to overcome increased work, there can be but little compensatory changes..

The right ventricle undergoes slight hypertrophy, and for a time the pulmonary circulation is fairly maintained; but the quantity of blood from the right auricle, being abnormally large, the right ventricle becomes enormously dilated, which causes thinning of its walls. As this progresses, its power becomes feeble, and, not being able to force the blood through the pulmonary artery, the right auricle, the venae cavae, and even the peripheral veins, become greatly dilated, cyanosis is marked, and the case ends fatally.

Symptoms.—The early symptoms are generally obscured by the primary lesion. If this lesion be the result of mitral insufficiency, the symptoms accompanying it will be present long before those relating to changes of the right heart, or, if due to wrongs of the respiratory

apparatus, the symptoms due to chronic bronchitis or pulmonary lesion will mask those relating directly to the tricuspid incompetency.

When fully developed, however, they become characteristic, and are suggestive of passive congestion of the lungs and marked engorgement of the systemic veins. Dyspnea at first, after slight exertion, soon becomes more or less constant. There is frequent cough and sometimes hemorrhage from the lungs. The pulse is small and irregular. Dizziness, with dull headache, may be attributed to passive hyperemia of the brain.

There is disturbance of the gastro-intestinal functions owing to engorgement of their structures, while the liver is found to be enlarged and indurated. The spleen shares in the general congestion, and a sense of weight and fullness is experienced in the left hypochondrium. The urine is scanty, high-colored, and contains albumen.

Physical Signs.—*Inspection.*—If the tricuspid insufficiency has been preceded by mitral regurgitation, the apex beat will be seen in the normal position or slightly to the right, owing to the increased hypertrophy of the right ventricle. Epigastric pulsation, with bulging of the lower sternal region, is not uncommon.

The most characteristic and pathognomonic sign is the visible pulsations of the veins of the neck with each cardiac systole; the lower portion of the jugulars first, and later in the disease throughout their entire course. It may also be seen in the subclavian, axillary, thyroid, and mammary veins in advanced cases. To bring this out more distinctly, the patient should be requested to hold the breath a few seconds before taking a full respiration.

Palpation.—If, when the patient lies on the back with the arms raised, we place the left hand over the right mid-axillary region, and the right hand over the upper abdominal region, we get an expansile pulsation of the liver synchronous with that of the right ventricle. A systolic thrill may sometimes be felt over the right ventricle.

The pulse depends to a great extent upon valvular lesions of the left heart which have preceded the tricuspid changes, though in most cases it is frequent, feeble, and irregular. Popoff has called attention to the greater weakness of the pulse of the right wrist, due to pressure on the

innominate artery by the enlarged right auricle and venae cavae.

Percussion.—Percussion reveals dullness extending an inch or more to the right of the sternum and downwards toward the epigastrium. It may also extend to the second interspace.

Auscultation.—A systolic murmur, soft and low in character, is usually heard at the lower part of the sternum near the ensiform cartilage, though not always, if the heart be weak. There is accentuation of the pulmonic second sound in the early stage, but as the incompetency increases, it loses its sharpness, and may finally disappear.

To the skilled ear, auscultation of the lower part of the jugular vein may reveal a venous sound, due to the closure of the valve at this point. This is before the valve becomes insufficient.

Diagnosis.—The venous pulse is the most significant of all physical signs, either as seen in the neck or observed by palpating the liver. If with this, we detect a systolic murmur, whose maximum intensity is heard over the lower sternum, and percussion reveals dullness to the right of the sternum, the diagnosis is complete.

TRICUSPID STENOSIS.

Definition.—Tricuspid stenosis is an obstruction of the tricuspid opening, usually congenital, though it may be acquired.

Etiology.—This is the rarest of all heart-lesions, and one that is generally congenital. It is also rarely, if ever, found as an independent disease, being usually associated with disease of the left heart. In these combined forms, rheumatism has been found to be a causal factor. It is very much more frequent in females than in males; thus of 114 cases recorded by Leudet, 80 per cent were found in females, while of 46 cases reported by Fenwick, 38 were in women.

The frequency with which the disease is found with other valve lesions may be seen by referring to Leudet's classification of 114 cases. Thus in 11 cases, the tricuspid valve was alone involved, the tricuspid, mitral, and aortic valves in 21 cases; while the tricuspid and mitral valves were found involved in 78 cases.

Pathology.—The valves may be thickened with a partial fusion of the segments. The other morbid changes are necessarily about the same as are found in mitral stenosis and tricuspid insufficiency. There can be only one result from such obstruction; namely, dilatation of the right auricle, with engorgement of the venous system, disturbing the various viscera. The right ventricle hypertrophies, owing to increased tension in the pulmonary circulation, through mitral stenosis.

Symptoms.—The general symptoms are those of venous engorgement, and are similar to those of tricuspid insufficiency. Hemorrhoids, enlargement of the liver, and cerebral congestion follow of necessity, with dropsy following as the engorgement increases.

Physical Signs.—*Inspection.*—Slight presystolic pulsation is seen in the jugulars and general cyanosis is pronounced.

Palpation.—Over the right ventricle may be noticed a presystolic thrill.

Percussion.—Dullness extending to the right of the sternum, due to the enlarged auricle, will be found to be characteristic.

Auscultation.—A presystolic murmur may be heard over the lower part of the sternum, terminating in a sharp first sound, if the case be uncomplicated; but in the very great number of cases there is such a confusion and combination of murmurs owing to the various complications, that there can not be said to be any characteristic murmurs.

Diagnosis.—Owing to the complications or associated valvular lesions, the differential diagnosis between stenosis and insufficiency is almost impossible.

PULMONARY INCOMPETENCY.

Synonym.—Pulmonary Insufficiency.

Definition.—Pulmonary incompetency is an imperfect closure of the pulmonary orifice of the right ventricle, due to a change in the pulmonary valves.

Etiology.—The disease is rare and usually congenital, though it may result from endocarditis, followed by similar changes that take place in other valvular diseases due to the same source; namely, sclerosis and adhesion of the segments.

Pathology.—In addition to thickening of the valves or partial union of the segments, we have hypertrophy and dilatation of the right ventricle, which, in turn, is followed by tricuspid insufficiency.

Symptoms.—There are no general symptoms that are characteristic, and the physical signs are not reliable. Although a diastolic murmur may be heard in the second right intercostal space and transmitted to the lower sternal region, it is difficult, if not impossible, to separate this from the murmur of aortic regurgitation.

Diagnosis.—The diagnosis is generally made during an autopsy.

PULMONARY STENOSIS.

Definition.—Pulmonary stenosis is an obstruction of the pulmonary opening of the right ventricle, due to congenital defects or to endocarditis.

Etiology.—The narrowing of the pulmonary orifice is generally due to congenital malformation, though endocarditis after birth may occasionally give rise to it.

Pathology.—There is generally fusion of the valve segments, leaving but a small opening, the valves not infrequently being covered with vegetations. Hypertrophy of the right ventricle naturally follows, to compensate the closure and maintain the pulmonary circulation.

Symptoms.—As in pulmonary insufficiency, the general symptoms are obscure and the physical signs uncertain. A systolic murmur, attended with a thrill, may be heard in the second right intercostal space to the left of the sternum. Other pulmonic murmurs, however, are to be heard, and the distinction is often not clear. The murmur of aortic stenosis, however, may be excluded, for it is transmitted to the cervical vessels, while the aforesaid systolic murmur is not.

Diagnosis.—As in pulmonary insufficiency, a positive diagnosis can only be made post-mortem.

COMBINED VALVULAR LESIONS.

In the majority of all cases of valvular diseases, combined murmurs or lesions occur after a few months or years. When a valve or orifice becomes affected, increased work is thrown upon some other part, and this naturally brings about additional changes. There may be a double lesion of a single valve, as stenosis and insufficiency; or there may be a single or double lesion of two or more valves. The combination most frequently found is mitral regurgitation with aortic regurgitation and obstruction. Mitral stenosis with regurgitation comes next in order, other combinations being of rarer occurrence.

Many of these combinations are compensatory in character, and really prove beneficial to the patient, being the only way nature has of coming to the rescue and preventing a fatal issue. A positive ante-mortem diagnosis is many times impossible, owing to a combination of murmurs and thrills, though generally some one murmur is more prominent and distinct, directing attention to the chief lesion, and associated murmurs will suggest the additional wrongs.

The state of the auricles and ventricles will be determined by the amount of hypertrophy and dilatation.

Prognosis in Chronic Valvular Diseases.—In making a prognosis, several factors are to be taken into consideration, such as age, sex, constitutional vices or defects, previous diseases, parts affected, etc. We are to remember that valvular diseases of the heart do not necessarily mean either a sudden or early death, many patients having lived to a good age and having died from some other disease, the cardiac lesion having been discovered during an autopsy.

When a patient is able to lead a quiet life, avoiding any severe exertion or strain upon the heart, and anything that would give rise to great emotional excitement, the valve lesion should not materially shorten life.

When the nutrition of the heart begins to fail and compensation is

disturbed and overcome, the prognosis is unfavorable, as death occurs sooner or later.

Osler says: "When the apex beat is in the normal situation and the rhythm is regular, the auscultatory phenomena may be practically disregarded."

Valvular lesions in children are more unfavorable than when occurring later in life.

The fact that women do not lead such strenuous lives as men makes the prognosis more favorable for the fair sex.

Valves Affected.—Aortic insufficiency is the most serious, while mitral insufficiency is the most favorable.

Previous Diseases.—Infectious fevers, where they have been of a malignant character, predispose to more serious lesions, than where there has been an entire absence of infection. Acute rheumatism, followed by the chronic form, also makes the lesion more serious. Tuberculosis, Bright's disease, and kindred lesions also render the disease unfavorable.

Treatment of Valvular Lesions.—There is no class of diseases that needs a more careful study with reference to treatment than chronic heart diseases. Recognizing some of the most prominent and common causes that lead to valvular lesion, the physician may, if thoughtful, either prevent or lessen their severity. Prophylaxis, then, will be first considered.

If there is a family history of heart disease, it is incumbent on the physician to outline a course of living that will not overtax the heart. While the patient should live in the open air and take well-directed exercise, any labor or exertion that would produce undue strain should be forbidden. The use of tobacco and alcohol should be absolutely restricted, and high altitudes avoided.

If rheumatism attacks such patients, great care should be taken during convalescence to prevent cold, and not overtax the heart. The patient should rest in bed for a week or two after all evidence of rheumatism has disappeared. The same precautions should be taken following the

infectious fevers.

In the selection of medicines great care should be taken to select the right remedy. There is no better field for the display of specific medication than in heart troubles, and I think there can be little doubt that many cardiac affections have failed to recover because the right remedy was not selected.

Digitalis has been the David selected to slay this Goliath of disease, and he is marched to the front for all cardiac diseases. The remedy certainly is a good one in some cases, but to expect one remedy to act as a stimulant in one case, sedative in another, tonic in a third, and, in fact, correct every diseased condition the heart is subject to, is certainly asking too much of it. Yet the almost universal treatment as soon as a diagnosis has been made is digitalis. The selection of the right remedy will be determined somewhat upon the stage of the disease, whether it is the period of compensation or non-compensation.

Stage of Compensation.—Unless of a very nervous or excitable temperament, the patient should be made to understand his true condition, that he may the more readily carry out the restrictions placed upon him; namely, to avoid severe physical exertion and great mental excitement, and to abstain from the use of coffee, tea, spirits, and tobacco, to avoid overloading the stomach, and to observe regular habits.

In some cases there is excessive force or power of the heart, as shown by the full, bounding pulse. This excess in power, if not overcome, will hasten the period of non-compensation, and should be controlled.

Veratrum acts nicely in such cases, but its action must be carefully watched, and as soon as the pulse begins to come under its influence, lessen the dose. The indication is the full, bounding' pulse.

Crataegus Oxyacantha.—This remedy has recently been very highly extolled in valvular troubles, some even claiming for the remedy wonderful solvent power in crustaceous and calcareous deposits on valves and in the lumen of arteries. While these claims no doubt are extravagant, it undoubtedly is a very good remedy in improving cardiac functions, and does lessen valvular deposits. It does not overstimulate, and thus is highly beneficial during this stage. It should be given in

from three to ten drop doses.

Cactus, when given in the small dose, is also a good remedy during this period, assisting to maintain the tone of the organ. Care must be taken not to overstimulate the heart and thus hasten its exhaustion, which is followed by dilatation. Whatever remedy is used, too early results must not be expected, but the remedy should be given a fair trial.

The diet should receive special consideration. Nourishing but easily digested food should be taken in moderate quantities. Fluids should be restricted to the smallest quantity compatible with health.

Stage of Non-Compensation.—This stage is recognized by dyspnea, nocturnal seizures of shortness of breath, and irregular action of the heart. It is the evidence of heart exhaustion to be followed by dilatation. Perfect rest in bed is of absolute necessity, the patient occupying the recumbent position. This measure alone will prove greatly beneficial, and may for a short time restore compensation. The diet should be nutritious and highly concentrated, and given in liberal quantities. All excitement is to be avoided; hence unnecessary conversation should be prohibited.

Digitalis.—As a stimulant to the overburdened heart, digitalis will be found one of the best remedies. It rests the heart by prolonging the asystole, thus allowing- the chambers to become better filled with blood. When dropsy is present, an infusion of the leaves, one or two drams to eight ounces of boiling water, of which one tablespoonful may be given every two hours, will give speedy results. It adds tone to the heart, and at the same time stimulates the kidneys.

Cactus may also be used during this stage as a heart-tonic. *Convallaria* and *strophanthus* have a similar action to digitalis, and are not so apt to disturb the stomach.

Apocynum will be very useful when dropsy is present for it not only adds tone to the weakened heart, but stimulates the kidneys and bowels to increased action, thus relieving the distended blood-vessels. Under its administration the dyspnea is greatly relieved. Should rheumatism be present, the anti-rheumatics, *bryonia*, *macrotys*, *rhamnus Californica*, *apocynum*, etc., should be given according to indications.

Carduus Marianus is highly extolled by the homeopaths, especially where the liver is involved, and should be tested.

Gastric complications will be relieved by *nux vomica*, *rhus tox.*, bismuth, and mint-water, and by regulating the quantity of nourishment taken. The bowels should not be allowed to become constipated. Hepatic congestion may be somewhat relieved by *uvedalia*, *leptandra*, *Podophyllin*, and *carduus marianus*.

Should there be hemorrhage and expectoration, much relief may be experienced from it, and unless profuse, needs no attention. For the pain, which is sometimes very severe, *crataegus*, *echinacea*, and *bryonia* should be tried before resorting to *morphia*. When the patient is unable to sleep, *passiflora* may give the desired result, or *trional* in ten-grain doses may be given. The old diaphoretic powder, in five or ten-grain doses, often gives relief. When absolutely required, *morphia* may be given.

Should compensation be restored, the patient should be carefully watched and medicated for several months. In no case should the patient be allowed to undergo physical or mental strain. Light exercise in the open air, and a good, liberal, nourishing, but easily digested diet recommended. Change of climate to one, where there is the least call for expenditure of force, is to be advised.

CARDIAC THROMBOSIS.

Definition.—Cardiac Thrombosis is the formation of blood-clots in the cavities of the heart.

Etiology.—Though alteration in the integrity of the blood may have some influence in bringing about cardiac thrombi, the chief causes undoubtedly lie in a diseased endocardium, and may occur in acute or chronic disease of this membrane. It may be due to the obstruction of a free circulation by roughened valves or mural coat, thus furnishing a resting-place for fibrous deposits. The acute diseases, most liable to prove the exciting cause are, rheumatism, pneumonia, diphtheria, pyemia, and puerperal wrongs.

Pathology.—The blood-clots are found most frequently in the right

side of the heart, in the auricular appendix more often than in the right ventricle. They vary in size, color, and consistency, and may be found from the size of a pinhead to that of a hen's-egg. If recent, they will be of a bright-red or reddish-brown color, and become gray or colorless as they age.

They are usually quite firmly attached to the endocardium by a sessile or pedunculated base, though the spherical or ball thrombi are free of attachments. They are firm in consistency in the earlier stages; but when degeneration takes place, softening follows, and sometimes particles become dislodged and float off to set up thrombi in other viscera. Calcareous degenerations have been noted.

They may occur singly or in groups. Thrombi have been known to project from one cavity into another, as from the left ventricle into the aorta.

Symptoms.—The symptoms are not very characteristic, and depend upon the size and rapidity of formation of the clot. The symptoms are those of obstructed circulation, the pulse being rapid and irregular, the apex-beat weak, and dyspnea more or less pronounced. Dizziness, vertigo, and attacks of syncope are common, and, later, cyanosis may appear.

In the advanced stage a clot may undergo necrosis, and blood-poisoning from absorption takes place. If a clot form suddenly and obstruct an orifice or coronary artery, sudden death results.

If the thrombi be in the right heart, and particles become dislodged, pulmonary embolism follows, with great dyspnea, pain, and cough, with bloody expectoration. Should the emboli be in the left ventricle, and a portion become detached, it would be swept into the general circulation, giving rise to cerebral, renal, or splenic embolism.

The physical signs are not very pronounced. The pulse is irregular and the apex-beat faint. The heart sounds are usually very feeble. Percussion gives negative results.

Diagnosis.—A positive diagnosis can only be made postmortem.

Prognosis.—The prognosis is always unfavorable, and sudden death

may be anticipated.

Treatment.—The treatment will be altogether symptomatic.

HYPERTROPHY OF THE HEART.

Synonym.—Hypertrophia Cordis.

Definition.—Hypertrophy of the heart is an enlargement of the organ, due to an increase in the volume of its muscular fibers, and usually also to dilatation of its cavities.

Etiology.—Hypertrophy of the heart is governed by the same law that applies to all development; namely, that size, strength, and activity of an organ depend upon its power and continued use. Increased activity gives increased development, whether of the biceps, the lung, or the heart.

The causes giving rise to the necessity for increased work are many, but may be divided into three classes: (a) Those that give rise to idiopathic hypertrophy; (b) those due to changes in the heart itself; (c) abnormal condition of the arteries.

Primary Idiopathic Hypertrophy.—This may occur from prolonged or prodigious physical exertion, as witnessed in athletes, bridge-builders, iron-workers, blacksmiths, etc. Mental excitation, where it is continued for a long time, as in some forms of hysteria, or the mental strain that keeps the patient keyed up to the highest tension, as in some forms of business, calls for over-activity of the heart and favors hypertrophy.

The excitation of the heart induced by drinking tea, coffee, and alcohol, also favors this condition. The increased expenditure of force from an overdistention of the blood-vessels by drinking large quantities of fluids, notably beer, is not to be overlooked as an exciting cause.

Lesions of the Heart.—By far the greater number of cases of cardiac hypertrophies, however, are due to endocardial changes affecting the valves or orifices of the heart. Just in proportion to the obstruction, is the heart called upon to do increased work, and the increased development is nature's method of prolonging the life of the individual.

Hypertrophy of the left ventricle is due to aortic insufficiency or aortic stenosis, also mitral insufficiency; while hypertrophy of the right ventricle is due to mitral stenosis and pulmonary obstruction. Hypertrophy of the left auricle follows mitral stenosis, and the right auricle will hypertrophy when there is tricuspid insufficiency.

Pericardial adhesions may give rise to hypertrophy as well as myocarditis. Bronchitis, empyema, pleural adhesions, and, in fact, any respiratory disease that gives to obstructed pulmonary circulation, will cause hypertrophy of the right heart.

Disease of the Blood-Vessels.—General arteriosclerosis, or sclerosis of the aorta, would give rise to hypertrophy, likewise all conditions of increased arterial tension, induced by contraction of the smaller arteries caused by irritating substances, chemical or infectious, such as syphilis, Bright's disease, etc. In fact, anything that increases the blood pressure in the larger or smaller vessels necessitates increased heart-action, and therefore acts as a causal factor in hypertrophy.

Pathology.—Hypertrophy of the heart exists in two forms, with and without dilatation of its cavities, the first being far more frequent. The morbid anatomy of the heart will therefore depend upon the form. The normal heart in the male weighs from nine to twelve ounces; in the female, from eight to ten ounces. In moderate hypertrophy it will vary from fifteen to thirty, and in exaggerated cases has been known to reach the enormous weight of sixty ounces, and is then known as the “cor bovinum” —ox-heart.

Simple hypertrophy exists where the cavities remain normal, and is usually confined to the left heart, though occasionally seen in the right ventricle where there is pulmonary obstruction. The ventricle walls may measure from one to two inches in thickness. It is doubtful if there is ever simple hypertrophy of the auricles, dilatation always accompanying the enlargement. The auricles never reach the thickness of the ventricles, there being less muscular structure to undergo change.

The altered shape of the heart depends upon the part or parts involved, and also the degree of enlargement. Thus, where the left ventricle alone is involved, the pear-shaped form is maintained, the heart being elongated. Where the right ventricle is hypertrophied, as in mitral

stenosis, the apex will be more blunt and composed largely of the right heart. Where both ventricles are involved, there is a marked broadening of the apex. Where greatly enlarged, the heart takes a lower position in the chest owing to increased weight.

When dilatation occurs, there may be a thinning of the hypertrophied walls. The papillary muscles and columnae carnae are often very much thickened. The color is dark red.

Symptoms.—It is quite difficult to separate the symptoms of hypertrophy from other lesions with which it is associated. In fact, during the period of compensation there may be an entire absence of subjective symptoms, especially when the hypertrophy is due to valvular lesions.

The compensatory changes take place so gradually that the stenosis or insufficiency is overcome by so delicate an adjustment of power that, unless some unusual strain is brought to bear upon the heart, the patient may be unconscious for months or years of any heart trouble.

As progressive changes occur, however, unpleasant sensations are experienced in the precordial region. Not a pain, but a sense of weight and oppression, especially when lying on the left side.

Overloading the stomach, active exercise, sexual excesses, or severe mental exertion bring on these symptoms in an exaggerated form, and the patient's attention is directed to the heart. Palpitation is not often present, though, if the patient be of a nervous temperament, he may complain of the "pounding of the heart against the chest wall." This may be quite noticeable if he has an irritable tobacco heart. Pain may be experienced following the excessive use of tobacco, alcohol, or coffee.

When the hypertrophy is excessive and the cerebral circulation is disturbed, the patient complains of dizziness, ringing in the ears, headache, flashing of lights before the eyes, uncomfortable flushing of the face, and a sensation as though the eyeballs were too prominent. This excessive hypertrophy may cause arteriosclerosis, and the heightened blood-pressure may result in rupture of the vessels, giving rise to apoplexy if in the brain, or to cough and hemorrhage if in the lungs (pulmonary apoplexy).

When dilatation takes place, the period of compensation gives way, and the symptoms are the same as in the last stage of valvular disease; namely, venous congestion; dyspnea now becomes a marked feature, the skin becomes cyanotic, and undulation of the cervical veins is noticeable. Engorgement of the liver, with gastric disturbance, follows, and the secretion of urine becomes scanty, albuminous, and high-colored, owing to congestion of the kidneys. Finally, dropsy makes its appearance, first in the extremities, but gradually extending to the body, till general anasarca is established.

Physical Signs.—*Inspection.*—In children there is a widening of the intercostal spaces, and quite a perceptible bulging of the precordia. The apex-beat is often visible, and extends as low as the seventh or eighth interspace and to the axillary line. If the right heart be enlarged, there will be bulging of the lower part of the sternum. An impulse may be seen to the left of the ensiform cartilage, and especially in the sixth interspace. It may also be seen to the right of the sternum in the third and fourth interspace.

Palpation.—In simple hypertrophy, the impulse of the apex-beat may be felt at the sixth intercostal space and to the left axillary line. When the enlargement is excessive, it may be felt as low as the seventh or eighth interspace and to the left axillary line. The impulse is slow and heaving, and lifts the fingers when lightly placed. If dilatation be present, the impulse is more sudden or abrupt.

Percussion.—Increased dullness is observed in every direction, as high as the second interspace and as low as the eighth; to the left, to the axillary line; and if the right heart is involved, to an inch or more to the right of the sternum, giving a transverse area of dullness seven or eight inches in extent.

Auscultation.—In hypertrophy without dilatation, the first sound is often prolonged and dull, becoming clear, sharp, and more sudden as dilatation takes place. In young persons this first sound is accompanied by a peculiar clinking sound, which Laennec designates as the “*cliquetis metalique*.” There is accentuation of the second sound, and in the aortic region is often reduplicated.

When the right is enlarged with dilatation, the first sound is loud, clear, sharp, and well denned. The second sound is sharply accentuated, and

may be reduplicated in the second left intercostal space. A systolic murmur of tricuspid insufficiency may be heard over the lower part of the sternum.

Diagnosis.—The increased area of cardiac dullness, the heaving impulse, and the character of the cardiac sounds, would suggest hypertrophy of the heart. We are to remember, however, that there are a variety of causes that give rise to increased dullness, notably pericardial effusion, retraction of the lungs, with consequent exposure of the heart, mediastinal tumors, and aneurisms, and a careful examination should be made that we may eliminate the aforesaid conditions.

If we remember that in pericardial effusion, palpation reveals a diminution and finally obliteration of the cardiac shock, while in hypertrophy the heaving impulse is characteristic, we will avoid confusion. The dullness due to an aneurism is upward and to the right or left.

Pleural effusions will be recognized by previous history and by the absence of the heaving impulse. The same may be said of mediastinal tumors. Retraction of the lungs, exposing the heart to the chest-walls, may be due to tuberculosis or cirrhosis of the lungs. The history, a careful study of the symptoms, and auscultation would reveal the diagnostic difference.

Not infrequently we meet hysterical patients and neurasthenics who suffer from exaggerated palpitation, but if we note the character of the beat, we note the absence of the heaving impulse, and repeated examinations will soon remove any doubt as to its true nature.

Prognosis.—The prognosis depends largely upon the conditions present and the stage of development. It is generally unfavorable as to a permanent cure, but where the exciting cause can be overcome or controlled, the patient may enjoy a good degree of health and live his or her allotted time of life. The prognosis is more favorable when it occurs in early life than when it comes on in later years. If the general health is good and the patient refrains from severe physical exertion, it may never be detected during the lifetime of the patient.

Where the patient is delicate and anemic, degeneration sooner or later

takes place owing to deficient nutrition, and the patient dies early. When due to valvular troubles, and the excitation continues calling for increased work of the valves, compensation finally gives way, dilatation occurs, and death terminates the disease.

Should the patient contract any of the infectious diseases, the heart would feel the force of the toxins, and to that extent be weakened, dilatation naturally following, when there may be a sudden termination of life. When the disease is idiopathic in the young, as in athletes, proper care, good hygiene, moderate exercise, and proper medication may result in a permanent cure.

If the physician can, by advice as to right methods of living and proper medication, maintain the period of compensation, the prognosis is favorable; but when this fails, the outlook is unfavorable.

Treatment.—The treatment of cardiac hypertrophy needs a careful study of each case, and will depend altogether upon the conditions present. When compensation is properly maintained, the treatment will be largely dietetic and hygienic. The diet should be plain, wholesome, and easily digested, and only as much as is consistent with health.

Coffee and tea should be used very sparingly, while alcohol and tobacco are to be absolutely prohibited. A quiet life should be enjoined, though moderate exercise in the open air is beneficial. All heavy work must be abandoned, and severe mental labor prohibited.

Regular habits are very essential, and eight, ten, or twelve hours' sleep encouraged. Sexual excesses must be prohibited.

The indication for remedies will depend upon several factors: thus respiratory wrongs would need remedies to control this phase of the disease. It may be bryonia for pain. lobelia for dyspnea, etc. Should dropsy be present, apocynum, digitalis, and like remedies, will be needed. Rheumatism would suggest the proper anti-rheumatics.

Where the hypertrophy is excessive, or rather where there is excessive power or force, veratrum may be used successfully, the indication being the full, bounding pulse. Its effects, however, should be watched carefully, and its depressing effects avoided.

Aconite adds tone to the heart, and encourages normal muscular action by removing irritation, and the *small, frequent, hard* pulse will be the symptoms calling for it. As the pulse is diminished in frequency, it gains in power.

Cactus encourages nutrition, adds tone to the organ, and is one of the best remedies in maintaining a stage of compensation. If it does not restore—and I am not sure but that it does to a certain extent—it at least retards for a long time further change, not only in the heart but in the blood-vessels as well. Of the specific tincture add ten to twenty drops to a half a glass of water, and give a teaspoonful every three hours. This should be taken for months.

One mistake in the treatment of these cases is in not continuing our remedies a sufficient length of time. Having selected the *right* remedy, patiently hold on to it till you get results; for in chronic heart troubles, beneficial effects are not seen in a few days.

Macrotys is an old and well-tried remedy where there is soreness and pain in the precordial region. The patient often complains of a bruised feeling; here macrotys will be found useful. If rheumatism be present, there will be additional reasons for its use. Crataegus has a similar action, and will often prove of great value.

Where dilatation occurs and compensation fails, quiet in bed must be strictly enjoined and nux vomica or strychnia given. Where the heart is feeble and dropsy appears, digitalis must not be overlooked. Apocynum will be indicated where there is general anasarca with feebleness; a decoction of the fresh root will increase the secretions from the kidneys and bowels, and at the same time add tone to the heart.

Lobelia will be called for to relieve asthmatic conditions where there is a sense of oppression, weight, and fullness in the precordial region, and where the pulse is of the same character.

Rhus Tox.—Frequently an irritation of the cerebro-spinal centers gives rise to irritation of the heart, which will be shown in the *small, frequent* pulse, with a *sharp* stroke. The patient is restless and starts in his sleep; in these cases, ten drops of rhus tox. to a half glass of water, a teaspoonful every one, two, or three hours, gives good results.

Echinacea.—Where there is septic infection, *echinacea* must not be forgotten.

CARDIAC DILATATION.

Definition.—Cardiac dilatation is an increase in the size of the cavities of the heart, which may be primary, with attenuation or thinning of its walls; or secondary, with thickening of its walls, hypertrophy, and dilatation.

Etiology.—Acute primary dilatation may be the result of undue exertion, without proper training, as excessive bicycling, mountain climbing, overwork on the running track, horizontal bar, or rowing, as witnessed in the enthusiasm of new members of athletic associations.

Heavy lifting by workmen who overtax their strength; strong emotional excitement, as intense anger or extraordinary fright, may also give rise to this form; in fact, any condition that gives rise to increased intra-cardiac pressure may produce dilatation. The right ventricle, in these cases, suffers most.

Dilatation from chronic valvular lesions have already, been considered, and are among the most frequent causes; here, however, there is always hypertrophy as well. Obstruction of the pulmonary vessels due to chronic bronchitis, emphysema, tuberculosis, and kindred diseases, will also give rise to dilatation of the right heart.

Any disease that weakens the heart is a possible cause; thus myocarditis, resulting from the infectious fevers, such as typhoid fever, scarlet fever, and diphtheria; also rheumatic endocarditis and pericarditis; in fact, any condition that impairs the nutrition of the heart is a possible factor in producing dilatation.

Pathology.—The left auricle and right ventricle are the cavities most frequently affected, though any one or all combined may be involved in the process. When dilatation and hypertrophy are combined, it is usually secondary and due to valve lesions. If there be aortic insufficiency or stenosis, the left ventricle is first involved, to be followed in turn by the left auricle, right ventricle, and right auricle. Mitral insufficiency or stenosis is followed by dilatation of the left auricle, to be

followed in turn by dilatation of the right ventricle and right auricle. Where chronic pulmonary diseases exist, the right heart will be the part affected.

The degree of dilatation varies: in extreme cases the auricles have been known to contain twenty or more ounces. If the ventricles are the cavities involved, the heart is broadened: but if all the cavities share in the dilatation, the organ assumes a globular form. Where there is extreme dilatation the venae cavae and pulmonary veins also share in the same progressive changes. Parenchymatous, fibroid, or fatty degenerations of the heart and endocardium take place with the progressive changes.

Other organs are impressed in the same manner. The liver becomes engorged, and then undergoes degeneration, jaundice being an accompanying factor. The mucous membrane of stomach and bowels becomes congested, giving rise to severe functional disturbance. The brain early feels the effect of the general venous congestion, as seen by engorgement of the pia mater and increase of fluid in the ventricles.

Symptoms.—Dilatation being associated with those of hypertrophy, valvular lesions, and other cardiac complications, naturally shares the symptoms of the various combinations, feebleness, and incompleteness being the most pronounced. In proportion to the amount of dilatation do we recognize the heart's waning power. The patient experiences an undefined, uneasy sensation in the cardiac region; not exactly a pain, yet oppression of a distressing character. Dyspnea is one of the prominent symptoms, increasing as the cavities enlarge.

Where the right heart is involved, pulmonary symptoms soon develop, a hacking cough, attended with a serous and sometimes sanguineous expectoration, follows. As the dyspnea increases, the patient is unable to lie down without bringing on a series of spasmodic coughs, commonly known as cardiac asthma.

The pulse, as well as the apex-beat, is feeble. The pulse is often irregular both as to power and rhythm. The extremities, and even the body, are apt to be cool. Any undue exertion, either mental or physical, aggravates all the symptoms, especially the dyspnea.

The veins, particularly those of the neck, are distended, and the patient

is more or less livid or bluish in appearance. Owing to congestion of the liver, there is a sense of weight and oppression in the right hypochondrium, and the patient takes on an icteric hue.

There is more or less gastric disturbance, resulting from the general congestion, and attacks of vomiting are not infrequent, digestion is imperfect, and the evidence of impaired nutrition is apparent. Diarrhea announces the congested condition of the intestinal tract, and adds to the debility.

The kidneys are also frequently involved, and nephritis is a common attendant, the urine being scanty, high-colored, albuminous, and contains casts.

Cerebral congestion is attended by headache, dizziness, and sometimes vertigo. Delirium rarely occurs. With general venous congestion, edema appears, first in the feet, gradually encroaching upon the body, till finally anasarca or general dropsy results.

Physical Signs.—*Inspection.*—If the patient be thin or emaciated, and the dilatation be of the left ventricle, the apex-beat will be seen to be displaced downward and to the left. If the dilatation be of the right ventricle, the pulsation will be seen in the epigastrium. If the patient be well nourished, the feeble apex-beat may not be seen, and other signs must be looked for to aid in the diagnosis. In advanced cases, visible pulsation of the jugulars is pronounced.

Palpation.—The feeble, undulating apex-beat may be felt, though not constantly. If the dilatation be of the right heart, a pulsation may be quite pronounced over the liver. The jugular pulse may be readily felt, even though not visible.

Percussion.—Dullness will be increased transversely to the left axillary line in dilatation of the left ventricle, and to the right nipple line when the right heart is dilated. If the auricles are involved, the area of dullness may extend to the first rib and transversely as far as when the ventricles are involved.

Auscultation.—Dilatation is always associated, more or less, with valvular insufficiency or stenosis, and, therefore, pre-existing murmurs will necessarily influence the sound made by dilatation. The first and

second sounds are quite similar, and are short and sharp, resembling fetal heart-sounds, the long pause being shortened. Irregular pulsations, both as to time and force, have been noted, and the canter rhythm is not uncommon.

Diagnosis.—The diagnosis of dilatation of the heart is less difficult than many other organic lesions. The wavy or undulating apex-beat, the first and second sounds being sharp and of the same length; the canter rhythm, or embryo-cardia; the frequent irregular pulse; pulsation in the epigastric region; the wavy pulsation in the jugulars, and the general cyanotic appearance, together with anasarca, make a group of diagnostic symptoms that can scarcely lead to a mistake.

Prognosis.—The prognosis of cardiac dilatation is always unfavorable, and though life may be prolonged by careful living, change of climate, and remedies that add tone to the organ, we are not to forget that the changes are progressive and the termination is death in most cases.

Treatment.—The patient must clearly understand that severe muscular or mental exertion, or *anything* that causes *unusual* excitement, *must* be positively forbidden. The patient should lead a quiet life, as much in the open air as possible, and have such diversion as will attract his attention away from himself.

The aim of all treatment in cardiac dilatation is to *increase* the *muscular power of the heart*. To make good muscle requires good blood, and this requires good digestion. Diet, then, will be an important factor in the treatment; this should consist of good, nutritious food, easily digested, and only enough fluids allowed as is compatible with health.

Any disturbance of the stomach should be corrected, constipation should be overcome, and hepatic derangements should be controlled. A few remedies will be indicated, and should be given to overcome special conditions. For the small, feeble, irregular pulse, aconite, in the small dose, will give good results. We give the remedy, not for its sedative effect, but to add *tone* to the heart. Five drops of the agent to four ounces of water, and a teaspoonful every three hours, will not disappoint you.

Cactus.—This remedy does not overstimulate and thus weaken the heart action, but its tendency is to increase the heart's nutrition and

add power to the muscle. Of the specific tincture, use twenty drops, to water four ounces. Goss says of this remedy: "If the patient suffers from a cramping pain, like a band around the heart, I have *always* found this agent gives quick relief."

Crataegus.—This is a remedy that is receiving a great deal of attention in cardiac lesions, and as a tonic and restorative has given excellent results. The remedy should be given in from five to ten drop doses.

Lobelia.—This will afford some relief to the distressing dyspnea, though we are not to forget the mechanical obstruction causing the difficult breathing and expect too much of the remedy.

Apocynum is the remedy when dropsical effusion appears. It not only stimulates the kidneys to carry off accumulations, but strengthens the heart at the same time.

Digitalis will be used for similar conditions.

Lycopus Virginicus will be a good remedy where pulmonary troubles arise, attended by cough and hemoptysis.

Spigelia, alternated with bryonia, will afford relief when there is pain, sharp and stabbing in character.

No matter what agents are employed, the patient should be kept as quiet as possible, saving the heart any unnecessary work. Smoking should be prohibited.

CARDIAC ATROPHY.

Definition.—Cardiac atrophy is a decrease in the size, strength, weight, and activity of the heart.

Etiology.—The causes depend somewhat upon the character of the atrophy, whether general or local. Thus mitral stenosis gives rise to a local atrophy, the left ventricle being the part affected. Other parts may atrophy when degenerative changes are confined to a portion of the heart, while general cardiac atrophy may arise from causes that impair or interfere with the nutrition of the body, the heart sharing in the

general atrophy, cancer of the stomach, phthisis pulmonalis, suppurative processes where long continued, Bright's disease, etc., being examples. Partial obstruction of the coronary arteries may give rise to atrophy by impairing the heart's nutrition. Senile atrophy is usually the result of failing nutrition. General atrophy may also be congenital.

Pathology.—The degree of atrophy varies, Bramwell reporting a case in an adult where the heart weighed only two and three-fourths ounces. The surface of the heart loses its regular outline, becoming wrinkled or rough, while the coronary arteries are quite tortuous. As the waste exceeds the supply, the sub-pericardial fat early disappears, and may be replaced by a granular or mucous tissue.

The color varies: it may be pale and firm, or it may be yellow and known as yellow atrophy: again it may be pigmented, soft, and granular, and known as brown atrophy. The muscular fibers may be replaced in part by granular or fatty material.

Symptoms.—The symptoms of cardiac atrophy are not very pronounced, feebleness of action being the most prominent; but as this is characteristic of all wasting diseases, we may not gain much information from this source. The apex-beat is quite weak and often invisible, and, though percussion shows the area of dullness diminished, it may be due to pulmonary emphysema.

Diagnosis.—While atrophy may be suspected, a positive diagnosis is only made post-mortem.

Prognosis.—The prognosis depends more upon the underlying disease than upon the extent of the atrophy of the heart; for, though diminished in size, there is diminished work required, and a proper balance is maintained.

Treatment.—The treatment will be tonic and restorative. Where the primary lesion can be overcome, our treatment will be directed to the accomplishment of that end. Gentle exercise in the open air, a good, nutritious, and easily digested food supplied, will be an important factor in the treatment. All measures that look to a better blood supply will aid in prolonging life. Incidentally cactus or crataegus may be given as heart tonics.

ACUTE MYOCARDITIS.

Synonym.—Carditis.

Definition.—Acute myocarditis is an acute inflammation, diffuse or circumscribed, of the interstitial or parenchymatous substance of the heart.

Etiology.—Two forms of myocarditis are recognized, the diffuse or parenchymatous and the circumscribed.

Diffuse or parenchymatous myocarditis is rarely ever idiopathic, but follows as a complication or result of some other disease. It occurs most frequently in the course of the infectious fevers, especially typhoid fever, diphtheria, and scarlet fever, and the sudden deaths in these cases from heart-failure can be traced in nearly every case to myocarditis.

Endocarditis and pericarditis, if of a severe type, nearly always extend to the myocardium. Rheumatism figures as a causal agent, as does gonorrhoea. We may be safe in saying that the toxins in any infectious disease may so influence the heart as to give rise to myocarditis.

Circumscribed myocarditis may arise from the same sources that give rise to the diffuse form; viz., the infectious fevers; or it may be due to emboli in the coronary arteries.

Pathology.—The heart, in the early stage, is of a dark-red color, swollen, softened, and injected; later it changes to a yellowish gray or mottled appearance, and is readily broken down. There may be dilatation of the cavities, and if there has been localized myocarditis, weakening the tissues, partial aneurism may result.

The more minute changes consist in an infiltration of the interstitial substance of round cells, and later fibroid degeneration. The muscular fibers undergo fatty or granular degeneration.

In the diffuse form the chief pathological changes take place in the connective tissue, and the left ventricle suffers more than the right.

In the localized or circumscribed form there are areas of necrosis, which

are followed by abscess formation. These abscesses may open into the cavities of the heart, and thus enter the blood-stream, giving rise to abscess formation in other portions of the body. A favorite location for an abscess is in the interior wall of the ventricle near the apex and septum. They may empty into the pericardium, resulting in suppurative pericarditis. When they perforate the heart cavities, in addition to poisoning the general blood with an accompanying septicemia, they often give rise to malignant endocarditis. This form usually terminates fatally, though, in rare cases, nature throws a covering around the abscess, the pus is incapsuled, and undergoes caseation or calcification.

Symptoms.—The symptoms of the primary lesion usually so obscure the true nature of the disease that subjective symptoms may be entirely absent. If, however, there is a sense of constriction of the chest, some palpitation, more or less dyspnea attended by slight pain, and the pulse is rapid, small, and easily compressed, evidences of cardiac enfeeblement, myocarditis would be suggested.

Should suppurative myocarditis exist, and the abscess perforate the cavities, embolic manifestations would appear in brain, lungs, or spleen. An increase of temperature, with the above symptoms, would naturally follow, and, if suppurative in character, the fever would be of a septic type.

Physical Signs.—The physical signs are similar to those of dilatation. The action of the heart, in the early stage, is tumultuous, but as changes take place it becomes small and irregular.

Auscultation reveals the first and second sound of nearly equal length, short and sharp, which soon becomes feeble. If dilatation has taken place, murmurs frequently develop. The most frequent is a systolic murmur suggesting mitral insufficiency.

Diagnosis.—The diagnosis may not positively be made during life; however, great heart enfeeblement as shown by the weak first sound or systolic murmur and the small irregular pulse would suggest myocarditis.

Prognosis.—The prognosis is favorable where it appears in a mild form, though the severer forms usually terminate fatally and the sudden termination of life during the infectious diseases, notably

diphtheria and typhoid, may not infrequently be due to myocarditis.

Treatment.—Absolute rest must be enforced in every case; the reclining position should be observed, and nutrition maintained.

The agents recommended for endocarditis and pericarditis will be found useful. In fact, the above mentioned diseases are nearly always attended by more or less myocarditis. Heart tonics should be used with great care. Cactus, in small doses, will be a good agent. When the heart flags, carbonate of ammonia as a diffusible stimulant will be found beneficial, as will also strychnia.

Septic conditions will require antiseptics, and when there is rheumatism with an acid condition of the blood, the acetate, citrate, nitrate, or lithiate of potassium will be found beneficial.

CHRONIC MYOCARDITIS.

Synonyms.—Fibroid Heart; Cardio-Sclerosis; Chronic Interstitial Myocarditis.

Definition.—A chronic inflammation of the heart muscle, resulting in induration, due to fibroid degeneration.

Etiology.—Sclerosis of the coronary arteries, Bright's disease, diabetes, rheumatism, gout, and the excessive use of tobacco and alcohol, are the most frequent causes of fibroid degeneration.

Sclerosis of the coronary arteries may be due to chemical irritants or syphilis, or may follow as a result of the infectious diseases. Each of these may be said to act as a causal agent. It may be an extension of chronic endocarditis and pericarditis; it may also follow acute diffused myocarditis. Chronic valvular lesions may so impair the nutrition of the heart as to give rise to myocarditis, or it may be due to a direct extension of the inflammation along the chordae tendineae, or valves, to their muscular attachments.

Age and sex predispose somewhat, as sclerotic changes occur more frequently in elderly people, and more males than females suffer from this lesion. When the disease occurs during fetal life, the right heart is

the seat of the disease.

Pathology.—While the anatomical changes may be diffuse, they are usually circumscribed, the wall of the left ventricle, the septum, and the papillary muscles being, even in the diffuse form, more extensively involved. In fetal myocarditis the apex of the right ventricle is the favorite site.

The muscular fibril is replaced by fibrous tissue, which is dense, hard, and of a grayish-white appearance. The weight of the heart is increased, both by the degenerative changes and the hypertrophy and dilatation that so frequently accompanies it. There may be a narrowing of the pulmonary and the aortic orifices due to contraction of the changed tissue.

The branches of the coronary arteries may be occluded, either by circumscribed areas or by emboli. These sclerotic changes in the coronary arteries are frequently due to syphilis, resulting in obliterating endocarditis. When aneurism of the heart occurs, it is usually due to arteriosclerosis. In advanced stages, fatty degeneration may replace the fibroid or be associated with it.

Symptoms.—The symptoms are not at all characteristic; in fact, they are so indefinite in many cases as not to cause a suspicion of the true difficulty, and the true nature is only revealed during an autopsy. The hypertrophy, that usually attends, so compensates that the patient is unaware of his condition.

Generally, however, there is evidence of enfeeblement, and this is accompanied by dyspnea and more or less palpitation. A sense of constriction is often present, and attacks of angina become quite frequent and distressing. The pulse is slow and often irregular, the pulse-rate being reduced to fifty, forty, or even less, per minute.

When there is a sudden failure of the cerebral circulation, or after unusual exertion, the patient may be attacked with syncope. Pseudo-apopleptic attacks may terminate in sudden death.

Physical Signs.—Where hypertrophy exists, the apex-beat is displaced downward and to the left, the dullness being in the same direction. Although the heart-sounds may be clear and strong early in the disease,

they soon become feeble and indistinct.

Diagnosis.—The recognition of this disease before death is extremely difficult, and though cardiac weakness may be easily determined, it is often indistinguishable from hypertrophy with dilatation or the many valvular lesions. Generally the absence of murmurs enables us to differentiate it from valvular lesions. To distinguish it from fatty degeneration is quite impossible. Frequent attacks of angina would excite suspicion more than any other symptom, and if the pulse were reduced to fifty or forty per minute, additional reason would exist for believing in chronic myocarditis.

Prognosis.—The prognosis is usually unfavorable as to a cure, though favorable as to life. The disease comes so insidiously in the majority of cases, and degenerative changes have so far advanced, that a complete cure is out of the question. If, however, the patient can refrain from severe mental or physical exertion, can live in an equable climate, and be much in the open air, the life may be prolonged to its allotted period. Where frequent attacks of angina occur and the cerebral circulation suddenly fails, death may occur quite suddenly.

Treatment.—The habits and methods of living should be thoroughly impressed upon the patient. No severe mental or physical exertion should be allowed, no tobacco permitted, while coffee and tea should be taken sparingly or not at all. Gentle exercise in the open air is advisable. The diet should be nutritious, and, as a rule, fluids should be restricted.

In the way of medication, strychnia as a stimulant, when there is great debility, will be found useful. In case of syncope, a hypodermic injection of camphor and ether will give good results.

Cactus and crataegus are agents that will improve the innervation and nutrition of the heart, and should be administered for a long time.

If syphilis exists, the anti-syphilitics will be used.

DEGENERATIONS OF THE MYOCARDIUM.

Anemic necrosis, anemic infarct, or white infarct, is the term given to that form of myocardiac degeneration due to occlusion of the branches

of the coronary artery. As the anterior coronary artery is most frequently the seat of the obstruction, the infarcts are most frequently found in the left ventricle and septum, parts supplied by this branch of the artery, and consist of patches of grayish white or grayish red, wedge-shaped masses. These may become softened and break down, forming what is known as myo-malacia cordis. This softened, and therefore weakened area, may cause a rupture of the heart.

At other times these spots, in place of softening, change to a hyalin appearance, which in turn become sclerotic. The minute changes in the muscular tissue are the replacement of the muscle fibers with granular material. At other times they assume a hyalin form, and finally become sclerotic.

The symptoms are obscure, unreliable, and of no diagnostic value. Death may occur suddenly, and an autopsy alone reveal the lesion.

Calcareous degeneration, or calcification of the myocardium, is of very rare occurrence, and consists of the displacement of the muscular fiber by limy deposits.

Calcification of the valves occurs more frequently. Following myocardial abscesses, chalky nodules may sometimes be found.

Amyloid Degeneration.—This, like calcification, is a rare disease, and results from the same causes that give rise to amyloid disease of any other part. It is more apt to be confined to the blood-vessels and interstitial tissues, and is frequently preceded by suppurative processes, tuberculosis, syphilis, etc.

Hyalin Degeneration.—This has already been noticed in connection with anemic necrosis, and may also be found in connection with amyloid degeneration. The connective tissue is replaced in limited areas by a glossy, transparent material.

It may occur independently of other degenerations and follows prolonged suppurative fevers. The fibers become swollen, and are changed into a homogeneous translucent material.

Brown Atrophy.—This is a form of degeneration that is frequently found in connection with other degenerations, especially fatty, and

consists of a deposit in the muscular tissue of a yellowish-brown, granular material. It is found in advanced valvular troubles sometimes, and in old people.

The diagnosis is made post-mortem.

Parenchymatous Degeneration has been considered in chronic myocarditis.

Fatty Degeneration.—This is not to be confused with fatty infiltration or fatty overgrowth. Fatty degeneration is the replacement of the muscle fiber by oil globules or fat. This is perhaps the most common of all degenerations and is due to faulty nutrition. This may arise from many conditions. We may say that all wasting diseases, such as tuberculosis, carcinoma, acute and chronic anemia, and the various infectious diseases, are causal conditions.

Syphilis, by its poisoning effect, may change the nutritive properties of the blood to such an extent as to render the fiber unable to reproduce itself. Pericarditis and the valvular lesions, which have already been considered, also favor this condition. Disease of the coronary arteries, by impairing nutrition, is a frequent cause. It is also associated with other degenerations, parenchymatous, white, infarct, etc. The disease is more frequently met with after middle life, and is more common in men than in women.

Certain poisons, such as phosphorus, arsenic, and alcohol, are followed by fatty degeneration. When due to phosphorus or arsenic, the degeneration is sometimes quite rapid, while if from alcohol it involves a much longer time.

Pathology.—The degeneration may be confined to local areas or the entire organ may be involved. Usually the left ventricle and papillary muscles are the seat of the tissue change. Yellowish patches are seen, which are friable and easily torn. Sometimes it is of a reddish-brown color, and frequently occurs with brown atrophy. Its friability favors rupture of the organ, which sometimes occurs. If the entire organ be involved, there is enlargement, its walls being flabby.

Fatty degeneration of the heart is not infrequently associated with fatty degeneration of other parts of the body. As a result of its friability,

dilatation of its cavities is not uncommon. Sclerosis of the coronary arteries is often found.

Symptoms.—The symptoms of this form of degeneration are not characteristic and may not be separated from myocarditis. Enfeeblement is always a prominent condition, and palpitation, dyspnea, and a quick, feeble, and irregular pulse, with cool, clammy extremities, are symptoms that would arouse suspicion of the true condition.

Cardiac asthma, with occasional attacks of angina pectoris, may also be present.

Physical signs are similar to those of cardiac dilatation.

Diagnosis.—The diagnosis is extremely difficult. The age of the patient, the enfeebled action, the small, irregular pulse, the evidence of dilatation, the dyspnea and palpitation, cardiac asthma and angina pectoris, and, in advanced stages, the Cheyne-Stokes respiration, would all suggest fatty degeneration, though a positive diagnosis should not be made.

Prognosis.—The prognosis is unfavorable as to a cure, though the patient may live for years after tissue changes have taken place. Undue exertion, or great mental excitement may result in rupture of the heart, and sudden death.

Treatment.—If one keeps in mind the conditions that give rise to degenerations, the line of treatment will suggest itself. Impaired nutrition, enfeebled vitality, and cell formation prevent normal tissue formation. To make a good blood, to renew the tissue, and to break down the old and imperfect tissue, is a difficult task, and one that many times is impossible to accomplish.

Hygienic measures are important. An equable climate, an out-of-door life, gentle exercise, regular habits, and the avoidance of severe exertion or mental excitement, will do much for the patient. A nutritious but easily digested diet, with a restriction of fluids, especially at meal-times, will assist largely in maintaining the health, and further stay tissue change.

Cactus, pulsatilla, crataegus, lobelia, etc., will be used as occasion

demands.

FATTY OVERGROWTHS.

This is frequently the result of general obesity and may completely envelop the heart, sometimes the layers of adipose tissue being an inch or more in thickness. When excessive, the pressure may give rise to atrophy and enfeeblement.

The fat in the auriculo-ventricular furrows may also be greatly increased, while infiltration between the muscular fibers is not uncommon, and may extend to the endocardium.

This condition is generally seen after middle life, and among high livers who lead a sedentary life.

Treatment.—The treatment is principally hygienic and dietetic. A rigid diet, with a sparing use of fluids, will generally accomplish wonders. No fluids at meal-times, nor for two hours after, should be made an absolute law. Exercise in the open air, as much as can be taken without unduly wearying the patient, should be rigidly enforced. Small doses of the juice of phytolacca berries may be given, but the effect should be carefully watched.

DISEASE OF THE CORONARY ARTERY.

Disease of the coronary arteries has been considered in anemic necrosis, and needs no further mention.

CARDIAC ANEURISM.

Aneurism may be confined to the valves—valvular aneurism; or may involve the walls of the myocardium—aneurism of the heart-wall.

Etiology.—Aneurism of the valve results from endocarditis, either acute or chronic, whereby the reflection of the endocardium upon the valve becomes softened or destroyed, and the intro-cardial blood-pressure produces dilatations, spherical in form, the convex side facing

the least resistance. Thus, when of the aortic valve, the most frequently involved, the aneurism bulges into the left ventricle; while if the mitral valve be the one involved, the aneurism projects into the left auricle. When rupture occurs, valvular incompetency is pronounced.

Aneurism of the wall is usually due to weakening of the tissue by myocarditis, though the various degenerations, which occur in the heart, as well as mural endocarditis, may be responsible for it. Knife-wounds have also been followed by aneurism. Pericardial adhesions may so weaken the wall as to favor dilatation.

Pathology.—The dilatation, followed by sacculation, is usually found near the apex in the left ventricle. In size, they vary from that of a marble to that of a croquet-ball. There is usually only one, though this may be sacculated, forming two patches in one cavity, and Peacock reports a case where there were three pouches. The endocardium is found to be opaque, while the myocardium may undergo degeneration or sclerosis.

Symptoms.—The symptoms are not sufficiently definite to excite suspicion of the true character of the disease. There is general enfeeblement, due to the lesions that give rise to it, and sometimes a bulging, pulsating tumor may be seen in the apex region.

Diagnosis.—A positive diagnosis is only made post-mortem.

Prognosis.—The prognosis is unfavorable, and, though death may occur suddenly from syncope and rupture, it is usually due to exhaustion depending upon the myocarditis or the degenerations which give rise to it.

Treatment.—The same line of treatment as was suggested in cardiac degenerations will be followed in aneurism. Hygienic, dietetic, and such cardiac remedies as may be required to meet special conditions, will compose the treatment.

RUPTURE OF THE HEART.

This very rare accident occurs after the heart has been weakened by degenerations, especially fatty degeneration. Quain has found this to be

the cause in seventy-seven out of a hundred cases examined.

Suppurative myocarditis and obstruction of the coronary artery resulting in anemic necrosis, are also responsible. It occurs most frequently after the age of sixty, the time of life when the degenerative changes culminate so disastrously. It occurs more frequently among males than among females, owing to the more strenuous life of males. The culmination may be the result of severe exertion, though it has occurred when the patient was at perfect rest.

The point of rupture is usually in the anterior wall of the left ventricle, more rarely in the right ventricle. The pericardium is generally filled with dark, coagulated blood.

Symptoms.—This accident usually results in sudden death. The patient is seized with a sharp, stablike pain, may cry out in his anguish, and fall unconscious, or, grasping the region of his heart with his hand, remain stationary, fearing to move. A deathlike pallor spreads over the face, the surface is covered with a cold sweat, the pulse is tumultuous, and the patient falls unconscious. If he survives a few hours, the dyspnea is marked and the pain agonizing in character. Mays reports a case that lived for seventeen days after the rupture.

Diagnosis.—Excruciating pain in the cardiac region, pallor of face, cold sweat, and a sense of suffocation, would suggest a rupture of the heart.

Prognosis.—The prognosis is always hopeless, for if death is not sudden, the patient's life is prolonged but for a few hours or a few days at most.

Treatment.—The only treatment that will be of great benefit will be prophylactic, for after the accident, opiates, to relieve the intense pain, is all that can be used.

In every case where there is reason to believe that myocarditis, obstruction of the coronary arteries, or degenerations exist, the physician should explain the danger and the result which is liable to follow, unless the patient leads a very temperate life.

All excesses should be avoided. The diet should be nutritious, but easily digested, and as little fluid as is consistent with health should be

allowed. Gentle exercise in the open air, and a residence in an equable climate is desirable; unfortunately but few can profit by such advice, though very much depends upon one's environment.

When rupture has occurred, complete rest in bed, hypodermic injections of morphine, with local applications to relieve pain, will constitute the treatment. Stimulants should be avoided, as they increase the hemorrhage.

MINOR CARDIAC AFFECTIONS.

New Growths.—While new growths may originate in the myocardium or endocardium, they are more apt to be an extension from the parts. Cancer, as a primary disease of the heart, is of very rare occurrence, though as an extension from the mediastinum, the lungs, or pleura, it is occasionally seen.

Syphilitic deposits are found on the valves, and in the ventricles, more frequently the left. Cysts are very rarely found. Myxomatous, fibroid, lipomatous, sarcomatous, lymphoid, and sometimes chondromatous growths occur, though they are not common. They are more apt to develop in the pericardium than in the heart substance.

The symptoms are negative, and the growths are only discovered post-mortem; hence the prognosis and treatment need no consideration.

Parasites.—The heart may be the habitat of four forms of parasites; the taenia echinococcus, cysticercus cellulose, actinomyces, and pentastomum denticulatum. The first two, by developing cysts, may produce serious results. They are more frequently found in the right ventricle. When they rupture, if it be in the right ventricle, secondary cysts of lung sometimes develop. As a result of rupture of the cysts, portions may float off in the general circulation, and give rise to embolic abscesses in various organs of the body. These cysts are generally found in the liver and other organs at the same time.

Misplacement.—Malpositions of the heart may be congenital or acquired as the result of excessive tympany, ascites, tumors, or disease of neighboring parts. The most important and remarkable congenital malposition is where the heart is located to the right, in place of the left

median line. The apex-beat is found in the fifth interspace of the right side, and the general boundaries are the same, otherwise, as if located on the corresponding side. The arch of the aorta curves over the right bronchus instead of the left, and the descending aorta is to the right of the spinal column instead of to the left. Usually there is an interchange of the other organs, though not always. Thus the liver takes the left side, and the spleen occupies the right.

In the fetus the heart occupies the median line, and occasionally this persists after birth. Another malposition is where the heart is found immediately beneath the skin, the sternum being missing. The heart has also been found misplaced in the abdominal cavity.

A rare and a serious displacement is where the heart occupies the cervical region. Floating heart is that condition where its attachments become weakened and relaxed and the heart becomes more or less motile.

Wounds and Foreign Bodies.—Although wounds of the heart are generally fatal, occasionally an injury of the heart occurs that startles the medical world. Bullets have been found encysted in the ventricles, and only recently a man was stabbed in the heart, severe hemorrhages followed, but the surgeon hurriedly opened the pericardium, stitched up the gaping wound, and, to the surprise of every one, the patient recovered. Pins have been found imbedded in the heart.

IV. NEUROSES OF THE HEART.

PALPITATION.

Definition.—Palpitation is the consciousness of the heart's action, which may be normal in power and in time of beat, though there is increased power and frequency, attended by more or less anxiety and distress.

Etiology.—The fact that “delirium cordis,” a condition of extreme disturbance, frequently exists in heart-troubles, and yet the patient is entirely unconscious of the existence of any disturbance, while at other times the patient complains of great distress from the severe palpitation when an examination reveals the heart's action to be normal in power

and frequency, shows conclusively that it may be nervous in character.

Among the causes that may be mentioned as giving rise to palpitation, are:

Dyspepsia.—Indigestion, I believe, is responsible for more cases of functional heart-disease than all other causes combined. The distention of the stomach by gas, presses against the sensitive nerves of the heart, which gives rise, not only to palpitation, but irregular heart action as well.

Nervous Lesions.—Emotional excitement, especially the distressing emotions, such as fear, grief, and despondency, are causes;

It also occurs in neurasthenia. Hysteria is not infrequently accompanied by palpitation. Sexual excess, especially masturbation, is another common cause of cardiac palpitation.

Stimulants.—The use or abuse of tea and coffee, as well as alcohol, will cause an irritable and excessive action. Tobacco may be included in the same list. The irritant action from a poorly elaborated blood, as found in anemia, will also give rise to this condition; prolonged fever gives rise to palpitation in the same way.

Reflexes.—A common, though often overlooked, source of irritation is rectal troubles. Hemorrhoids, fissures, fistules, and papillae may give rise to such nerve-waste as to produce their reflex influence on the heart. Disease of the uterus, ovaries, and urethra may act as indirect factors in the same way. Chronic valvular lesions, as well as some other organic lesions, may give rise to palpitation, though not frequently.

Symptoms.—If the attack be mild in character, there may be but little change in the character of the pulse, the force and frequency being found, upon examination, to be normal. The consciousness of the patient, of his heart's action, however, renders him nervous, and a worried or anxious look tells of his mental discomfort.

Where the attack is severe, the heart beats tumultuously, and the patient feels the impulse against the chest-walls, and often hears the sound of his own heart-beat, while the physician may see the pulsation if the chest-walls be thin, and, by placing the hand over the heart, detect its

throbbing character. There may be a choking or smothering sensation, especially if the subject be hysterical, and the patient, alarmed and anxious, fears impending dissolution.

Sometimes a cold sweat occurs; there is pain in the precordial region, rapid breathing, the extremities become cold, the eyeballs protude, there is a ringing in the ears, vertigo, and an attack may terminate in unconsciousness. If the patient be neurasthenic, an attack may be followed by the passing of a copious quantity of clear urine.

The attacks are usually intermittent and paroxysmal in character, though they may be more or less constant.

Physical Signs.—Inspection will often reveal a visible and forceful impulse, and also throbbing of the superficial vessels.

By placing the fingers or the hand over the region of the heart, a heaving and throbbing impulse is felt, while the pulse at the wrist is full and bounding or sharp and frequent, sometimes quite irregular.

Auscultation shows the first sound magnified; it is loud, abrupt, short, and may be heard without applying the ear to the chest.

Percussion may reveal an increased dullness, though usually the dullness is natural.

Diagnosis.—The condition, palpitation, is readily recognized by the complaint of the patient, and the symptoms already named, and a careful physical examination will reveal whether it be a neurosis or due to organic disease.

Prognosis.—The prognosis is favorable as to life, and also as to permanent results, where the causes can be removed. When due, however, to epilepsy, prolonged masturbation, spinal irritation, and such causes as frequently do not yield to treatment, the outlook is not encouraging.

Treatment.—The patient's mental disturbance must be quieted by positive assurances that an unfavorable termination will not occur. It is well to put the patient to bed, enjoin quiet, and have anything that would tend to excite him removed from his presence.

After a hopeful and reassuring talk, place the patient on pulsatilla, if the sensation of impending death predominates, or if there be a constant dread of an indefinable character present; in these cases pulsatilla will give good results. Ten to twenty drops of the specific tincture in a half glass of water, a teaspoonful every one, two, or three hours, as the case may demand. If there be precordial oppression, with a sense of suffocation, add ten drops of specific lobelia to the above remedy, and give every twenty, thirty, or sixty minutes. If there be pain present, alternate the above with bryonia or macrotys, ten drops of each to water four ounces, and give teaspoonful every hour.

Where there is great excitement, the patient is flushed, nervous, and restless, gelsemium, ten to thirty drops to half a glass of water, will be of good service. Cactus is a good remedy when the heart's action is weak. When the force is violent, veratrum, thirty to sixty drops to half a glass of water, will be the better remedy.

After an attack is over, the treatment will be directed to overcoming the exciting cause.

When due to indigestion, the diet should be regulated, fluids at meal-times forbidden, and the various dyspeptic conditions corrected.

If anemia is the exciting cause, our attention is directed to furnishing a better quality and quantity of blood.

If alcohol, tea, coffee, or tobacco are responsible, they are to be absolutely prohibited. Where due to rectal irritation, or ovarian or uterine trouble, these wrongs must be corrected before a cure can be effected.

Regular habits should be enjoined, all forms of dissipation corrected, and for persons of sedentary habits an out-of-door life advised. A trip, lasting several weeks or months, where the attention of the patient is turned from self to new objects, new faces, new scenes, will often accomplish far more than will the administration of drugs.

TACHYCARDIA.

Synonyms.—Rapid Heart; Synchosphyxia; Paroxysmal Hurry of the Heart.

Definition.—Tachycardia is a rapid action of the heart, either constant or paroxysmal. It is usually associated with palpitation, though it may be entirely independent. There are generally no subjective symptoms of forcible action in tachycardia.

Etiology.—Occasionally we meet with a physiological tachycardia, the normal pulse running a hundred or more per minute, and still more rarely is found a patient who can increase the pulse-rate at will.

The rapid heart seen in all fevers is not usually considered under the head of tachycardia, but is limited to that form which is paroxysmal in character. It is generally due to a neurosis, though it sometimes occurs as the result of a growth or clot in or about the medulla. Also pressure of the pneumogastrics by growths or tumors may give rise to it. The peripheral neuritis of the pneumogastrics produced by the toxins of the various infectious fevers may be attended by heart hurry.

The more frequent causes, however, are reflex, and may be due to wrongs of the rectum, the uterus, ovaries, bladder, or urethra. Fright, grief, and emotional excitement are frequently the exciting causes of a rapid heart. Severe physical or mental exertion may be followed by a rapid heart-beat, which may continue for days. Sexual excesses and masturbation will also give rise to this condition, some cases being especially stubborn in yielding. Often associated with palpitation, the same causes may give rise to it. Anemic and chlorotic females are subject to rapid heart, while the victim of hysteria or neurasthenia may suffer with paroxysms of tachycardia. It sometimes occurs in females during the menopause.

Pathology.—No characteristic lesions are found, though neuritis of the pneumogastric and myocardial degenerations have been discovered post-mortem.

Symptoms.—Where tachycardia is permanent, there are few pronounced symptoms aside from rapid pulse-beat; but in the paroxysmal form, true tachycardia, the symptoms are more varied and

pronounced.

As a rule, the attacks come on suddenly and often without any premonitory symptoms. In other cases an attack is announced by vertigo, ringing in the ears, and a sense of danger impending.

The subjective symptoms also vary. Sometimes there is almost an entire absence of unpleasant sensations, the patient being unaware of the increased movement of the heart; in fact, he is inclined to believe that the heart-beat is not sufficiently rapid. At first the face is pale, but it soon becomes flushed and sometimes turgid.

The pulse is small, weak, easily compressed, and sometimes irregular. At the beginning of an attack the pulse rapidly increases to one hundred and fifty or two hundred beats per minute, and has been known to reach three hundred per minute.

The respiration may remain normal in frequency, though it is usually somewhat increased. There may be dyspnea and sometimes a feeling of uneasiness, or even pain, in the precordial region. It is not infrequently associated with palpitation, and the patient becomes extremely anxious as to his condition. Vertigo, headache, and ringing in the ears may continue through an attack. An attack may last for but a few minutes, or it may last for hours or sometimes days.

Physical Signs.—If the chest-walls be thin, the rapid, diffuse, and irregular impulse may be perceptible on inspection. Palpation but confirms the visible signs. There may be no enlargement of the heart, and nothing is learned by percussion.

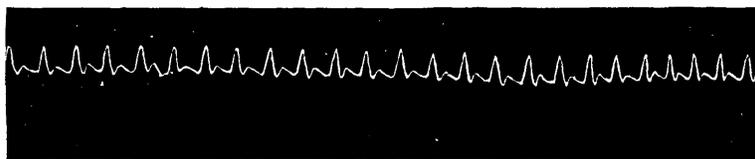


FIGURE 23. PULSE-TRACING IN A CASE OF TACHYCARDIA.—(Goodno.)

Auscultation may show the heart-beats somewhat modified. Since a less amount of blood is thrown into the aorta with each ventricular systole, the first sound will be slightly accentuated, while the second sound will be diminished. The second pulmonic sound may sometimes be increased, as may also the first systolic sound. Murmurs are seldom heard.

Diagnosis.—The diagnosis is readily made unless complications exist. The great rapidity of the heart's action, with an absence of subjective symptoms; is characteristic. Dyspnea, pre-cordial oppression, pain, and a sense of impending danger are generally associated with palpitation, and where present in tachycardia, are much milder than in the former.

Prognosis.—The prognosis depends somewhat on the causes giving rise to it, though at best it yields but slowly. It seldom proves fatal, though in elderly patients there may be rupture of the cerebral vessels, or death may result from heart exhaustion.

Where the tachycardia is due to reflexes, a cure may result in a removal of the cause. The various orifices should be carefully examined for sources of irritation, and when found they should be promptly removed.

Treatment.—A successful treatment will necessarily be the one which corrects the wrongs that give rise to this disease. In one, it will be wrongs of digestion that need attention, and the patient will need to have his diet restricted and fluids prohibited at meal-times. The bitter tonics will assist in bringing about a cure in these cases. Another will need to have hemorrhoids, pockets, fistulas, fissures, and papilla removed, or a uterus curetted or urethra dilated, before the trouble is overcome. To overlook these points is to court defeat.

During a paroxysm, the patient should be put to bed, his mind quieted as to the result of his case, and such remedies given as are especially indicated.

Gelsemium, if there be undue excitement, may be administered in full doses, a half dram or a dram to a half glass of water, and a teaspoonful every hour.

Aconite, in the small dose, is indicated where the pulse is small and rapid, and as this is the most characteristic symptom, we will find this remedy often beneficial.

Pulsatilla will be used where there is a sense of impending danger. The patient will have an anxious and frightened appearance.

Passiflora.—Where the patient is sleepless and uneasy, *passiflora* in

half or teaspoonful doses will give good results.

Morphia.—Where the rapidity is extreme, and the patient alarmed, a hypodermic of morphia will give the quickest relief. As a rule, the bromides and opiates had better be omitted.

BRADYCARDIA.

Synonyms.—Brachycardia; Slow Heart.

Definition.—Bradycardia is slowness of the heart, either normal, physiological (bradycardia), or pathological, which may be symptomatic and due to some other lesion, or it may be due to a neurosis.

Etiology.—There are some cases where the slow heart is seemingly physiological, the patient being apparently free from disease, though the pulse-rate may be only sixty. However, as many cases of organic heart disease are only made post-mortem, it may be a case of mistaken diagnosis.

The most frequent cause is the toxemia caused by the infectious fevers, especially typhoid fever, pneumonia, diphtheria, erysipelas, influenza, and acute rheumatism. Riegal, who analyzed 1,047 cases of bradycardia, where the pulse was less than sixty, found more cases from the acute fevers than from any other cause. Baumgartner cites a case where the pulse was only twenty-five, in the latter stage of diphtheria.

Next in frequency, according to Riegal, are wrongs of the digestive tract, 397 cases having, as a cause, some wrong of the gastro-intestinal tract. Chronic dyspepsia, either due to functional wrongs or from ulceration or cancer of the stomach, gives rise to more or less general enfeeblement, attended by a slow pulse. Wrongs of the liver act in the same way.

Bradycardia not infrequently follows confinement, owing, no doubt, to the severe exertion and the loss of so large an amount of fluid during delivery, causing exhaustion.

A slow pulse is found in diseases of the coronary arteries, sclerosis, stenosis of the aorta, and in degenerations of the myo-cardium.

Wrongs of the urinary apparatus may also be followed by bradycardia. Thus, in one case of nephritis with bladder complications, I rarely found the patient's pulse as high as sixty, it usually running about fifty.

Although tea, coffee, and alcohol usually give rise to tachycardia, they may also be responsible for bradycardia.

Chlorosis, anemia, diabetes, and kindred lesions, are sometimes the causes of slowing of the pulse.

Lesions of the nervous system are sometimes accompanied by slowing of the pulse; thus apoplexy, meningitis, tumors of the cerebrum and medulla, and injuries to the cord, give rise to bradycardia.

Bradycardia is sometimes associated with a neurosis, as seen in epilepsy, hysteria, mania, and paresis.

Pathology.—There are no changes characteristic of bradycardia, and when purely a neurosis, tissue changes are absent; and where structural change is found, it is rather a coincident than a result.

Symptoms.—The slow heart-beat, which is the characteristic symptom, may be temporary or permanent, and, when temporary, usually comes on suddenly and terminates in the same way, though a gradual return to the normal heart-beat is not uncommon. During an attack there may be twitching of the muscles, especially where it is epileptiform in character.

An attack may come on unannounced, or be preceded by ringing in the ears, dizziness, and a sense of impending danger. There may be great prostration, which is quite pronounced when myocardial degenerations have taken place.

The pulse-rate varies, and may be from fifty, to as low as eight or ten per minute, and is weak and small.

When the heart is very feeble, the impulse may not be felt regularly at the wrist, and sometimes the radial pulse intermits every other beat, the pulse at the wrist numbering but half the number of the regular heart-beats; for this reason auscultation should always be performed while taking the pulse-rate in bradycardia.

During an attack, the patient may have repeated attacks of syncope, or he may lose consciousness early in an attack, and remain so for hours, consciousness being followed by prostration and a sense of weariness.

Diagnosis.—The diagnosis is readily made by comparing the pulse-rate at the wrist with the heart-beat, which is determined by auscultation; they should be synchronous, and less than fifty in number per minute.

Prognosis.—The prognosis depends altogether upon the cause; thus, if the result of myocardial or cerebral degenerations, the case is hopeless, death usually occurring suddenly.

Treatment.—The treatment consists of two parts: First, to overcome the paroxysm; and, secondly, to prevent its return. For the first condition we usually resort to stimulants; ammoniated tincture of valerian, from thirty to sixty drops in a little sweetened water, is often beneficial. Strychnia, from the sixtieth to the thirtieth of a grain hypodermically, will also give prompt relief.

During the interval between attacks a careful study of the patient's condition should be made, and our treatment be directed to overcoming the exciting causes.

When there are no structural changes, such as degenerations of the heart and brain, the patient may be assured that his life is in but little danger. When the slow heart is due to reflex conditions, a careful search should be instituted, and the cause removed. In one case it may be due to sexual excesses; in another to rectal troubles; while a third may be traced to wrongs of the genito-uterine system.

Some cases do better under the influence of electricity than upon internal medication.

Where the bradycardia is permanent, tonics will replace stimulants. The patient's general health will need to be restored. Good digestion must be secured in order to make a good blood. For eighteen years I have taught my classes the benefits of a dry diet in correcting wrongs of the digestive apparatus in order to secure a good blood supply. Cactus will prove beneficial in these cases.

ARHYTHMIA.

Synonyms.—Irregular Heart; Allorhythmia; Delirium Cordis; Intermittent Heart.

Definition.—Arhythmia is a condition where there is either a drop, or skip in the beat—intermittent pulse; or where the volume or force of the pulse is not regular—irregular pulse.

Etiology.—The causes of arhythmia are many, and may act directly on the heart and its innervation, or indirectly, the irritation being at some distant part. Thus, of the first, disease of the heart and blood-vessels may be named. Pericarditis may so disturb cardiac innervation as to result in an irregular heart action; while an irregular or intermittent pulse may be the first symptom to direct our attention to myocarditis, either acute or chronic myocardial degenerations are often attended by arhythmia, though a morbid condition of the cardiac ganglia may not be detected.

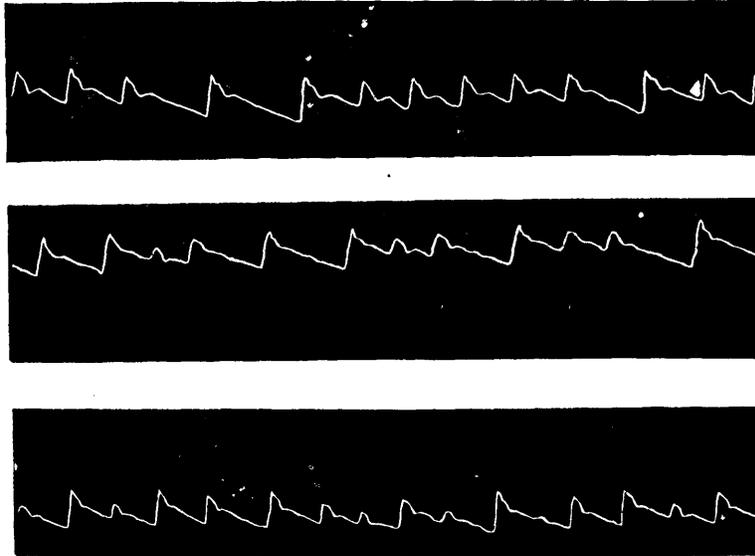
Gouty subjects suffering with arteriosclerosis also frequently have an irregular pulse, while valvular lesions are common causes. Cardiac innervation may be disturbed by organic lesions of the brain, such as concussion, hemorrhage, meningitis, or growths, or physical influences, such as grief, fear, melancholy, etc.

Perhaps the most frequent causes are the reflexes, most prominent of which is dyspepsia, especially when flatulency is a prominent feature. Wrongs of the liver and intestines may also give rise to arhythmia. In late years the attention of the profession has been turned to wrongs of the genito-uterine and rectal systems as being responsible for irregular action of the heart. Laparotomies are not infrequently followed by arhythmia.

The toxic influence of tea, coffee, alcohol, and certain drugs, as well as the infectious fevers, may give rise to an irregular or intermittent pulse. The excessive use of tobacco, especially cigarette-smoking, is responsible for arhythmia, and is known as the tobacco heart.

Occasionally we meet with persons, apparently in good health, whose heart action is very irregular. Such a case I have in mind, the first knowledge of which was obtained during an examination for life

insurance. That was some three or four years ago. I have examined the pulse and heart a number of times since, and always with the same result—arhythmia. The patient is the picture of health, and suffers no inconvenience from the trouble.



FIGURE; 24. EXAMPLES OF ARHYTHMIA.—(Goodno.)

Symptoms.—The irregularity may be in force, space, or time. The most common form is where an occasional beat is missed; it may be due to feeble ventricular contraction, the pulse-rate at the wrist being too weak to be perceptible. In other cases the intermission occurs with great regularity, each second, third, or fourth beat being absent. This pulse is known as the “*pulsus bigeminus*,” “*pulsus trigeminus*,” “*pulsus quadrigeminus*”

In the bigeminal pulse, the first beat is usually the stronger, and in some cases is the only one felt at the wrist.

The *pulsus alternans* is where every other beat is strong, followed by a weak one.

The *paradoxic pulse* of Kussmal is where the beats are more frequent and feeble during inspiration than during expiration.

Delirium cordis is where the pulse shows irregularity and inequality.

The fetal heart-rhythm, or embryocardia, is where the long pause is shortened, so that the first and second sounds are almost identical.

The *canter* or *gallop* rhythm is where, in a rapid pulse, the first, though more commonly the second sound, seems split, or a reduplication of the sounds. It has been likened to the triple sound of a horse at canter.

The arhythmia may impress the system so slightly that the condition is often discovered accidentally.

The diagnosis will be made by auscultation, at the same time the pulse is being taken at the wrist, or, what is better, by the use of the sphygmograph.

Prognosis.—The prognosis depends altogether upon the conditions giving rise to it. Many times the patient's health remains undisturbed for years, the arhythmia being discovered accidentally. When due to causes outside the heart, the prognosis is more favorable; but when due to valvular or myocardial changes, it is always more or less grave.

Treatment.—While the treatment depends upon the conditions found in each case, there are certain general instructions to be given in all cases, whether functional or structural.

Tea, coffee, alcohol, and tobacco are to be discontinued, the last two especially, while the former, if taken, should be used sparingly.

The diet is of importance. Only such food as is easily digested should be permitted, and all fluids at meal-times should be discontinued. Light exercise in the open air should be practiced daily, though, should there be structural wrongs, this should be carefully regulated.

Sexual excesses should be avoided, and all habits that produce exhaustion must be abandoned.

When the arhythmia is purely functional and due to reflexes, a thorough examination must be made to find the exciting cause.

Orificial surgery many times gives splendid results by removing exciting causes. If due to mental worry or overwork, a cure may be effected by change of occupation, change of climate, and change of scenes and mode of life.

Cactus.—Where the heart's action is feeble and the impulse at the wrist scarcely perceptible, cactus will give good results.

Veratrum.—Where the heart's action is wildly irregular and the pulse full, veratrum 10-15 drops, in half a glass of water, and a teaspoonful every one, two, or three hours, will be found beneficial.

Scutcllaria is a useful remedy, where the irregularity depends upon undue nervous excitation. If there is tumultuous action it may be combined with macrotys or lobelia.

Crataegus.—This remedy helps steady the heart's action, whether the trouble is due to functional or structural wrongs.

Digitalis.—This old heart remedy would be used in similar conditions to those calling for cactus; viz., feebleness.

Strychnia.—When due to excessive venery or masturbation, strychnia, one-sixtieth grain three or four times per day, will give good results.

Macrotys, *viburnum*, and *pulsatilla* will be found useful when the irregularity is clue to uterine derangements.

ANGINA PECTORIS.

Synonyms.—Stenocardia; Breast-Pang; Neuralgia of the Heart.

Definition.—Angina pectoris is an affection characterized by paroxysms of excruciating pain in the precordial region, extending into the neck, shoulder, and down the left arm, and attended by a sense of impending death. Nearly all writers agree that angina pectoris is not an independent disease, but is symptomatic of various cardiac affections, though it is occasionally due to some disturbance of the nervous system.

Etiology.—Angina pectoris is a rare disease, occurring usually after the fortieth year, and affecting mostly males. It may be divided into symptomatic, or true angina, and essential angina.

Symptomatic stenocardia is associated with cardiac affections, such as chronic myocarditis, various degenerations of the heart, aortic valvular

insufficiency, stenosis of the aorta, arteriosclerosis of the coronary arteries, and adhesive pericarditis.

Essential stenocardia is due to some disturbance of the nervous system, and is reflex. Thus it may be associated with wrong's of the stomach and bowels, the liver, uterus, ovaries, and rectal irritation. The excessive use of tobacco, and the inordinate use of coffee, tea, and alcohol, may act as producing causes. Influenza, Bright's disease, gout, and syphilis are also to be reckoned as disturbing forces.

It also occurs in hysterical patients and in emotional subjects.

Pathology.—While organic heart disease is found in all cases of true angina pectoris, there is no one lesion that is characteristic, and the same structural lesions that exist in angina, are often found without there ever having been any anginoid attacks.

The physiological explanation of anginoid paroxysms is summed up by Pepper in his work on practice as follows:

“1. Changes in the cardiac nerves have been noted by a number of observers, and angina is sometimes regarded as a neuralgia of the cardiac plexus. The close relation of the latter to the root of the aorta and of its continuation, the coronary plexus, to the coronary arteries, seems to offer an explanation of the frequency of angina pectoris in diseases of the aorta and coronary vessels. Lauceraux, Haddon, Leroux, and Rokitansky demonstrated pathological lesions of the plexus, the vagus, and the phrenic nerve, and Putjakin found alterations in the intercardial ganglia.

“2. Spasm or cramp of the cardiac muscle naturally suggested itself to Heberten and the older observers generally, but convincing proof of the existence of such a condition is lacking.

“3. Increased arterial tension and intracardiac tension seem undoubtedly the occasion of paroxysms in certain cases, as in aortic regurgitation and in the vasomotor angina of Nothnagle. The explanation would likewise apply to the cases of angina occurring in association with sclerosis of the aorta.”

Symptoms.—One of the distinguishing features of angina pectoris is

its paroxysmal character. In rare cases there may be premonitory symptoms, such as dizziness, ringing in the ears, nausea, or hot and cold flashes: but usually it comes on suddenly, and without the slightest warning. The patient suddenly experiences excruciating pain of a lancinating or rending character in the precordial region, which extends to the shoulders and neck and down the left arm to the finger-tips, which sometimes become numb and cold. Occasionally the pain extends to both arms. To the intense, stablike pain is added the undefinable and fearful sense of impending death, which is unmistakably written on the face, in the drawn features, the leaden, ashen gray, or livid color, and the surface covered with a cold, beady sweat. The patient rarely cries out, though suffering untold pain. He is afraid to make the slightest movement or outcry for fear of sudden death.

The breathing is shallow and irregular. The pulse is usually tumultuous and irregular, though it may be but slightly disturbed. The paroxysms usually last but for a few seconds, though they may last for an hour or more. Generally the pain subsides as abruptly as it began, though the paroxysms frequently terminate with nausea and vomiting, or eructations of large quantities of gas; or the patient, not infrequently, voids a large quantity of pale urine or has a loose bowel movement.

Following the paroxysm, the patient seems quite exhausted, and may remain so for several days. The attacks vary in frequency from a few days' interval to several years.

In rare cases the paroxysm terminates in unconsciousness, and in still rarer cases in sudden death.

In false angina, the patient is more apt to be restless and to cry out with pain.

Diagnosis.—The diagnosis is usually not difficult. The sudden onset, the excruciating character of the pain with no outcry, the agonized expression portending death, the ashen-gray color and bedewed face, the shallow, irregular breathing, and the equally sudden termination of the paroxysm, the age and sex of the patient (the case being usually that of a male past forty years of age),—are characteristics that can hardly be mistaken for any other disease.

Prognosis.—The prognosis depends almost entirely upon the causes

giving rise to it. True angina, however, is always a grave disease, and may terminate fatally during a paroxysm. The extent of the cardiac changes would, of course, determine largely the outcome. If there be extensive sclerosis of the coronary arteries, the prognosis would be unfavorable, and we might reasonably expect a sudden termination of life. On the other hand, if it be neurotic in character, the prognosis will be favorable.

Treatment.—The treatment consists of two parts. I. Prophylactic ; 2, to relieve the paroxysm.

Prophylactic.—The conditions that give rise to the paroxysms should be carefully studied, and the remedies directed, as far as possible, to correct such wrongs. Crataegus, cactus, strophanthus, nux vomica, digitalis, apocynum, and the iodide of arsenic should be thought of in this connection.

The diet should be nourishing and easily digested, and all fluids restricted at meal-times. Severe physical work or exercise should be abandoned, and everything tending to greatly excite the emotions, as well as the heart's action, avoided. Dissipation of all kinds should be stopped; late hours and irregular habits corrected. Change of climate and an out-of-door life, such as would improve the general health, would be beneficial.

Lobelia.—During a paroxysm, a teaspoonful of the specific tincture of lobelia will give prompt relief, or we may combine with it a half-teaspoonful of macrotys. As to local applications, both hot and cold are recommended. Nitrate of amyl also gives prompt relief when inhaled. A perle containing two to five drops, may be crushed in the handkerchief and inhaled.

A physician of my acquaintance who suffers with angina tells me that he gets greater relief from iodide of arsenic than from any other remedy. He makes a tincture by adding ten grains of the crude drug to one ounce of alcohol, and of this he adds ten drops to a half glass of water, and takes a teaspoonful every twenty, thirty, or sixty minutes.

Nitroglycerin will also give speedy relief in some cases. The patient should be quiet for a few days following an attack.

V. DISEASES OF THE ARTERIES.

ACUTE AORTITIS.

Definition.—Acute aortitis is an acute inflammation of the intima of the aorta, similar to that met with in the endocardium.

Etiology.—Nearly all cases of aortitis are preceded by some one of the infectious diseases,—typhoid fever, diphtheria, scarlet fever, tuberculosis, and others of the same class. Syphilis, rheumatism, and alcoholism are also important factors in producing' the disease. It may be an extension of endocarditis.

Pathology.—The morbid changes are so similar to those of acute endocarditis that a minute description of the pathological changes are unnecessary. As a result of increased cell proliferation, there is a thickening of the intima, some points of which are more pronounced than others, and these local spots may be covered by fibrinous deposits. These excrescences vary from the size of a shot to that of a cherry. Ulceration may follow, and portions of these fibrinous masses floating off may give rise to embolic infarcts.

Symptoms.—The local symptoms are usually characterized by pain, more or less severe, in the precordial region, or it takes the form of a tenderness and soreness on pressure in the sub-sternal region.

Sometimes the pain is intense, resembling angina pectoris.

There is usually some fever, though never marked unless embolism occurs; in such cases, rigors, night-sweats, and a high temperature curve, with prostration, will be the additional symptoms.

Palpitation is not uncommon, and a cardiac murmur may be heard over the region of the aorta.

Diagnosis.—A positive diagnosis' is almost impossible. The thoracic pain, more diffuse than in endocarditis, and the high seat of the murmur, would suggest aortitis.

Prognosis.—The prognosis is always unfavorable, as a fatal embolism

or rupture of the aorta may occur without warning at any time.

Treatment.—The treatment will be along the same lines recommended for acute endocarditis, and will consist of rest, an easily digested diet, and the indicated remedy.

ARTERIOSCLEROSIS.

Synonyms.—Endarteritis Chronic Deformens; Atheroma; Arterial Sclerosis.

Definition.—Arteriosclerosis is an inflammatory and degenerative condition of the arterial system, primarily of the intima, although later degenerative changes may involve the whole structure. Calcarine deposits are quite common.

Etiology.—The predisposing causes of arteriosclerosis are old age and heredity. We may say that it is essentially a disease of old age, the large majority of cases occurring in persons past the age of forty.

When we remember the work of the arteries, however, day and night, awake or asleep, it is not surprising that, in the feeble, the arterial system ages rapidly. Occasionally we find the disease in persons between the ages of twenty-five and thirty. The inheritance bequeathed to the offspring is frequently a feeble circulatory apparatus, and it is not uncommon to find several members of a family suffering from the same disease.

In some cases it may almost be said to be a physiological condition, the result of the constant work of many years.

The causes that figure most frequently in bringing about this condition of the blood-vessels are, first, the toxins generated by certain diseases.

Syphilis may head the list, and following close in its train may be named alcoholism, its twin brother. Rheumatism, gout, and tuberculosis act in the same way, while typhoid fever, scarlet fever, diphtheria, influenza, and the malignant diseases may so impregnate the blood as to be considered important factors in producing the disease.

Overeating and Drinking.—The overfilling of the blood-vessels, that follows the ingestion of large quantities of fluids and solids, is regarded by many writers as an important factor in producing the disease. Overwork, whereby increased vascular tension results, also contributes to this condition. Athletes, boiler-makers, miners, and all who perform great and prolonged physical exertion, invite this condition.

Renal Disease.—Quite a difference of opinion exists as to the part Bright's disease plays in the etiology of arteriosclerosis. Some believe that, by increasing the blood-pressure, these secondary results follow, and there seems good reason to believe that in some cases Bright's disease is the primary lesion from which the sclerosis can be traced. On the other hand, we find some cases of chronic nephritis which seem to be due to arteriosclerosis.

Pathology.—The tissue-changes of the coats of the vessels may be divided into two kinds—the localized or nodular, and the diffuse—though, in most cases, there is a combination of the two.

The most frequent seat of the election is in the aorta and coronary; the splenic, iliac, femoral, cerebral vessels; the uterine, bronchial, internal spermatic, common carotid, and hypogastric following next in frequency, according to Rokitansky. The vessels of the stomach and mesentery are but seldom affected. When there is impairment of the pulmonary circulation, as in mitral stenosis, the pulmonary vessels may become sclerotic.

Localised or Nodular Arteriosclerosis.—As a result of proliferation, infiltrated areas begin in the middle and outer coats. These nodules vary in size from that of a small shot to that of a large coin. As they increase in size, the intima loses its smoothness and becomes thickened and rough, and appears yellow over the seat of the lesion. As these changes progress, the middle and outer coats are weakened, but compensatory changes occur in the intima, which result in thickening of the intima, already noted.

Later, necrosis may occur within these atheromatous spots, giving rise to atheromatous abscesses. When these rupture upon the intima, an atheromatous ulcer is the result. In place of this, calcification may occur in these plates. Should the intima undergo softening or liquefaction, dilatation is apt to follow, giving rise to aneurism.

While these changes usually occur in the aorta, they may also occur in the smaller vessels.

Diffuse Arterio-sclerosis.—In this form the change in the coats of the vessels extends throughout the greater part of the arterial system, and in some cases invades the capillaries and veins—angina sclerosis. Even in the diffuse form, however, there is apt to be nodular areas in the aorta. The intima, though smooth, is much thickened by proliferation of the sub-endothelial tissue, while the muscle fibers in the media and adventitia may almost entirely disappear, being replaced by fatty, necrotic, and hyalin degeneration.

In senile arterio-sclerosis calcareous deposits occur, which render the vessels rigid. Where these tissue-changes involve the capillaries, there may be complete obliteration of their lumen in some places, notably the kidneys.

As a result of the narrowed caliber of the vessels, nutrition is defective, and atrophy of the liver, kidney, and spleen may result. The increased work thrown upon the heart, however, generally results in hypertrophy of this organ.

Symptoms.—The disease may come on so insidiously, and the general health be so little disturbed, owing to compensatory change in the heart, that the disease may never be suspected during life, and only revealed on autopsy. At other times, while examining our patient for some other disease, the increased tension of the pulse, the accentuated aortic second sound, will draw our attention to the existing change in the vessels.

A uniform picture of arteriosclerosis can not be given; for the symptoms depend largely upon the vessels involved, and we will have to consider various types depending upon the parts involved, as cardiac, cerebral, renal, and peripheral arteriosclerosis.

Cardiovascular Type.—The symptoms will depend, upon the degree of the arterial tension. The pulse at the wrist, as a result of thickening of the arterial walls, is hard and incompressible, and, if calcification has taken place, can not be felt on palpation. The artery in such cases feels like a rigid cord, or, if nodulated, feels like a bird's neck.

The pulse-rate is usually diminished, and, when compared with the apex-beat, shows a decided retardation, due to want of elasticity of its coats. This slow pulse is known as the “pulsus tardus.”

The sphygmograph shows a characteristic tracing in the gradual ascent, the broad top, and equally gradual descent, with the dicrotic notch almost, if not entirely, obliterated. The increased arterial tension, caused by inelasticity of its walls and increased action of the heart to propel the necessary blood supply, causes hypertrophy of the left ventricle, which may be recognized by increased dullness downward and to the left, and by the accentuated ringing second sound.

When the hypertrophy is sufficient to compensate for the resistance due to rigid walls, the health is but little affected, and the disease may be overlooked. When myocardial degenerations take place, the first sound of the heart is very weak, and often a systolic murmur can be heard at the apex. Palpitation often occurs, and if slight exertion is made, dyspnea becomes marked. There is more or less constriction, and if the coronary arteries are involved, angina pectoris is not uncommon.

Cerebral Type.—The first evidence of this form may be headache, more or less intractable, melancholy, dizziness, with ringing in the ears. Attacks of vertigo, especially on slight exertion, are quite frequent. As the disease advances hemiplegia or aphasia may occur. The memory becomes treacherous and the intellectual faculties generally fail.

Renal Type.—The symptoms differ but little from those of atrophy of the kidneys, and result from a diminished blood-supply due to the sclerotic vessels.

Peripheral Type.—In this form the arteries leading to the extremities become so obstructed as to practically cut off the blood-supply, the extremities become cold and lifeless, and gangrene follows.

Diagnosis.—Where the disease is well marked, the diagnosis is usually comparatively easy. The increased arterial tension, thickening of the temporal, radial, bronchial, and femoral arteries, which may be recognized by the hard, cordlike feel; the hypertrophy of the left ventricle, as shown by dullness to the left and downwards; and the accentuation of the second aortic sound,— make a group of symptoms that can hardly be mistaken for those of any other lesion.

Prognosis.—As to completely curing or removing the sclerotic condition, the prognosis is unfavorable, but the patient may be assured of fairly good health and probably years of life. Nature provides against starvation of the tissues by compensatory changes in the heart, which compensation may be maintained for years. Finally, however, degenerations may occur, and the blood supply is not sufficient for the purpose of the body, and tired nature succumbs to the inevitable.

Treatment.—In the treatment of this disease we need the cooperation of the patient, and it is best to explain to him his true condition, that he may the more readily acquiesce in the restrictions placed upon him.

Alcohol and all intoxicating liquors should be absolutely forbidden, as well as the use of tobacco. Dissipation of all kinds must be avoided, and regular habits enjoined. The diet should be nourishing, but easily digested, and fluids should be restricted at meal-times and for one or two hours thereafter.

When possible, the patient will do better if taken to an equable climate, where there is plenty of sunshine, and he can be much out of doors. The altitude must not be too great. A quiet life should be enjoined.

Where syphilis is present, Donovan's solution of arsenic, phytolacca, and echinacea will be found useful, while the iodides will be the favorite remedies with many, especially the iodide of potassium and the iodide of lithium. The lithiates for the kidneys. with an occasional saline for the bowels, will give some relief. Cactus, collinsonia, carduus marianus, strophanthus, and like remedies may be of some benefit when myocardial degeneration? have taken place, though medicines in most cases will but feebly influence the disease.

ANEURISM.

Definition.—An aneurism is a circumscribed dilatation of an artery, formed by the giving away of one or more of its coats, and may be sacculated, fusiform, or cylindrical in form. Several forms or types are recognized.

(a) The *true* aneurism is where the sac is formed of one or more of its

coats.

(b) The false aneurism is where there is a rupture of the coats, and the blood is found in the adjacent tissues.

(c) A dissecting aneurism is where there is a rupture of the intima, and the blood burrows, or dissects, between the walls of the vessel. The aorta is the usual seat, and may be traversed its entire length.

(d) A miliary aneurism, so named from its minute size, is found in the cerebral vessels.

(e) An arterio-venous aneurism is where the dilatation occurs in veins, the result of a communication being established with an artery.

These various forms are termed axial, when the entire circumference of the vessel is involved in the dilatation, and peripheral when the dilatation involves but one side of the artery.

Etiology.—Age and sex are predisposing factors, the disease occurring between the ages of thirty and fifty, and mostly in men. An enfeebled condition of the walls of an artery is necessary for the development of an aneurism. This may be congenital or it may be acquired. The most frequent cause is arteriosclerosis, especially during the early stage, before compensatory changes have taken place.

Syphilis, gout, rheumatism, alcoholism, lead poisoning, uric acid, Bright's disease, and diabetes, are conditions that enfeeble all tissues, the arterial coats not excepted.

Severe exertion or strain is also responsible for a weakening of the vascular walls, and how much is due to strain, and how much is due to syphilis, in soldiers and sailors, is difficult to state, though each figures quite extensively.

Traumatism should be considered as an etiological factor, and heavy body bruises or blows upon the chest would tend to weaken vascular walls.

Embolism.—The plugging of an artery by an embolus is apt to result in the development of an aneurism on the proximate side of the

obstruction.

Mycotic Aneurism.—More recent examinations tend to show that, in some cases, the weakened condition of the walls is due to micro-organisms; at least an abundant product of micro-organisms have been found present in the aneurismal sac.

Pathology.—There is generally degeneration of the arterial walls, arterio-sclerosis being frequently present. In some the intima and media have been destroyed, the adventitia being the retaining wall. The blood in an aneurismal sac may become laminated and of a fibrous character, thus restoring the arterial wall. There is generally hypertrophy of the heart due to arteriosclerosis.

Aneurism of the Thoracic Aorta.—Of over nine hundred cases of aneurism collected by Crisp, forty-five per cent were found in the thoracic portion of the aorta, and eleven per cent in the abdominal aorta, or about seventy-five per cent of all aortic aneurisms were located in the thoracic portion, over fifty per cent being found in the ascending portion, and diminishing in frequency as the distance from the heart increases.

Symptoms.—The symptoms depend largely upon the size and location of the aneurism. If small, the disturbance is so slight that it is not recognized; but as it increases in size and interferes by pressure, characteristic and distressing symptoms make their appearance. Should the aneurism be located where there is plenty of room for development without pressing upon important parts, it may attain a large size without local or systemic disturbance. Since location determines the symptoms, we will consider them according to the part affected.

(1) *Aneurisms of the Ascending Portion of the Arch.*—If the aneurisms are small and near the sinuses of Valsalva, they may remain unsuspected till a sudden termination of life reveals a ruptured aneurism in the pericardium.

When located above the sinuses, earlier and more pronounced symptoms are present. Thus, if the aneurism be located on the right or convex border, the pressure would be against the superior venae cavae, which would result in congestion and edema of the upper extremities, or the pressure may involve only the sub-clavian, resulting in enlargement

and edema of the right arm.

They may attain very great size, pushing out into the pleura or forward against the sternum and ribs, causing erosion, and finally appearing beneath the skin as pulsating, bluish tumors. They may press against the right recurrent laryngeal nerve, which will be followed by dyspnea and apnea.

Should the aneurism be located on the left or concave border, the pressure would cause displacement of the heart, forward, downward, and to the left. Pressure on the inferior venae cavae would cause dropsy of the lower extremities.

Death is usually sudden, the warning symptoms being intense pain, cyanosis, and dyspnea. It may rupture into the pleura, pericardium, superior venae cavae, or externally, according to location.

(2) *Aneurism of the Transverse Portion of the Arch.*—The most pronounced symptoms occur when developed in the transverse portion, owing to the small amount of space afforded for their development, and consequently they exert greater pressure upon neighboring parts.

It may extend in the usual direction, backward, and press in against the trachea, causing a ringing, paroxysmal, metallic cough, with more or less dyspnea, or, pressing against the esophagus, cause difficulty in swallowing. The tumor may press against the bronchi, which embarrasses respiration, and is attended by severe attacks of paroxysmal coughing, with watery or muco-purulent expectoration. Marked dilatation of the bronchi may follow. When very large, the tumor may press against the lung, giving rise to severe pulmonary symptoms, and, in time, to suppuration, termed by Osier, aneurismal phthisis.

The left recurrent laryngeal may be affected with the same result as where the right was involved; viz., cough, dyspnea, and aphonia. The aneurism may encroach upwards, involving the carotid and subclavian on the left side, or the carotid and innominate on the right. The sympathetic may be involved by pressure, resulting in dilatation of the pupil where the irritation is slight; but where more severe, paralysis may follow, with the contracted pupils. Where the thoracic duct feels the encroaching tumor, general atrophy follows.

When the aneurism develops on the anterior portion of the arch, it encroaches upon the sternum, and by continued pressure may cause severe erosions. The aneurism may develop to an enormous size, encroaching upon both the right and left pleura, crowding both the lungs.

(3) *Aneurism of the Descending Portion of the Arch.*—The pressure is mostly backward against the vertebrae, extending from the third to the sixth dorsal, oftentimes causing erosions. It may make its way to the scapula, and project as a pulsating tumor. There is sometimes compression of the cord, which is attended by great suffering. The esophagus and bronchi may be pressed against, with dysphagia and bronchiectasis, to which reference has already been made.

The descending portion of the thoracic duct is generally involved near the diaphragm, the tumor lying against the lower dorsal, which may be severely eroded.

Wherever located, pain is always a distressing and prominent symptom. If the aneurism is of sudden development, as sometimes occurs under great physical exertion, the patient experiences a sudden “giving way,” due to rupture of the tunica media, and attended by a sharp pain in the upper part of the chest.

The pain in later stages may be the result of stretching of the nerve filaments in the Walls of the aorta, or it may be due to pressure upon the adjacent parts. Where there is erosion of the bone, as of the sternum or vertebrae, the pain is of a boring character, and causes great suffering. In rare cases the tumor may develop to great size, with but very little pain, even where there has been erosion of the bone.

Physical-Signs.—The physical signs, like the general symptoms, depend upon the size and location of the tumor; if small and deep-seated, a physical examination may fail to reveal the tumor.

Inspection.—One of the most important and characteristic signs is a pulsating tumor of the chest, and, though there may be no protrusion of the walls of the chest in the early stage, by standing at the patient's side, and having the light strike him obliquely, a pulsation synchronous with the systols of the heart may be revealed.

If the aneurism be of the ascending arch, the pulsation will be to the right of the sternum and in the second or third interspace. If located in the transverse portion, the sac will be behind the manubrium, though it may be seen pulsating at the supra-sternal notch. Where the innominate artery is the seat of the aneurism, the pulsation will be seen above the second rib extending into the neck.

If the descending portion be involved, the pulsating tumor will be seen to the left of the spinal column, extending into the scapular region.

In some cases there is marked bulging of the tumor, caused by erosion of the sternum or ribs or perforation of the back. They vary in size from a billiard-ball to a cocoanut. The skin covering the tumor is thin, smooth, and stained a dark-red color. If the aneurism be large, the apex-beat will be displaced downwards and to the left.

When there is compression of the superior cava, there will be cyanosis of the head, upper chest, and arms. Where there is compression of the inferior cava, the abdominal walls and legs will be congested and dropsical.

Palpation.—When inspection fails to reveal pulsation, the tumor is deep-seated, and may be recognized by palpation. By placing one hand over the sternum and the other over the spine, a strong, heaving pulsation is imparted to the hand, radiating in every direction, and known as the expansile pulsation. When the tumor has perforated the chest, and the hand can grasp the tumor, this expansile character is much more marked.

One of the signs of great value in aneurisms is the diastolic shock imparted to the hand on palpation, and is synchronous with the closure of the aortic valves. This usually occurs when the aneurism is at the root of the aorta. Where there is dilatation of the arch, a systolic thrill is sometimes present.

Percussion.—Where the aneurism is deep-seated, percussion will most likely give negative results; when the tumor reaches the chest-wall, however, an abnormal area of dullness is heard, the location depending upon the part affected: thus, if the aneurism be located in the ascending portion of the arch, the dullness will be to the right of the sternum and

above the third rib. If situated on the transverse portion, the dullness will be over the sternum and to the left; while if on the descending portion, it will be heard in the left interscapular region. The sound is peculiarly flat.

Auscultation.—Auscultation may reveal murmurs that are characteristic, or give negative results, depending upon the thickness of the laminae of fibrin. The most characteristic sign is a systolic murmur heard over the area of dullness, and transmitted to the carotids, and if there be aortic insufficiency a diastolic murmur will also be heard. In large aneurisms of the arch a loud, ringing, accentuated second sound is a sign of diagnostic value.

Drummond speaks of a systolic murmur heard in the trachea, due to the expulsion of air at each distention of the sac.

A physical sign of importance is a slowing of the pulse in the arteries beyond the aneurism, the sac acting as a reservoir, breaking the force of the systole. When the sac is very large, there will be an absence of pulsation in the abdominal and femoral arteries from the same cause.

The two radial pulses may show a marked difference in time; thus, if the aneurism be situated in the transverse portion of the arch and the innominate is not involved, the pulse at the right wrist and in the neck is strong and almost synchronous with the systole of the heart, while the pulse of the left wrist is small, weak, and retarded.

Surgeon-Major Oliver described what at one time was regarded as a very valuable sign, a *tracheal tugging*. The patient is directed to sit or stand in an upright position, close the mouth, and elevate the chin. The cervical cartilage is then grasped between the finger and thumb and elevated till the trachea is tense, when there will be a downward dragging or tugging at each systole. When taken with other signs it is valuable: but alone, it will not be of much weight, as it is sometimes found in health and in other diseases

Diagnosis.—The diagnosis of an aneurism is sometimes quite difficult, if not impossible. In some cases the symptoms are so obscure and the aneurism so deep-seated that, after the most careful examination, the lesion may not be discovered. This is especially true if the aneurism be small and located in the sinuses of Valsalva.

If the patient's occupation has required great physical exertion, and his age is between thirty and forty-five, and should there be a history of arterio-sclerosis coupled with obscure thoracic pains, sudden attacks of intense pain and anginoid in character, or pain radiating along the bronchial plexus or intercostal nerves, we would think of aneurism.

If to these symptoms are added dyspnea, dysphagia, aphonia, and cough, either of a dry, ringing, metallic character—laryngeal, or loud, and hoarse,—bronchial, with profuse expectoration; or if there be edema and congestion of the upper extremities, and if a physical examination reveals dullness in the aortic region, a systolic murmur, and a systolic and diastolic accentuation of the second sound, change in the character and time of the pulse, and the marked difference between the left and the right pulse, and if to all these symptoms be added, the tracheal lugging, the diagnosis is assured.

A differential diagnosis has to be made between aneurisms and solid tumors, pulsating empyema, pulmonary tuberculosis, and abnormal pulsations of the aorta. The tumors that are most likely to be confused with aneurisms, are cancers, sarcomas, and 'glandular enlargements of the mediastinum.

If we bear in mind, however, that while tumors and enlarged glands may give rise to all the pressure'symptoms of aneurisms, abdominal pulsation is either lacking, or, if present, is quick, not slow and expansive.

There is also an absence in growths, of the systolic thrill on palpation, and the diastolic shock is missing. On auscultation, if there be a tumor, the systolic murmur is either absent or very faint, and accentuation of the second sound is not perceptible. There is also uniformity in the radials, and tracheal tugging is not present. If the growths be malignant in character, or a sarcoma, there will be greater evidence of malnutrition, more emaciation, and that peculiar appearance designated as cancerous cachexia. The pain is more constant, and there is enlargement of the axillary and cervical lymphatics.

Pulsating Empyema is not so circumscribed, but covers a more superficial area, is not expansile, and is produced by respiratory movement.

Auscultation fails to reveal the characteristic murmur or diastolic shock and there is no retardation of the radial pulse. On the other hand, chills, hectic fever, night-sweats, and emaciation, characteristic symptoms of empyemia, render the case a plain one.

Pulmonary Tuberculosis.—Where an aneurism presses a bronchus, causing dilatation, attended by cough and profuse expectoration, followed by fever and emaciation, the symptoms may be mistaken for phthisis; in the latter, however, the history, the night-sweats, greater fever, and emaciation, will enable us to distinguish the one from the other.

Abnormal Pulsation in the Aorta.—We meet with abnormal pulsation in neurotic patients, generally females, and occasionally in retraction of the lungs.

Curvature of the spine may give rise to displacement of the aorta, with forcible pulsation. When these conditions are present, there is one marked difference from aneurisms; namely, absence of retardation of the pulse and the characteristic expansile pulsation. The perfecting of the use of the X-ray will most likely render the diagnosis of aneurism positive.

Prognosis.—The outlook in thoracic aneurism is always serious, and though recovery may take place spontaneously, and by treatment, the tendency is towards a fatal termination, and recoveries are very rare.

Rupture and sudden death may occur at any moment. According to Lebert, who examined a large number of cases, the duration of life, from the time the first distinct symptom made its appearance until death, was from fifteen to eighteen months. The patient's occupation and habits will determine to a certain extent the length of life. Where an even, quiet life is led, the patient may live for years.

Treatment.—To effect a cure we must resort to such measures as will promote coagulation of the blood and bring about contraction of the sac, and any treatment that favors this condition will be highly beneficial.

To accomplish this, the late Dr. Tufnell, of Dublin, advocated rest in bed and a dry diet. By these means the fluids in the system are reduced to

the minimum, arterial tension is lessened, the amount of fibrin increased, and the number of heart-beats greatly lessened. Tufnell's diet list was very rigid and consisted of the following bill of fare: For breakfast, two ounces of bread and butter and two ounces of milk; for dinner, two or three ounces of meat and two ounces of milk or claret; for supper, two ounces of bread and two ounces of milk.

There is no doubt that rest in bed and the above rigid diet would greatly lessen the number of heart-beats and lessen the quantity of fluids in the body, but there are few patients who would submit to such a quiet life and so rigorous a diet; indeed it is not necessary to go to quite such extremes, though the patient must lead a quiet life, and his fluids should be restricted.

Tea, coffee, and alcohol should be forbidden, and his allowance of water reduced to eight or ten ounces per day.

To produce coagulation of the blood, the introduction of fine wire, horsehair, or fine catgut has been practiced with some degree of success. Galvano-puncture, electrolysis, and the injection of styptics have been used for the same purpose. There is always some danger, however, attending these local measures, since coagulated particles may float off and give rise to embolism. Ergotine dissolved in water or glycerin has been injected directly into the sac, in the hopes of inducing contraction in the smooth muscles of the walls of the aneurism.

Iodide of potassium has been largely used by the old school as a remedy for aneurism.

The pain, when very severe, will call for morphia. The calcium salts are thought to influence the process of clotting, and may be used, though too much reliance must not be placed in their efficacy. The natural mineral waters may be freely used to prevent constipation.

ANEURISM OF THE PULMONARY ARTERY.

Aneurism of the pulmonary artery is very rare, and is usually sacculated or fusiform; extreme dilatation, however, is not uncommon, and may result from mitral stenosis, phthisis, emphysema, or any affection that obstructs the lesser circulation. When extreme, there will

be insufficiency of the semilunar valves.

Symptoms.—If the aneurism be large, the symptoms will be similar to those of aneurism of the thoracic aorta; usually, however, the aneurism is small and the symptoms are negative. If there be extreme dilatation, there will be regurgitation into the right ventricle, which will be attended by cyanosis, dyspnea, and more or less cough.

Physical examination reveals a pulsation in the second or third interspace and to the left of the sternum. On palpation, the systolic pulsation is attended by a thrill and diastolic shock.

Percussion reveals a dull or flat sound, over the area of pulsation (second interspace). We are to remember, however, that where there is retraction of the lung, the percussion note will be dull, though neither dilatation nor aneurism exist.

Auscultation may reveal a loud, superficial, systolic murmur to the left of the sternum, over the second interspace.

The prognosis is unfavorable, and the treatment will be on the same principles as outlined for thoracic aneurism.

ANEURISM OF THE CORONARY ARTERY.

The coronary artery may be the seat of aneurism due to weakened condition of its walls, the result of arterio-sclerosis. It is very rare, however, and not discoverable during life.

ANEURISM OF THE ABDOMINAL AORTA.

Aneurism of the abdominal aorta arises from the same causes that give rise to aneurism of other parts. It occurs far less frequently than thoracic aneurism, however, and is found far more often in men than in women. It is usually saccular or fusiform, and located near the celiac axis.

Symptoms.—The most common as well as constant symptom, is pain, which may be sharp and lancinating in character, extending to the back

and around the abdomen, or it may be of a dull, boring character, when there is erosion of the vertebra. There is, nearly always some gastric disturbance, attended by vomiting and pain, and jaundice is a common symptom. When the pressure is upon the liver, spleen, or kidneys, congestion of these organs follows, with the usual accompanying symptoms.

If there is erosion of the vertebra, paraplegia may follow. In rare cases the aneurism may perforate the diaphragm, and rupture into the pleura or lung. When it arises from the anterior wall, it may form a well-defined tumor. There is generally retardation of the femoral pulse.

Physical Signs.—Inspection may reveal pulsation in the epigastric region; and if the aneurism be on the anterior wall, a well-defined tumor is visible. The pulsation is heaving and expansile. and if near the diaphragm it is double; a diastolic shock from the heart may be recognized. A systolic thrill is sometimes noticeable.

Percussion reveals an abnormal area of dullness.

On auscultation, a soft bruit or murmur can usually be detected.

Diagnosis.—If we bear in mind that the pressure of a tumor that can be grasped in the hand, and which has a heaving, expansile pulsation, is the only positive diagnostic symptom, we will avoid many errors. The throbbing aorta occurs in neurasthenia and in anemia, and must not be mistaken for aneurism. It sometimes happens that a tumor of some of the abdominal viscera will be lifted with each abdominal pulsation, and may simulate aneurism.

If the patient be examined in the knee-chest position, however, the tumor will drop forward, when the pulsation ceases. It is also noticeable that the pulsation is not expansile.

Prognosis.—The prognosis is exceedingly grave, though very rarely, a case heals spontaneously. Sudden death may occur from rupture into the pleura, peritoneum, or intestines, or it may occur by obliterating the lumen by clots. Sometimes death is the result of paralysis induced by erosion of the vertebra.

Treatment.—The treatment of abdominal aneurism will be on the

same principle adopted for thoracic aneurism. Firm pressure for hours, the patient being anesthetized, has been practiced with some degree of success, though there is always danger of rupturing the aneurism by this means.

ANEURISM OF THE CELIAC AXIS.

This sometimes occurs in connection with aneurism of the upper portion of the abdominal aorta. Aneurism of its branches may occur, though it is quite rare. The symptoms of aneurism of the splenic and hepatic arteries are somewhat vague or similar to wrongs of the spleen and liver.

The tumor rarely ever reaches a large size, and the diagnosis is made post-mortem.

ARTERIO-VENOUS ANEURISM.

Definition.—Arterio-venous aneurism is an abnormal communication between an artery and vein, and known as *varicose aneurism* when a sac lies between the two.

When the communication is direct, the sac being absent, it is known as aneurismal varix. Venesection is generally responsible for this condition, the piercing of an artery and vein occurring during the operation. This accounts for the frequency with which it occurs at the bend of the elbow. Accidental puncture may occur at other parts.

Symptoms.—Sudden distention of the veins of the upper part of the body, cyanosis, and edema are the most characteristic symptoms.

Auscultation reveals a continuous thrill and buzzing, whizzing or humming murmur, intensified during systole.

Treatment.—When the aneurism is superficial, surgical measures will correct the difficulty; but if it be a thoracic arterio-venous aneurism, the same treatment will be pursued as recommended for the arterial variety.