PART II
DISEASES OF THE CIRCULATORY SYSTEM

GENERAL DISCUSSION

The heart is subject to the effects produced upon the circulation of the blood both through variations in its nervous control and through variations in the resistance offered to the expelling force of the systole. The manner in which the nervous mechanism may be deranged is best understood by reference to physiological relationships of the cardiac nerve centers.

The vagus nerve carries inhibitory impulses to the cardiac ganglia. It carries sensory fibers which may reflexly affect the heart's action, and also the tension of the blood vessels over the body, especially of the splanchnic region.

The white rami of the upper thoracic region carry impulses which increase the speed and the force of the heart's beat to the superior and the middle cervical sympathetic ganglia. The gray fibers from these ganglia join the vagi and are carried with them to the heart, where they are distributed to the muscle fibers. The same pathway which carries the augmentor and accelerator impulses to the heart carries also, probably, impulses which influence the size of the cardiac blood vessels. This matter is not proved. Also, viscero-sensory nerves are carried from the heart, upward with the vagi, and thence in the path of the cardiac accelerators, to the heart center in the upper thoracic segments.

Both the vagus center and the upper thoracic centers are controlled by a general heart center in the medulla, which may or may not be identical with the vagus center.

The heart may be diseased by the following conditions: abnormal pressure in the blood vessels; poisonous substances in the blood stream and starvation through poor blood; local infection; abnormal positions of the first to the fifth thoracic vertebræ and ribs; and muscular tension in the cervical region, of such a nature as to press the pulsating carotid more closely against the vagus fibers; abnormal sensory impulses carried to the centers from other parts of the body, usually viscera innervated by the vagus.

The diagnosis of many obscure cardiac conditions is often very much facilitated by the aid of the Roentgen ray. Relative increases in size, shape, and density are shown positively. Abnormalities of position and sometimes the adhesions causing them can be determined in no other way as accurately. Stereoscopic radiographs are especially valuable in conjunction with the fluoroscopic examination.

CHAPTER XIII

DISEASES OF THE PERICARDIUM

ACUTE PERICARDITIS

(Acute plastic or dry pericarditis; acute fibrinous pericarditis)

Acute pericarditis is an inflammation of one or both layers of the outer covering of the heart.

Etiology. The exciting causes are rheumatism and gout, eruptive fevers; pneumonia; septicemia and pyemia from whatever cause; tuberculosis, nephritis, and extension of disease processes from neighboring parts.

Subluxations of the fourth and fifth thoracic vertebræ, the first to fifth ribs, the clavicles, the atlas and axis, are found very constantly in pericarditis. The reflex muscular contractions involve most constantly the third, fourth and fifth thoracic and the upper cervical segments. The lesions mentioned must be considered important etiological factors.

Pathology. In fibrinous pericarditis, the usual form, there is hyperemia with loss of luster. The exudation of fibrin gives the pericardial surfaces a peculiar shaggy or "bread and butter" appearance. The process often stops at this stage, constituting the plastic form. In pericarditis with effusion, the serous or sero-fibrinous fluid may amount to two litres. This type is most frequently associated with acute rheumatism, tonsillitis, tuberculosis, and septicemia. The absorption of the exudate may result in slight or extensive adhesions which may permanently hinder the cardiac action.

Suppurative pericarditis is due to pyogenic infection as in pyemic or local processes and the fluid is generally purulent from the beginning. Hemorrhagic effusion is generally due to tuberculosis or malignant disease. In nearly all cases the myocardium shares in the inflammation.

Diagnosis is difficult, especially in fat people. The subjective symptoms are often obscure and are masked by the preëxisting disease. In the plastic form, there may be no symptoms or at most precordial distress or pain most marked at the xiphoid cartilage. In pericarditis with effusion, the presence of the fluid may cause the pain above mentioned; moderate fever, 101° to 103° F. or exacerbation of an already existing fever, often irregular, at the onset of pericardial complications; rapid heart action with feeble irregular pulse; pressure symptoms; dyspnea, dysphagia; irritative cough; aphonia; hiccough; nausea and vomiting; distention of the veins of the neck and duskiness of the face. There may be great restlessness, melancholia, delirium, or acute mania, and more rarely the pulsus paradoxus. Absorption is usually rapid but the heart may remain irritable for a long time. In purulent

pericarditis the diagnostic points are the same as in the fibrinous form with the addition of septic phenomena. The only positive evidence is by exploratory puncture.

More rarely, the onset of pericarditis is indicated by rigors, remittent fever, frequently nausea and vomiting, precordial distress and tenderness, acute shooting pains, increased respirations and a dry, suppressed cough, increased cardiac action, sometimes violent palpitation; this lasts a few hours to a day or two. In children, the onset is often insidious and may precede any obvious signs of rheumatism.

The physical signs are more important than the subjective symptoms for diagnosis.

Before the effusion of fluid the excited cardiac action with precordial friction fremitus is evident. The fremitus becomes less pronounced as the effusion increases but is rarely entirely absent until complete resolution takes place.

The characteristic "to-and-fro" friction rub may cause scratching, grating, or "new leather" creaking; is both systolic and diastolic; has its point of maximum intensity in the third or fourth interspace along the sternum and this varies with the position of the patient; is localized although it extends more or less over the whole cardiac surface; seems near to the ear, is modified by the pressure of the stethoscope, position of the patient, and by respiratory movement.

During the stage of effusion, no friction sounds are present. The heart sounds are muffled and feeble.

Inspection shows precordial prominence with widening and bulging of the lower intercostal spaces, most marked in the fourth interspace over the right ventricle and increased when the patient leans forward. The bulging increases with the effusion. When there is a large amount of effusion, the clavicle is elevated with bulging of the left retroclavicular space so that the first rib can be palpated to the sternum. The cardiac impulse may be feeble or absent. Tenderness may be present. The apex beat is displaced, according to the amount and location of the effusion. The cardiac dullness is increased vertically and laterally.

Many "signs" are described, but the exceptions are many, and their recognition more difficult than is a diagnosis based upon a recognition of the actual condition present in each case.

Reflex muscular contractions usually appear first in the region of the third and fourth inter-spinous areas. These increase in area, until almost the entire interscapular region is involved. Sometimes only the deeper spinal layers are contracted; more often the superficial layers also, and the intercostals, especially of the left side, are contracted and hypersensitive. The relief of these reflex contractions gives comfort, and this is diagnostic of pericarditis as distinguished from endocarditis.

Treatment. Absolute mental and physical rest in bed to relieve the heart of all work possible is essential. Slow the heart action by deep, steady pressure through the cervical and upper dorsal areas especially at the fourth and fifth dorsal on the left side. Correct any subluxations found. Pay particular attention to the atlas and the structures along the course of the vagus. The ribs and clavicle must be carefully attended to and the intercostal muscles relaxed. Relax the diaphragm. Deep, steady pressure at the fourth, fifth and sixth cervical areas quiets the heart action for some hours. The pain about the heart is relieved by the general treatment but if especially severe, deep, steady pressure at the first, second, and third cervical and the fourth, fifth, and sixth dorsal areas is indicated. Dyspnea is allayed by the treatment as outlined and by raising the ribs. When the pulse becomes weak and cyanosis occurs, stimulation of the heart and lungs is required, but this does not occur unless conditions are very unfavorable. In the early stages an ice bag to the precordium will aid in giving much relief.

Liquid diet, principally milk, is usually given at first. Later other light nutritious food may be added. Dry food is sometimes advised.

Free elimination with the bowels, kidneys, and skin active must be secured to promote absorption of the fluid after the acute stage subsides and the elimination of the poisonous waste products.

If the effusion is very great, producing urgent cardiac symptoms, paracentesis or incision may need to be performed. Drainage is necessary in purulent cases.

Prognosis. This depends upon the cause. In simple serofibrinous types the outlook is good for recovery in one to three weeks. The greater the amount of fluid, the more grave is the prognosis. Permanent damage may result from complication with endocarditis or myocarditis or from the formation of extensive adhesions. In purulent or hemorrhagic pericarditis the prognosis is grave. Pericarditis complicating pneumonia or renal disease is often fatal. Relapses are not infrequent.

Sequelæ. Greater or less degree of adhesion occurs in most cases. These adhesions may lead to hypertrophy, or may merely add to the cardiac difficulty in later pathological states.

CHRONIC ADHESIVE PERICARDITIS

(Adherent pericardium)

Chronic adhesive pericarditis in any degree may result from acute pericarditis, varying with the amount of effusion. If there are simply adhesions between the visceral and parietal layers usually there are no recognizable symptoms. When the inflam-

mation becomes more chronic and extends to the mediastinum and pleura, the parietal pericardium may become adherent to the pleura and chest wall. It is more often found in young people. The subjective and objective symptoms are interdependent.

Diagnosis. The precordia is prominent. There is an indrawing of the interspaces, at the time of the ventricular systole, most marked at the apex and synchronous with the systolic shock. There is also a systolic retraction of the left back in the region of the eleventh and twelfth ribs.

Diaphragm Phenomena of Broadbent. There is a visible systolic tug communicated through the diaphragm to its points of attachment, the seventh or eighth rib in the left parasternal line and on the left side behind the eleventh and twelfth ribs. The apex is displaced outward and the area of impulse is increased, both due to the cardiac hypertrophy. The apex is fixed, not changing with the different positions of the patient. The impulse is undulatory, wavy and in the apex region. Diastolic rebound of the chest wall is characteristic of pericardial adhesions. **Friedreich's Sign**—(the collapse of the cervical veins during diastole) and **pulsus paradoxus**—(the pulse becoming smaller at the end of inspiration) are characteristic. The area of cardiac dullness is increased, usually upward even to the first interspace; this is not modified by respiration.

When the membrane over the right ventricle is most affected, there may be very marked systemic disturbances. The hepatic engorgement may simulate cirrhosis of the liver—pseudo-cirrhosis—there may be gastro-intestinal disturbances which cloud the diagnosis greatly. When the pericardium over the left ventricle is most affected, pulmonary disturbances—dyspnea, cough, etc., may be so marked as to simulate pulmonary disease.

PERICARDITIS CALLOSA. This is a form of chronic pericarditis which appears insidiously during childhood. It is very hard to recognize before death, but should be suspected in children with symptoms of hepatic cirrhosis, edema or ascites, full jugulars, or cyanosis. The treatment is that of pericarditis in adults, when the condition is recognized. Its prevention depends upon care during convalescence from the acute fevers, and upon the maintenance of a suitable diet and hygienic regime for children; rheumatic tendencies are perhaps especially to be avoided. Bony lesions of etiological importance include those affecting the cardiac centers, and these should be corrected by very gentle movements, avoiding any undue irritation of the nerve endings of the affected articular surfaces. Recovery is rare; even when death is delayed the heart rarely regains full functional activity.

Treatment. In most cases no particular treatment is needed for chronic pericarditis since the heart adapts itself to the conditions as found and no particular symptoms are produced. When the condition is recognizable treatment may be necessary. It must be remembered that the adhesions may embarrass the heart's

action to such an extent as to lead to hypertrophy or to disturbances of the circulation. These adhesions are connective tissue and are more or less completely covered by a secreting and endothelial membrane. If attempts are made to stretch or to break these adhesions the irritation upon this membrane may set up an acute exacerbation and a chronic inflammation. For this reason great care is necessary in giving treatments or exercises which might throw tension upon the pericardial and mediastinal tissues. In order to relieve the cardiac embarrassment the following procedures may be modified to suit individual cases: The ribs should be raised and held spread apart while the patient takes a long breath. This should be done with the patient lying upon the side, then upon the back and then upon the face in order that the influence of gravity may be allowed to act from as many different directions as possible. The lower ribs may be pulled out while the patient lies upon his back or side with the knees drawn up in such a way as to relax the abdominal muscles. This may be done with the patient in the knee-chest position.

Exercises in moderation are also helpful. The patient may be taught to breathe slowly and deeply while the arms are extended strongly upwards, outwards and downwards; while he is in the knee-chest position lying upon the right side, the left side, the face and the back. All of these exercises and manipulations tend to bring gradual and gentle tension upon the pericardial adhesions and also to facilitate the cardiac hypertrophy. The hygienic conditions are those indicated in any condition requiring cardiac hypertrophy. The general health should be kept as good as possible and all structural, infectious, and environmental causes for diminished vitality should be removed.

When the adhesions seem limited to the region of the apex, and are very strong, embarrassing the heart's action seriously, their surgical section may be considered.

HYDROPERICARDIUM. Pericardial dropsy is an accumulation of water in the pericardial sac without inflammation, occurring always secondary to cardiac or renal disease, pneumothorax, pressure of an aneurysm, mediastinal tumors, or diseased cardiac veins, clinically marked by pressure symptoms, precordial distress, disturbed cardiac action, dyspnea, dry cough, and dysphagia, and the physical signs, those of effusion without any friction sound ever being present.

The fluid varies from an ounce to one or two pints, is clear, yellowish or straw-colored, and alkaline in reaction.

The diagnosis is made by the history and by aspiration of the fluid. The treatment is that of the underlying cause. If the cardiac action is greatly embarrassed, paracentesis may be performed.

HEMOPERICARDIUM. Hemopericardium is found in rupture of aneurysm of the first part of the aorta, of the cardiac wall, of the coronary arteries, and in rupture and wounds of the heart.

Death usually follows before there is time for the production of symptoms other than those of rapid heart failure due to compression.

PNEUMOPERICARDIUM. (Air in the pericardium.) Rarely wounds, fistulæ, gas-producing bacteria, may result in the presence of atmospheric air or carbon-dioxid in the pericardial sac. The symptoms are those of pericarditis, purulent or with effusion. A tympanitic note over the heart, which changes with the position of the patient; splashing, "water wheel" sounds, synchronous with the pulse, make the diagnosis fairly positive.

The treatment is the same as for purulent pericarditis, with which it is usually associated. The prognosis is always very grave, and death is to be expected within a very few days.

CHRONIC POLYSEROSITIS (Pick's disease). In this rather rare disease the serous membranes become thickened through the proliferation of the cells of their connective tissue framework. The pericardium becomes greatly thickened, and its layers adherent. The pleura and peritoneum undergo similar changes; the splenic and hepatic capsules are greatly thickened, with symptoms of cirrhosis of both organs. The cardiac symptoms are usually first noticed and most conspicuous.

Treatment is limited to palliative measures. Such spinal corrections as are indicated on examination facilitates the best circulation and elimination possible under the circumstances. Paracentesis may be indicated, if the ascites becomes annoying.

Prognosis. These patients may live for years, with varying discomfort. Recovery is probably impossible. Death is usually due to some intercurrent disease, as pneumonia.

CHAPTER XIV

DISEASES OF THE MYOCARDIUM

ACUTE MYOCARDITIS

(Acute interstitial myocarditis; carditis; abscesses of the heart)

Acute myocarditis is rapid degeneration of the cardiac muscle or an extension of a septic pericarditis or endocarditis usually occurring in connection with the infectious fevers, clinically characterized by the sudden appearance of cardiac failure and usually quickly fatal. Rarely the infectious agent is introduced into the heart by trauma.

Diagnosis. During convalescence from some acute disease, or after the occurrence of pericarditis, dyspnea, sighing, syncope and precordial oppression occur; the pulse becomes rapid and weak, but rarely irregular; the face is pale; the hands cold, and other signs of depressed circulation are noted; occasionally collapse and coma follow; the heart sounds are feeble, sometimes the first sound is accentuated, the cardiac impulse and the apex beat may be imperceptible.

Treatment. This depends upon the conditions as found on examination. Absolute rest, physical and mental, is indicated. Even turning in bed, or lifting an arm, or slight excitement, may be immediately fatal. Very gentle, steady pressure near the spines of the tenth thoracic to the second lumbar vertebræ relieves the cardiac strain; this should be repeated two or three times each day.

The most important therapeutic consideration is **prevention.** During the course of the infectious fevers, or in any pyemic process, the condition of the heart should be watched carefully; any violent exertion during convalescence is to be avoided. The correction of the bony lesions associated with the cardiac nerve centers should be a routine procedure in all acute fevers, pneumonia, the puerperal state, rheumatism, or other conditions liable to affect the heart in any way.

Prognosis. The disease is usually rapidly fatal; death is to be expected in a few hours to six days after the first symptoms are noticed.

CHRONIC INTERSTITIAL MYOCARDITIS

(Fibrous myocarditis; fibroid heart; chronic carditis; cardiosclerosis)

Chronic interstitial myocarditis is a slowly developing change in the heart musculature due to hyperplasia and induration of

the connective tissues. It is characterized by more or less dyspnea on exertion, tachycardia or bradycardia, precordial distress and pain, and symptoms of anemia of various organs. It often presents no symptoms.

Etiology. The conditions responsible for general arterio-sclerosis may also be responsible for interstitial myocarditis. The inorganic poisons such as phosphorus, lead or mercury; the overuse of stimulating foods and drinks, especially alcohol; and the organic toxins of syphilis, gout, rheumatism, malaria, diabetes, nephritis, carcinoma, and so on, are all important etiological factors. Bony lesions are indirectly responsible through causing increased blood pressure or preventing the proper elimination of the products of katabolism.

Pathology. This condition bears a direct relation to the arteries throughout the body and especially to the coronary arteries. The primary changes in the coronary arteries may be either acute or chronic arteritis, atheroma, or endarteritis obliterans of syphilitic origin, both forms being factors in causing thrombosis of a large branch. Embolism of the coronary artery results in sudden death.

The heart is enlarged and dilated, the structural changes being either diffused or localized in the walls of the left ventricle, the papillary muscles and the septum.

Diagnosis. The symptoms are nearly all cardiac in character. Heart-tire is out of all proportion to other evidences of old age. It is indicated by breathlessness on exertion, slight cyanosis, puffiness or edema of the ankles and weakness out of proportion to the appearance of the patient. Cerebral symptoms include giddiness, vertigo, syncope, insomnia, pseudo-epileptic attacks on rising from the recumbent position, pseudo-apoplexy, rarely mania, delusional attacks, or dementia. Dry hacking cough; dyspepsia and constipation; scanty albuminous urine and dropsy indicate the generally impaired circulation.

Tachycardia, 150 to 180 per minute, may be a terminal cardiac sign of myocarditis. Bradycardia is much more frequent, between 30 and 50 per minute, and may be associated with Stokes-Adams syndrome, rarely with angina or severe arrhythmia. Death results from syncope. Arrhythmia with feeble pulse may be the only clinical symptom. Anginal attacks vary from great distress to true angina. (See Angina pectoris.) There is a sallow, pallid complexion, and evidences of premature age as shown by hair, baggy eyelids and abundance of wrinkles.

The examination of the heart shows a feeble impulse at times scarcely felt, the apex beat not palpable or displaced to left; area of absolute dullness increased; dilated hypertrophy. The sounds are feeble, the first sound more or less valvular. There is a characteristic irregularity of force and rhythm. Murmurs are frequent and due to valve lesions. There may be gallop rhythm or reduplication of the systolic sounds best heard at fourth rib in parasternal

line and more marked in the recumbent position and after exercise. Added signs are found when hypertrophy or dilation become marked.

The arteries are palpable, tortuous vessels with thick walls and high tension; the temporal artery is prominent and the arcus senilis is often present. Blood pressure is increased.

Exercise has little effect upon functional disturbances, but increases the pain, dyspnea, cardiac disturbances when the myocardium is degenerated.

Treatment. There is no remedy for the fibroid change, but the fibrosis may be held in check and the symptoms met or prevented by improving nutrition, preventing constipation, guarding against any mental strain or physical exertion, by diet, and by careful systematic treatment.

The removal of every factor responsible for abnormally high blood pressure or for the retention of the toxic products of metabolism may prevent the more rapid course of the disease. The symptoms may be treated as they arise. Stimulation of the accelerators in the upper dorsal and the sympathetics in the cervical region increase the strength of beat and tone of the heart muscle. The heart must be watched very carefully during this treatment, and for some hours afterward. The palpitation, dyspnea, arrhythmia, and depressed circulation are relieved by raising the ribs, and by long, slow, gentle movements of the lower thoracic spinal column. Inhibition of the splanchnics is often useful. For pseudo-apoplexy, the patient is placed recumbent with the head slightly raised, and the whole spine treated with careful relaxation and correction, paying particular attention to the cervical area. The blood pressure may be equalized by deep, steady pressure to the splanchnics and to the solar plexus direct. All secretions must be kept active. Pay attention to the kidney area.

Diet must be generous and of easily digested foods. Little or no tea or coffee must be given. Tobacco and alcohol are absolutely forbidden. The patient should lie down several hours during the day. Exercise must be carefully gauged by the effect upon the pulse, blood pressure, and the cardiac signs. The following may be used, beginning with the first and, as followed by improvement, each advance may be taken: (a) massage; (b) resistance movements; (c) moderate walking on the level; (d) light gymnastics.

The Nauheim treatment may be of benefit but must be given very carefully and in selected cases.

After improvement, the patient should return for osteopathic examination every few months. A little occasional attention may be of very great value in prolonging a comfortable life.

Prognosis. This is largely determined by the habits of the patient. The disease is incurable but the patient may live fairly

comfortably for many years if he will observe due care. This is one of the most common causes of heart failure in the course of acute pneumonia, typhoid fever, and from overexertion.

Prevention is important. The paragraphs on etiology and treatment suggest the best methods for avoiding the disease.

FATTY DEGENERATION

Fatty degeneration of the heart is a change of the cardiac muscle fibers, the transverse striæ being replaced by granules or globules of fat. It is clinically characterized by feeble cardiac action, venous stasis, and dyspnea.

Etiology. It is a disease of elderly people and follows those diseases which induce fatty changes, as carcinoma; tuberculosis; chronic gout; prolonged anemia; kidney diseases; prolonged administration of chemical poisons as arsenic, phosphorus or alcohol; lack of out-door exercise; and chronic intestinal toxemia. It may follow fatty infiltration.

Diagnosis. The symptoms are often obscure. There may be coldness of the feet, drowsiness after meals, dyspnea on exertion, syncopal or even epileptiform attacks. Angina pectoris may occur. Cheyne-Stokes breathing, sighing or oscillating respiration may cause fright. Cardiac asthma and arcus senilis are sometimes present.

The physical signs include a weak, irregular cardiac impulse; cardiac dullness normal or very moderately enlarged, first sound feeble, toneless, and almost inaudible; second sound normal or weak; pulse often remarkably slow, compressible, sometimes irregular. It is associated with atheromatous changes in the vessels.

Diagnosis is rarely more than probable. It is to be distinguished from fibroid degeneration.

Treatment. The removal of the etiological factors is important, when this is possible. The treatment outlined for chronic myocarditis should be adapted to the needs of each individual patient. A quiet life is imperative. Errors in diet must be avoided.

Prognosis. Recovery is not to be expected, and death may occur at any time, from cardiac paralysis, rupture of the heart, or exhaustion. Life may be prolonged and made more comfortable by correct hygiene and such treatment as is indicated by the condition of the patient.

FATTY INFILTRATION

Fatty infiltration myocarditis is an excess of fat between the muscle fibers and around the heart. It is associated with general obesity and evidences of cardiac weakness.

Diagnosis. There are no symptoms until the muscle fibers are so weakened that dilatation occurs with its particular signs. The heart sounds are weak and muffled; a murmur may be present at the apex and the pulse is feeble and regular; these conditions exist for years. Diagnosis is made by the presence of obesity and the evidences of cardiac weakness, without signs of other cardiac disease.

Treatment. In this case much can be done if the heart muscle has not been too badly weakened. The main object is to reduce the fat. (See Obesity.) The treatment for chronic myocarditis may be adapted to the condition of the patient.

Prognosis. In young people the prospect is good for symptomatic recovery, though such persons must be careful not to allow too great increase in the body fat, during their remaining life. Elderly persons must be careful not to reduce weight too rapidly; such individuals may suffer serious cardiac symptoms, even death, as the result of rapid loss of weight. If the fatty deposit is continued, or if the patient refuses obedience to hygienic laws, permanent injury to the cardiac muscle follows. Sudden death may occur from rupture, or with symptoms of heart block.

DILATATION OF THE HEART

(Cardiac dilatation)

Dilatation of the heart is an increase in the size of one or more of its cavities without hypertrophy. It is characterized by feebleness of the circulation, terminating in venous stasis, cyanosis, edema, and exhaustion, and is most typically seen in the broken compensation of aortic and mitral regurgitation.

Etiology. It is due to causes which directly affect the myocardium as bacterial toxins, chemical poisons or prolonged pyrexia. Increased pressure within the walls, emotion, shock, and physical exertion, especially running or bicycling uphill, mountain climbing, etc., cause sudden or acute dilatation. In those of feeble resistance slight causes are effective. It sometimes seems to be idiopathic. It occurs secondarily in chronic valvular lesions, chronic bronchitis, chronic interstitial nephritis, alcoholism, and syphilis. Among the predisposing causes are fatty and fibroid degenerations of the muscle, and inadequate nutrition.

Diagnosis. The general symptoms are referable to enfeebled circulation; feeble pulse, headache aggravated by the upright position, attacks of syncope, cough, dyspnea, jaundice, dyspepsia, constipation, scanty often albuminous urine, mental dullness, vertigo often relieved by copious epistaxis, and finally dropsy beginning in the lower extremities. If these changes take place slowly, it is termed "gradual failure of compensation."

The precordial throbbing and extended, wavy impulse; the small, weak, irregular pulse; the increased dullness of square outline, the diminution or loss of the muscular element of the first sound, should make the diagnosis apparent. In general, if there are no valvular lesions, the cardiac sounds are weaker, the systolic sounds sharper, short, and of high pitch. The X-ray shows large "thin" heart-shadow with characteristic shape distorted according to cavity involved.

Treatment. The object of treatment is to secure hypertrophy, if possible. Correction of the subluxations is important, especially those affecting the cardiac and vasomotor centers. Active elimination by all emunctories must be maintained. The diet must be liberal and as nutritious as the patient can assimilate. Digestive disturbances must be avoided. Rest from business, excitement, and physical strain is essential. There are various systems of exercise devised, among which may be mentioned: Swedish or Ling plan of passive exercise and massage, Schott movements against limited resistance, and Oertel's climbing method.

Prognosis. The outlook depends upon the amount of hypertrophy to be secured. Otherwise the prognosis is very unfavorable, death resulting sooner or later from exhaustion or from cardiac paralysis.

CARDIAC HYPERTROPHY

(Hypertrophy of the heart)

Cardiac hypertrophy is an increase in the number and size of muscle cells of the heart, induced by overwork of the heart from whatever cause and characterized by forcible cardiac impulse and accelerated circulation.

Etiology. Among the predisposing causes are: valvular and especially aortic lesions; adherent pericardium; diseases of the lungs; increased peripheral resistance as a result of arteriosclerosis or chronic interstitial inflammations; aneurysm of the aorta; overexertion of the healthy heart (athletic heart); long-continued stimulation due to the neuroses, exophthalmic goitre, or the long-continued use of large quantities of tea, coffee, or tobacco.

Pathology. The hypertrophy is usually limited to the ventricles, the left side being more commonly involved. The auricles have not so much muscular tissue and so dilate more readily than hypertrophy. The shape is altered. If the left ventricle is chiefly involved, the heart is elongated and the cavity dilated; if the right ventricle is the more implicated, it is widened transversely and the apex blunted; if both ventricles, the shape becomes globular. From increase in weight, the heart may drop back when the patient is recumbent, but on sitting or standing it sinks lower in the chest and to the left causing more or less prominence of the abdomen.

The varieties are (1) simple hypertrophy which is a simple increase in the thickness of the cardiac walls; and (2) eccentric or dilated hypertrophy which is increase in the walls with dilatation of one or more cavities.

Diagnosis. If the hypertrophy is only sufficient to compensate for defects, there are no symptoms. The degree of hypertrophy depends largely upon the age of the patient. If it is disproportionate to the obstacle, there is increased and forcible cardiac action, precordial discomfort, headache, dizziness on exertion, tinnitus aurium, flushes, flashes of light, dyspnea on exertion, congestion of the face and eyes, dry cough, epistaxis, restless nights, and more or less jerking of the limbs.

When the hypertrophy is concomitant with general arteriosclerosis, the arteries become full and the pulse firm and bounding, the carotids and superficial arteries pulsate markedly so that the patient complains of throbbing sensations.

If the disease began early in life, there is bulging of the precordium; if after adult life, fullness and prominence with a distinct impulse is seen. The cardiac impulse is felt one or two interspaces lower and to the left, stronger, slower, more or less diffused and forcible, more "heaving," than normal. The apex beat may be felt in the sixth, seventh, or eighth interspaces even three inches outside of the mid-mammary line. The pulse is full, strong, regular, and of increased tension. It is modified according to the valvular lesions present.

The increased area of dullness extends vertically and transversely to left of sternum; if the right ventricle involved, dullness is increased to right of sternum. X-ray shows enlarged "thickened" shadow.

If there are no valvular changes, the first sound is loud, prolonged, of low pitch, and of a somewhat dull or metallic quality. The second sound is strongly accentuated, clear and loud. Associated valvular disease causes varying murmurs.

The sequelæ of left ventricular hypertrophy are cerebral hemorrhage, miliary cerebral aneurysms, fatty degeneration or cardiac dilatation.

Hypertrophy of the Right Ventricle is due to chronic valvular lesions of right or left heart, or pulmonary diseases as emphysema and cirrhosis. Bulging over the lower part of sternum and occasionally over the sixth and seventh left costal cartilages is present. Epigastric pulsation may be seen in third and fourth interspaces to right of sternum. The radial pulse is of small volume. The cardiac dullness is moderately increased transversely and to the right.

There is accentuation of the second pulmonic sound due to increased tension of the pulmonary artery. Reduplication of the second cardiac sound may occur.

Auricular Hypertrophy is always combined with dilatation. In the left auricle the signs are few and indefinite; dullness to left of sternum, in the second and third interspaces with a presystolic

impulse or wave in the second space. This is inferred, if mitral stenosis or regurgitation is present. In the right auricle, hypertrophy is always with dilatation and is secondary to incompetency or stenosis of the tricuspid valve and associated with right ventricular hypertrophy and dilatation. The main signs are: dullness in the third and fourth interspaces to right of sternum, with often a presystolic wave in same area, systolic jugular pulsation and evidences of venous engorgement.

Treatment. Remove the cause if possible. If excessive, lessen the force and number of cardiac pulsations by deep steady pressure at the third and fourth dorsal vertebræ, correcting any lesions present. Note the position of first ribs, clavicles, and lower ribs. Correct the habits of the patient, according to conditions as found. All active exertion should be restricted and the recumbent position assumed several hours during the day if possible. The diet must be carefully regulated, nutritious, yet all kinds of stimulating foods interdicted.

Prognosis. The outlook depends upon the original cause. If the hypertrophy is compensatory for valvular lesions, the duration and comfort of life may not be affected. Further hypertrophy can usually be prevented by active and persistent treatment, unless the original cause increases in severity.

CARDIAC MISPLACEMENTS. These may be congenital or acquired. Transposition of the heart may be associated with transposition of the abdominal viscera or it may exist alone. Transposition does not in the least interfere with perfectly normal function; it is rarely recognized ante mortem and no treatment whatever is possible or necessary.

The position of the heart may be changed by variations in the position or the structure of other thoracic viscera. The right lung may be destroyed by abscess or otherwise and the left lung may increase in size to such an extent as to pull the heart well over to the right side.

Rarely a weakness of the suspensory tissues permits the heart to change its position when changing position of the body of the patient. This possibility must be kept in mind when making a diagnosis of cardiac hypertrophy and dilatation. The diagnosis rests upon finding the different locations of the cardiac dullness when the patient assumes different positions. Roentgen ray gives valuable geography and is positive.

INJURIES OF THE HEART

Although very trivial injuries to the heart usually result in death, yet it occasionally happens that recovery occurs from wounds of considerable size. In addition to bullet and stab wounds and other gross trauma, a number of cases have been reported in which the heart has been invaded by needles and other sharp objects. In several cases a needle has been found embedded in scar tissue in the wall of the heart, sometimes with a part of its length projecting into the ventricle. Surgical repair of the injured heart is possible.

CARDIAC NEOPLASMS

It is very rare that the heart or its membranes are the seat of new growths of any kind. The diagnosis is frequently impossible ante mortem, although it occasionally happens that metastatic growths may be expected. The vegetations upon the valves of the heart are not to be considered in any sense as neoplasms.

Increase in the cardiac dullness with a weak heart beat and other signs of cardiac embarrassment, together with cachexia and other systemic indications, may give the diagnosis, especially if the cardiac neoplasm is secondary to recognized malignancy elsewhere. No treatment is of any value and death is speedy, in all cases in which any cardiac symptoms are present.

CARDIAC ANEURYSM

Aneurysm of the wall of the heart may occur as the result of disease of the coronary vessels, myocardial degeneration or sudden increase in blood pressure in a heart whose walls are weakened. The sac may be barely perceptible or it may be as large as the patient's head. Attempt at repair is made by the coagulation of the blood in the sac and the organization of the clot. The condition is not usually recognized before death and the only treatment is that of the predisposing causes.

Aneurysm of the valves may occur in endocarditis. The sac may be of considerable size without producing any recognized symptom.

If aneurysm of the wall of the heart or of another large vessel becomes ruptured sudden death occurs.

DISEASED CORONARY ARTERIES

The symptoms associated with disease of the coronary arteries and to a certain extent of the vessels of Thebesius are fairly typical. Any of the ordinary degenerations affecting the walls of blood vessels may be seen in the branches of the coronary arteries and when any of these pathological changes result in the occlusion of any of the arterial twigs an infarct is produced whose after history may follow either of two definite paths. Coronary arteries are terminal and there is very slight opportunity for overlapping of areas of distribution. When the circulation is partially supplied and perhaps under certain other conditions the muscle cells undergo gradual atrophy and there is a multiplication of the connective tissue elements throughout the infarct area, the condition resembles a little mass of scar tissue in the middle of the cardiac muscle. This condition is known as the white infarct, and when a number of such accidents occur the heart assumes the mottled

appearance which sometimes receives the name of "marble heart." Under other circumstances the cardiac muscle undergoes softening and may be absorbed; in any of these cases the wall of the heart is greatly weakened.

At the time of the occlusion of the vessels the patient suffers very severe pain in the precordium and the symptoms of angina pectoris may occur. At other times there is a dizziness or perhaps syncope. The symptoms depend upon the size of the arteries occluded.

The condition terminates by sudden death. Sometimes the wall of the heart ruptures, more frequently on the anterior aspect of the left ventricle, and sometimes the occlusion of one of the larger branches of one of the coronary arteries produces death.

Treatment. At the time of the shock the patient should rest for several hours or several days in bed. He must avoid sudden exertion, violent emotion, excitement and all the conditions which ordinarily raise blood pressure.

The prophylaxis is far more important. It consists in avoiding all of those factors which cause arteriosclerosis or myocarditis.

ANGINA PECTORIS

(Breast-pang; stenocardia; neuralgia of the heart)

Angina pectoris is an affection characterized by sudden attacks of agonizing pain in the cardiac region and a sense of impending death.

Etiology. The predisposing causes include all conditions which interfere with the nutrition of the walls of the heart, particularly in men past middle life; among these nutritive influences being mentioned affections of the cardiac ganglia and plexuses. Syphilitic aortitis is a factor in men under thirty-five years. Spinal luxations which have been found in these cases are those of the atlas, the cervical region and the upper dorsal area. Lesions of the fourth thoracic are reported in connection with coronary spasm.

The exciting causes are sudden strain, over-distended stomach, powerful emotional disturbances, gout, diabetes, and influenza.

Disturbance of the coronary circulation is the one factor to be considered. This is due to some obstructive lesion in the typical case. Spasm of the coronary arteries produces exactly the same symptoms, and is not to be differentiated from the effects of obstructive lesion, in most cases. Probably functional spasm is associated with the true obstructive lesion very often; there is no doubt that functional spasm predisposes to coronary disease and thus to obstruction.

Diagnosis. There is a sudden seizure with acute intensely agonizing pain with a sense of constriction across the chest or of

suffocation, the patient stops whatever he is doing, grasps something if handy, and stands perfectly still. The terrible feeling of anxiety and anguish is shown in the face. The **pain** is most marked at the lower end of the sternum, radiates into the neck and down the left arm, more rarely into the right arm. It follows the course of the ulnar nerve. There is referred pain in the region of the fifth, sixth, and seventh, even the eighth and ninth dorsal vertebræ and also pain in the cervical region. Muscles in this region are tense and hypersensitive. The respiration is very shallow and difficult although there is no obstruction to the entrance of air into the lungs. The face is very pale, even gray, with no cyanosis. The whole body is covered with a cold sweat. The pulse may show increased tension. The attack may last a few seconds to many minutes, leaving the patient prostrated. An excessive flow of urine follows the attack. It may terminate fatally at the first attack or may recur at intervals, the first being mild and those following increasing in severity.

Some cases present all the symptoms except pain **(angina sine dolore)**. Other cases are associated with coldness and pallor of the extremities (angina pectoris vasomotoria), the pain being comparatively slight.

Angina vera is most common in men past middle life; is often brought on by exertion; is rarely nocturnal or periodic; is not associated with other symptoms; the pain is agonizing and attended by a sense of constriction, and is of short duration. The patient presents an attitude of silence and immobility; arteriosclerosis is present and the prognosis is grave; attacks often proving fatal.

Pseudo-angina is hysterical. It is most common in women of any age from childhood, attacks are spontaneous, often periodical and nocturnal, and associated with other hysterical symptoms. The pain is less severe, with a sense of distention instead of constriction, duration is one or more hours, is attended by agitation and activity, and is never fatal.

Treatment. The patient should be put to bed as soon as an attack seems impending. The first object is to relieve the pain by raising the left lower ribs over the cardiac area, pressure being made at the same time over the upper three dorsal transverse processes. Apply deep steady pressure to the vagus in the neck and relax the precordial intercostal tissues. If the attack is exceedingly severe or continued for some time, a few whiffs of chloroform may be necessary to secure relaxation. An ice bag over the heart usually gives relief; hot applications are more effective in occasional cases.

Interval treatment. Careful attention to the general nutrition and elimination is necessary to improve the nutrition of the heart muscle. Cases with spasm of the coronary arteries due to lesions of

the fourth or other thoracic vertebræ are apt to recover after correction of the lesion. If the condition has not been too long present, recovery is complete; in cases of long standing recovery may be slower, or the changes in the vessel walls may not permit complete recovery, though the symptoms are greatly relieved. This correction is best made during the intervals of the attacks—the shock of sudden correction is apt to perpetuate the attack if attempts are made at that time.

In obstructive cases, the effects of the associated spasm are not to be neglected, so that the same corrective measures are indicated, whether there is or is not reason to infer a true obstructive angina. Passive exercises and general massage may be gently given by the nurse.

A strict milk diet may be necessary at first until the general nutrition is improved and toxins removed. The general diet should include plenty of fresh vegetables and fruits, eggs, dairy products, lean meat in great moderation, and plenty of pure drinking water unless other symptoms indicate water restriction. Tobacco, alcohol, tea and coffee are to be forbidden. Rest in bed for some weeks is sometimes useful. Mental and muscular overstrain are to be avoided in all cases. Cold baths are dangerous, but hot ones are useful. Hot foot-baths on retiring may give full night's sleep. The bowels should receive careful attention. Strenuous enemas are harmful. Purgative drugs should be absolutely forbidden.

The **prognosis** is fairly good for recovery from the attack but is ultimately fatal in organic cases. Cases due to spasm of the vessels often recover, apparently completely.

CHAPTER XV

THE CARDIAC NEUROSES

It is often extremely difficult to draw the line between those cardiac symptoms due to nervous disturbances and those due to organic or mechanical causes. Purely neurotic heart action is usually associated with disturbed vasomotor activity. Probably every case of organic disease of the heart or blood vessels is complicated by more or less marked disturbances in the nervous control of the cardiac and vascular muscles.

Etiology. The etiology of the cardiac neuroses depends upon those factors which modify the action of the heart centers in the medulla and in the upper thoracic spinal cord or over the nerve trunks and ganglia by means of which nerve impulses are transmitted to the heart. The place of the bony lesions has already been mentioned.

Neurotic inheritance is usually present and the stigmata of hysteria are frequently found. The use of alcohol, tobacco, tea, coffee, or of excessive meat or starch eating are etiological factors of varying importance. Emotional disturbances beyond the normal limits are responsible for the palpitation especially.

Diagnosis. The diagnosis of the cardiac neuroses must be made by the exclusion of all organic diseases, not only of the heart and blood vessels, but also of other organs capable of affecting the heart. The ductless glands, the kidneys, the blood itself, the liver, the stomach, the pelvic organs, must also be carefully examined before the diagnosis of a cardiac neurosis can safely be made.

Treatment. The treatment has already been indicated by what has been said of the etiology. The character of the bony lesions as found upon the examination of each patient is by far the most important factor in uncomplicated cases. Increased nutrition of the body, and especially better circulation through the spinal cord and the lower brain centers, are also important. Complete rest is often necessary for the acute attacks. In patients with hysteric or neurasthenic symptoms the treatment suitable for these diseases must be employed.

Prognosis. In uncomplicated cases the prognosis for speedy recovery is very good indeed, provided suitable treatment is given.

Without treatment the life of the patient is not in danger but his comfort and efficiency are considerably lessened by the disturbed heart action. Whether these functional disturbances may lead to

organic diseases of the heart later in life is a question which is not yet answered; a priori, it is to be supposed that the effects of functional disturbances might predispose to organic disease.

Palpitation of the heart is characterized by increased force of the heart beat which causes uncomfortable sensations in the cardiac region, often throbbing in the temples, the throat and abdomen. It is more frequently associated with bony lesions of the upper cervical region although lesions in the interscapular spinal column are not rare. Digestive disturbances may be exciting causes.

BRADYCARDIA

(Unusually slow heart beat)

Extremely low pulse, 35 to 50, may be found in persons who are otherwise normal. Purely functional bradycardia must be distinguished from the slow pulse which is present in certain forms of cardiac hypertrophy and especially in diseases characterized by the presence of toxic substances in the blood stream. Jaundice and toxemia from the absorption of the toxic productions of intestinal putrefaction are especially to be noticed in this connection. The bony lesions usually found in the purely functional bradycardia include especially those of the second to the fourth thoracic vertebræ and the regions associated with these. Upper rib lesions and upper cervical lesions are occasionally found in these cases.

Treatment. Bradycardia due to nervous disturbance, bony lesions, and the milder forms of toxemia can be relieved by pressure upon the vagus nerve in the neck, or by stimulating movements applied to the tissues near the third and fourth thoracic spinous processes and to the deeper spinal muscles of these segments.

For relief of attacks, the patient should be given warm or hot drinks, hot applications to the abdomen and limbs, and all constricting clothing should be removed. Correction of the bony lesions as found should be thorough, both for the effect upon the heart itself, and for the relief of the underlying toxemia, nerve reflexes, and other causative factors which may be present. Stimulating manipulations to the mid-thoracic region and the suboccipital triangles may relieve the attack.

TACHYCARDIA

(Unusually rapid heart beat)

This may be present as an individual idiosyncrasy, as a cardiac neurosis, in exophthalmic goiter, or as a symptom of disease of the heart muscle. Not rarely a pulse of 100 to 140 may be found in individuals apparently in perfect health. The speed may be due

to shortened rest period or to increased rapidity of the entire cardiac cycle. In **polysystole,** the interval between the first and second sounds is normal, while the rest period is shortened. In **embryocardia,** the interval between the first and second sounds, and also the rest period, are shortened.

If tachycardia can be relieved by stimulating the vagus nerve in the neck, or by steady pressure over the suboccipital triangles, or near the spinous processes of the third and fourth thoracic vertebræ, it is of extracardiac origin—which may include exophthalmic goiter, the effects of bony lesions, or other cardiac neurosis. If these manipulations do not modify the pulse rate, after thorough testing, the heart muscle is probably diseased. In some cases of exophthalmic goiter, also, it is not possible to affect the heart by these manipulations.

Treatment. Correction of the lesions of the third and fourth thoracic vertebræ and the associated ribs, and of the cervical vertebræ, the first, second, fifth and sixth ribs, especially on the left side, when these are found, is of importance. In reflex or systemic nervous cardiac disturbances, the relief of the cause of the neurosis is of primary importance. For the attacks, the patient should be taught to lie with the head low, take a full breath, and hold the glottis closed to the limit of comfortable endurance. Compressing the abdomen gives relief. Pressure upon the tissues near the third and fourth vertebræ is palliative.

ARRHYTHMIA

(Arrhythmia cordis; irregularity of the pulse)

Normally, the contraction of the heart originates at the sino-auricular node, at the mouth of the superior vena cava, is conducted to the auricle, and hence to the ventricle by way of the auriculo-ventricular bundle (bundle of His or Gaskell's ridge). Under conditions of abnormal stimulation, contractions may originate in the auriculo-ventricular node in the wall of the right ventricle near the coronary sinus; or in the auriculo-ventricular bundle on the ventricular side of the node; or in the auricular tissue itself.

Etiology. The main causes are valvular and myocardial diseases; rheumatism and chorea; long-continued excessive use of tobacco, tea, or coffee; flatulent dyspepsia; the nervous conditions of neurasthenia, hysteria, and melancholia, or of organic disease of the nervous system.

Subluxations especially at the anterior ends of the first to the fifth ribs, or the fourth and fifth dorsal vertebræ may irritate the heart and cause a simple arrhythmia.

There are several types of irregularity.

Sinus Irregularity begins at the sino-auricular node. The beats are irregular, equal in size, but with a variable diastolic period.

Extra-Systole is the commonest form. There is premature contraction of the auricle or ventricle or both, independent of the sinus rhythm. The sinus stimulation then occurs during the refractory period and the diastole is prolonged until the next sinus stimulation is due. This accounts for many cases of intermission, pulsus bigeminus or trigeminus and delirium cordis.

Auricular Fibrillation is a condition in which the auricular muscle fibers contract rapidly and inco-ordinately producing an arrhythmia with no regularity or sequence. A ventricular venous pulse can be seen in the neck. The condition is especially frequent in mitral stenosis and in the senile heart. Irregularity occurs when there is a failure in the conducting power of the primitive bundle, the ventricular systole being occasionally omitted.

"**Pulsus Alternans** shows itself by a comparatively regular alternation of strong and weak beats. It needs be but briefly considered here, as it can rarely be determined except by instrumental means. When recognized it is generally accepted as an ill omen. As a prognostic sign it compares with such others as albuminuric retinitis, signifying that but a brief period of life can be expected." —M. W. Peck.

It is due to depression or failure of the contractile power of the ventricle. Many intermissions are functional and may be habitual and unrecognized.

The treatment and prognosis depend upon the cause of the arrhythmia and not upon its severity. Organic disease with almost unrecognizable symptoms may occasion speedy death; functional arrhythmia which is very severe may be followed by perfect recovery under suitable treatment of the causes.

STOKES-ADAMS DISEASE

(Heart block)

This is the term applied to the effect produced by diseases of the muscular bundle of His.

Pathology. The bundle of His which transmits the impulse from the auricles to the ventricles is composed of muscular tissue which retains embryonic characteristics throughout life, it is therefore somewhat more easily subject to disease than is the rest of the heart muscle. When it undergoes degeneration, the contraction wave arising around the roots of the great veins in the auricles passes with difficulty to the ventricular walls; for this reason the auricles may beat twice or even three or four times to every single ventricular systole. The condition is most easily recognized by the jugular pulsation.

Diagnosis. Simultaneous tracings taken of the jugular and radial pulsations usually give the diagnosis without question.

Treatment. The cause of the cardiac degeneration should be removed if this is possible. Stimulating foods and drinks, overwork and emotional strain must be avoided, all structural causes of cardiac malfunction must be removed. Rest in bed at the time of the exacerbations is necessary.

Prognosis. The prognosis is grave for all cases of Stokes-Adams disease, usually death is imminent when the diagnosis is made.

Congenital heart-block is a rare condition, probably due to the imperfect development of the bundle of His. It is recognized by the history of congenital defect; the missed ventricular beats associated with rhythmical jugular pulsation, and the absence of findings diagnostic of other cardiac defects. In one case recorded (P. C. O. Clinic) development or compensation occurred, so that at least a symptomatic recovery prevented further symptoms.

CARDIAC ASTHMA. These attacks are characteristic and apt to come on in the early morning hours. The patient is suddenly seized with dyspnea; the respirations are labored, not much increased in frequency but the distress is extreme; rattling sounds come from fluid in the bronchi; clear fluid pours from the mouth or is easily discharged; if death is near, it is discharged with difficulty, if at all. The patient sits upright with pallid face, sweat-covered forehead, and cold pallid hands. The temperature is subnormal, the blood pressure low, the pulse is rapid, thready, irregular and often scarcely perceptible at the wrist, the patient being in a state of profound shock. The heart sounds are heard only as a confused jumble with irregular or ineffectual systoles, the lung sounds overshadowing the cardiac. In the interval after recovery from attack the patient will remain fairly free from dyspnea. The pulmonary edema present is usually due to impairment of the pneumogastric nerve. The cause is not known. It is often associated with chronic nephritis.

With this form, death usually results within a year although patients have lived several years.

CHAPTER XVI

DISEASES OF THE ENDOCARDIUM

ACUTE FIBRINOUS ENDOCARDITIS

(Exudative endocarditis; valvulitis; endocarditis verrucosa)

Acute fibrinous endocarditis is an inflammation of the lining membrane of the cavity of the heart, the valves being the most commonly affected. It is characterized by the formation of fibrous nodulations upon the surface called vegetations; the clinical symptoms may be practically negative. Murmurs variously placed upon the cardiac cycle give the diagnosis.

Etiology. It is rarely primary. Secondarily, it is found most commonly in acute rheumatism, also in chorea, tonsillitis, scarlet fever, pneumonia, phthisis, kidney diseases, and occasionally in gonorrhea, in the cachexias and in subluxations affecting the cardiac centers. Pyorrhea alveolaris may be important.

Pathology. Cloudiness is followed by edematous thickening of the valvular endocardium, superficial erosions and the formation of small granulations, deposit of layers of fibrin, blood corpuscles, and a few organisms. The whole process results in the formation of small friable warty outgrowths—vegetations. These are most marked one or two millimeters from the free borders at the line of contact of the leaflets. In the course of time, they are transformed into fibrous tissue. The myocardium is always involved. The vegetations are upon the side of the valve opposed to the blood stream; at the aortic valve they project into the ventricle; at the mitral, into the auricle.

The location is significant. Congenital endocarditis attacks the right side of the heart; simple endocarditis attacks the left side; malignant endocarditis attacks both sides, but more often and more severely the left side.

Diagnosis. The subjective symptoms are usually negative or vague, since in most fevers the heart is somewhat dilated and a murmur may be present without endocarditis. In severe cases, there may be irregular, rapid, feeble pulse, faintness, pallor, increased perspiration, dyspnea, precordial pains, throbbing carotids. Later the pulse becomes less frequent and more or less venous stasis and pulmonary congestion appear. Usually a sharp rise in temperature in the course of the primary disease without other causes, leads to observation of the heart. Reflex contractions of the deeper spinal muscles of the second to the fifth thoracic segments may be found occasionally, but are not always present. Their occurrence probably depends upon an associated myocarditis.

The most reliable physical sign is the development of a roughened murmur, like soft blowing, with the first sound into a well-

marked systolic mitral murmur, during rheumatism or acute fevers. It may change in character from day to day or be accentuated; the second sound may be reduplicated at apex and accentuated. Reduplication and accentuation of the pulmonic second sound is frequent. The new sound may disappear at first when the patient is sitting but later it persists. There is more or less dilatation and cardiac irregularity. All cardiac signs may be latent.

Complications. As the vegetations are very friable, they may become detached. These emboli produce additional symptoms according to the location of the end-artery in which they lodge.

Embolism of the kidneys is marked by sudden deep-seated lumbar pain, albuminuria, even hematuria. Embolism of the brain is shown by sudden palsies, convulsions, aphasia, or sudden disturbances of consciousness. Embolism of the spleen usually causes sharp pain and tenderness in the splenic region. Embolism of the skin shows in petechial or purpuric spots. Embolism of the lungs is known by local pain, hemoptysis, possibly localized dullness, bronchial breathing and rales.

Treatment. Prevention of endocarditis in patients with the primary diseases is important, and consists in guarding them from cold and undue exertion. The correction of any lesion found in the cervical and upper five dorsal vertebræ and the ribs at their anterior and posterior articulations, relaxation of contracted muscles, raising and spreading the ribs, and deep steady pressure in the sub-occipital fossa to lower the fever and quiet the heart, are some of the measures indicated. Cold over the cardiac area aids in keeping cardiac action slower.

Absolute bodily and mental rest for weeks or months is necessary. The diet should be light and liquid. The bowels and kidneys must be kept active to eliminate the waste metabolic products.

Prognosis. Simple endocarditis without complications is not dangerous to life. The affected valve usually remains damaged and later becomes the seat of chronic endocarditis.

ACUTE AORTITIS. This disease is usually associated with acute endocarditis with which it is usually confused in diagnosis. It is due to any of the infectious diseases, to alcohol, syphilis, rheumatism, etc. The pain occurs at a somewhat higher level than is the case in endocarditis. The palpitation may be very severe but symptoms may resemble those of angina pectoris. The treatment is the same as that of endocarditis; recovery is usually to be expected so far as the aortitis is concerned.

MALIGNANT OR ULCERATIVE ENDOCARDITIS

The malignant form may be primary although it is more frequently secondary to septic processes, usually in connection with a suppurating external wound, puerperal sepsis, acute necrosis, or

gonorrhea, which may be latent. It often attacks the valves which are the seat of chronic inflammation.

Pathology. The initial changes are similar to the simple form but ulcerations may completely replace the vegetations. The vegetations when present are larger and fungating; the underlying tissues are necrotic, show loss of substance and round-celled infiltration and contain masses of bacteria. When the vegetations become detached, they form septic emboli giving rise to metastatic abscesses. Ulcerative process causes great destruction of the valves and may even lead to perforation of the curtain. If the vegetation touches the mural endocardium, the part touched becomes affected by contact.

Diagnosis. The patient is, and appears, extremely ill. Local symptoms may be entirely lacking, and cardiac disturbance may be unsuspected until death occurs. In the **"typhoid,"** the most frequent type, the symptoms include varying degrees of vomiting and diarrhea, pain in the hepatic or the splenic region; dimness of vision; hematuria and albuminuria; irregular chills and fever (sometimes to 106° F.); ecchymoses and sweating; and the "typhoid state."

The **"cardiac type"** (recurring malignant endocarditis) is characterized by recurring attacks of septic endocarditis in a heart with valvular lesions. This condition should not be confused with the simpler attacks of recurring simple endocarditis.

The **"cerebral type"** symptoms are practically those of cerebral meningitis.

The physical signs are similar to those of the simple form. Variations from day to day in the character of the murmurs seem to be more frequent. Leucocytosis is usually, but not always present; there may be blood changes indicative of the primary disease. Local infection should be sought for as the heart disease is often due to a latent gonococcus infection.

The treatment is similar to that of simple endocarditis. Removal of the sources of infection is a major consideration. The excretion of the poisons formed must be aided by every possible means. Stimulation of the liver aids bowel elimination and raises the opsonic index. To increase the comfort of the patient and delay death is the most that can be expected.

Prognosis. Cases engrafted upon an existing cardiac disease may last for several months; complete recovery is not to be expected. Death may occur at any time, or being delayed, may result from intercurrent disease.

CHRONIC ENDOCARDITIS

(Sclerotic endocarditis; interstitial endocarditis)

This is frequently limited to the valvular endocardium, though the mural form is by no means rare. It produces various forms of valvular defects, according to the various etiological and localizing

factors present. It may be chronic from the beginning, or may result from the acute form.

Etiology. Primarily, the disease results from syphilis, malaria, rheumatism, lead, alcohol, and other infections and poisons; or from arteriosclerosis, interstitial nephritis, muscular strain, and such mechanical agencies. Hereditary and congenital defects are to be considered. Secondary chronic endocarditis is associated with rheumatism and tonsillitis, most frequently; and with chorea, pneumonia, and the exanthematous fevers.

Pathology. The left heart is far more commonly affected, the valvular more often than the mural membrane. The myocardium usually is also affected to some extent in the latter case especially.

There is sclerosis of the valves, with formation of small firm nodular prominences, and of yellowish opaque, fatty patches, often deposition of calcium salts, the cusps may be adhered or rigid and curled, and the chordæ tendineæ are shortened and thickened or adhered.

As a result of the changes mentioned, the valves become variously thickened and adherent. The contraction of the chordæ tendineæ modify the structural changes. Thus is produced the "funnel mitral," the "buttonhole mitral," the "annular mitral," etc. The term "chronic endocarditis" should be limited to those cases in which there is a progressive inflammation, and not be applied to valvular lesions in which no further change is occurring.

Diagnosis. The location of the most serious changes is usually determined by the recognition of the valves most affected; mural endocarditis can only be strongly suspected when symptoms of cardiac distress follow acute endocarditis. The symptoms are mostly those of a slowly increasing valvular defect, with increasing hypertrophy which seems hardly able to compensate for the defect. Mild leucocytosis is present in nearly every case. Mild secondary anemia is to be expected. Other blood findings, and the urinary findings, depend upon the effects produced by the circulatory disturbance on other organs, and this depends upon the valve most seriously affected.

Reflex muscular contractions are not present, or are negligible, so long as the inflammation is limited to the endocardium. Pericardial and myocardial inflammations may complicate the endocarditis, and thus reflex contractions be produced in the deep spinal muscles innervated from the second to the fourth thoracic segments. The intercostals are not usually affected unless the pericardium is inflamed also.

Treatment. First, is to be considered the prophylaxis. Patients with conditions ordinarily considered etiologic factors in endocarditis should be guarded against further heart disturbances. Patients recovering from rheumatism, tonsillitis, pneumonia, or the exanthematous fevers should be watched, and not permitted to sit up, or after leaving bed, to undertake any strenuous exercise until the heart is well strengthened and the nerve centers have regained their normal activity. Correction of the lesions affecting the heart

centers, if any are found, is important. Lesions affecting the action of kidneys or liver or intestinal tract may permit toxemia—this must be guarded against in all cases.

The treatment of acute endocarditis, as already given, may also be considered prophylactic for the chronic type. When chronic endocarditis is recognized, the treatment depends to some extent upon the extent and the location of the valvular injury. There are certain factors that are applicable to all lesions. These include properly graded rest and exercise; suitable diet, according to the patient's needs, and his digestion and elimination; and the maintenance of correct structural conditions of the body.

Prognosis. The outlook is doubtful, so long as the endocarditis persists. After inflammation ceases, the nature of the valvular lesions permits a more or less hopeful prognosis according to the conditions as found. In the mural type the injury to the myocardium may be serious; this is not easily determined. The possibility of mural injury should be kept in mind in every case in which the cardiac embarrassment presents unusual features. (See also valvular lesions.)

VALVULAR LESIONS

Injury to the valves of the heart varies from changes so light as to cause no recognizable symptoms, to those so severe as to result in sudden death.

Etiology. Congenital weakness may permit a sudden rise in blood pressure, due to muscular strain, emotional storms, or other causes, to rupture a valve not previously diseased. Such conditions are rarely found. In practically every case injury to a valve is primarily due to inflammation. Rheumatism, tonsillitis, pyorrhea, chorea, the infectious fevers, are the most common causes of endocarditis, with resulting valvular lesion; the cardiac valves may share the effects produced upon the circulatory system by the presence of poisonous substances in the blood stream, such as are present in alcoholism, autointoxication of any type, syphilis, etc., or to poisoning by lead, mercury, and other drugs, or as the result of chronic infections in the body anywhere. Overwork, too little exercise, too little fresh air, too much food and faulty food, and all the unhygienic habits usually considered causative of arteriosclerosis and of acute and chronic endocarditis are also causative of valvular lesions.

Diagnosis. According to the structural changes produced, each valve may present three variations in normal action; narrowed orifice, causing stenosis (obstruction); imperfect closure, permitting regurgitation (incompetency, insufficiency), or the valves may

close imperfectly over the narrowed orifice, causing symptoms of both states.

The diagnosis of the different valves must be considered separately; the treatment of valvular lesions will be considered together, since many factors are alike in all cases.

MITRAL REGURGITATION

This is the most frequent form of valvular lesion. The contraction and narrowing of the leaves of the valves, with or without contraction of the chordæ tendineæ, results in a reflex of blood into the right auricle. This, becoming distended, hypertrophies. Ventricular hypertrophy results from the associated muscular activity. If the left auricle is unable to overcome the back flow, it dilates, the lungs become congested, and the right ventricle hypertrophies. The somewhat congested condition with embarrassed action of the right heart, engorgement of the lesser circulation and congestion of the systemic veins may exist for several years but gradually leads to tricuspid incompetency, cyanotic induration and anasarca.

Diagnosis. The first symptom noticed is shortness of breath on exertion or on going up stairs. Disturbed compensation is marked by dyspnea, cyanosis, palpitation, frequent attacks of bronchitis due to pulmonary congestion, hemoptysis, and persistent cough with blood-stained sputum containing alveolar cells and pigmented granules. Cardiac **"sleep start"** is a disturbing feature —just as the patient falls asleep, he wakes gasping for breath and feeling as if the heart were stopping. There may be a sense of emptiness or cardiac distress, the pain not usually being severe. The face is pale and pinched, the lips and ears dusky, the cheek capillaries are enlarged, the fingers are clubbed, especially in children.

In a child or in older persons in whom the lesion has dated from childhood there is often visible precordial bulging. The apex beat is at first forcible, diffuse and heaving; as compensation fails it becomes feeble or unrecognizable; it is displaced to the sixth intercostal space to the left of nipple line. Thrill is rare. The pulse is at first full, regular, small and soft (low tension). After broken compensation, it becomes irregular, no two beats of equal force or volume.

The area of cardiac dullness is increased transversely and vertically. A blowing **murmur,** systolic in time, heard loudest at apex, propagated to axilla and under the angle of the scapula, modifies or replaces the valve sound. If the murmur is loud it may be heard at the back close to left side of spine, or in the left axilla, or just beneath the angle of the left scapula. The second

pulmonic sound is markedly accentuated particularly in the pulmonary area. The recumbent position makes this murmur clearer.

The **urine** is scanty and albuminous, with tube casts and sometimes blood corpuscles.

The **sphygmogram** shows a wavy irregular line, with occasional normal waves among irregular waves of low and variable amplitude.

Prognosis. No ill effects result as long as compensation can be maintained. There are apt to be many short attacks of apparently failing compensation, from which the patient easily recovers, with rest and other suitable treatment (q. v.). When compensation fails, pulmonary congestion may lead to death. Pneumonia or other pulmonary disease is apt to be fatal, at any time. Dropsy or exhaustion may be the immediate cause of death. Sudden death is less frequent than in other valve lesions.

MITRAL STENOSIS

This lesion is usually associated with mitral regurgitation. Hypertrophy of the left auricle is more marked than in regurgitation alone.

Diagnosis. During compensation (which may last for years) there are no symptoms. When compensation fails, there is a small, rapid, irregular, feeble pulse, dyspnea, cough, signs of pulmonary engorgement, bronchorrhea, frequently hemoptysis, followed by dilatation of the right heart, with general venous stasis, liver greatly enlarged, and death.

Any time during the duration of the lesion, vegetations may loosen and enter the general circulation. If these emboli reach the brain, aphasia, hemiplegia, or other symptoms of cerebral embolism may occur.

The enlarged auricle may press upon the recurrent laryngeal nerve and cause paralysis of the vocal cord of the same side. During hypertrophy of the left auricle an undulatory impulse is seen over its area, with bulging over the lower part of the sternum and fifth and sixth costal cartilages. This is most marked in children. There is a **thrill** or fremitus rough and grating in quality, presystolic in time, terminating in a sharp sudden shock, synchronous with the cardiac impulse, felt best during expiration and in a limited area to the left of the sternum in fourth and fifth interspaces within the nipple line. During dilatation, the cardiac impulse is diffused, feeble and irregular, left near the xiphoid cartilage. During compensation, the **pulse** is slow, regular, small, and of good tension. If the orifice is much narrowed, it is small, weak, and irregular in force and rhythm. If compensation fails, and the right heart is dilated, it is rapid, weak, small in size and

irregular in force and rhythm. The systemic arterial **blood pressure** is lowered because the whole volume of blood cannot be sent out. There is an increased area of **cardiac dullness** on the right side of sternum and along left margin as the hypertrophy is on the right side. It may even extend up to the second rib. A rough blowing vibratory, sometimes rasping or purring **murmur,** high in pitch, is heard in the mitral area, to the inner side of the apex beat or along the left sternal margin, presystolic in time, running up to the first sound in which it abruptly ends, synchronous with the thrill. The first sound is short and clear and abrupt. The second pulmonic sound is accentuated. In the later stages, when the narrowing becomes considerable, a mid-diastolic murmur may appear occupying the whole of diastole. The second sound may disappear. As compensation fails, the murmur may disappear, leaving only a flapping or snappy first sound at the apex of a gallop rhythm. The **sphygmographic** tracing is an irregular line, with somewhat shorter waves than in regurgitation alone. Normal ascents and descents are less frequent, and the irregularity is more marked than in the first lesion.

Prognosis. The outlook is about the same as in mitral regurgitation. Cerebral emboli are apt to occur. Pregnancy and the puerperium are thought to be detrimental, but many women go through repeated pregnancies without rupturing compensation.

AORTIC REGURGITATION

This is the lesion of athletic and able-bodied, vigorous men, and stands next in frequency to mitral regurgitation. The orifices are more or less dilated resulting in the non-closure of the aortic valves, allowing a part of the blood to flow back into the left ventricle. This causes overfilling of the ventricle, hence dilatation of its cavity. The muscle hypertrophies to compensate which may cause the heart to attain an enormous size, the **"cor bovinum"** of the old writers.

Diagnosis. The earliest signs are due to cerebral anemia, headache, dizziness, flashes of light, feeling of faintness on sudden rising, these followed by congestion of the face and eyes, tinnitus aurium, and insomnia. Precordial **pain** is usually present and may be severe, often with anginoid attacks. True angina pectoris is more often associated with this than with other valvular lesions. On the slightest failure of compensation, the cardiac action becomes excessive and distressing, palpitation on slight exertion causing anxiety and fear in the patient. There is gradual or rapid appearance of dyspnea, increased on exertion, cyanosis, hepatic enlargement, renal congestion with scanty, albuminous **urine** and edema of the feet. Ascites is rare. **Mental** disturbances are fre-

quent. Cases of neurasthenia in athletic persons, and of suicide during apparently vigorous health, are more often due to this lesion than is generally recognized. Syncope is frequent. Secondary **anemia** may be marked; the red count may be lower than 3,000,000 per cu. m m.

The cardiac **impulse** is forcible, displaced downward and to the left, pulsation far beyond normal apex to sixth or seventh interspace in left anterior axillary line and accompanied by a characteristic jerking. A diastolic **thrill** may be felt over the base of the heart and adjacent to the large vessels. The apex beat is found in the seventh or eighth interspace in anterior axillary line. The pulse is rapid and characterized by a sudden rise and a sudden fall producing a peculiar kicking sensation to the finger—**"water-hammer"** or Corrigan's pulse. There may be retardation of the pulse so that there is an appreciable interval between the heart beat and the radial pulse. The **sphygmographic** tracing is characterized by sharply rising anacrotic wave, to great height; the katacrotic wave drops with a needle point; the dicrotic is very sharply marked. With failing compensation or the development of other valve lesions, the line becomes variably irregular. **Capillary** pulse is well-marked in the finger nails or in the lips as an alternate flushing and paling. Pulsation in peripheral vessels is more common in this than in other forms.

There is increase of **cardiac dullness** downward and to the left, occasionally upward and to the left of the sternum from hypertrophy of the left auricle, and associated with massive hypertrophy. A soft blowing prolonged **murmur,** of low pitch, and a churning or rushing character, is heard in the aortic area, most distinct at junction of sternum and fourth left costal cartilage, diastolic in time, and transmitted down the sternum and toward the apex. It may modify or replace the second sound which is usually absent. Auscultation over the carotid artery may reveal the second sound when it is not found at the aortic cartilage. This indicates a small amount of regurgitation, hence a better prognosis. Double murmurs may be heard on auscultation over the carotids and subclavians.

The persistent and uniformly high systolic blood pressure with the low diastolic pressure, gives high pulse pressure; this is pathognomonic.

AORTIC STENOSIS

This is chiefly a disease of advanced life, associated with arteriosclerosis. There is usually simply a slow sclerotic change in the valves, usually with some regurgitation. Hypertrophy of the left ventricle follows the gradually diminishing orifice.

Diagnosis. There are no noticeable symptoms as long as the hypertrophy keeps pace with the stenosis. Later, syncope, vertigo,

headache and insomnia or bad dreams occur. Anemia is present; emboli may lead to serious complications. The apex beat is slow, heaving, forcible, displaced to the left according to the hypertrophy. A marked **thrill** at the base of the heart and of maximum force in the aortic area is felt. In no other condition is there such an intense thrill. The pulse is small, hard, slow **(pulsus tardus)**, often interrupted, and the tension often increased depending upon the obstruction and degree of hypertrophy. The **sphygmogram** shows a blunt ascent, often with a notch near the summit; a plateau marks the height of the anacrotic wave. The katacrotic wave descends slowly, and the dicrotic wave is rarely visible. The base line may be prolonged. The area of dullness is never as wide as in aortic regurgitation. The first sound is replaced by a harsh, loud, rasping, sometimes whistling or often musical **murmur**, heard best in the aortic area at junction of the second right costal cartilage with the sternum. It is systolic in time, and transmitted into the carotids. When associated with aortic regurgitation, there is a double or see-saw murmur. There are other murmurs in this area not due to stenosis; the hemic murmur has a soft bruit; calcareous plates in the aorta or on a cusp produces a sound very similar to the stenotic murmur.

The prognosis is comparatively favorable, as hypertrophy is usually of good degree and easily maintained with a quiet life. When the stenosis is solitary, it is often due to atheroma, with danger of cerebral hemorrhage.

TRICUSPID REGURGITATION

The general symptoms are due to retarded pulmonary circulation and visceral congestion, marked as follows: In the lungs, as dyspnea, bronchitis, or pulmonary edema; in the digestive tract by dyspepsia, hematemesis, ascites and slight hepatic enlargement, tenderness and icterus; in the kidneys by scanty high-colored urine, varying amounts of albumin, few hyaline casts, isolated red blood cells and general dropsy and uremia; in subcutaneous tissues by edema beginning in the feet and ankles and extending upward, and in grave cases by ascites, hydropericardium or hydrothorax.

Jugular pulse-wave is observed more often in the right than in the left vein. The cardiac impulse is feeble and extends downward. The rhythmical expansile liver pulsation is best obtained by laying one hand over the fifth and sixth costal cartilages and the other hand over the lower border of the liver in the mid-axillary line. The jugular vein, when obstructed by the palpating finger, fills up from below during systole. There is hypertrophy of the right ventricle. A soft blowing **murmur**, low in pitch, systolic in time, is heard distinctly over the xiphoid cartilage, or the head of fourth rib to right of sternum, and within an inch of the

apex, thus limiting the area of transmission. The second pulmonary sound is weak. There may be either a single or a double sound in the crural or other large superficial veins. The pulse is markedly irregular.

The prognosis is always grave. Each case must be considered on its own merits.

TRICUSPID STENOSIS

This is a rare congenital or acquired affection occurring secondary to disease of the left heart or associated with acute endocarditis. Clinically, cyanosis of the face and lips is commonly seen, this becoming pronounced when dropsy occurs. The physical signs are transverse enlargement of the heart, particularly the right side; a presystolic murmur heard at the base of the ensiform cartilage, and a presystolic thrill. Dilatation of the auricles soon follows with venous stasis, and venous pulsations as in tricuspid regurgitation, with which it is usually associated.

The prognosis is always very unfavorable; death is usually imminent when the diagnosis is made.

THE PULMONARY VALVES

Murmurs heard in the region of the pulmonary valves are extremely common, but lesions of the valves are exceedingly rare. It is often called the "area of auscultatory romance," of Balfour.

A systolic murmur is heard in healthy, thin-chested individuals, particularly children, during expiration and in the recumbent position; with rapid heart action as in fevers or after exertion; in most anemic states. These functional, anemic, or hemic murmurs are always at the base of the heart; always systolic; not transmitted away from heart; soft in character; low in pitch; variable in intensity, now heard, now absent.

PULMONARY STENOSIS is the commonest of the congenital murmurs and may be associated with constriction of the pulmonary artery; patulous foramen ovale; patulous ductus Botalli or its stricture; imperfection of the ventricular septum.

Hypertrophy of the right ventricle may follow and establish compensation. The child is weak, markedly cyanosed, with flabby tissues, soft bones, and a generally poorly nourished condition. The physical signs are marked enlargement of the right ventricle, a loud systolic murmur with a thrill heard best to left of sternum in the second interspace and not transmitted to the vessels. The second pulmonic sound is weak or absent or replaced by a diastolic murmur.

The prognosis is unfavorable, the little patients dying in a few days to a few months. Sometimes, compensation is established so that they live longer but they are always weakly.

PULMONARY REGURGITATION is a rare affection usually of congenital malformation; the changes are similar to those of aortic insufficiency.

The general symptoms are referable to dilatation of the right heart and consequent pulmonary congestion. Suffocative attacks are marked. On examination, the cardiac dullness extends to the right of the sternum, a loud blowing diastolic murmur is heard most distinctly at the junction of the third left costal cartilage and the sternum, transmitted down the sternum. Death occurs from dropsy and exhaustion.

Treatment of Valvular Lesions. Functional recovery is to be expected—this is due to compensatory hypertrophy of the myocardium. This is to be secured only when the conditions which control the action of the heart are normal—or approximately so. Modification of the heart's rate and force to meet varying physiological states depends upon normal activity of the cardiac nerve centers and upon the maintenance of fairly normal blood and blood pressure. In order that the most speedy and perfect compensation can be secured, all factors that might interfere with the normal activities of the heart centers must be removed. This includes lesions of the upper thoracic and cervical spine, the clavicles, and the upper ribs; all factors which raise the blood pressure unduly must be removed; this includes lesions of the lower thoracic spine, especially; all strenuous exercise, all emotional storms and all causes of gastric disturbances or flatulence. All factors which diminish the nutritive qualities of the blood, or which add to its toxic elements, must be removed. This includes the use of alcohol or tobacco, the overuse of meats and carbohydrates, any dietetic errors, constipation, and lesions which might interfere with the activities of skin, kidneys, lungs, liver, or any other of the organs concerned in nutrition or elimination. Relief may be given by raising the lower ribs and elevating the abdominal viscera. Forced expiration, with contraction of the diaphragm, may give relief.

In case of pain or sudden dilatation with cyanosis, cold locally over the heart is indicated, such as an ice bag, Leiter's coil, or compresses kept cold. Insomnia is often relieved by a cup of hot gruel or a tepid bath at bed time. Massage to limbs at bed time may encourage sleep. The condition of the heart and respiration must be noted, and the blood pressure variations watched constantly at first; each patient presents more or less peculiar idiosyncrasies.

"In giving a summation of the osteopathic treatment for heart conditions in general we must aim to reduce the work of the heart to the minimum. The disturbed circulation must be controlled by careful attention to the vasomotor nerves at the various centers along the spine. Treatment must be given to correct any lesion found in the ribs or vertebræ from the first to the tenth dorsal. See that the tenth cranial nerves are not obstructed anywhere along their course. The ribs must be spread, the sternum raised and the chest expanded to the utmost to give the lungs free action. The importance of the lung is often overlooked in the treatment of heart troubles. We must obtain the maximum amount of oxygen to nourish the blood. Relax the muscles all along the line. Remove the lesions of the cervical area, and free up the cervical ganglia of the sympathetic chain. A general treatment to quiet the patient may be indicated. Remember the possibility of a reflex irritation at some remote point. The excretory organs must be kept active and a proper diet ordered.

"It is difficult to outline with exactness the treatment for a given heart lesion on account of the varied causes that may contribute to the condition. In general, look for all the causes and remove them as far as possible. Try to lessen the work of the heart and aim to build up compensation by giving the heart good nourishment for its work."—L. J. Bingham.

"The existence of the lesions as found is held to interfere with the progress of the compensatory hypertrophy.

"The treatment of these cases includes the correction of whatever bony lesions are found which might interfere, (a) with the innervation of the heart, (b) with the nutrition of the body, (c) with the maintenance of a normal blood pressure. The correction of any abnormal habits of eating, sleeping, resting, etc., which may be present is held to be an important part of the osteopathic treatment, as is also the cessation of whatever drugs the patient may have been using. Usually the drugs are stopped at once, but sometimes, when the patient is very weak and nervous, the drug-habit is stopped gradually. In every case recorded the drug has been discontinued within the week."—P. C. O. Clinic Report.

"The treatment of valvular lesions in general and mitral lesions in particular, is not confined to the lesions at the valves, but concerns the resulting complications. The symptoms of cardiac lesions do not manifest themselves until the heart muscle is worn out, when compensation is lost. . . .

"The fact that most cases recover compensation under proper treatment and the heart muscle is able to do its work fairly well, even after it had been apparently completely exhausted, is evidence that the weakness was not due to degeneration, but to fatigue. . . . First, we must have absolute rest in bed to relieve the heart of all unnecessary labor; second, daily osteopathic treatment practically the whole length of the spine; in the cervical and upper dorsal regions to improve the tone of the cardiac muscle and the tone of the peripheral arterioles; in the middle dorsal, for a tonic effect upon the lung tissues and the great splanchnics, and to increase the action of the sweat glands; in the lower dorsal, for a diuretic effect upon the kidneys; in the lumbar region, for the bowels. . . .

"The diet should be confined to milk and milk products, both for nourishment and for its diuretic effect. . . . After compensation has been successfully reëstablished, the patient should be kept under observation for at least a year, and the same hygienic and dietetic rules laid down for the stage of compensation must be strictly adhered to."—C. J. Muttart.

The prognosis of any cardiac valve lesion depends upon the efficiency of compensation. This in turn is dependent upon several factors. In children under ten years, the outlook is grave, although sudden death is rare. Lesions acquired during puberty are more apt to be permanently compensated. Women bear valvular disease better than men.

To justify a favorable prognosis, several factors must be present; a good general health, good habits, the existence of no exceptional liability to rheumatism or catarrh, that the origin of the valve lesion is independent of the degenerations, the existence of the valve lesion without any change for over three years, the ventricles acting with moderate frequency and general regularity, the presence of sound arteries, and freedom from pulmonary, hepatic, or renal congestion.

CHAPTER XVII

DISEASES OF THE BLOOD VESSELS

GENERAL DISCUSSION

The lining of the arteries, veins and heart is continuous and practically identical. Through this channel, with its lining membrane of a single layer of endothelial cells the blood flows with constantly varying pressure and carrying varying combinations of food and of waste materials.

The walls of the blood vessels are nourished by the vaso-vasorum; these are controlled by vasomotor nerves, as are other tissues of the body. The muscular walls of the vessels are also controlled by nerves which vary their caliber. Both these sets of nerves carry impulses from nerve centers; these, like other nerve centers, are influenced by the impulses reaching them over sensory nerves, and from related centers in other parts of the nervous system. Thus, bony lesions affect the nutrition and the functional activities of the vascular walls. The blood vessels are nourished by the blood, as are all other tissues of the body. Thus, they are subject to the toxic and nutritional variations in the blood. They carry the blood under pressure, and so are liable to injury from improper changes in this pressure. These three factors—variations in nervous control, variations in the quality of the blood, and variations in the pressure of the blood, are the factors which make up the etiology of diseases of the blood vessels and which also determine the methods most efficient in securing recovery from these conditions.

The practical aspect of this question is of great importance. In the first place, the etiological value of the bony lesion in causing disturbances in the pressure of the blood must be recognized. Slight malpositions of vertebræ and ribs, or other forms of peripheral irritation anywhere, may affect the chief vasomotor centers, or the subsidiary centers in the cord or medulla. If, as the result of these lesions, or of other factors, the blood pressure falls too low, the nutrition of the entire body may be affected; this malnutrition, with the associated accumulation of more or less toxic waste products of metabolism, predisposes to vascular disease no less than does the abnormally high blood pressure which may be due to bony lesions as well as to the causes given for arteriosclerosis in general.

The **treatment** of diseases of the blood vessels depends upon the removal of these factors—those which interfere with normal

nervous control; those which interfere with the normal pressure or the normal flow of the blood, and those which interfere with the normal quality of the blood in the vessels.

ARTERIOSCLEROSIS

(Arterio-capillary fibrosis; atheroma; endarteritis chronica deformans; arterial sclerosis)

Arteriosclerosis is a rigid condition of the arteries produced by fibrous thickening of their walls; it is associated with fibrous changes in other organs and is marked clinically by hypertension and functional disturbances dependent upon the location and extent of the fibrosis.

Etiology. The hardening of the connective tissues, especially of the arteries, in old age is so constantly observed in man and the higher animals, that it may be considered a factor in normal living. Among the factors producing this hardening prematurely, may be included heredity; poisons such as lead, mercury, alcohol, drugs; syphilis, nephritis, malaria, rheumatism, gout, diabetes, and other diseases with toxic characters; the products of intestinal putrefaction and fermentation, especially indol and related substances; and the continued high blood pressure due to overeating and drinking, excitement, worry, too great and frequent muscular effort, etc. It is seen that these causes fall into two groups—the toxic and the mechanical.

The spinal column is invariably rigid, especially in the lower thoracic region. This abnormal rigidity may be due in part to the hardening and rigidity of all tissues not kept supple by use, but it is certainly an important causative factor. Rigid thorax is common. Lateral curves are less frequent than slightly posterior positions, or the "ramrod" spine.

"Where the sclerosis has attacked the upper limbs, the most frequent lesions have been from the third to the fifth dorsal vertebræ, and as has been the case of an unequal blood pressure, the lesions were rotations of those vertebræ. In two incidents, bi-lateral sclerosis of the radials existed without apparent involvement of other palpable arteries. The lesion was of an impacted anterior nature extending from the second to fifth dorsal.

"Cases exhibiting unequal tortuosity of the facial and temporal arteries, in my experience, have invariably shown lesions of the second, third and fourth cervical vertebræ and likewise, invariably these have been rotations toward the sclerosed side.

"There is another type of sclerosis common to osteopathic practice, the etiology of which is apparently auto-intoxication."—F. C. Farmer.

Pathology. The aorta is first in frequency of disease, next come the coronary arteries. The cerebral, temporal, radial, brachial, ulnar, femoral are found affected in the order named. The change seems to begin in the intima, with slightly raised thickenings, due to multiplication of the endothelial cells. This condition is called "atheroma" or "gruel-tumor" on account of its consistency. The media and adventitia undergo inflammatory changes as a result

of the pressure or toxins, muscle fibers are absorbed, and the connective tissue overgrowth results in the formation of thick, weak, inelastic walls. The atheroma may soften and the wall of the vessel become perforated, or may dilate greatly, with the formation of an aneurysm. Calcification may occur in the areas affected most by the disturbed circulation.

Diagnosis. The symptoms are hypertension of the pulse, hypertrophy of the left ventricle, accentuation of the second sound in the aortic area, and thickening of the peripheral vessels. Early there may be a prolonged first sound. The arterial walls are palpated by obliterating the pulse when they appear like cords under the finger. Sometimes irregular thickenings or the edges of calcareous plates in the walls can be palpated. Arteriosclerosis may be advanced in the aorta and other great vessels without symptoms. Later stages are marked by rigidity, visibility, and tortuosity of the peripheral arteries. When the heart is failing, the sounds are feeble, often irregular and intermittent. Among the general symptoms of arteriosclerosis are periods of mental lassitude, irritability with headache occurring after mental or physical excitement, digestive disturbance, momentary attacks of dizziness accompanied by nausea and followed by profuse sweating and temporary weakness. Insomnia, loss of memory, melancholia, fear, show gradual loss of mental vigor and bodily tone.

Sclerosis of the renal arteries produces urinary changes and the arteriosclerotic kidney, sometimes chronic interstitial nephritis. When the cerebral arteries are involved, there may be transient attacks of hemiplegia or monoplegia or aphasia with recovery within twenty-four hours and recurrences later; aneurysm, rupture or thrombosis with their attendant phenomena. Sclerosis of the coronary arteries is a factor in producing narrowing, thrombosis, angina pectoris, or aneurysm of the heart. When the arteries of the extremities are affected thrombosis and senile gangrene or intermittent claudication or lameness (crural angina) may occur.

Urinary changes are characteristic. There is intermittent albuminuria, urea and nitrogen are normal, uric acid is low, xanthin bases are increased, sediment is scanty and hard to find, casts are rare, and uric acid and calcium oxalate crystals are common.

The blood pressure is increased. There may be only a constant slight raise of 20 to 30 mm. of Hg. above normal or it may run high; 160 to 180 mm. of Hg. is Faught's average.

Treatment. Increasing the mobility of the dorsal spine and of the chest with elevation of the ribs is indicated. Whatever luxations are present should be corrected. Elimination by every avenue is to be stimulated. Renal efficiency is to be maintained by careful work on the renal splanchnics, diet, and hygienic regulations. Direct abdominal work assists in equalizing the circulation and lowers the blood pressure.

"Judgment must be used in attempting to correct faulty blood pressure as well as other conditions. Relaxing treatment must not be adopted merely on the strength of sphygmomanometer readings; a thick vessel with a comparatively low reading, say 135, may require to have its tone raised, while a thinner-walled vessel, with a reading of 125, may advantageously have its tone lowered.

"The chief difficulty, it seems to me, is that most people confuse arterio-sclerosis with high manometric readings. A manometer measures the resistance of an artery to outside pressure, which is often due to hypertonicity of the vessel, itself in turn due to some form of autointoxication. The high reading is not necessarily due to arterio-sclerosis."—C. M. Bancroft.

The corrective movements indicated in these cases should be given slowly and gently. No increased sensory stimulation should be sent into the centers from the articular surfaces. The lesions must be corrected without stimulating the nerve centers. Directions as to hygienic measures must be given with due regard to the pressure changes liable to occur. With care, the blood pressure may be kept within reasonable limits even while considerable corrective work is being done. Careful watching of the pulse may prevent undue rise of blood pressure in the aged and arterio-sclerotic. The same considerations are important in outlining the treatment and the hygienic advice for patients with cardiac lesions, aneurysms, and all conditions in which a rise of blood pressure is harmful or dangerous.

An exclusive milk diet is often of service in reducing a dangerous hypertension. Loss of strength is to be avoided. In obese cases reduction of weight must be gradual. Fruit and vegetables with the milk products should be freely used. Meat and starch are to be kept low. Sodium chloride, spices, tobacco, alcoholic drinks, are to be omitted or greatly moderated. If any particular food advised is found to cause renal irritation it must be stopped. A water intake of about 1500 cc.—3 pints—is best in most cases.

A quiet life is best, but the patient must not be unhappy. Baths must not be of extreme temperatures either hot or cold. Warm baths and sponging daily are of the best use. Exercise should be taken regularly, moderate out-door sorts being best. Too sudden changes in altitude are not advisable, nor residence above 3,000 feet suitable. An even climate is desirable.

Prognosis. Structural changes can be prevented but not removed. If the case is recognized early before renal changes are apparent, diet, hygiene, and a general lower plane of living instituted, the promise of a comfortable life for many years may be made. The condition is not favorable for recovery but not incompatible with life. In the later stages, some circulatory accident is apt to occur on slight provocation. These include apoplexy and sudden death.

PHLEBOSCLEROSIS

(Sclerosis of the veins)

This condition may accompany arterio-sclerosis, hepatic sclerosis, or mitral lesions. The pathology and treatment are those of arterio-sclerosis. It is not

often diagnosed ante-mortem. The treatment consists in relieving the high venous blood pressure and usually in the increased elimination of toxic substances from the blood stream.

ANEURYSM

An aneurysm is a persistent localized dilatation of an artery. It may be fusiform, saccular or cylindrical in form; it is called axial when the entire circumference of the artery is affected; peripheral when only one side forms the sac. Miliary aneurysms are so called from their small size; they are found on the cerebral arteries. A false aneurysm is produced when rupture of the arterial coat, usually very small, permits the blood to escape into the perivascular tissues. The connective tissues, with the coagulation of the blood and the organization of this clot, form a sac which may become very strong. A dissecting aneurysm is formed when the blood penetrates into space between the arterial coats, separating them. This blood may coagulate and organize with no further harm. Aneurysmal varix is formed when an arteriovenous connection is made through the rupture of weakened arterial wall into an adjacent vein. When this connection is made through an intervening sac, the structure is called a varicose aneurysm.

Etiology. There are two main causes; damage to the vessel wall from arteriosclerosis particularly when of syphilitic origin, or from causes acting from without the vessel; and increased vascular strain as a result of laborious occupations. Among the more general causes are toxemias and conditions affecting the innervation of the vessels from spinal or other subluxations. It is a disease especially frequent in middle life.

Diagnosis. In terminal arteries, serious symptoms may be produced by aneurysm even of small vessels; otherwise no recognizable symptoms are apt to follow unless the affected vessel is an arterial trunk. Tumor, pulsation, systolic murmurs audible over the dilatation, pain, pressure symptoms, and the results of the impaired circulation are the symptoms most commonly present. Other symptoms occur, according to the location of the aneurysm. The X-ray gives accurate information concerning thoracic aneurysm.

Aneurysm of the Aortic Arch. This is the most common form. The onset is gradual with arteriosclerosis and generally failing health. Pain, dyspnea and cough may vary in degree and be either constant or intermittent. The tumor may produce visible bulging with pulsation. Corrigan's sign—expansile pulsation—with thrill and diastolic shock, tracheal tugging, and tenderness over the affected area, may be found. When the aneurysm is in

the transverse arch or in the subclavian artery the pulse and blood pressure vary in the right and left carotid or radial arteries.

The abnormal area of dullness with increased resistance is often evident. Over the tumor there is a murmur or bruit synchronous with the first sound, louder than systole, lower in pitch, and of a blowing character. When the aortic valves are intact, the aortic sound will be markedly accentuated.

Thoracic Aorta. This is most frequent among men who are prematurely aged or engaged in occupations which tend to increase normal aortic strain; syphilis is the most constant factor and septic emboli and traumatism are worthy of mention. The symptoms are due to pressure, and depend upon the direction of protrusion. They may include dysphagia; dyspnea, "aneurysmal asthma"; alterations of the voice, as stridor, aphonia, "leopard growl," "goose cough," "gander cough"; hemoptysis; severe hiccough; dilatation or contraction of the pupils, unilateral or bilateral; pallor or flushing of the face, unilateral or bilateral; cardiac irregularity; vomiting, nausea; edema of the arms and face, sometimes unilateral; and other symptoms due to pressure upon nerves or vessels. When the thoracic duct is involved fatty stools and rapid emaciation may veil the diagnosis. Erosion of the bodies of the vertebræ and pressure upon the cord cause severe boring pain, followed by paraplegia and death.

The expansile pulsation, systolic thrill and diastolic shock are found above the third costal cartilage. Tracheal tugging may be present. Percussion adds a dull flat note over the tumor with increased resistance. The apex beat is displaced.

Sometimes differences are found in the radial pulses or the pulse may be retarded in the vessels beyond the aneurysm.

Over the dull area a ringing accentuated second sound and a systolic bruit are characteristic.

Descending Thoracic Aorta. When beyond the arch, aneurysm is often latent. In other cases, there is pain in the back from erosion of the vertebræ, sometimes dysphagia, and occasionally a pulsatile tumor is found to the left of the spine.

Abdominal Aorta. This occurs most commonly near the cœliac axis; it may grow upward and push the diaphragm before it, or backward and erode the vertebræ. Characteristic dull boring pain and neuralgias are thus produced.

Palpation reveals a definite tumor with aneurysmal characteristics. The bruit is heard to the left of the median line and the femoral pulse is retarded. Compression paraplegia, embolism of the superior mesenteric artery, complete obliteration of the lumen or rupture lead to death. Diagnosis of aneurysms must be made from mediastinal tumors, pulsating emphysema necessitatis, aortic

insufficiency, cardiac displacements by other thoracic conditions, neurotic pulsation of the aorta, and abdominal tumors.

Aneurysm of the Pulmonary Artery is rarely diagnosed from thoracic aneurysm. It is rare, and is caused by phthisis, mitral disease, emphysema, or other causes of obstruction to the pulmonary circulation.

Aneurysm of the Splenic Artery is rarely recognized. The symptoms include deep-seated abdominal pain, hematemesis, sometimes hepatic disturbances. A pulsating tumor with systolic murmur, may lead to a diagnosis. Gastric ulcer or gastric cancer may be confused with this aneurysm.

Aneurysm of the Hepatic Artery is very rare. Severe pain, vomiting, hematemesis and jaundice are present.

Aneurysm of the Mesenteric Arteries cause pain and vague intestinal disturbances. They are apt to rupture into the peritoneal cavity, causing sudden death. Ante-mortem diagnosis is practically impossible.

Miliary Aneurysms of the Renal and the Cerebral Vessels are not infrequent. They may produce no symptoms, or various vague pressure symptoms.

The **Treatment of Aneurysm** depends largely upon the location. Every manipulation must be very carefully considered as there is no way of estimating the strength of the aneurysmal wall. Relaxation of the contracted spinal muscles, and very careful treatment to tone up the general nutrition, secure elimination and quiet the circulation are some of the measures necessary. Absolute rest of body and mind is essential when the tumor is marked. The diet must be nourishing but limited and the liquids reduced to a minimum. Alcohol and its allies are strictly prohibited.

Rest in bed, ice over the affected area, long, steady pressure over the tissues near the tenth to the twelfth thoracic spines may relieve the pain and lower the blood pressure. The symptoms are to be treated as they occur; much relief can often be secured by careful treatment according to the spinal conditions as found on examination each day.

If the blood pressure can be kept rather low, the coagulation of the blood is hastened and the danger of embolism lessened. The restoration of normal nerve stimulation to the injured arterial walls may cause increased tone and a tendency to recovery. This is best secured by spinal treatment.

Very low liquid intake lowers the blood volume and increases its viscidity. The free use of gelatine is supposed to increase the coagulation of the blood. The "pressure treatment" of abdominal aneurysm has been of value in selected cases; steady, increasing pressure over the proximal portion of the dilatation for twenty-

four hours beginning under anesthesia is the method usually followed.

Surgical treatment should be considered. In selected cases the injured artery may be repaired very efficiently. Each patient requires special consideration as to the technique to be employed.

Prognosis. Recovery may occur in the smaller arteries, in the dissecting aneurysms, and sometimes in the peripheral sacular forms, through thrombosis or occlusion. In terminal arteries, the infarct thus formed may cause serious symptoms or death. The danger of embolism during the formation and organization of clot must be remembered; this danger is slightly lessened by preventing sudden variations in the blood pressure or in the position of the body. The outlook is always grave, and sudden death may occur from rupture, embolism, pressure upon vital organs, nerves, or from disease in distant organs resulting from the circulatory disturbance.

VARICOSE VEINS AND HEMORRHOIDS

(Phlebectasia; varix; varicosities)

Veins which lose their elasticity and become permanently dilated from the pressure of the blood within them are called varicose. The condition is characterized clinically chiefly by pressure symptoms. When the venous walls break down, or when the nutrition of surrounding tissues become lessened by the disturbed circulation, varicose ulcers are apt to occur. These are usually very indolent, obstinate and not particularly painful unless nerve trunks are involved.

Etiology. Phlebitis and phlebosclerosis weaken the vessel walls, and thus predispose to varicosities. The most important factor is an impediment to the onward flow of venous blood. Veins which have no valves, and those subjected to the effects of gravity are most apt to be affected; this places the largest number of varicosities in the legs and the rectum. Women suffer more than men; probably partly on account of dress; partly the puerperal state, and partly lack of exercise. Cardiac and hepatic disease especially delay the flow of blood and lead to hemorrhoids as well as to varicosities of the legs.

Bony lesions affecting the centers controlling the vessels affected must be considered important in etiology. This is especially found to be true in hemorrhoids.

Hemorrhoids are dilated hemorrhoidal veins. Especially when the portal circulation is obstructed, collateral circulation is apt to be reëstablished partly by means of the relations of the hemorrhoidal veins with the vena cava. Thus the veins are subjected to greatly increased pressure, and hemorrhoids result.

Internal hemorrhoids—those which do not protrude beyond the external sphincter—are more painful and more urgent causes of the neuroses than are the external—those which do protrude beyond the sphincter.

The passage of feces over the tumors usually erodes the membranes; these areas become infected with tubercle bacilli or other bacteria, and varying degrees of fistulous and burrowing abscesses result. Very obstinate and serious tissue destruction may thus be initiated. The condition is much worse when the fecal masses are hard and dry and when defecation is attended with straining. The relation of constipation (q. v.) to hemorrhoids is evident.

The rupture of the veins into the surrounding subcutaneous tissues may be followed by the coagulation and organization of the blood, and the formation of nodular masses of scar tissue which may be responsible for serious nervous disturbances. Or the hemorrhagic blood may become infected, and thus abscesses of varying extent may result.

The treatment of dilated veins includes the correction of the causative factors, when this is possible. Hemorrhoids require relief of the constipation, and the use of such oil or water enemas as are necessary to secure very soft feces and easy defecation. The hepatic and cardiac diseases must receive suitable treatment. Uterine mal-positions, neoplasms, and enlarged prostates must receive suitable care. Rest in bed is most helpful.

Patients must not sit to rest when the recumbent position is possible; they must not stand for long times under any circumstances. Correction of the coccygeal, sacral, innominate and lumbar lesions is very important. Unduly contracted sphincters should be treated by very slow and easy dilatation. Pain is to be avoided as much as possible in this work.

VARICOSE ULCERS

These result from the breaking down of the tissues in the neighborhood of a varicose vein—this may or may not be associated with rupture of the vein. The tissue injury is marked, and the constant pressure exerted by the varicosity makes such ulcers chronic and resistant to the usual therapeutic measures.

Treatment. Correction of innominate and lumbar lesions is of first importance. Leg movements which facilitate the flow of blood upward should be frequently given, even if no apparent tension exists around the groin. All tissues around Poupart's ligament and the sciatic notch must be watched and kept relaxed. The ulcer itself is not to be handled, though very careful crowding of the surrounding tissues toward the ulcer, thus filling it with fresh blood and lymph which is immediately drained away when the

pressure is again relieved, is helpful. The patient should lie down several times during the day, even if he can only rest for a few minutes this relief of the tension is useful. The use of the elastic stocking or of elastic bandages depends upon the individual conditions. If the patient is able to spend considerable time lying down, or to remain in bed most of the day, the most rapid recovery occurs with no pressure upon the leg at all. If he must be on his feet for long intervals, some support is usually required.

In all cases, the correction of whatever structural conditions may be found, which could interfere either directly or indirectly with the circulation or with the nervous control of the legs, is a very important factor in the treatment of the varicose veins as well as of the varicose ulcers.

EPISTAXIS

(Nose bleed)

Bleeding at the nose may occur as the only symptom in any one of a number of distinct diseases. Wounds of the nasal mucosa do not usually present any difficulty in diagnosis. Nasal polyps, ulcers, or merely hyperemia of the mucous membrane may be responsible for the condition. The latter factor is no doubt associated with bony lesions in the upper cervical or the upper thoracic region. Fracture of the base of the skull usually produces rather obstinate nasal hemorrhage. Influenza, syphilis and a number of other infectious diseases may so act upon the blood as to prevent coagulation; for this reason children who are liable to attacks of nose bleed upon slight provocation should be very carefully protected. Hemophilia, pernicious anemia and leukemia may show as the first symptom an attack of nose bleed. Vicarious menstruation should be mentioned. Changes in atmospheric pressure, especially rapid ascent of a mountain or other rapid elevation, may cause nose bleed. Nose bleed may be symptomatic of high blood pressure.

Treatment. For the relief of the attack an ice bag or a cloth wet in cold water may be placed around the neck. The nose may be packed in obstinate cases; this is rather to be avoided if possible. Steady pressure in the suboccipital region lowers the general blood pressure; steady pressure over the region of the eighth to the tenth transverse processes dilates the splanchnic vessels, and mechanically draws the blood from the head region. During the intervals, in habitual cases, the cause of the weakness should be found and removed, if possible. The prognosis depends upon the cause of the bleeding.