free diuresis is set up, and dropsy lessens and disappears.

In mitral incompetence, not only is the muscle stimulated, but the regurgitation is lessened owing to the more perfect closure of the auriculo-ventricular ring. In stenosis the lengthened diastolic period allows more time for blood to pass from the engorged auricle through the narrowed valve.

The action of digitalis is somewhat slow and it is not until the third day that the full effects of the drug are seen. When marked slowing of the pulse to 60 or under, or a fall in the quantity of urine occurs, the dose should be lessened or stopped, owing to the danger of toxic manifestations, which reveal themselves in the form of nausea and vomiting, weakness and irregularity of the pulse, or a double beat of the heart to one of the pulse.

The drug should be given freely for from forty-eight to seventy-two hours when the symptoms are at all urgent—say in doses of ♏ xv. of the tincture or ʒ iv. of the infusion every three or four hours. Smaller doses are usually sufficient (♏ x.–xv. t.i.d.) in the absence of severe symptoms. Nausea and vomiting are sometimes produced by even moderate doses, and in such cases rectal administration of one or more large doses of the fluid extract are often extremely beneficial. Five or ten minims (♏ i.–gr. i. digitalis) administered with starch in this way often act rapidly, and I have seen the pulse slowed and dyspnœa relieved in the course of a few hours. An old and reliable preparation is Guy's pill, consisting of gr. i. each of digitalis, squills, and pil. hydrarg.

Strophanthus is a valuable substitute for digitalis. It acts on the heart like digitalis, but has not the same tonic effects on the arteries. It is consequently sometimes preferred in arterio-sclerotic cases with increased arterial tension. It is inferior to digitalis in removing dropsy, but is sometimes of value in alleviating cardiac symptoms when this drug fails. Sparteine acts in a similar manner to strophanthus, but as a rule is inferior to it.

Caffeine is occasionally useful as a substitute for digitalis. It sometimes induces restlessness and insomnia.

Suprarenal extract has recently come into use as a cardiac tonic; it acts on the muscular substance of the heart. Some of the clinical reports are very encouraging and it seems worthy of further trial.

Strychnine is a valuable adjunct to digitalis and may be administered in doses of gr. $\frac{1}{30}$ three to six times daily.

Diffusible stimulants such as alcohol, ether, and ammonia are of value in emergencies, and until the more slowly acting stimulants affect the heart.

A dose of calomel, gr. v. with gr. x. of bicarbonate of soda, is often administered with benefit preceding the course of digitalis. It relieves portal congestion and engorgement of the right heart, and increases the efficacy of digitalis.

In arterio-sclerotic cases the long-continued administration of iodides often proves of much value in controlling palpitation and other symptoms, possibly by lowering peripheral resistance. Nitroglycerin is also a valuable agent in lessening peripheral resistance and is often advantageously given with digitalis in doses of ♏ i. of the one-per-cent. solution.

Obstinate or persistent symptoms often require special measures for their mitigation or relief.

Dyspnœa continuing after the use of digitalis, and especially when nocturnal in character, is usually relieved in a remarkable manner by a hypodermic injection of morphine, and in advanced cases this drug is invaluable.

Sleeplessness sometimes yields to paraldehyde, trional, or sulphonal, but, as it is often dependent on dyspnœa, these remedies are much inferior to morphine.

Pain over the cardiac region is often alleviated by the iodides, especially in arterial cases, or by nitroglycerin.

Dropsy.—Dropsy may be diminished by free purgation. For this purpose calomel, salines, and hydragogues, especially compound jalap powder, are most serviceable.

Needle puncture of the distended limb with strict aseptic precautions allows considerable quantities of the fluid to drain away. Southey's tubes are also very efficacious, but there is considerable danger of inflammatory changes taking place in the subcutaneous tissues.

Theobromine in doses of gr. lx. to gr. xc. daily may be used with occasional benefit as a diuretic when the urinary secretion is persistently small.

When considerable quantities of fluid collect in the abdominal or the pleural cavity paracentesis is necessary and may be repeated as often as required.

Palpitation is much relieved by an ice-bag over the precordial region. A diffusible stimulant, or strychnine in full doses often proves of signal service. If the attack is severe, a hypodermic injection of morphine may be given. Strict attention to diet together with quiet and rest is also essential.

Vomiting.—Food must sometimes be withheld for some hours, or replaced by sips of hot water, brandy, or champagne. After the vomiting has yielded or lessened, liquid food may be cautiously tried. Milk may be given freely diluted with lime water, Vichy or effervescing waters, or peptonized beef essences are often tolerated when other foods are rejected. In obstinate cases rectal alimentation may become requisite. The frequent action of digitalis in inducing vomiting must be remembered in this connection. It must often be withheld for a period, but should not be given up without good reason.

Venesection is occasionally required when marked cyanosis or dyspnœa is present, and ten, twenty, or even thirty ounces of blood may be abstracted from the arm. A valuable measure which has largely fallen into disuse is the application of a few leeches to the precordial region followed by hot fomentations to encourage bleeding.

During the past few years much has been written on the Nauheim methods, consisting in bathing in aerated water, and movements with resistance. The baths lessen the peripheral resistance, and the movements tend to empty the veins. Percussion and skiagraphs show a lessening of cardiac dilatation after application of these methods. They seem to be more successful at the natural springs than when applied in hospitals or other localities.

Frederick G. Finley.

HEART, RUPTURES OF.—These are divided into two classes: *First*, Those due to degeneration of the myocardium, the so-called spontaneous ruptures; and *second*, those due to traumatism.

Instances of the first class, while not frequent, are more often observed than are those of traumatic origin.

Morbid Anatomy.—Spontaneous rupture of the heart generally occurs in the substance of the left ventricle owing to the greater frequency of degenerative changes in this locality, and to the greater strain which this part of the heart has to bear in lifting the blood through the systemic circulation.

The anterior face of the ventricular wall somewhat above the apex is usually the site of the tear. Although it is said that the tear usually begins from within and proceeds to the outer face, the reverse may happen, and it is impossible to tell from inspection upon which surface the lesion originated. The sites of the rupture in 55 cases collected by Elleaume were as follows: 43 in the left ventricle, 7 in the right, 3 in the right auricle, and 2 in the left auricle.

The tear may be complete or incomplete, and may vary in extent from a centimetre to the length of the ventricle. It is usually single, but as many as five have been noted, sometimes intercommunicating. The rent is generally ragged, irregular, and sometimes ecchymosed at the edges. Its course through the ventricular wall may be tortuous so that the endocardial and epicardial openings do not correspond. Usually the fissure runs parallel to the course of the muscular fibres. Those ruptures which are due to an abscess of the heart wall or to hemorrhagic softening more frequently are in the form of an ulceration or perforation. The immediate result of this accident is the distention of the pericardial sac with blood which generally forms clots, leaving the serum in the pericardium.

Cases have been noted in which a plug of fibrin has more or less completely occluded the tear in the heart wall.

Etiology.—This form of rupture is always the result of disease of the myocardium, softening, fatty degeneration, fatty infiltration, etc., dependent generally upon occlusion or fibrosis of the coronary arteries. This degeneration may be local or general, and may result primarily in an aneurism of the heart or aorta, which may burst. Very rarely we have abscess of the heart wall, which bursts and leads to rupture. The *exciting* cause of the lesion may be a sudden and perhaps an unusual motion like stumbling or falling, lifting or straining. A comparatively frequent cause is running for a train. Anger, excitement, and mental or physical shock may determine the accident. Not infrequently it occurs while the patient is in perfect rest. He may be asleep in bed. The male sex is considerably more liable to this accident than the female. It seldom occurs before the fiftieth year, more often after the sixtieth.

Symptoms.—Seldom are the premonitory signs of this disaster distinctive. There would naturally be signs of a diseased and feeble heart. Comparatively few diseased hearts, however, go on to rupture. On the occurrence of the lesion, death is usually instantaneous; the patient not infrequently being found dead in bed with the clothes not even disturbed. In case the rupture is oblique in direction and small in extent, the patient may survive several hours, or even days.

Dr. Langman has recently reported a case of a man who had two ruptures of the heart wall, one having happened two weeks before the other. He died four hours after the occurrence of the second, which was in the left ventricle, the first was in the right. The heart walls were thin, but not fatty.

The special symptoms denoting approaching dissolution are intense precordial pain, restlessness, dyspnœa, rapid, feeble, and fluttering pulse; vomiting, cyanosis, loss of consciousness, and convulsions.

Hampeln has observed that there is greater pain in rupture of the pericardial portion of the aorta than in that of the heart substance proper.

The physical signs, even when the patient's life is sufficiently prolonged, are more or less indefinite. The pulse is weak and intermittent. The heart sounds are muffled, distant, and imperfectly developed, with probably some increase of pericardial dulness.

Diagnosis.—This is so uncertain that it cannot as a rule be relied upon, although in some cases the condition has been made out ante mortem. A diagnosis ought certainly to be made in those cases in which, in addition to the rational symptoms already detailed, percussion affords evidence that the pericardial area has recently undergone enlargement.

Prognosis.—This is uniformly unfavorable; no case of spontaneous heart rupture having ever been known to recover, although life may be prolonged for several days and even weeks, by judicious treatment and nursing.

Treatment.—This is mainly palliative and can have no effect on the diseased condition of the heart. Cold applications, morphine hypodermatically, a strict maintenance of the recumbent posture, perfect quiet, and warmth to the extremities may prolong life for a limited period. Stimulants are contraindicated.

Traumatic Ruptures of the Heart are quite rare. This organ from its shape and position, its mobility and the protection afforded it by the elasticity of its walls, and by its overlying and surrounding structures, is seldom ruptured—except in cases in which the entire chest wall is crushed in. A certain number of cases of rupture of the heart walls have occurred with comparatively little injury to the chest wall, even without penetration of the pericardial sac. The writer was able, in 1899, to collect forty-five cases of heart rupture, not due to stab or gunshot wounds, and generally speaking caused by an injury from the effects of which the heart walls would be expected to escape scot free. In the majority of the cases there was evidence of disease or abnormality of the organ. But in a few cases the heart muscle was healthy.

The writer reported the case of a vigorous man of twenty-eight who was riding his bicycle rapidly, when the forward wheel struck a hose pipe which was filled with water and which lay across the road. As a result of this sudden strain the bicycle broke at the "head," and the rider was thrown to the ground with great violence, the detached handle-bar post interposing itself between his body and the hard road. This impact fractured the sixth costal cartilage somewhat to the left of the sternum and drove the distal end attached to the rib into the pericardial sac. The sac did not tear; but the force of the blow was so great that the right auricle was torn entirely through. A triangular flap (see accompanying photograph, Fig. 2596) of the heart wall had been lifted up,

Fig. 2596.—Showing Traumatic Rupture of Heart at Apex of Right Ventricle.

and when it was turned back the ventricular cavity was exposed. The heart weighed eleven and one-quarter ounces, and its valves and muscular substance were healthy and competent. The man lived for an hour and a half after the accident and partly recovered from shock. The cause of death was apparently the distention of the pericardium with blood, of which from eight to ten ounces, mostly coagulated, was found in the sac.

Of traumatic ruptures a majority occur in the right ventricle near its apex. Of Gamgee's 28 cases, in 9 there was no fracture of ribs or sternum and either no bruise of the parietes or a very slight one. The pericardium was intact in at least one-half of the cases, and in 22 in which the principal seat of the injury was noted, the right ventricle was torn in 8; the left in 3. The left auricle was injured in 7 and the right in 4. The apex of the right ventricle is a favorite spot for traumatic rupture, first, on account of its exposed position as it comes in contact with the chest wall in systole, and second, because the myocardium is thinnest at this point. Ruptures of the muscular fibres most frequently occur in systole when they are tense and firm and offer the greatest resistance to the impact of the opposing force. If struck in diastole when the ventricles are filled with blood, their walls being flaccid, the seat of rupture will be in the resisting valves, or on the interventricular septum.

So far as ascertained the longest time that a person has survived after traumatic heart rupture is seventeen hours. The symptoms of intense pericardial pain, weak, fluttering or intermittent pulse, dyspnœa, lividity, cold extremities, sweating, and convulsions, are much the same as in cases of spontaneous rupture. With the history of a blow upon the chest, especially if the ribs or their car-

tilages are found fractured, the diagnosis of heart rupture might be made out and bold surgery might save a life. The technique of the operation would be the same as that laid down for wounds of the heart substance. No other treatment could be anything more than palliative.

Richard Cole Newton.

HEART STRAIN.—This may be defined as the untoward effects of overexertion on the previously normal heart.

While the metabolic changes during violent exercise doubtless produce toxins—"fatigue products"—affecting the nutrition of the heart, and causing relaxation of its tonus, the effects are largely mechanical. The heart is called upon to do more work than when at rest, to receive and send out more blood, and to do this under different conditions of blood pressure and nutrition.

The work done by the heart in a given interval of time depends on two factors—the amount of blood sent out and the resistance against which it is pumped. The heart's arterial output in turn is dependent upon its venous input,—that is, the heart normally sends out the same amount of blood per second as it receives. The resistance or aortic blood pressure depends on the balance between the output of the heart and that of the arteries—the heart endeavors to overcome the peripheral resistance. The output of the heart remaining the same, in other words, the resistance would depend upon the state of contraction or dilatation of the arterioles.

The peripheral blood pressure shows, during active exercise, a temporary increase, which is followed, even if the exercise continues, by a very marked decrease. In a recent examination of the contestants in a twenty-five-mile running race with which the writer was associated, this fall was invariably present in men examined at the finish and was shown by direct study of the blood pressure and by the sphygmograph. The temporary rise is due to increased output of the heart. The pressure of the muscles of the extremities on the large veins, the similar action of the muscles of the abdomen and diaphragm on the portal system, and the increased activity of the respiratory apparatus, all favor the flow of blood to the right auricle, and through the lungs to the left heart. For a while the increased output causes an increase in arterial tension and increased work for the left ventricle, but there is soon a dilatation of the peripheral vessels, especially in the muscles, and a consequent fall of pressure with relief to the left heart. According to Allbutt, when this adjustment is accomplished the phenomenon of "second wind" occurs.

Training.—When the heart is frequently called upon to do a large amount of work changes take place through which it is able to meet larger demands upon it. In trained athletes, especially in those engaged in sports requiring long-continued violent exertion, as in long-distance runners or oarsmen, the heart area shows marked and progressive enlargement. Inspection frequently shows visible bulging of the left chest and percussion shows more or less enlargement. The left border may be outside the nipple, and the right correspondingly changed. The impulse is often diffuse and powerful.

The explanation of these findings is doubtless that the heart muscle undergoes hypertrophy exactly like any other muscle. Perhaps there is some dilatation as well, the capacity of the organ as well as its strength being increased. The respiratory apparatus also becomes more efficient, but the changes here are less easily determined than in the heart. It cannot be too strongly insisted upon that these changes are physiological. More than that, it is probable that without more or less cardiac hypertrophy a man cannot with impunity engage in violent athletic contests, while with the heart properly trained he will rarely do himself any immediate damage.

What are the remote results of normal training? Are these hearts more subject in after years to functional disorders or degenerative conditions than others? Important as this question is in connection with modern college athletics there is much doubt about its answer. It is probable that a heart in which simple hypertrophy has occurred as a result of training, which has never suffered from any of the acute or chronic disorders presently to be described, which has, in short, enabled its owner to perform tasks beyond the power of other men without at any time causing any untoward symptoms—such a heart will, after the cessation of training, return quietly to its original size. The decrease is, however, slow. Darling found that after a year the Harvard rowers still had large hearts. But, as Darling remarks, hypertrophy of the heart from training is no more undesirable and gives the patient no more trouble than the corresponding hypertrophy of the biceps. Perhaps the most thorough investigation of this matter was that of Morgan (1873) who studied the after health of the 294 men who rowed in the Oxford-Cambridge races between 1829 and 1869. But seventeen claimed to have been injured, and in only two or three would the evidence convince us to-day that previously healthy men suffered permanent damage to the heart. The mortality was less than comparison with insurance statistics would lead one to expect, and Morgan concluded that in the majority of cases training does great and lasting good. On the other hand Stengel mentions cases in which trained men several years after the cessation of active athletics, with no intervening symptoms, began to complain of sensations of oppression and distention in the chest with "consciousness of the heart." Certainly, if in the course of training the heart has suffered any of the forms of strain, permanent damage may remain. Too little attention has been given the subject of training by medical men and the matter cannot be said to rest at present on very firm scientific foundations.

Acute Heart Strain; Acute Dilatation.—When the heart is examined immediately after an athletic contest it is common to find enlargement of the area of dulness over and above what was already present as a result of training. So commonly is this the case, and so rapidly does the temporary enlargement subside that some dilatation or relaxation would seem to be physiological after sufficient severe exercise.

Under constant conditions of intraventricular blood pressure the total stress on the walls of the ventricle varies with the size of the cavity, and the increase in total stress is as the cube of the radius of curvature, assuming that the ventricle is approximately spherical. It is evident that as dilatation advances the heart muscle has to bear a constantly increasing strain, and that should it continue a point would be reached where it could no longer contract at all (paralysis from overdistention.)

There are, however, certain things which limit this process: in the first place, up to a certain point the muscle fibres act to better advantage when stretched than when partly contracted; secondly, the chordæ tendineæ and trabeculæ tend to prevent excessive stretching; thirdly, the fall of blood pressure as exercise continues relieves the strain; finally, the auriculo-ventricular orifices share the dilatation, or the mitral and tricuspid sphincters relax and regurgitation occurs. In regard to this last point, the normal mitral and tricuspid valves are able to close an orifice considerably dilated, and the action of the so-called sphincters is not fully admitted by physiologists. According to Albutt sudden stress affects chiefly the aortic area, prolonged exertion the right heart.

Acute dilatation may result from severe exertion in any form, though it is said to be most frequent when the legs are used, as in running, rowing, forced marching, bicycling, and mountain climbing. It has resulted from fright and other emotional shocks, and from sudden peripheral vaso-motor contraction the result of cold bathing. It is much more apt to occur in untrained individuals, but even in the Harvard University crews each race is followed by temporary increase in the area of dulness. It is more easily produced in older persons, in those with beginning degenerative conditions, and after convalescence from acute diseases.

The symptoms due to severe physical exertion are familiar to every one—rapid pulse, pallor, and dyspnœa. In extreme cases these symptoms are severe and the skin is cyanosed or grayish. There may be a sense of severe oppression in the chest, dizziness, nausea, or nosebleed. The man may fall to the ground unconscious or even dead. But recovery is generally prompt, so that the athlete who falls exhausted at the finish of a hard-fought race may be well in an hour and ready to win another event.

But symptoms may persist for some days or weeks, during which there are dyspnœa on slight exertion, palpitations, præcordial oppression, pain or "side-ache," with vertigo, and albuminuria. The heart is rapid and irregular. Exacerbations of these symptoms may occur after a meal, without exciting cause or may even waken the patient at night. The patient may die in a few days, compensation not being established. Sometimes there is permanent damage and he remains "broken-winded" to the end of his days—unable to perform any unusual exertion without dyspnœa and cardiac irregularity.

The most important physical sign of pathological dilatation is sudden and persistent increase in the area of cardiac dulness. It must be remembered that the heart of the athlete is generally already enlarged from hypertrophy and that some further temporary dilatation is so common that it cannot alone constitute a disease. The impulse is often feeble and the rhythm irregular. There may be a murmur, usually systolic, at the base to the left of the sternum. The murmur is fugitive, generally disappearing within a few minutes. Its nature and position of maximum intensity are somewhat variable.

The explanation of this murmur which is frequently present in the normal cases of slight temporary dilatation is a question that has excited considerable discussion. Stengel believes that it is due to dilatation of the pulmonary conus arteriosus. In a study of the runners in the Boston Athletic Association's Marathon race (twenty-five miles) of 1899, Williams and Arnold found fugitive systolic murmurs at the base to the left of the sternum in eleven out of thirteen cases examined. In some it was transmitted along the left border of the heart to the apex and in some to the back. They consider that the heart shared in the general muscular exhaustion, and that the mitral sphincter and papillary muscles relaxed, giving a true regurgitation into the auricle. Darling found similar murmurs in the Harvard crew after racing and accepts Williams' and Arnold's explanation, which appears to be widely adopted.

The writer has assisted in examining the hearts of the Boston Marathon runners for the past two years, during which murmurs were not found with the same regularity as by Williams and Arnold. Some might be described as mere "murmurishness" of the first sound. In two cases murmurs were found at the right of the sternum. In some they were present both before and after the race, and in one or two they were present at the start and not at the finish. If any considerable mitral regurgitation had occurred, evidences of pulmonic engorgement would be expected. These were not present, nor were they reported by either of the other writers mentioned. Possibly some murmurs were cardio-respiratory, and certainly some—those that were present at the start only—were due to nervousness. Such murmurs from nervous excitement are matters of common observation. Further study of the matter is needed.

The diagnosis of acute dilatation is generally obvious. The enlargement and murmur which are the normal result of severe exertion will soon disappear, while in the serious dilatation they, together with the symptoms, will be more persistent. Persistence of the murmur with the other signs of mitral insufficiency after the disappearance of the symptoms will usually mean a pre-existing valvular lesion. If the heart is known to have been previously normal a permanent murmur with pain, œdema, and gradual recovery with hypertrophy may indicate a ruptured valve.

Treatment should be directed to relieving the heart from work as much as possible. Rest in bed is imperative in the severe cases. When cyanosis is extreme and the right heart distended, venesection may be called for, more often in older full-blooded men than in young athletes. For drugs, strychnine and alcohol may be used, with digitalis if the pulse is weak and rapid. The latter should be used cautiously especially in the early days; later it may be of the greatest value. During and after convalescence the patient must be very careful not to overexert himself. An athlete should return to active exercise, if at all, very gradually and under close medical supervision.

In prophylaxis the most important point is training, the object being to produce a hypertrophy of the heart comparable to the accompanying development of the peripheral muscles. This is illustrated by the cases of Williams and Arnold. The only two of their thirteen who failed to show enlargement dropped out of the race before reaching the finish. The avoidance of alcohol, tobacco, and other excesses is important.

The question as to what findings should exclude a man from participation in a race or game is difficult to answer. We are not justified in excluding a man because his heart does not show the requisite hypertrophy. If he has been under constant supervision there is no difficulty—persistent murmurs with dyspnœa and irregularity after preliminary trials should exclude him. But a murmur just before the start is of no significance.

Rupture of a Valve is a rare result of overexertion in the normal organ. The aortic and mitral valves are most frequently involved. There is generally severe pain at the time of the accident, often with a sense of something giving way in the chest. The usual signs of regurgitation are present. Immediate death may occur or recovery, according to the severity of the lesion.

Rupture of the Heart is even more rare and probably never occurs in the normal organ. A number of cases have occurred in degenerative conditions as a result of sudden strain. Death is usually instantaneous.

Chronic Heart Strain.—By this is meant those forms of cardiac disturbance which result from frequently repeated exertion—the functional condition known as "irritable heart" and the hypertrophied or dilated organ with symptoms of overaction. The pathology of these conditions and their relation to one another are obscure. J. M. Da Costa, who studied soldiers in the War of the Rebellion, considered that irritable heart was an early stage whose continuance resulted in hypertrophy and overaction. Treadwell studied the same class of men seven years after the war and concluded that the symptoms were due to dilatation when compensatory hypertrophy had failed.

While repeated overexertion is the chief element in the production of many of these cases it is certain that, especially in the functional cases, syphilis, and excesses of alcohol, tobacco, tea and coffee, play an important part. They often commence after convalescence from acute diseases—diarrhœa, digestive troubles, typhoid, or malaria. Improper food, insufficient sleep, and exposure or sexual excesses are occasional factors. In athletes worry over an approaching contest and in soldiers the excitement of an active campaign may have a direct or indirect influence. Constriction of the chest by heavy and improperly adjusted accoutrements has been given a prominent place.

Most of the "soldier's hearts" occur in men under twenty-five, of tall, lanky build. In civil life it is commoner in pedestrians, mountain climbers, and bicyclists. It may result from excessive dancing. J. Burney Yeo describes similar conditions in girls at puberty as a result of rapid growth and educational strains. In college athletes who are kept in the best physical condition and whose training is carefully managed it is apparently uncommon. Still, the cause of "staleness" or overtraining is very obscure and probably the heart (Darling) is a factor.

The symptoms and course of "soldier's heart" are thus summarized by Da Costa: "A man who had been for some months or longer in active service would be seized with diarrhœa, annoying, yet not enough to keep him out of the field; or, attacked with diarrhœa or fever, he

rejoined his command and again underwent the exertions of a soldier's life. He soon noticed that he could not bear them as formerly; he got out of breath, could not keep up with his comrades, was annoyed with dizziness and palpitations; his accoutrements oppressed him, and all this though he appeared well and healthy. Seeking advice from the surgeon of the regiment, it was decided that he was unfit for duty and he was sent to the hospital, where his persistently quick-acting heart confirmed his story, though he looked like a man in sound condition. Any digestive disturbances which might have existed gradually passed away, but the irritability of the heart remained, and only very slowly did the excited organ return to its natural condition. Or it failed to do so, notwithstanding the use of remedies which control the circulation: thus the case might go on for a long time, and the patient, after having been the round of the hospitals, would be discharged, or, as unfit for active service, be placed in the Invalid Corps."

The important symptoms are precordial pain, usually paroxysmal; palpitations, tachycardia, the heart being characteristically more rapid in the upright position or after slight exertion; dyspnœa; and nervous disturbances—headache, dizziness, and sleeplessness. The symptoms may be absent except during exercise or they may come on paroxysmally during rest or sleep.

Examination in the functional cases shows little or no enlargement, the chief physical signs being irregularity or rapidity of action on slight exertion or on suddenly assuming the erect posture. Systolic murmurs may be present, but they are inconstant and variable. The first sound is described as short and valvular. In the cases with permanent dilatation the area is large, the impulse labored and diffuse, the first sound dull and prolonged. Constant murmurs may be present; due to regurgitation from relatively insufficient valves.

In the differential diagnosis it must be remembered that a true valvular lesion from endocarditis may exist without causing a murmur, especially in the case of mitral stenosis, and that the patient with such a lesion may correctly date the onset of symptoms to a period of over-exercise. Renal, pulmonary, and vascular degenerations must be borne in mind. Some of the cases are clinically identical with myocarditis.

The prognosis is uncertain. Complete recovery may occur, but as a rule more or less irritability remains. The quotation from Da Costa given above indicates what seems to be the common result in military cases. It is probable that many cases become virtually ones of valvular disease—relative insufficiency—or of myocarditis. Some go from bad to worse and die within a few years.

For prophylactic treatment training is again of great importance, or at least occupations requiring severe exertion should be entered into gradually so that hypertrophy of the heart muscle may occur before the full strain is put on the organ. Active exercise should be resumed with great care after recovery from acute disease. Excesses, especially of tobacco and alcohol, should be avoided permanently by those whose occupations call for a large amount of work on the part of the heart.

When the condition of irritable or dilated heart is once established prolonged rest is imperative with gradual return to active life after recovery. Among drugs digitalis will be of greatest use. The bromides will also be of service. Iron and other tonics may be called for.

In conclusion the statement must be reiterated that the danger from over-exercise in healthy hearts is not great, especially if the subject be well-trained. Indeed, Darling mentions one oarsman in whom, at the beginning of the season, the heart showed obvious signs of mitral regurgitation from previous organic disease of the valve. As the season advanced the symptoms and signs entirely disappeared, and the man did valuable work in the final hard-fought intercollegiate boat race. Such instances may be rare, but the heart certainly has a vast reserve power.

Ralph C. Larrabee.

Allbutt, T. C.: System of Medicine, vol. v.
Altschul: Münch. med. Woch., December 6th, 1898.
Bäumler: Deut. Arch. f. klin. Med., vol. lxvi., 1899.
Cautley: Brit. Med. Journal, 1893.
Christ: Arch. f. klin. Med., Bd. iii., Hefte i. and ii.
Da Costa, J. M.: Toner Lectures, May 14th, 1874.—Am. Journ. Med. Sciences, January, 1871.
Darling: Boston Med. and Surg. Journal, August 31st and September 7th, 1899; also June 6th, 1901.
Friedreich: Wien. klin. therap. Woch., 1898, Hefte 2 and 5.
Herschell: Lancet, March 2d, 1895.
Herz: Deut. med. Woch., February 22d and March 1st, 1900.
Hill, Leonard: Shäfer's Physiology, 1900, vol. ii.
Jamieson: Intercol. Med. Journal Australia, November 10th, 1896.
McCarthy: Thesis for Degree M.B., Camb., 1898. Quoted in Allbutt's System of Medicine.
Morgan: University Oars, London, 1873.
Powell: Allbutt's System of Medicine, vol. v.
Prince: Med. Record (N. Y.), April 20th, 1889.—Am. Journ. Med. Sciences, February, 1901.
Roy and Adami: Brit. Med. Journ., December 15th, 1888.
Sherrington: Allbutt's System of Medicine, vol. v.
Stengel: Am. Journ. Med. Sciences, November, 1899.
Treadwell: Boston Med. and Surg. Journal, September, 1872.
Weber: Lancet, October 28th, 1893.
Williams and Arnold: Phila. Med. Journal, June 3d, 1899.
Yeo: Edinburgh Med. Journ., July, 1899.
Zuntz: Verh. d. Berl. physiol. Ges. Arch. f. Physiol., p. 378, 1895.

HEART, THROMBOSIS OF.—The blood clots found within the heart cavities may be divided into three classes: *post-mortem clots, ante-mortem* or *agonal clots*, and *true thrombi*. The post-mortem clot is formed after the heart has ceased to beat; it is a soft, dark-red, jelly clot filling up the cavity of the auricle or ventricle and not adherent to the heart wall. It is commonly known as cruor or the currant-jelly clot. Such coagula are found usually in the right auricle and ventricle, but may be present in all four of the cavities. When coagulation occurs rapidly after death the clot is uniformly red throughout; but if coagulation is delayed the uppermost portion of the clot may be light-colored or yellowish from the sinking of the red cells. In themselves the post-mortem clots are of significance only in so far as the character of the blood and its coagulability are concerned. If coagulation takes place while the ventricles are contracted in rigor mortis the clots will be found in the auricles only, but, if coagulation be delayed until the contraction of the ventricles has passed, the ventricular cavities may again become filled with blood which later coagulates.

The ante-mortem or agonal clot is a white or mixed clot found in the periphery of the large veins, right and left auricles, and right ventricle, but rarely in the left ventricle. Such clots consist of fibrin with few or many leucocytes, while that part of the clot toward the axial stream may contain red blood cells. They develop during the slowing of the blood stream in cases of slow death, and are formed only in the peripheral stream where there are no red cells. As the current becomes slower red cells are caught in that portion of the clot toward the central stream. As soon as death occurs and the current comes to a standstill, the blood in the central stream coagulates forming a post-mortem currant-jelly clot. Combinations of agonal and post-mortem clots are thus formed and are distinguished by the presence of a central red clot surrounded by a peripheral layer of white clot. The agonal clot is soft, but much firmer than the currant-jelly clot. It is not attached to the endocardium and is easily removed from between the trabeculæ of the ventricular wall. It is further distinguished from true cardiac thrombi in its moistness, translucency, and greater elasticity. On microscopical examination the agonal clot is found to consist almost entirely of a coarse reticulum of fibrin; on the other hand the fibrin in the post-mortem jelly clot is in the form of fine fibrillæ. The significance of the agonal clot is only that of a slowly progressive heart failure; in cases of sudden death without previous impairment of cardiac efficiency the white peripheral clots are either very small or absent, while in cases of slow death in chronic valvular disease, cachexias, etc., they may be very large. Care should be taken to differentiate between these and the true heart thrombi for which they are frequently mistaken.

The true heart thrombi are yellowish, reddish, or reddish-gray or brown, opaque, dry, inelastic, brittle, and

in the great majority of cases more or less adherent to the endocardium. The conditions under which they are formed are practically the same as those of thrombosis of the blood-vessels, the chief factors being lesions of the endocardium, disturbances of the circulation, and changes in the chemical composition of the blood. Of these the endocardial changes are by far the most important. At the point of lesion blood plates are first deposited, followed by the processes of conglutination and coagulation as in the formation of thrombi in the vessels. The resulting thrombus is usually a mixed clot, very frequently laminated. When formed in the peripheral stream with rapid circulation it may be entirely white; if the current is very much slowed it may be red throughout. In old red thrombi the diffusion of the hæmoglobin from the red cells may give a washed-out reddish or brown color. The latter color is also the result of the formation of blood pigment from the disintegration of the red cells. The surface of the thrombus is not infrequently ribbed; this appearance has been explained as being due to wave-like movements in the blood, the process being analogous to the formation of wave marks on sandy bottoms. The general external appearances of cardiac thrombi are dependent upon the manner of formation, rapidity of development, and the local conditions of the circulation. The size and shape of cardiac thrombi show great variation. They may be so small as to be hidden between the muscle columns, or so large as completely to fill one of the heart cavities. They may appear as small globular masses projecting from between the muscle trabeculæ (globular vegetations); or as flat, circumscribed masses firmly attached to the heart wall. On the valves they may form bead-like excrescences or vegetations. Not infrequently the thrombus is attached to the endocardium by a pedicle (heart polyp); as the result of the tearing of such attachment free globular thrombi may be formed (ball thrombi). In rare cases the thrombus may be spread over the endocardium like a false membrane.

The thrombus when cut into may be found to be made up of dry and brittle laminations, in some instances the central portion of the thrombus is under slight pressure easily crumbled to dust. In other cases the central portion may be cystic, the cavity filled with a thick reddish-brown or pus-like substance. Such secondary degenerations are of frequent occurrence. The most common is a simple softening or liquefaction; when many leucocytes are present a grayish pus-like fluid may be formed; if the degenerating portion is made up of red cells alone a grumous reddish-brown fluid results. Calcification of the thrombus may also take place, resulting in the formation of a cardiolith. As a more favorable sequela of cardiac thrombosis organization of the thrombus may occur, the latter becoming ultimately converted into a nodular or diffuse fibrous thickening of the endocardium. In some cases both the degenerating and the organized thrombi have been mistaken for cardiac neoplasms, cysts, and fibroma respectively. It is also not improbable that some of the growths on the endocardial surface described as myxoma were recently organized thrombi.

The microscopical examination shows the cardiac thrombi to be composed of fibrin, disintegrated blood cells, blood pigment, masses of calcification, etc. In case of simple softening of the central portion the cavity thus formed contains granular débris, blood pigment, etc. In recent thrombi the leucocytes present may still show traces of their nuclei. If organization has begun fibroblasts and new capillaries may be seen extending from the endocardium into the thrombus.

Thrombi are more frequently found in the right side of the heart than in the left. In the auricles the appendix, and in the ventricles the neighborhood of the apex are the favorite seats of location. The pedunculated thrombi (cardiac polyps) are of frequent occurrence in the right ventricle, while the free ball thrombi, in the majority of recorded cases, were found in the dilated left auricle in cases of mitral stenosis. Cardiac thrombi may be single or multiple, and may be found at any age. According to their location in the heart they are also designated as parietal and valvular.

Etiology.—The changes in the endocardium leading to thrombosis are either inflammatory, or degenerative, or necrotic in nature. These lesions may be the result of the local action of pathogenic bacteria or due to general intoxications, disturbances of nutrition, changes in the blood, etc. The pus-forming organisms, gonococcus, and tubercle bacillus have been found in cardiac thrombi under such conditions as to make it very probable that they were the exciting factors of the thrombosis. The valvular vegetations are in the majority of cases due to bacteria. In the acute infections fatty degeneration of the endocardial endothelium not infrequently leads to thrombosis. Local changes in the myocardium, such as anæmic infarction, myocarditis, etc., are usually associated with the formation of thrombi on the overlying endocardium. Aneurismal dilatations of the cardiac wall are very likely to be partly or wholly filled by a thrombus. In many chronic diseases in which there are general disturbances of nutrition, systemic intoxication, etc., as in chronic nephritis, pulmonary tuberculosis, chronic valvular disease, etc., heart thrombi are frequently found. In pulmonary tuberculosis they are of such frequent occurrence as to give rise to the suspicion that in many cases they may be due to the local action of tubercle bacilli which have lodged upon the endocardium. Cardiac thrombosis often occurs in cases of general cachexia (marasmic thrombi). In severe superficial burns of the skin the changes produced in the blood may lead to the formation of thrombi, both in the right side of the heart and in the pulmonary arteries. Small emboli coming from venous thrombi and lodging upon the valves or between the muscle trabeculæ of the right ventricle may give rise to secondary or induced thrombi. Sclerotic changes in the valves or mural endocardium followed by atheromatous degeneration or calcification may also become the seat of thrombosis. Dilatation of the heart and slowing of the blood stream are favoring factors in all of the conditions named above, but are probably not in themselves sufficient to cause thrombosis; the changes in the endocardium must be regarded as of prime importance.

Symptoms.—Thrombi of large size may exist indefinitely without serious impairment of cardiac efficiency. In the majority of cases the symptoms produced cannot be differentiated from cardiac insufficiency due to other causes. The physical signs are those of dilatation; in case of the blocking of a valvular orifice by the thrombus the heart's action is usually too weak to produce audible murmurs. The valvular vegetations may give rise to symptoms of stenosis or insufficiency. In the case of the ball thrombi so frequently formed in the left auricle in mitral stenosis sudden death or the most serious disturbances of circulation may be caused by the complete or partial blocking of the mitral orifice. The occurrence of embolism, either of the pulmonary or of the systemic arteries, is the symptom of greatest value in the diagnosis of cardiac thrombosis. Embolic abscesses may be produced throughout the body in the case of emboli arising from thrombi caused by pyogenic bacteria. In the case of a patent foramen ovale paradoxical embolism from thrombi in the right side of the heart may occur. In general it may be said that the diagnosis of cardiac thrombosis is usually impossible, having been made in but few cases. Aside from the occurrence of embolism the irregularity of the symptoms and physical signs may excite suspicion of a movable body within the heart.

The Prognosis is bad, not only because of the interference of the heart's action and the danger of embolism, but also because of the conditions which have led to the thrombosis. The treatment is directed chiefly to the latter and to the support of the heart.

Aldred Scott Warthin.

HEART, WOUNDS OF.—Wounds of the heart may be either non-penetrating or penetrating—injuring the cardiac wall or opening a cavity. The chief dangers