of energy is capable of liberating latent energy in complex labile substances—the proteids, breaking them down into simpler, more stabile bodies. We have had the statements that pepsin actually loses its activity in this process, and that it remains unaltered. There can be no doubt, however, that pepsin undergoes no change in exerting its energy, for this is readily demonstrated.

A pepsin having been subjected to the usual digestive test—upon acid albumin mixture—may be made repeatedly to exhibit its action upon the addition of fresh volumes of water and albumin and when the requisite percentage of acid is maintained, thus preventing saturation of the digestive fluid with the soluble products formed. In this way the writer has found pepsin to digest several hundred thousand times its own weight of albumen without exhaustion of energy. Interesting as this may be as to the marvellous power of the enzyme, it seems, however, to be without therapeutic bearing, in view of the fact that the gastric juice is normally discharged into the intestine with the completion of the stomach digestion.

Pepsin in the dry form, if non-hygroscopic, retains its vitality for years at ordinary temperature. In solution it may be readily preserved without serious impairment; alcohol and glycerin combined form the best preservatives, from both the medicinal and the pharmaceutical standpoints; for general use, about fifteen per cent. absolute alcohol and glycerin being the amount required. The anhydrous glycerin does not readily take up the enzyme; when diluted with water, to the extent of about forty per cent., it affords a useful vehicle. Elixirs, essences, and glycerites are commonly and very conveniently made with pepsin. There is no pharmacopœial method or standard for these products; they are variable, and distinctly inferior in therapeutic utility to fluids made directly from the fresh stomach.

Chemical preservatives—salicylic acid, boric acid, etc.—are obviously objectionable. Absolute alcohol precipitates pepsin, and by long contact distinctly weakens it; but the presence of alcohol up to about fifteen per cent. exerts no sensible influence upon the ferment, the enzyme being freely extracted by such hydro-alcoholic menstruum. *In vitro* alcohol, when it constitutes ten per cent. of the medium, sensibly affects the *digestive* action of pepsin; and it checks this digestive action, not by altering the ferment, but simply for the reason that alcohol is not a competent medium for the ferment in any particular. The products of enzymic action are insoluble in alcohol and are of lessened solubility in hydro-alcoholic media in direct ratio to increased percentage of alcohol. This, however, concerns digestion *in vitro* solely, for alcohol as present in any proper peptic fluid becomes in this respect a negligible quantity by dilution with gastric content and its free absorption. The inert or feeble nature of many of the vinous and alcoholic preparations has been due to the insignificant amount of pepsin actually contained in them and not simply to their alcohol content. The intimate relation of pepsin to hydrochloric acid has naturally led to the impression that this and other mineral acids may be freely admixed with the ferment; but this is a serious error. The acid of the gastric juice is bound up in a peculiar manner with the proteids, and thus the enzyme seems to be protected from the acid, whereas pepsin in solution with pure water and the normal content of absolute HCl (0.2 per cent.) rapidly deteriorates at ordinary temperature. This constitutes a striking evidence of the fact that a solution of pepsin and water and HCl is not gastric juice; it represents the proteolytic ferment, and exhibits absolutely the proteolytic action only of the gastric juice. Other mineral acids are distinctly injurious to the ferment in any fluid form. It is not possible to mix a mineral acid in medicinal quantity with pepsin in a fluid mixture of convenient volume of dosage without distinctly injuring the ferment even for extemporaneous use; while combined in a percentage much above that of the gastric juice content, such mixtures are distinctly incompatible with the normal activity of the ferment and unsuited for pharmaceutical products. For instance, if we take five minims as a moderate dose of official dilute HCl, this in two fluidrachms would yield an acidity of 0.42 absolute HCl, which is twice the acidity of normal gastric juice and fatal to the enzyme.

Pepsin fluids should have an acid reaction; but when acids *per se* are indicated medicinally, it is the best practice to exhibit them separately by such vehicle and means as are most desirable in conjunction with the pepsin preparation. If the preparation has an alkaline reaction, this is conclusive evidence of its inertness.

Pepsin is incompatible with bismuth ammonio-citrate in solution. If the mixture has a neutral or an alkaline reaction, the ferment cannot retain its activity; on the other hand, if it is acid, it is impossible to maintain the bismuth in solution. Owing to the unstable and insoluble nature of the salt, its solution is usually effected by the addition of ammonia, which is obviously incompatible with pepsin. The therapeutic value of elixirs of pepsin, bismuth, and strychnine necessarily cannot be attributed in any degree to pepsin; devitalized pepsin cannot in any way add to the value of a medicinal compound.

Pepsin and pancreatin are incompatible in solution, for the reason that if the menstruum be of such acid nature as to preserve the pepsin, the pancreatic enzyme will be in time destroyed; while if it is neutral or feebly alkaline, the pepsin will be destroyed. Acid-pepsin fluids are unsuitable for the admixture of all ferments except the milk-curdling; the pepsin will be the only enzyme of all those originally combined which will retain its activity. Mixtures of the ferments in solution are readily subject to recognized tests for the presence of any one of the ferments. If a fresh and feebly acid infusion of the stomach, or solution of pepsin, be mixed with an aqueous infusion of the pancreas, each one of the ferments contained in this mixture may be immediately made to exert its peculiar action under the proper conditions; the mixture will exhibit the digestive action of pepsin, of trypsin, and of diastase. But upon keeping this mixed ferment solution at ordinary room temperature for a few weeks, it will be found upon systematic assay to have gradually and rapidly deteriorated in respect to one or another of its ferments.

The fact that a number of ferments are mixed in any fluid does not in the least interfere with the method of assay for testing or determining the presence of any one or of each separate ferment in the fluid mixture. When it is desired to combine gastric and pancreatic ferments in solution, they are best directed in extemporaneous mixture of preparations which have been obtained directly from the stomach and the pancreas gland, and thus they will maintain their individual action for such length of time as will ordinarily be required by the patient.

The therapeutic use of pepsin is prejudiced and complicated by the prevalence, in the past, of inefficient products and incompatible combinations, a condition which does not exist concerning any other agent or class of agents of the materia medica, owing to the fact that with the definite and standardized chemicals and galenical products there has been presented no such obstacle in establishing therapeutic action and scientific dosage. The increasing knowledge and application of physiological chemistry in therapeutics, and the accumulation of laboratory and clinical observations with regard to the action of animal gastric juice, have strongly confirmed its rational and obvious utility and promise, and have advanced its repute and use as a therapeutic agent.

Pepsin exhibited in adequate doses aids gastric digestion, with effects apparent in the relief of various dyspeptic symptoms and in the promotion of nutrition; it affords a rational remedy to which the physician may have recourse in cases of feeble and readily disturbed digestion. The beneficial effects of pepsin are not restricted alone to the improvement of gastric disorders; there is abundant evidence that each step in the chain of digestive action is of essential importance, and defective stomach digestion cannot but influence the entire digestive process.

The pancreatic ferments attack with great facility the soft and partially converted proteids and starches in the form in which they normally reach the intestinal tract, breaking them down into their most soluble and diffusible forms, and the development of the latent pancreas enzymes waits upon the influence of constituents of normal gastric juice. Coagulated protein food in masses is but very slightly attacked by pancreas juice in contrast with peptic action, and thus the complete conversion of food is absolutely dependent upon the interaction of both gastric and intestinal digestion. Disorders even of the intestinal tract are frequently benefited by the administration of pepsin. Pepsin in doses so small as to seem a slight factor in the physiological process, and administered either just before or immediately after eating, produces results which can be attributed only to the theory advanced that it imparts an impetus to peptic secretion and action. Pepsin is used to promote the toleration of drugs which impair the appetite and disturb digestion. For all these purposes the gastric juice, extracted directly from the fresh stomach in proper pharmaceutical form, is found most generally useful. This preparation renders available at once all the properties of the gastric juice, both its enzymes (the peptic and milk-curdling) and its acid in proteid combination—the entire organic and inorganic content in natural association. These enzymes are thus less susceptible to unfavorable influence than is the precipitated ferment.

Pepsin is given in scales, powder, tablets, and capsules, ordinarily in doses of from one to five grains; the scales are readily soluble in water—plain or with acid; the glycerite, especially the glycerin extract from the stomach, is useful, and if properly prepared is far more agreeable than the scale itself taken in solution. The essence prepared from the gastric juice is the most efficient and agreeable preparation, and its grateful qualities enhance the effect of the digestive principles contained therein. The desired dose of drug, for instance sodium salicylate or iodide of potassium, is prescribed in the proportion of, say, five grains to each teaspoonful of the essence of pepsin, and this added to two or three tablespoonfuls of warm milk gives instantly a firm curd. The milk may be previously sweetened or flavored if desired, the object being to present the drug in a small bulk for convenience; even this serves well to disguise the medicine.

When pepsin is given simply to promote digestion, it should never be administered in a disagreeable form, and when given to facilitate the exhibition and therapeutic action of disagreeable drugs, the essence is not only valuable as a vehicle, but should be given immediately after the drug if it is desired to obtain its best effect.

The essence of pepsin is much used in combination with savory, soluble, and diffusible food products, the prepared peptonized foods, and it should be mixed in about equal quantities therewith; this combination proves of peculiar value in acute forms of indigestion and intolerance of food; in seasickness, for instance, it is especially useful. Essence of pepsin is also much used in combination with pure phenol, which is thus well masked and well borne, and this mixture, which is both antiseptic and sedative to an irritable gastric mucous membrane, does not in medicinal proportion unfavorably influence the gastric enzymes.

The gastric juice essence is more especially found serviceable as a drug vehicle, and in conjunction with it maximum doses of mercurials, iodides, salicylates, etc., are peculiarly well tolerated. It is also valuable for the production of junket—a jelly-like, diffusible form of pure milk—which is also a carrier of drugs which blend with it and thus lose much of their disagreeable taste and effect. Junket affords an agreeable and wholesome variety of food, and is serviceable in convalescence where liquid foods have become distasteful and are no longer required. It is made as follows:

Junket.—Into a clean saucepan put one-half pint of fresh, cool milk, heat it lukewarm (not over 100° F.); then add one teaspoonful of essence of pepsin, and stir just enough to mix; divide quickly into small cups or glasses and let stand until firmly jellied, when the junket is ready for use, just as it is, or with sugar; it may be placed on ice and taken cold.

Whey.—After preparing the junket by the above method, let it stand until firmly jellied, then beat with a fork until it is finely divided; now strain and the whey (liquid part) is ready for use; keep in a bottle near ice.

Pepsin digestion has long been observed to effect the solution of dead tissue, pus, necrosed bone, etc. The availability of the gastric juice in an active, sterile, and stable extract of great potency, has recently led to its application as a surgical solvent in the bladder, urethra, eye, ear, nose, and throat, and in pus cases in general—sores, abscesses, carbuncles, gangrene, leg ulcers, etc. Gastric juice is thus found to possess peculiar and valuable properties as a solvent, healing, antiseptic, deodorizing, and sedative agent. It is painless in its action and incapable of attacking normal tissue, and has caused a speedy cure in cases which were so aggravated as to have resisted other treatment, thus rendering surgical interference unnecessary. In genito-urinary diseases it promises, from the most conservative estimate of the clinical trial which it has already received, to afford a remedy of great importance.

Benjamin T. Fairchild.

PEPTONURIA. See *Urine, etc.*

PERFORATING ULCER OF THE FOOT.—This is a rare affection, caused by pressure or injury where there is a degenerated nerve supply. It is found in leprosy, locomotor ataxia, lues, and alcoholic and diabetic neuritis. The most common location is where there is great pressure, as over the metatarso-phalangeal articulation of the great or little toe, or over the ball of the toes. Occasionally there are several lesions existing at the same time in one or both feet. A similar condition may also occur on the hands.

The process is very slow. It begins as a thickening of the skin resembling a corn, under which suppuration occurs; and later, when the horny plug is cast off, an ulcer is left. The destructive process extends downward until it reaches the bone, which may also become affected. The condition now is more that of a sinus than of an ulcer. The skin surrounding the opening is usually much thickened, and there may be granulations at the orifice. The diseased parts are generally painless and the neighboring parts are usually anæsthetic. Distortion of the toes, as well as trophic changes in the nails, may occur later; they are usually accompanied by an increased growth of hair, pigmentation, and hyperidrosis. The patients frequently complain of cold feet and neuralgic pains.

The prognosis is unfavorable, even if the lesions should heal, on account of the liability to recurrence, which in turn is due to permanent nerve lesions.

Perforating ulcer has to be differentiated only from a suppurating corn, which latter is painful and is accompanied by abnormal sensitiveness of the surrounding skin. In the case of a suppurating corn the results of surgical treatment are always satisfactory.

Prolonged rest will occasionally lead to healing of the lesion in the early stages, but exercise will cause the sore to recur. Packing the sinus with lint wet with a saturated solution of salicylic acid in glycerin, and the employment of mechanical devices to prevent pressure will frequently produce a temporary cure. Free opening of the sinus or stretching of the nerves which supply the part has been followed by good results in some cases. In the later stages excision of the ulcer is useless and amputation of the foot is necessary. Even then the ulcer may recur in the stump, unless the limb is removed at a point far from the lesion and above the line of anæsthesia.

Howard Morrow.

PERICARDIUM, DISEASES OF THE.—HISTORY.—Anatomical alterations in the pericardium were known long before diseases of the heart proper received careful

study. Galen was familiar with pericardial effusion in animals, and suspected it in men.

The fables about the occurrence of hairy hearts in men of remarkable strength and daring are doubtless based upon the discovery of stringy fibrin deposited upon the heart. Rondelet described pericarditis as having symptoms of fever, dyspnœa, pain under the sternum, and attacks of syncope.

In the eighteenth century the authors of works upon diseases of the heart made numerous anatomical observations upon diseases of the pericardium.

Vieussens often met with adhesion of the heart to the pericardium at autopsies, and assigned certain functional disturbances which occur during life to the existence of this condition. In earlier times it was thought to be a congenital defect. Albertini appreciated the difficulties of a symptomatological recognition of pericardial effusion. Morgagni believed that on account of the difficulty of diagnosis, the day was yet remote when we should have recourse to the puncture of the pericardium as suggested by Riolan. Senac realized that it was impossible to make a diagnosis from the indefinite symptomatology, but thought that in hydropericardium he recognized an undulatory movement between the third and the fifth ribs. Corvisart thought that he could feel this. Both were in error. However, Corvisart first distinguished between inflammatory exudations and dropsical effusions, but could not lay down any fixed rules for differential diagnosis. Avenbrugger was the first to state any accurate physical signs; these were bulging of the precordium and increase in the area of percussion dulness. Laënnec, however, doubted the possibility of diagnosing pericarditis with certainty.

The discovery of the pericardial friction rub by Collins in 1824 made certain the recognition of dry pericarditis. Since this time our knowledge of the physical signs of all varieties of pericarditis have increased remarkably; but none are so pathognomonic or of so much assistance in diagnosis as this peculiar friction rub.

Anatomy.—Before launching upon a description of the diseases of the pericardium, it will be well to devote a few lines to its normal anatomy. It is a fibro-serous sac, somewhat conical in shape, surrounding the heart and the origin of the great vessels. Its base is directed downward, rests upon the diaphragm, and is firmly attached to its central tendon, and more loosely to its muscular structure by areolar tissue. Its narrower portion is directed upward, and surrounds the great vessels. The fibrous layer is continued for some distance along the coats of the great vessels, in the form of prolongations, which gradually become incorporated in their coats. The inferior vena cava passes through the floor of the pericardium to reach the heart. The serous membrane lines the fibrous sac and is reflected over the surface of the heart, thus constituting its parietal and visceral portions. They are continuous along the great vessels, about an inch to an inch and a half above the base of the heart.

Externally, the pericardium is in contact anteriorly and laterally with the pleuræ covering the lungs, with the exception of a triangular space, behind the lower sternum, which remains uncovered. It is attached by fibrous bands to the manubrium and ensiform cartilage. Behind it are the œsophagus, descending aorta, bifurcation of the trachea and left bronchus, and the other structures which form the root of the left lung. The phrenic nerves pass down, one on each side of the pericardium, on their way to the diaphragm.

In health the serous surfaces are kept moist by a secretion normal to serous membranes. The amount is always small, but varies from a few cubic centimetres to an ounce or two. It is common to find at autopsy several ounces of pericardial fluid. In most cases, however, this is a post-mortem transudate. As a result of the presence of this fluid the serous surfaces glide smoothly over each other during the various phases of the heart's action without producing audible or palpable signs.

The pericardium of an adult man with a healthy heart is capable of holding from fourteen to twenty-two ounces of fluid; that of a boy between six and nine years old, about six ounces when the sac is distended to the full by injecting water into it, by means of a syringe, through an opening made into the anterior part of the pericardium (Sibson).

The following are the important diseases of the pericardium:

1. Pericarditis: (*a*) dry or plastic (pericarditis sicca); (*b*) wet pericarditis, or pericarditis with effusion; (*c*) suppurative pericarditis; (*d*) chronic adhesive pericarditis.
2. Hydropericardium.
3. Hæmopericardium.
4. Pneumopericardium.
5. New growths in the pericardium.

Morbid Anatomy.—I. *Abnormal Conditions of the Pericardium* not of clinical interest, and which do not furnish physical signs.

(*a*) *Absence of the Pericardium.*—This occurs in ectopia cordis. It is usually only partial, there being a slit in the pericardium through which the heart protrudes. Very rarely the heart and the left lung lie in the same serous sac. The heart is covered by the visceral layer of the pericardium; at the origin of the great vessels there are usually found rudimentary portions of the parietal layer in the form of fringe-like reduplications.

(*b*) *Diverticula.*—Hernia-like pouches are rarely found. They are due to the pressure outward of fluid. This occurs in chronic conditions in which the fibrous layer has become weakened, and either yields or separates and allows the serous layer to be pushed through by the exudate.

Such pouches are usually small, but they have been known to contain as much as from three to four ounces. The opening into the pericardium may be wide or narrow. These conditions are not recognized during life.

(*c*) *Milk Spots*, also called soldier's spots and tendinous spots.

By these terms are meant those circumscribed, whitish, slightly thickened spots which are so frequently found upon the pericardium. Some authorities look upon them as evidences of an old pericarditis, and as such they have influenced the statistics of pericarditis.

Most writers now believe them to be areas of chronic hyperplasia of connective tissue. Friedreich believed that they resulted from a continual mechanical irritation of the surface of the heart, and were found most frequently on those parts of the heart which were continually brought into contact with the more resisting portion of the chest wall. These spots are nearly always found on the visceral pericardium and on the anterior surface of the right ventricle along the coronary arteries. They are much more common in advanced age than in youth, and in men than in women. They are of no clinical importance and cannot be recognized during life.

(*d*) *Thinned Pericardium.*—The wall of the sac may be thinned as a result of distention from an enlarged heart or from the pressure of fluid.

(*e*) *Foreign Bodies.*—These have been found lying free in the sac, and have been regarded as polypi detached from the inner surface of the pericardium, or as results of fibrous or calcareous deposits about foreign substances.

(*f*) *Calcareous Deposits.*—In cases of prolonged pericarditis there may be more or less calcareous deposit in the pericardium.

II. *Acute Plastic Pericarditis.*—In this variety both layers of the pericardium are covered with a yellowish, sticky layer of inflammatory lymph of varying thickness. As a result of the constant friction of these two surfaces during the heart's action, this material is thrown into ridges, and at times presents a ragged appearance (so-called bread-and-butter adhesions of Laënnec), resembling the appearance of two slices of bread and butter, which have been stuck together and then drawn apart. It has also been likened to tripe. The involvement of the pericardium may be universal or only partial. If only partial it is more common at the base of the heart than elsewhere.

The various changes occurring in pericarditis are sometimes described as following a definite order of succession: (1) Increased vascularity; (2) fibrinous exudation; (3) fluid effusion; (4) absorption; (5) adhesion. It is very seldom, however, that these stages can be recognized, as there is likely to be a mixture of two or more—as, for instance, the association of a plastic exudate with fluid effusion, or even the combination of adhesions and fluid.

It is possible for absorption to take place, followed by resolution. Very often, however, especially in the pericarditis sicca, the plastic material becomes organized into firm adhesions which pass from parietal to visceral pericardium. In the early stages of an inflammation the adhesions may be very fine and delicate and easily broken with the fingers. Later on, however, they become exceedingly strong, and the pericardium cannot be separated from the heart without tearing the heart substance.

The presence of adhesions may be partial or universal. If universal, there will be entire obliteration of the pericardial cavity. Adhesions may also exist between the pericardium and pleura, or between the pericardium and chest wall, as a result of mediastino-pericarditis (pericarditis externa).

Effusion.—As previously stated, there is present normally in the pericardial sac enough fluid (a few cubic centimetres) to keep the surfaces well lubricated. In diseased conditions this fluid may be enormously increased in quantity and greatly altered in character. Roberts says that the average quantity of fluid in pericarditis is from eight to twelve ounces, but it may range from an ounce or two to two or three pints or more. Balfour says several pints; Broadbent says that any large amount is exceptional.

The statements of different authorities vary much as regards the size of exudates. Sibson states that the effusion is likely to be large in rheumatic pericarditis, while John Broadbent says that it is the exception to find a large effusion in this condition. The truth is, there is no definite rule. Roberts states that the quantity is likely to be small in Bright's disease. However, Dr. Herman Allyn (*American Medicine*, October 18th, 1902) reports a case of pericarditis which occurred as a terminal infection in Bright's disease, and in which he removed by paracentesis, on the day previous to death, forty-four ounces of bloody serum. At autopsy the pericardial sac was found to contain about two hundred and fifty ounces of bloody serous exudate.

It is generally stated that the exudate is large in cases secondary to scurvy. In the Russian epidemic referred to later, the quantity amounted to from four to five pints.

Character of Fluid.—This varies much, depending on the nature of the inflammation. In the typical case of pericarditis it is a clear yellow, in which shreds of fibrin and leucocytes may be present. In rheumatic pericarditis it is usually clear, but may be blood-tinged and may occasionally become purulent. In new growths and tuberculosis the fluid is likely to be blood-tinged, but not necessarily so. In scurvy the exudate is usually bloody; in fact, it may be almost clear blood.

The specific gravity of a pericardial exudate, like that of other serous cavities, is usually above 1.015, though there are rare exceptions to this rule.

Absorption.—The natural tendency in most cases of serous or sero-fibrous effusion is toward absorption sooner or later. This is especially true in rheumatic cases. In fact, after reaching the acme, in a day or so there may be a distinct diminution in amount, and in from four to six days the quantity may fall to normal. In rare cases an ordinary inflammatory exudate does not undergo absorption, but remains as a chronic collection, or may become hemorrhagic or purulent. Many authorities believe that even a fibrinous exudation may be absorbed, up to a certain amount, after undergoing a fatty change.

The fibrous patches left from pericarditis are larger, thicker, and have a more irregular distribution than the so-called milk spots, and as a rule are associated with adhesions.

A consideration of the morbid anatomy of pericarditis would not be complete without reference to the changes which are produced in the heart. Broadbent says: "The heart is usually found to be dilated to a varying degree. In the subacute or chronic cases in which the pericardium has become adherent, the dilatation is often extreme, and the heart muscle soft and flabby, showing evidence, on microscopical examination, of well-marked, inflammatory changes. Dr. Poynton has shown by a series of sections of the heart wall, in cases of rheumatic pericarditis, that the cardiac muscle, as well as the pericardium, is almost invariably attacked by the inflammatory process, and that there are foci of small round-cell infiltration between the muscle fibres throughout the thickness of the heart wall. The myocarditis which accompanies pericarditis is therefore not simply an extension of the inflammation from the pericardium to the myocardium. There is granular and fatty degeneration of the cardiac muscle due to the toxic effects of the rheumatic poison, as well as actual destruction of muscle fibres by inflammatory exudation."

Eichhorst applies the name *Zuckergussherz—frosted heart*—to cases in which the epicardium is thickened by chronic pericarditis, so that it gives the organ the appearance of being covered by a sugar icing, as in the case of a frosted cake.

Calcareous Pericarditis has been referred to above. In chronic cases the heart may be completely invested by a calcareous coat which may in places be 1–1.5 cm. thick (Osler).

Etiology.—Pericarditis is almost always a secondary infection. Its etiology resembles very closely that of endocarditis. The more careful our examinations and the greater our bacteriological knowledge, the fewer will be the cases of idiopathic pericarditis discovered.

Rheumatism is by far the most common cause of pericarditis. Roberts, in "Allbutt's System," states that pericardial inflammation is to be looked upon not as a mere complication of rheumatism, but as an essential part of the disease.

Sibson noted that in the large majority of cases of rheumatic pericarditis endocarditis was also present. Broadbent states that pericarditis must not be regarded as a separate entity, but as part of a general inflammation of the heart, the myocardium being almost invariably and the endocardium frequently affected.

There is no definite relation, when a large number of cases is considered, between the severity of the joint affection and the severity of the pericarditis. It may develop at any time during the attack, even preceding the joint affection, or late in the disease.

Pneumonia, pleurisy, the various acute infectious diseases, especially scarlet fever—during the stage of desquamation or that of nephritis,—chorea, pyæmia, purpura, scurvy, are all causes of pericarditis. Especially interesting is its association with Bright's disease. Taylor found that pericarditis occurred in about ten per cent. of his cases of Bright's disease. Sibson, in an analysis of 1,691 cases of Bright's disease collected from various sources, found that pericarditis existed in 8.17 per cent. Tuberculosis, carcinoma, extension from contiguous tissues, traumatism, are also causes of pericarditis.

Aneurism of the aorta causes 2.6 per cent. of all cases, a very high figure when one recalls the comparative infrequency of aneurism (Preble).

Scurvy is frequently accompanied by hemorrhagic pericarditis. Seidlitz and Kyber report an epidemic occurring in Russia in 1840, in which 30 out of 60 fatal cases showed hemorrhagic pericarditis. The fluid was dark, and amounted to four or five pints.

Sears, in a study of 100 cases at the Boston City Hospital, assigns rheumatism as a cause in 51 cases, pneumonia or infection with the pneumococcus in 18; in 7 chronic nephritis, and in 5 pleurisy was the primary disease.

It is now generally accepted that the pericarditis of Bright's disease is usually an infection, often a terminal infection. (Banti believes in the uræmic theory.) Chronic disease lessens the resisting power of the tissues and invasion by micro-organisms becomes easier.

Bacteriology.—In acute pericarditis Flexner, in a limited number of cases, found bacteria present in the following order of frequency: M. lanceolatus, 11; streptococcus, 4; staphylococcus, 1; B. pyocyaneus, 1; B. influenzæ, 1; M. lanceolatus and B. coli communis, streptococcus, staphylococcus aureus and B. coli communis, 1; staphylococcus and B. coli communis, 2; unidentified, 1.

Infection Atrium.—Pneumonia, 8; bronchitis, 2; erysipelas, 1; leg ulcer, 1; tonsils, 1; peritoneum, 1; cancer of stomach, 1; sloughing myoma, 1; doubtful, 7.

Tubercle bacilli have rarely been found in cases of tuberculous pericarditis.

Symptomatology.—It was early recognized that the symptomatology of this condition was so indefinite, obscure, and inconstant, and resembled so closely that of other cardiac conditions, that a diagnosis could not be based upon it. This is only partly because the disease comes on secondarily to other conditions. The symptoms are often so indefinite that pericarditis is entirely overlooked, and may alter almost not at all the symptomatology of the primary disease. Even in the so-called idiopathic cases the subjective phenomena are very indefinite.

The following important symptoms may be mentioned:

Pain is present in many cases of pericarditis, especially early in their course, when friction sounds are heard, but it is not pathognomonic, and bears no special relation to the severity of the attack. It may be extremely severe in localized pericarditis and entirely absent in large effusions or extensive adhesions. When present it is usually in the precordial region. It varies much in its character, from a dull ache to a stabbing or tearing pain. It usually disappears with the advent of the effusion. There may be pain or tenderness in the epigastric region, especially when upward pressure is made at one or other of the costal angles. This pain usually comes on later than the precordial pain. The patient may complain of only a sensation of distress, a pressure or tightness about the heart, especially if a large effusion is present. There may be dyspnœa and palpitation.

The *pulse* has no special characteristics. The heart action in the early stages is rapid and energetic. Subsequently, as a result of the mechanical embarrassment superimposed by the presence of a large amount of fluid, and the involvement of the myocardium and its nerves, the heart action becomes weak and rapid, possibly irregular. In the early stages it may vary from 90 to 120; later, in rare cases it may reach 160. In some cases there is very little alteration from the normal, and rarely the pulse rate is below normal. Dr. Ewart says that in many cases of effusion the pulse is quick, resembling the Corrigan type. Pulsus paradoxus is said to be more marked in adherent pericardium than in any other known condition.

Friedreich's sign of collapse of the cervical veins during diastole is not considered important.

The respiratory symptoms vary much. Respirations are usually somewhat increased in frequency. If the effusion becomes large, dyspnœa and even orthopnœa may supervene. The patient usually prefers to lie on his back or on the left side. In large effusions there is often a short irritative cough, and in rare cases distressing hiccough due to involvement of the phrenic nerve.

There may be pain or difficulty in swallowing as a result of pressure of an effusion upon the œsophagus, or due to nerve irritation.

Inasmuch as pericarditis is a secondary affection, there is usually present the fever of the primary disease. The pericarditis may cause a slight extra elevation of temperature. It may be practically normal throughout, or only slightly elevated, to 100° or 101°; rarely to 103° F.

It must be remembered that the symptomatology of pericarditis may be altered by the associated primary disease. Thus, a case secondary to Bright's disease would differ from a case secondary to rheumatism.

Enormous effusion, by interfering with the action of the heart and aëration of the blood, may produce the most grave symptoms of dyspnœa, cyanosis, very rapid and weak heart action, which if not relieved may eventuate in death.

The rapid heart action, the pulsus paradoxus, and the asymmetry in the size of the pulse of the radials, the irregular type of temperature, the paralysis of the recurrent laryngeal nerve, the unequal pupils, the disturbed mental state, may all be important signs, if present (Billings).

Physical Signs.—*Acute Plastic or Dry Pericarditis.*—Fortunately the signs of this condition are very distinct; the danger is that they may disappear before the physician's attention is directed to the precordium. In some cases they are very evanescent, disappearing in a few hours; in others they persist for days.

Pericardial Friction Rub.—This is the pathognomonic sign of dry pericarditis. It is a superficial, dry, scraping or rubbing sound, distinctly dependent upon the heart movements. Frequently it is a to-and-fro friction corresponding to systole and diastole. It does not correspond exactly with the first and second sounds of the heart, but may occupy a place between them. At times it is only systolic. It is said to be first heard in most cases over the base of the heart, but may be heard first as a single systolic scratch at the apex. It is usually heard best just to the left of the sternum, between the third and fifth ribs. As the inflammation progresses it may be present over most of the precordium, even to the right of the sternum, in children. It is due to the rubbing of the inflamed pericardial surfaces over each other.

Palpation.—In well-marked cases a friction fremitus can be felt.

Clinical experience has proven that the most common cause of the disappearance of the friction sound is the advent of an effusion which separates the pericardial surfaces. Another fairly common cause is the development of adhesions, uniting the pericardial surfaces. Again, there may be absorption with resolution. The friction sound may reappear after the absorption of the fluid.

Differential Diagnosis.—A pericardial friction rub is usually so characteristic that little difficulty is experienced in its detection. It must be distinguished from organic and functional murmurs and from pleuritic friction sounds. Cardiac murmurs almost never have this superficial scraping, to-and-fro sound; they are likely to be more distinctly systolic or diastolic, and have special lines of transmission. They are not so much altered by changes in the patient's position and are more permanent.

Pleuritic friction sounds are very similar in their character, but are dependent on respiratory movements, and can be eliminated by causing the breath to be held.

Pleuro-pericardial Friction.—A friction sound resembling very closely the true pericardial friction is heard when there is inflammation of that part of the pleura which overlaps the pericardium. With each contraction of the heart the external surface of the pericardium is forced along the under surface of the inflamed pleura, and a friction sound is produced. Forced expiration might tend to eliminate this sound.

Signs of Effusion.—The fact that a friction sound has been heard is of great assistance in diagnosing an effusion. It is fair to suppose that an increased area of cardiac dulness, which develops subsequently to the detection of a pericardial friction rub, is due to an effusion. But one always keeps in mind the fact that a cardiac dilatation must be reckoned with.

Percussion.—It is usually stated that less than 100 c.c. of fluid cannot be recognized by percussion.

In an effusion of moderate degree the outline of dulness is quite characteristic, being of an irregular pear shape, or that of a triangle with rounded angles, whose base faces the diaphragm, and whose apex is above, to the left of the sternum. (Cabot states that he has not been able to make out this characteristically shaped area.)

More important is the fact that the dulness extends much farther to the left, beyond the apex beat, than it does in hypertrophy or dilatation.

Rotch has drawn special attention to the blunting of

the cardio-hepatic angle in the right fifth intercostal space in pericardial effusion. In health this cardio-hepatic angle is approximately a right angle. In pericardial effusion it is more obtuse. Rotch lays great stress upon the presence of absolute dulness in the fifth intercostal space, extending one and one-eighth to one and a half inches to the right of the sternum. However, this is found in dilatation of the right heart; for instance, in a case of advanced mitral insufficiency it is a common experience to obtain dulness in this area, and a consequent blunting of the cardio-hepatic angle. In fact, Broadbent speaks of two cases of dilated heart in which paracentesis was resorted to, because the signs seemed to point conclusively to pericardial effusion.

It is also a difficult matter to decide where liver dulness ends and heart dulness begins.

The explanation of the difference of opinion in regard to the value of Rotch's sign in the differential diagnosis between pericardial effusion and dilatation of the heart is due, I think, to the fact that one authority has in mind absolute heart dulness, the other only relative dulness.

With a large effusion, signs of compression of the left lung develop. Above the line of dulness is an area of tympany or hyper-resonance, with increased vesicular or broncho-vesicular breathing. Ewart has called attention to the presence, in large effusions, of a circumscribed patch of dulness or impaired resonance just below the angle of the left scapula, over which there are increased vocal fremitus, bronchophony, and bronchial breathing. Broadbent, I believe, is correct in saying that these signs might also be present in any case of enormously enlarged heart.

Position of the Apex.—It has been held by some of the authorities (Sibson) that the apex is pushed upward and outward by the fluid. Others state that it is tilted upward and inward, that is, is floated by the effusion. Rotch believes from his investigation that this is an erroneous view. It seems impossible for a fluid of a lighter specific gravity than the heart to float it upward.

Ludwig and Bowditch have observed that the impulse of the heart, as seen normally in the fifth left interspace, need not be caused by the heart's apex, but may be caused by a portion of the heart above the apex striking against the chest wall. This fact I have observed many times. It should also be remembered that in children the apex beat is normally in the fourth interspace. The presence of a high pulsation could be accounted for by the tumultuous action of that portion of the right ventricle.

A very important sign is the gradual weakening of the apex beat with the increase of fluid. It may be entirely obliterated. In pericardial effusions the pulse may be strong and the apex beat weak; in heart lesions the apex beat may be heaving and the pulse weak. The effusions may become enormous and thus hinder the heart's action.

The presence of adhesions may prevent a pericardial effusion from assuming the typical shape. Rotch speaks of a case in which dulness failed to appear in the fifth intercostal space (right side), because of the presence of adhesions binding the lung tightly to the right edge of the sternum.

Purulent Pericarditis.—This disease furnishes the same physical signs as the serous effusion. It might be suspected from the etiology, *i.e.*, if secondary to suppuration in the other parts of the body, especially of the lungs or pleura. The leucocyte count would very likely be higher than in the simple serous pericarditis. Paracentesis is the only positive means of determining the nature of the fluid. The temperature curve is of the septic type, resembling that of empyema. The process may come on insidiously. It is usually purulent from the outset, though a serous effusion occasionally becomes purulent.

Adherent Pericardium.—The *symptoms* are not at all characteristic, and cannot be distinguished from those of organic heart lesions.

The *physical signs* in marked cases may be quite distinctive. They depend upon whether the adhesions exist between only the parietal and visceral layers of the pericardium, or whether they include also adhesions between the external pericardium and the chest wall or pleura. The following are the most important: (1) Fixation of the apex beat. Under normal conditions the apex beat moves a considerable distance with change of position of the patient and forced inspiration. (2) Systolic retraction of one or more interspaces. This sign, however, is unreliable, especially if the retraction is present in only one interspace, and at the apex; for it is occasionally seen in greatly hypertrophied hearts, and it may also be due to atmospheric pressure.

If there be distinct dragging in of the costal cartilages at the lower end of the sternum, one can be almost certain of adhesions.

Broadbent's Sign.—"Systolic retraction of the posterior or lateral walls of the thorax may indicate the presence of a universally adherent pericardium. Such retraction may, however, be seen even when the pericardium is not adherent to the heart, but only to a larger extent than normal to the central tendon of the diaphragm, to the muscular substance on either side, and to the chest wall as well. In such cases the heart is usually greatly enlarged and hypertrophied from old valvular disease. The explanation seems to be that the portion of the diaphragm to which the pericardium is adherent is dragged upward at each systole of the heart, so that the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inward and retracted."

If pericardial adhesions are present between the heart and the diaphragm and the chest wall, the descent of the diaphragm would be much diminished. Fluoroscopic examination would be very useful in demonstrating this point. Litten's phenomenon would be diminished or absent.

Enlargement of the heart is common in adherent pericardium. There are both hypertrophy and dilatation. The hypertrophy results from the obstruction offered to the regular contraction of the heart.

Broadbent states that the heart becomes dilated during the acute pericarditis, and before it regains its original size, becomes anchored in this position by adhesions.

Friedreich's sign of diastolic collapse of the cervical veins is not considered useful by the majority of modern writers.

The pulsus paradoxus has generally been held to be a sign of pericarditis. It is characterized by the disappearance of the radial pulse during inspiration. Kussmaul believes that it is of diagnostic importance in indurated mediastino-pericarditis; it is due to the fibrous cord dragging on the aorta during inspiration.

Most of these signs are dependent upon the presence of adhesions between the pericardium and the chest wall. When such adhesions are absent signs are very indefinite.

Signs and symptoms of incompensation may come on which cannot be distinguished from those of incompensated organic lesions.

An important condition is that of *pseudo-cirrhosis* of the liver due to obliterative pericarditis. In this condition there is an enlarged liver, associated with ascites, but without œdema or enlarged spleen. Autopsies have shown, according to Becker, in all recorded cases, that ascites is due to a passive congestion of the liver, causing a connective-tissue formation with subsequent contraction and obstruction of the portal circulation, the result of obliterative pericarditis.

Differential Diagnosis.—The chief difficulty lies in distinguishing between dilatation of the heart and pericardial effusion, when, as occasionally happens, neither friction sound nor murmur can be heard. This may be better appreciated when it is remembered that on several occasions the right ventricle has been punctured by the paracentesis needle with fatal results, the diagnosis of effusion having been made. In many cases in which the effusion is only of moderate degree it is difficult to be absolutely certain of a diagnosis.

Massive pericardial effusions, amounting to one and a

half or two litres, have been mistaken for pleuritic effusions.

The following excellent table is taken from Rotch's "Pediatrics," p. 761:

DIFFERENTIAL DIAGNOSIS BETWEEN A DILATED HEART AND A PERICARDIAL EFFUSION.

Case I.—Endocarditis; dilated heart.	Case II.—Pericarditis; effusion.	Case III.—Endocarditis; enlarged heart; pericardial effusion.
Girl, eleven years. Attack followed acute articular rheumatism.	Boy, six years. Attack followed acute articular rheumatism.	Girl, eight years, August 3d. Attack followed acute articular rheumatism.
Orthopnœa; precordial pain; heart's impulse feeble, but perceptible a little to left and below left nipple, fifth interspace.	Orthopnœa; precordial pain; heart's impulse feeble but perceptible a little to left and below left nipple, fifth interspace.	Orthopnœa; precordial pain; heart's impulse feeble, but perceptible all over cardiac area, with apex beat a little below and to left of left nipple, fifth interspace.
Vertical absolute dulness not increased.	Vertical absolute dulness not increased.	Vertical absolute dulness not increased.
Absolute dulness under the sternum, and to left of sternum; identical with cases II. and III.	Absolute dulness under the sternum and to left of sternum; identical with cases I. and III.	Absolute dulness under the sternum and to left of sternum; identical with cases I. and II.
Absolute dulness did not extend to right of sternum.	Absolute dulness in fifth right interspace 2 or 3 cm. from edge of sternum.	Absolute dulness in fifth right interspace 3 or 4 cm. from edges of sternum.
Systolic murmur at apex.	Pericardial friction rub at base.	Soft systolic murmur at apex, transmitted to axilla; pericardial friction rub at base.
Recovery.	Recovery.	August 6th. Less dulness in fifth right interspace; apex murmur much louder and harsh.
		August 11th. Dulness only to right edge of sternum.
		August 18th. Dulness only to middle of sternum; friction rub ceased.
		December 1st. Physical examination the same as on August 18th, showing enlarged heart and mitral systolic murmur.

The following points, mentioned by Osler, may assist one in differentiating between dilatation of the heart and pericardial effusion:

In dilatation the impulse in thin-chested people is usually visible and undulatory; the shock of the cardiac sound is more distinctly palpable in dilatation; the peculiar area of dulness in effusion, especially if the upper limit shifts with change of position of the patient.

In dilatation the heart sounds are clearer, often sharp, valvular or fœtal in character; gallop rhythm is common, whereas in effusion the sounds are distant and muffled.

Rarely, in dilatation, is the distention sufficient to compress the lung and produce the tympanitic note in the axillary region.

Fluoroscopic examination is extremely useful for differential diagnosis. The opaque area does not pulsate as it does in enlarged heart or aneurism. The upper level can be seen to move with changes of position.

Diseases of the Pericardium in Children.—Only a few special observations need be mentioned under this heading, as the signs of pericardial disease are practically the same at all ages. Rotch states that so far as he could determine by the dissection of sixteen infants of different ages the relation of the infant's pericardium does not differ from that of the adult. The amount of fluid normally present is of variable quantity, but is probably under 5 c.c. When pericardial friction sounds are absent, the diagnosis of pericarditis in a young child is attended with great difficulties. Some writers (Warthin) state that an accentuated pulmonary second sound is characteristic of pericarditis. In infancy, however, the pulmonary second sound is normally much accentuated. Owing to the greater flexibility of its thorax the child is much more likely than the adult to manifest a bulging of the precordium as a result of the pressure of the fluid. It must be kept in mind that on account of the smallness of the child's thorax the heart and pericardium are both brought nearer the surface than in the adult; and as a result the heart's impulse can be felt, and the heart sounds heard, in much larger effusions than would be possible in adults.

Pericarditis sicca is uncommon in childhood. Exudation takes place more frequently than in the adult, and with greater rapidity, and is more likely to be purulent (Rotch). Exudation tinged with blood is not uncommon in early life, and is not so significant of tuberculosis as is a pronounced hemorrhagic exudation.

Holt states that pericarditis is rare in infancy and early childhood, only two cases having been seen in seven hundred and twenty-six consecutive autopsies at the New York Infant Asylum. In later childhood the disease is more frequent. According to Jacobi, Holt, and other authorities diseases of the lung and pleura, especially of the left side, take first rank as etiological factors in infancy and early childhood. After the fourth year rheumatism takes precedence and the pericarditis is then usually associated with endocarditis. Pericarditis may develop in the new-born as a result of infection of the cord. In children pericarditis may develop and become very pronounced, while the articular complaint is mild.

In young children pain seems to be generally absent.

Prognosis.—Pericarditis should always be looked upon as a serious disease, chiefly because of the myocardial degeneration which accompanies it. Death may take place in a few days in the acute cases associated with rheumatism and pneumonia, but this sequel is very uncommon. The immediate prognosis in these cases is generally good. The probability of repeated attacks, the likelihood that adhesions will form, and the presence of myocarditis render the prognosis for a long life unfavorable. When associated with Bright's disease the prognosis is bad.

Patients occasionally die from syncope as a result of embarrassment to the action of the heart from pressure, by very large collections of fluid.

Suppurative pericarditis is nearly always fatal if associated with a general septicæmia; if it is secondary to an empyema or other localized collection of pus, there may occasionally be recovery, with the adoption of early and proper surgical treatment. Of thirty-five cases of suppurative pericarditis treated by incision, fifteen recovered and twenty died (Roberts, *Am. Jour. Med. Sc.*, December, 1897).

In adherent pericardium the prognosis is serious if there are adhesions to the chest wall, or if the heart is enlarged, or especially if these adhesions are associated with valvular lesions. Universal adhesion of the pericardium to the heart, provided the heart is not enlarged, does not necessarily tend to shorten life.

Treatment.—Pericarditis must always be considered a serious disease, even if the symptoms are slight. The patient should be put to bed. The diet should be chiefly liquid, milk forming the major part. The stomach should not be overloaded. In the milder cases soft articles of food may be given. Pain should be relieved chiefly by the application of the ice-bag. In children hot applications may be more satisfactory. Morphine may be required in some cases. Restlessness and sleeplessness should be controlled by suitable doses of bromide or trional. The heart action, pulse, respirations, and color of the patient should be closely watched, and heart tonics, such as strychnine and digitalis and ammonia, given when indicated. The time may come when the heart is overwhelmed by the obstacles presented by the enormous effusion; the pulse becomes extremely weak and rapid, marked dyspnœa and cyanosis develop; then heart tonics are useless, and one must resort to paracentesis to save the patient.

Unless the symptoms are moderately urgent a serous effusion should not be evacuated, because many times the absorption is very rapid. If the effusion is large and has existed for many days, and shows no signs of resorption, it should then be evacuated without hesitation.

Paracentesis Pericardii.—The pericardium is tapped with a Potain aspirating set in the same way as is the pleura. The only difference is in the selection of the points of puncture. An excellent method is first to freeze the part with an ethyl chloride spray, make a very small incision through the skin with a bistoury, then insert the needle at right angles to the chest wall for a distance of from one and a half to two inches. After consulting a large number of authorities upon the correct site for puncture, and finding that each writer advises a different location, I have come to the conclusion that if a fairly large effusion is present it is safe to puncture anywhere from an inch to the right of the sternum to an inch or so beyond the left nipple line, between the fourth and sixth ribs. One is cautioned to avoid wounding the internal mammary artery. Little attention need be paid to the intercostal arteries.

Rotch very strongly recommends the fifth right interspace 4 cm. (1½ in.) outside the right border of the sternum. He states that an effusion of even as little as 100 c.c. can be found at this point, and that there is no danger here of wounding the heart, or the right internal mammary artery, or the pleura. Osler recommends the fourth left interspace, either at the sternal margin or 2.5 cm. (1 in.) from it. He also speaks of the fifth left interspace an inch and a half from the left sternal margin, and close to the costal margin in the left costo-xyphoid angle, as the point where the needle may be thrust upward and backward.

Purulent effusions should be treated like any other abscess, by early free incision and drainage. Irrigation of the sac is not advisable except in selected cases.

The treatment of adherent pericardium is practically that of organic heart lesions (myocardial and valvular). It is necessary to keep up bodily nutrition by proper exercise and diet, and at the same time guard against overtaxing the weak heart. If symptoms of incompensation develop they should be treated by rest, diet, and cardiac medicines, as indicated elsewhere.

HÆMOPERICARDIUM and HYDROPERICARDIUM have been considered in detail in Vol. IV.

PNEUMOPERICARDIUM, because of its extreme rarity and hopeless prognosis, is of very little practical importance. By it is meant the presence of gas or air in the pericardial sac. As a matter of fact, gas is never present alone, but is in combination with fluid, usually pus, *i.e.*, pyopneumopericardium. The fluid may be ichorous. It is always secondary to some very serious destructive disease in which a communication is established between the pericardium and a cavity or tube containing air—as, for instance, perforation from the œsophagus, especially in connection with cancer; rupture into the pericardium of a lung cavity, or pneumothorax, or perforation of a gastric ulcer. It may occur as a result of penetrating wounds, such as fractured ribs, concussion or crushing of the chest, or injury from the side of the œsophagus. The gas in pneumopericardium varies in amount and in composition, and is generally offensive. It may be under so great pressure that when the pericardium is punctured the gas escapes with a hissing noise.

Symptoms.—These are very indefinite, and difficult to dissociate from the primary disease. If the gas is present in abundance there will be dyspnœa, cyanosis, attacks of syncope, collapse, a feeble and irregular pulse, and occasionally dysphagia and precordial distress.

Physical Signs.—There may be bulging of the precordial region. The apex beat is weak or absent. The heart movements may produce a very peculiar crackling sensation due to the bursting of bubbles.

Percussion signs are very striking. A metallic tympanitic note is heard over the distended pericardium. Because of the presence of fluid and air a freely movable area of dulness is detected in the dependent part, upon change of position. The quality of the tympanitic note may also vary with the change of position.

Auscultation.—The heart sounds are unusually loud and may have a metallic ring. If murmurs are present they take on the same quality. The cardiac movements and deep breathing agitate the fluid and gas present in such a way as to produce unusual adventitious sounds. They have a metallic ringing quality, and have been likened to the sound of a water wheel.

Treatment is expectant and supportive. It may at times be wise to allow the gas to escape through a fine trocar, or even to incise and treat surgically. Little can be done in a medical way.

New Growths and Parasites.—Under this heading are included tuberculosis, carcinoma, and hydatids. Tuberculosis is much more common than the latter two. It is very unusual to find the tubercles of acute miliary tuberculosis on the pericardium. In most cases tuberculosis of the pericardium is chronic and secondary to tuberculosis in other parts of the body, especially of the lungs and mediastinal lymph glands. In many cases of pulmonary tuberculosis the complicating pericarditis is of the simple serous type. The exudate in tuberculosis and carcinoma of the pericardium is likely to be blood-tinged, and may be purulent or ichorous.

Carcinoma of the pericardium is extremely rare and is always secondary, the sac being involved by extension from neighboring organs.

Hydatids of the pericardium are extremely rare. Clinically we have no means of recognizing the presence of a new growth in the pericardium, except as we infer its presence from the detection of similar disease in neighboring tissues.

James Rae Arneill.

PERINEORRHAPHY. See *Obstetric Operations.*

PERINEUM, SURGICAL ANATOMY OF THE.—I. THE MALE PERINEUM.—In the skeleton the perineum corresponds to the outlet of the pelvis. It is a diamond- or lozenge-shaped space bounded in front by the pubis and subpubic ligament, behind by the coccyx, and on each side, from before backward, by the rami of the pubis and ischium, the great tuberosity of the ischium, and the great sacro-sciatic ligament.

The whole space measures about three inches and a half from side to side, and four inches antero-posteriorly. At the posterior part it is from two to three inches deep; anteriorly it only reaches the depth of one inch. The perineal space is separated from the pelvic cavity above by the recto-fascia and levatores ani muscles. A line drawn across from one ischial tuberosity to the other, and passing immediately in front of the anus, would divide the space into two parts (see Fig. 3794), the anterior of which is called the *urethral* triangle or true perineum, and the posterior the *anal* or rectal triangle. The anterior triangle contains the bulb and urethra, with the muscles of the perineum proper; the posterior triangle has in it the rectum and the two ischio-rectal fossæ.

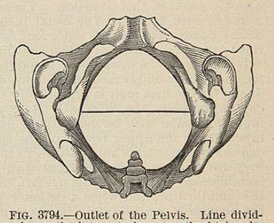

FIG. 3794.—Outlet of the Pelvis. Line dividing outlet into anterior or urethral triangle, and posterior or rectal triangle.

SURFACE ANATOMY.—In the undissected subject the superficial area of the perineum is very limited, especially when the thighs are brought together; it then consists of a narrow space or groove reaching from the coccyx behind to the symphysis pubis in front. In the centre of this groove is an elevation of the skin, called the median raphé, which runs from the front of the anus, over the scrotum, to the under surface of the penis. No vessels cross this line, and in this situation incisions may be made without any fear of hemorrhage. The osseous boundaries of the perineum may be easily made out through the skin; the great sacro-sciatic ligaments, however, being covered by the gluteal muscles, can be felt