of the heart are frequent secondary changes dependent upon the myocardial condition. On microscopical examination the gummata of the heart are distinguished from those in other regions of the body by the cellular nature of the process, the rarity of giant cells, and the relatively slight degree of caseation. Obliterating endarteritis of the coronary arteries may or may not be associated with gummatous myocarditis.

Slight degrees of interstitial myocarditis and gummata of small size may give rise either to circulatory disturbances or to general symptoms. In the majority of cases the onset is slow and insidious, the symptoms of serious myocarditis developing only when extensive involvement of the heart muscle has occurred. Consequently sudden death from acute cardiac insufficiency is very common, occurring in two-thirds of the cases. In those cases which are observed for some time before death dyspnœa, venous stasis, ascites, and general œdema gradually developed without the presence of the physical signs of valvular disease. Extensive fibroid change or gummata of large size may lead to aneurismal dilatation of the heart wall, general hypertrophy, or dilatation accompanied by the gradually developing symptoms of cardiac insufficiency. Rupture of a softened gumma into the blood stream very frequently leads to thrombosis and embolism. In the reported cases the emboli were found in the vessels of the retina and choroid, and in the coronary and pulmonary arteries. A form of syphilitic angina pectoris has been described by some writers who have attributed it to a specific action of the syphilitic poison upon the neuro-muscular apparatus of the heart wall or upon the sympathetic nerves. The progress of cardiac syphilis is usually very slow, periods of apparently good health may alternate with extended attacks of cardiac insufficiency. In either period slight accidents may cause sudden death. This may be due to rupture of the heart or to occlusion of the coronary arteries, either through fibroid changes, or through thrombosis or embolism.

The diagnosis of syphilitic disease of the heart rests upon the exclusion of other causal factors and the establishment of the existence of a syphilitic infection, either past or present. In doubtful cases the therapeutic test should be employed. The prognosis in undoubted cases of syphilitic myocarditis is very unfavorable if the condition is well advanced. Only in the earliest stages, before much structural change of the heart wall has occurred, is there hope of a cure. The treatment is that pursued in cases of syphilis in general, with special treatment directed to the myocardial condition. It is very probable that systematic treatment of all cases of syphilis, if continued faithfully for four or five years, would greatly diminish the number of cases of myocardial syphilis. *Aldred Scott Warthin.*

HEART DISEASES: TUBERCULOSIS.—Tuberculosis of the myocardium is very rare. The first recorded case is that of Townsend in 1832. In 1894 Valentin collected thirty-six cases from the literature, and estimated that the condition occurred but once in one thousand tuberculous bodies. Hand, in 1898, collected forty-two cases, and in 1899 Thiry was able to gather sixty-three cases from the literature up to that year. With the exception of a few cases the condition was secondary to tuberculous disease elsewhere in the body. Demme and Knopf have reported cases thought to be primary. The majority of the cases occurred before the fifteenth year, several in infants. The ventricles are more often affected, the wall of the right auricle next in frequency. In the majority of cases the lesions are characterized by the scarcity of tubercle bacilli found in them.

The infection of the myocardium occurs either by direct extension from the pericardium or endocardium, or by hæmatogenous or lymphogenous metastasis. According to some writers a retrograde lymphogenous metastasis from the bronchial lymph glands forms the most common means of infection. This is denied by others, inasmuch as the lack of collateral anastomosis of the cardiac lymph vessels does not favor retrograde metastasis. In the miliary cases the avenue of infection is usually through the veins.

The disease may manifest itself in several forms: (1) Scattered miliary nodules through the muscle; (2) large caseous nodules varying in size from a pea to a hen's egg, usually multiple, but occasionally single; (3) diffuse tuberculous myocarditis in the form of a fibroid change with scattered caseous nodules of small size; (4) fibroid myocarditis with small non-caseating tubercles. The third and fourth forms are very rare; and are with difficulty distinguished from syphilitic processes, as the bacilli are found in such scanty numbers as to be very difficult of demonstration. The miliary tubercles are usually most numerous just beneath the epicardium, occasionally beneath the endocardium. They rarely extend far into the muscle. The large caseous tubercles represent a more chronic infection; they may be encapsulated or show extensive signs of healing. In these cases the pericardium is almost always involved, the endocardium only rarely when the lesion is in the ventricles. In the case of a tubercle of large size developing in the auricular wall both peri- and endocardium are soon involved. Perforation into the auricular cavity may take place and give rise to a general miliary tuberculosis. In a case reported by Püschmann there was an acute miliary tuberculosis which seemed to have taken its origin from a thrombus containing numerous tubercle bacilli which was attached to a caseating tubercle in the auricular wall.

With slowly developing tubercles there is always associated an interstitial myocarditis. In the case of the large caseous tubercle this process is confined to its immediate vicinity. This form of interstitial myocarditis is much less important than the diffuse form associated with scattered tubercles of slow growth and showing little tendency to caseate. In spite of the weakening of the cardiac wall through replacement of the muscle by connective tissue and through caseation of the tubercle, a rupture or aneurism of the wall is not likely to occur in these cases because of the diminished blood pressure. In none of the recorded cases of myocardial tuberculosis has this event been observed. Further, there is usually extensive fatty degeneration of the heart muscle in the neighborhood of the tuberculous process, and this diminishes to a still greater degree the efficiency of the heart's action. Calcification or encapsulation of the caseous tubercles may occur here as elsewhere. Complete healing may result, the tubercle being wholly replaced by connective tissue.

Within very recent years the presence of tubercle bacilli in parietal thrombi and endocardial vegetations has been many times demonstrated, and it is very probable that tuberculous endocarditis is of much more frequent occurrence than is usually supposed. The great frequency of cardiac thrombi in pulmonary tuberculosis suggests the possibility of their formation by the lodgment of bacilli upon the endocardium. Secondary involvement of the myocardium may follow the development of such endocardial tubercles.

Tuberculosis of the myocardium presents no characteristic symptom complex, and in the great majority of cases cannot be diagnosed. The symptoms depending directly upon the condition would be those of a myocarditis. Involvement of the pericardium may give rise to such definite signs and symptoms as to permit of the diagnosis being made. In case of pericardial effusion a bacteriological examination of fluid obtained by puncture could be made. The termination of the cases is by death, due usually to the general tuberculosis rather than to the condition of the heart. Rarely solitary tubercles of small size and slow growth may heal. For the great majority of the cases there is no treatment beyond that of a purely symptomatic nature. *Aldred Scott Warthin.*

HEART DISEASES: VALVULAR LESIONS.—General Etiology.—The great majority of cases of chronic valvular lesions originate in acute or chronic endocarditis. A single attack of acute rheumatism may leave the valves in a seriously damaged condition, but more frequently contraction and thickening of the valvular apparatus continue after the acute stage has passed off, and the leaflets gradually become deformed and defective in function. Recurring attacks of endocarditis are liable to occur in valves once damaged by this cause, and slight defects may thus terminate in serious and irreparable injury.

Valvular deformity may also originate in a chronic sclerotic process, comparable to the changes which take place in sclerosis of the vessels. Although this chronic process may originate from rheumatic influences, yet there are a number of causes which, when taken collectively, exert a more frequent influence.

That high arterial pressure is capable of producing valvular lesions is shown very clearly by the experiments of Roy and Adami. These observers narrowed the aorta of the dog by clamping and thus induced a fringe of swollen tissue at the free edge of the valve, due to infiltration of cells and œdema.

Clinically it is well recognized that long-continued high arterial pressure may result in sclerosis and thickening of the valves. More particularly is this noticed in the aortic segments which have to bear the brunt of increased strain in the arterial system; the mitral cusps often suffer as well, and thickening of their free borders is very frequent in cardiac hypertrophy, a condition in which they are subjected to increased strain.

The proportion of cases originating in acute and in chronic endocarditis, has been variously estimated. It is more common to trace an origin in acute endocarditis, resulting from rheumatism or other infectious processes, in the mitral than in the aortic segments. In aortic lesions the process is frequently chronic from the outset.

Guttmann regards three-fourths of all valvular cases as beginning in a chronic fashion, and Worobjew's (*Deut. Arch. f. klin. Med.*, 69, 466) analysis of 180 cases also confirms this view. Most writers have, however, regarded the rheumatic cases as the most frequent; Schott attributes 54 per cent., and Gibson 63 per cent. of cases to a rheumatic origin.

Aortic Incompetence.—*Etiology.*—Although occurring at all ages and in both sexes, it is far more commonly met in males about the middle period of life. Under the age of twenty-five most cases are due to a rheumatic origin, and in these it is usual to find the mitral valve also affected. Anatomically, a large majority of cases originate in a chronic fibrosis of the segments of the valve with resulting retraction and deformity. All conditions leading to continued or even intermittent high pressure tend to the development of fibroid thickening. Incompetence is consequently often seen in arterio-sclerosis, chronic Bright's disease, gout, plumbism, and in chronic alcoholism. Those engaged in laborious physical work constitute the greater number of cases. It is consequently frequent in laborers, and is occasionally seen in athletes. Old soldiers often suffer from the disease, owing partly to laborious physical exertion, partly to chronic alcoholism and to specific infection, which has a well-marked but obscure relation to the disease. Tabetics are not infrequently attacked by aortic insufficiency, a fact attributed by Gowers to a common origin in syphilis. Owing to the frequent involvement of the coronary vessels in the sclerotic processes at the root of the aorta, the arterial cases are usually more serious than those due to stationary rheumatic endocarditis.

Morbid Anatomy.—In the great majority of cases aortic incompetence is due to a chronic sclerotic process in the valves. Their free edges become thickened and retracted, and the cusps fail to support a column of water when poured into the aorta. Their edges frequently adhere to each other, causing more or less obstruction. Patches of atheroma are often present on the valves, and in old-standing cases there may be an abundant deposit of lime salts, giving a hard bony feel to the margins of the orifice. When extreme adhesions and stenosis are present, a transverse bar is sometimes seen running from the united cusps to the wall of the aorta, and represents the margins of the original segments. The mitral valve is frequently involved in the sclerotic process, particularly in the rheumatic cases. The root of the aorta often presents atheromatous and calcareous plates and in many instances it is dilated. Freshly raised atheromatous plaques in the aorta, involving one or more of the aortic segments, are occasionally met with. Fresh endocarditis is frequently added on to older lesions, and it is not uncommon to find a row of warty granulations along the edge of the valve. Vegetations are found in the course of acute rheumatism and in malignant endocarditis. They may interfere mechanically with the closure of the valves, or in the latter instance cause incompetence from rupture of one of the segments.

Dilatation of the root of the aorta, the result of atheromatous changes, is a rare cause of aortic insufficiency. A series of cases has been recorded by Pitt (Trans. Path. Soc., 1898) in which incompetence was induced in this way, without marked changes in the valve. In aneurism it is very common to find aortic regurgitation, but in such cases it is usually due to a coexisting sclerotic change in the valve. With advancing age the aortic orifice becomes wider, but the comparative rarity of aortic incompetence in old people would seem to indicate that the dilatation is insufficient to allow of regurgitation.

Osler (Trans. Am. Phys.) has pointed out the frequency with which sclerosis and insufficiency occur in those cases of malformation in which two, instead of three cusps, exist.

Rupture of the aortic cusps occurring spontaneously, or more commonly from violent efforts, or as the result of severe direct injury to the chest has been recorded in a number of instances, and is a rare cause of aortic incompetence. Barié has shown that rupture may be produced experimentally, and that the aortic cusps suffer more readily than other valves. Although perfectly healthy valves may suffer in this way, yet in most instances there is evidence of previous sclerotic change. The average age at which rupture occurs is thirty-nine, a period of life at which degenerative changes are common, and is suggestive of previous disease.

Important secondary changes take place in the cardiac chambers as the result of aortic incompetence. The left ventricle is obliged to accommodate not only the usual amount of blood from the auricle, but also that which regurgitates during diastole of the ventricle. The consequence is that this chamber becomes dilated to an extent corresponding with the volume of blood passing from the aorta, and the degree to which dilatation takes place is thus an index to the extent of regurgitation. In the severer types of the disease dilatation is extreme, and the heart reaches a size seldom attained under other circumstances, *the cor bovinum*. Although dilatation is primary and the cavity remains greatly enlarged, some hypertrophy of the ventricular wall also results, and thus balances or compensates for the diseased valve. Owing to the difficulty it experiences in propelling blood into a ventricle already distended, the auricle in turn dilates and hypertrophies, and eventually the right heart becomes involved as in mitral disease.

Symptoms.—This lesion is most frequently discovered during the routine examination of patients in apparent health, or in the subjects of other maladies. Compensation is often well maintained for years, and during this period there are few or no subjective symptoms, and indeed there is usually much less discomfort than in mitral lesions. Dizziness, palpitation, and precordial pain are sometimes noticed, and dyspnœa on exertion is often an early symptom of failing compensation. The facies presents in many instances a pale and anxious aspect, contrasting with the cyanosis of mitral affections.

An exception to the insidious onset is seen in cases of ruptured valve. Here the abrupt onset with severe precordial pain and dyspnœa, the development of a diastolic murmur, sometimes so loud as to be perceptible to the patient himself, the occurrence immediately or shortly after some unusual strain or external violence to the chest, form a clinical picture which is very striking. The severer cases may terminate fatally in a few weeks, but

for the most part they survive for a longer period. Two of Barié's cases lived for eleven and thirteen years respectively.

Failing compensation is marked by dyspnœa, palpitation, and swelling of the feet. Dyspnœa, as in other forms of cardiac disease, comes on from exertion, especially in mounting a hill or climbing a stair. As the malady advances there are often attacks of paroxysmal dyspnœa, coming on especially at night, and known as cardiac asthma. Loss of sleep and a moderate loss of flesh give the patient a worn and anxious aspect. Œdema, if present, is usually moderate in extent, except in advanced cases in which there is secondary mitral incompetence, when it may attain a high grade.

Pain is more commonly present in aortic than in other valvular lesions. It varies much in intensity, from a sense of weight and uneasiness to acute pain, often radiating to the left arm and shoulder, seldom to the right. In arterio-sclerotic cases, in which the coronary arteries are involved, all the characters of true angina may be present.

In advanced stages of the disease orthopnœa and Cheyne-Stokes breathing, either with or without general anasarca and the visceral congestion of mitral lesions, may be present.

Mental disturbance develops more commonly in aortic than in other valvular affections, and shows itself in restlessness and change of temper. In some instances delusions are prominent and the patient may require restraint.

The symptoms in many instances are dominated by those of arterio-sclerosis or chronic renal disease, in which the incompetent valve is merely an incident. Sudden death is an accident which may ensue even in apparent health. Such an occurrence is more liable to happen when there is disease of the coronary vessels. Death occasionally comes rather unexpectedly after the symptoms of ruptured compensation have lasted for some time, but the more common termination is by gradual failure, dropsy, and asystole.

Physical Signs.—Visible pulsation in the arteries often suggests the existence of aortic incompetence. Throbbing of the carotids, visible pulsation in the radial and temporal arteries, and, in severe cases, even in the digital branches of the hands and feet are distinctly seen. Pulsation of the palate and uvula has been observed, and the pulse may be counted in the retinal arteries with the ophthalmoscope. Capillary pulsation is seen in the finger nails, and is also readily observed in the lips when a glass slide is pressed on them. The pulse has certain characters which at once reveal the nature of the affection to the practised finger. The wave is large, it strikes the finger suddenly and as rapidly recedes, these characters being more marked when the arm is raised. Since the appearance of Corrigan's paper, this pulse has gone by his name, and is also known as the water-hammer or collapsing pulse. It is regular in volume and rhythm except in the later stages, when slight irregularity may develop. The arteries are frequently thickened and this feature is seen even in young people suffering from the rheumatic form of the disease.

Examination of the heart shows the presence of very distinctive signs, the more important being the increased size of the organ and the diastolic murmur at the base. These signs, taken in conjunction with the arterial phenomena, render the affection one of the easiest of cardiac lesions to recognize.

Inspection often shows a slight degree of fulness in the cardiac region, particularly in children and young adults, in whom the chest wall is more elastic. Visible pulsation is seen below and outside the normal apex site, and retraction may be seen in the third and fourth spaces due to atmospheric pressure during systole.

On palpation the cardiac impulse is forcible and heaving, and the apex is felt in the nipple line or as far out as the mid-axilla, in the fifth, sixth, or even the seventh space. The increased area of the heart is also shown by percussion, the dulness extending down and to the left side.

The most characteristic sign is found on auscultation in a diastolic murmur, due to the reflux of blood to the ventricle. It is usually a soft prolonged soufflé, beginning with the second sound, and occupying the greater part of the diastolic interval; it is sometimes shorter or its character may be harsh. Although sometimes having its site of maximum intensity at the aortic cartilage, it is usually best heard about the pulmonary region. It is transmitted down the sternum, especially to its left side, and is occasionally heard only at the lower sternal region. It is frequently transmitted to the apex, and when loud is heard all over the front and back of the chest. The diastolic murmur in question is one of the most persistent of cardiac murmurs. It occasionally disappears during periods of asystole, to reappear with increasing cardiac power. Instances have been reported in which the murmur disappeared from closure of the orifice by a vegetation, and Musser has recorded a remarkable case in which the leak ceased owing to wearing down of the thickened corpora Arantii. A systolic murmur commonly precedes the diastolic sound, and with it gives the well-known to-and-fro murmur.

The second sound is often dull and indistinct and may be entirely lost. When absent it indicates serious deformity of the valve segments and is usually a sign of extensive regurgitation. The sound should be listened for in the arteries of the neck, as it may be present, but obscured by murmurs at the base of the heart.

The gradual development of aortic incompetence may sometimes be traced from an accentuated second sound in arterial sclerosis to the diastolic murmur of the incompetent valves.

A presystolic murmur at the apex was first described by Flint in cases of aortic incompetence, and commonly goes by his name. It exactly resembles the rumbling murmur heard in many cases of mitral stenosis. Thayer in a recent study of this murmur states that it was present in sixty per cent. of his cases, and that thrill, snapping first sound, and the tapping impulse of mitral stenosis were also occasionally noticed. The pulse characters form the best guide in distinguishing the two conditions, but when they are combined it is often difficult or impossible to state whether the murmur is associated with mitral stenosis or is due to aortic incompetence.

The temperature in cardiac disease is usually subnormal. Elevation of temperature is sometimes the first evidence of a fresh endocarditis. It may, however, signify some rheumatic or other inflammatory complication. In the advanced stage of cardiac disease it is most often due to a broncho-pneumonia, and is frequently seen a few days preceding death.

Diagnosis.—The physical signs of a well-marked case of aortic incompetence are unmistakable. The diastolic murmur is a very constant feature, only seldom disappearing in advanced cases with asystole. Temporary diastolic murmurs are occasionally heard in arterio-sclerosis, the valve probably yielding to the increased blood pressure. Allbutt regards such cases as the early stage of the affection. A diastolic murmur is sometimes heard in mitral stenosis at the pulmonary area, and represents a temporary incompetence of the pulmonary leaflets from increased pressure in this vessel. Such a murmur is distinguished by the small pulse of mitral disease, instead of the large collapsing pulse of aortic incompetence.

The degree of regurgitation is estimated by the size of the ventricle and the character of the pulse. Disappearance of the second sound at the base and in the arteries of the neck also indicates free regurgitation.

Aortic Stenosis.—*Etiology.*—Although occurring at all ages, and sometimes as the result of changes in early life, aortic stenosis is most common in old men with calcareous arteries.

In high degrees of the affection the valves are thickened and calcareous and the orifice may become so narrowed as hardly to admit a probe. In rare cases the obstruction is subvalvular and the result of an attack of fœtal endocarditis. Stenosis, apart from the slighter

grades of the affection, and unassociated with aortic incompetence, is a rather rare condition.

The symptoms are similar to those found in aortic regurgitation, and depend rather on associated conditions, such as generalized arterio-sclerosis, sclerosis of the root of the aorta, disease of the coronary vessels, and myocarditis, than on the aortic lesion itself.

The onset of the affection is insidious, and there is a long period of latency. Uneasiness or pain in the cardiac region, sometimes assuming the characters of true angina, cardiac oppression, palpitation and dizziness, dyspnœa and syncopal or even epileptic attacks, may be amongst the earlier symptoms. In the later stages dilatation of the left ventricle takes place and relative mitral insufficiency ensues. Dropsy and the usual evidence of venous stasis set in, and the clinical picture assumes the type seen in the more advanced stages of mitral disease.

The diagnosis rests on the physical signs. The peripheral arteries are usually thickened and calcareous, whilst the characters of the pulse afford valuable evidence of the nature of the lesion. It is small, often of high tension, the upstroke is slow and gradual, the wave is well sustained and it falls away slowly. These features are well shown in a sphygmographic tracing; the upstroke is oblique and is sometimes interrupted by an anacrotic notch, like the dicrotic notch in the down stroke. This notch has been regarded as a double effort on the part of the ventricle during systole, but it occurs in other conditions and has no special diagnostic significance; the apex of the curve is rounded and the downstroke gradual. The pulse rate is often infrequent, falling to 60 or below. (*Vide* Clin. Soc. Trans., 1897.)

Evidence of hypertrophy of the ventricle is present, but the enlargement does not reach the degree seen in aortic incompetence. Inspection and palpation reveal an apex beat below and outside the normal site, with a strong heaving character. Emphysema of the lungs, however, is frequently present and prevents the apex site from being determined, rendering it weak and indistinct. Under these circumstances cardiac dulness is often lessened. A systolic thrill about the aortic cartilage is commonly present; it is often well marked and of great intensity, and is sometimes transmitted to the vessels of the neck, or felt in the episternal notch. With failing cardiac power the thrill may lessen or disappear. On auscultation a rough harsh murmur is heard in the aortic region, occasionally having a musical character. It is prolonged through the whole of systole, often completely obscuring both sounds of the heart, the second being lost from the rigid character of the valves. It is best heard about the aortic cartilage and transmitted to the vessels at the root of the neck. It is heard over the cardiac area with diminishing intensity toward the apex, and in many cases it is so loud that it is heard all over the chest and even at a short distance from the patient. In the terminal stages of the malady, with failing cardiac action, the murmur may lose something of its intensity and roughness.

Diagnosis.—A common error is to regard all aortic systolic murmurs as due to stenosis. Murmurs in this region are most frequently due to atheroma of the root of the aorta, to slight sclerotic changes in the valves, or they are of a functional character. They are then often less loud and rough than in cases of stenosis. Coarse atheromatous changes in the aorta may, however, cause both a rough murmur and a thrill, and it may prove difficult or impossible to distinguish such a condition from true stenosis. An accentuated second sound is common in atheromatous states, and is not likely to be present in stenosis, owing to the rigid state of the valves.

The peculiar characters of the pulse, the marked characters of the murmur, and the presence of thrill form a combination of signs which are highly characteristic, and, when distinct, are unmistakable.

Mitral Incompetence.—This may result from acute or chronic endocarditis, from rupture of the chordæ tendineæ, or from dilatation of the ventricle preventing the apposition of the valve segments.

In young people a large proportion of cases originate in acute endocarditis; hence the frequency with which a history of acute rheumatism occurs in the clinical history. It is by no means infrequent to obtain no history of rheumatic conditions, even in children and young people, whilst in older individuals a chronic sclerotic process is often the primary change.

The mitral valve is much more liable to rheumatic inflammation than the aortic, and in fact the latter valve seldom suffers from this cause without involvement of the former.

In malignant endocarditis, rapid ulceration or necrosis of the valves, or rupture of the chordæ, is occasionally responsible for serious destruction of the valve. Rupture of the chordæ also occurs, apart from acute endocarditis, as a result of strain or external violence, although such cases are very unusual. Cardiac dilatation, resulting from strain or from myocardial changes, often produces relative mitral incompetence, even with perfectly healthy valves. This condition is frequent as a temporary manifestation in the acute dilatation of infectious disease and in anæmia. In aortic lesions and in chronic dilatation or myocarditis, incompetence occurs owing to this mechanism.

Morbid Anatomy.—The changes in old-standing cases consist in thickening and rigidity of the valvular segments. In cases due to endocarditis, there is almost invariably some degree of narrowing. The orifice is naturally wider than normal when relative incompetence is present.

Secondary changes of a compensatory character take place in the various cardiac cavities. The left auricle dilates to accommodate the extra current of blood passing through the leaking valve, and also hypertrophies to cope with the increased work thrown upon it. The ventricle undergoes similar changes, being stimulated by the increased flow from the left auricle. The circulation in the pulmonary circuit is impeded by the overfilled auricle, and in consequence the right ventricle, and ultimately the right auricle, dilate and hypertrophy.

The tricuspid orifice, owing to dilatation, may become incompetent, a feature which accounts for certain clinical phenomena in the advanced stage of the affection. Sclerotic changes occasionally occur both in the trunk and in the smaller branches of the pulmonary artery, owing to the increased blood pressure.

A deposit of fibrin amongst the musculi pectinati or chordæ tendineæ is seen in the dilated cardiac chambers, and forms the usual source of emboli.

Venous stasis in the various organs is frequent in mitral lesions. The lungs are affected earliest, and undergo the change known as brown induration. The capillaries are overfilled and consequently thrown into loops, whilst blood pigment passes to the alveolar walls, and a slight increase of interstitial tissue, the result of chronic congestion, takes place. The epithelial lining in the alveoli is somewhat swollen, and contains specks of pigment.

Hemorrhagic infarcts are common in the advanced stages. They are often attended by hæmoptysis and a localized pleuritis. Œdema of the lungs and hydrothorax are frequent as manifestations of part of a general dropsy, and a terminal broncho-pneumonia is a common cause of death.

The liver shows dilatation of the radicles of the hepatic veins in the centres of the lobules, with an exudation of pigment in the surrounding cells, whilst the periphery of the lobule undergoes fatty changes owing to deficient oxygenation. To the naked eye the cut surface presents a mottled appearance, the dark-colored centres contrasting with the pale peripheral portions of the lobules, and giving the organ the name of the nutmeg liver. When congested it is enlarged and extends below the costal border, and in the later stages fibroid changes may supervene, the organ becoming indurated and uneven on the surface, a condition known as cardiac cirrhosis.

Somewhat similar changes take place in the kidneys; these organs are enlarged and congested in the earlier stages, and eventually may terminate in cirrhosis and contraction.

Œdema and congestion of the gastro-intestinal tract, and subcutaneous œdema with effusion into the serous sacs, are also present in the advanced stages of the affection.

Symptoms.—These vary greatly according to the character and stage of the disease.

In acute endocarditis the development of a mitral incompetence is revealed only by the physical signs; dropsy and dyspnœa are only exceptionally present even in the graver forms of endocarditis, and the clinical picture is that of the primary infectious process. In chronic cases compensation may be maintained for a long period, even when the serious nature of the lesion is shown by a greatly enlarged heart and an apex murmur. Although some individuals pass through a long life with little or no discomfort, it is more usual to find some dyspnœa on exertion. The facial appearance is often suggestive. The lips and cheeks may be of a deeper tinge than usual, the veins of the neck are distended, and there is stellate enlargement of the veinules of the cheeks, and occasionally clubbing of the finger nails. Attacks of palpitation are often induced by exertion, but they may also result from flatulence. A dry hacking cough or recurring attacks of bronchitis are frequently found.

Amongst the graver symptoms are a weak, irregular pulse, dropsy, and increasing dyspnœa. Breathlessness is brought on by even slight exertion, ultimately becoming constant even at rest. The respirations are deep and labored, accompanied by contractions of the extraordinary muscles of respiration, and the patient rests propped up by pillows, or leaning forward in an arm-chair. Attacks of Cheyne-Stokes breathing are often seen, or again paroxysms of dyspnœa, often occurring at night and interfering with rest.

Accompanying the dyspnœa is œdema, which begins about the feet and ankles, noticed first after being up, and disappearing with a night's rest. Unless checked by suitable treatment it gradually increases, and extends to the thighs, scrotum, and serous sacs. The face remains free, but the upper extremities sometimes share in the progressing œdema. The œdema is firm and hard, and is accompanied by coldness and cyanosis of the extremities, and lividity of the face. When the abdomen becomes distended with fluid it still further embarrasses the breathing.

Hydrothorax is usually bilateral, but in some instances occurs chiefly on one side. In some cases the dropsical effusion affects principally the serous sacs, particularly the peritoneum, and requires periodic removal. It sometimes collects with extraordinary rapidity, after a period of absence.

Sleeplessness, starting from sleep, and bad dreams occasionally occur, even apart from the severer symptoms of failure of compensation.

Of the various organs the liver is enlarged and somewhat tender, frequently reaching as low as the umbilicus. A slight tinge of jaundice is very common, and bile may be detected in the urine. The secretion of urine is greatly diminished, sometimes falling to three or four ounces, or even being suppressed. It is of high specific gravity, it deposits a sediment of urates, and shows albumin in small or moderate quantities, and often granular and hyaline casts. With the disappearance of dropsy diuresis sets in, and the urine may run up to two or three times its normal quantity, with disappearance of albumin, and later of casts. Gastric symptoms such as flatulence, and sometimes nausea and vomiting, are not uncommon.

With appropriate treatment dyspnœa and œdema may disappear, and the patient be restored to comparative health. Sooner or later, however, the same symptoms recur, particularly in individuals who are unable to obtain the necessary rest. It is frequent to find several such attacks before the fatal issue.

Eventually a period is reached when the heart, either in consequence of extreme dilatation or of myocardial degeneration, fails to respond to treatment. The terminal illness is frequently very prolonged, and for several months the unfortunate individual struggles with dyspnœa and dropsy, and eventually succumbs to exhaustion.

Physical Signs.—The most characteristic sign is a systolic murmur, heard best at the apex. It is variable in character, being sometimes loud and bellows-like, sometimes having a musical character, or again being extremely soft and distant. Faint systolic murmurs at the apex also occur with slight defects in the valve, and in conditions of temporary dilatation, whilst a loud bellows or a musical murmur usually indicate more definite changes in the valve. The murmur of incompetence may, if faint, be localized at the apex, but it is usually transmitted to the axilla, or heard in the back at or above the angle of the scapula; it is often heard up to the pulmonary cartilage and near to the ensiform, and it may even have its site of maximum intensity at the pulmonary area. The first sound is often obscured or replaced by the murmur, whilst the second sound is accentuated at the pulmonary area. With failing power of the right ventricle accentuation disappears, and is thus of unfavorable omen.

A thrill is of the rarest occurrence in mitral incompetence. When there is any considerable degree of regurgitation there is enlargement of the left ventricle. The impulse is strong and heaving, and lies in a line with, or outside the nipple; a well-marked epigastric pulsation is often present owing to hypertrophy of the right ventricle. As dilatation or weakness supervenes, the impulse becomes weak and diffuse. Fulness and distention are sometimes seen, particularly in the yielding chest walls of children. Percussion shows an increase in dulness to the left and often to the right, reaching the right sternal border or even the parasternal line. Upward, dulness often begins a space higher than normal.

Diagnosis.—It is not uncommon to find a systolic murmur at the apex in the absence of any other sign of cardiac disease. Its chief importance is the mental disquietude it may cause in nervous patients, and the unfavorable light in which it is usually regarded by life insurance companies. Such murmurs are sometimes evidence of slight valvular damage, but in the absence of symptoms and of other signs of disease, such as enlargement and pulmonary accentuation, they may be disregarded. A murmur heard toward the end of inspiration, and attributed to the entry of air into the pulmonary vesicles during systole, is not uncommon, and is known as the cardio-respiratory murmur. It has usually no cardiac significance. It is not always possible to distinguish regurgitation due to a valvular lesion from that of muscular dilatation. In elderly individuals, in Bright's disease or arterial sclerosis, or in anæmia, the latter condition may be suspected. A history of rheumatism, on the other hand, suggests endocarditis. In some instances, as in anæmia and in acute dilatation from strain or infectious fevers, any doubt is often cleared up by the gradual disappearance of the morbid signs.

Mitral Stenosis.—Narrowing of the mitral valve is for the most part a disease of the first half of adult life, often starting from an acute endocarditis in childhood. It is much more common in females, owing doubtless to the greater frequency of rheumatism in young girls than in males of the same age. Although the lesion is often regarded as of purely rheumatic origin, it is remarkable how often a history of acute rheumatism is absent. In children, however, rheumatism, as pointed out by Cheadle, is frequently an ill-marked condition, the articular manifestations being slight, or replaced by such symptoms as tonsillitis, growing pains, or erythema, and yet in these apparently slight cases severe forms of endocarditis may develop.

Cases of mitral stenosis are occasionally found in a first attack of acute rheumatism, a fact strongly suggestive of an old-standing latent rheumatic influence. An absence of a history of acute rheumatism is therefore no evidence of a non-rheumatic origin.

Mitral stenosis is occasionally found in arterio-sclerotic cases and in interstitial nephritis, a fact which has suggested an arterial origin for some of the cases. In 542 autopsies, in cases of interstitial nephritis, Pitt found mitral stenosis 33 times, and other writers have recorded the association of the two conditions.

In most if not all instances of rheumatic endocarditis, there is some narrowing of the mitral orifice, but it is only when this is considerable that the signs become distinct.

Anatomically the valve curtains are rigid, thickened, and adherent, and form a diaphragm perforated by the mitral orifice, which is reduced to a narrow slit-like opening, often termed the "button-hole" orifice. Less commonly the valves project in a funnel-shaped fashion toward the ventricle. The chordæ tendineæ are thickened and adherent, whilst a deposit of calcareous salts is frequent in the valve and even in the chordæ themselves.

The secondary changes are seen first in the left auricle which becomes hypertrophied and often dilated. According to Samway's observations hypertrophy is the primary change, dilatation occurring later. He found that cases of stenosis dying from cardiac failure presented, in addition to hypertrophy, a marked degree of dilatation, whereas those dying from surgical diseases or affections apart from the heart usually showed hypertrophy only.

The state of the left ventricle varies greatly. Theoretically it is argued that the diminished quantity of blood passing through the narrowed orifice cannot induce hypertrophy or dilatation, and that the ventricle remains of normal size or even undergoes a certain degree of atrophy. This view is certainly correct for some cases; at the same time hypertrophy is seen in a considerable proportion of cases, and according to Baumbach, who enters into this question very fully, a moderate degree of hypertrophy is present in almost all cases. Mitral regurgitation may be regarded as responsible for this hypertrophy in some instances, whilst in others there are pericardial adhesions or arterio-sclerosis. The cavities of the right side undergo a marked degree of dilatation and hypertrophy, and the contrast between the enlarged right ventricle and the small left is sometimes very striking.

Symptoms.—Some degree of dyspnœa is almost always present on exertion, and this with palpitation may continue for years. When the disease dates from childhood there is frequently a stunted growth, the chest is ill-developed, and there is diminished physical and mental vigor. The lips are often of a deep blue tinge, and in old-standing cases there is sometimes clubbing of the nails of the fingers and even of the toes.

As compensation fails dyspnœa becomes more and more troublesome, and signs of venous stasis similar to those seen in mitral incompetence are found in the various organs. Pulmonary symptoms are common and these patients are very subject to recurring attacks of bronchitis.

Attacks of hæmoptysis occasionally occur, and, unless due to infarction, often have a beneficial effect, relieving pulmonary congestion and the embarrassed right heart. Other signs of venous congestion are observed in recurring attacks of epistaxis from which some patients suffer, whilst hemorrhoids or menorrhagia occur in others.

Dropsy of the extremities is seldom a prominent feature, and any œdema present is usually slight. Signs of a venous obstruction often fall chiefly or exclusively on the portal system, and hepatic enlargement or even cirrhosis may be present with ascites, in cases which show little or no œdema of the lower extremities.

Welch (Trans. Am. Phys., 1900) has recently called attention to venous thrombosis in cardiac disease, occurring in serious failure of compensation, and more particularly in mitral stenosis. The veins affected are most frequently those conveying blood from the neck and upper extremity, and more commonly on the left than the right side. The peculiar localization of the thrombosis is to be attributed to the greater length and obliquity of the left innominate vein, and to pressure, direct or indirect, on the left subclavian vein from the dilated left auricle and pulmonary veins.

Symptoms of ruptured compensation often first appear during pregnancy or labor, or follow parturition; alarming or even fatal attacks of pulmonary œdema sometimes appear, or persistently rapid action of the heart leading to asystole. Pregnancy is a complication in which there is always serious risk, although in favorable cases there is little or no disturbance. Subsequent pregnancies are sometimes passed without special difficulty, when earlier ones had caused great disturbance.

Hysterical manifestations are not uncommon in the course of mitral stenosis, both conditions being common in young women. Cardiac symptoms often predominate, and severe attacks of pseudo-angina, of palpitation, or of dyspnœa may occur in well-compensated cases. Without a correct appreciation of the neurotic element in such individuals these symptoms may lead to undue alarm.

Embolism is more common in this than in other forms of valvular lesions. If it occurs in the brain this accident shows itself in the form of hemiplegia, and if the embolus lodge on the left side, there will be aphasia.

The physical signs when well marked are extremely characteristic, but in the advanced stage of the disease they are often indefinite or equivocal. The more important are: the presystolic thrill and murmur, the snapping first sound, the accentuated pulmonary second, and evidence of enlargement of the right heart.

The pulse is small, somewhat increased in rate, and often of good tension; it is usually regular until a late stage of the malady, when extreme rapidity and arrhythmia may set in. The thrill, which is one of the most characteristic signs of the disease, is felt at the apex, and when the heart is not unduly rapid it is readily made out distinctly to precede the systolic impulse. It is sometimes prolonged, running through the whole diastolic interval, or it may immediately precede the impulse, and is then strictly pre-systolic. When present it is pathognomonic of stenosis. The cardiac impulse often gives the impression of a jet of liquid being projected against the hand. The murmur in typical cases is loud and rough, and is commonly compared to the sound "rrrb." It runs up to and terminates in a loud, abrupt, and snapping first sound. The murmur may be prolonged through the diastolic interval, but in the great majority of instances it occurs at the end of this period. In either case it is usually spoken of as the pre-systolic murmur. The murmur is in most instances localized at or about the apex, although, as pointed out by Griffiths, it is sometimes transmitted to the axilla or even heard in the back. The murmur is sometimes represented by a mere rumble, and in the absence of the snapping first sound is easily missed by the untrained ear. With advancing cardiac weakness both thrill and murmur often disappear. A systolic murmur is frequently present, due to associated incompetence. It may persist when the thrill and murmur have disappeared.

Hardly less characteristic than the murmur is the abrupt snapping first sound in which it terminates. It is often mistaken for the second sound, but the distinction can be made by noting that it is synchronous with the apex impulse. The second sound is often lost at the apex, and is sometimes reduplicated at this region; in the pulmonary area it is accentuated and even ringing, losing these characters with failing power in the right ventricle. With high tension in the pulmonary artery, a faint, whiffing, diastolic murmur is occasionally heard. This is regarded by Gibson and others as due to a temporary incompetence at the pulmonary orifice.

Evidence of enlargement of the right heart is often present, as shown by the increased impulse over the lower sternal region, and by an extension of dulness to the right border of the sternum or beyond, reaching in extreme cases as far as the right nipple line. With failing compensation and particularly in advanced stages of the disease the thrill and murmur often disappear, and the