Graves' disease, with a heart's action little or not at all accelerated. In a well-marked case of thyroid cachexia, however, the action of the heart is very rapid, amounting to 120 or more beats to the minute. This is often accompanied by a violent throbbing, which extends even to the small arteries. The sounds of the heart are also greatly augmented in volume, being audible in some instances at a distance of several inches from the chest. This peculiar action of the heart differs from simple palpitation by its continuity and by its embracing the small arteries as well as the heart itself in the throbbing. At first there is no structural change in the heart, but the rapid and perturbed action is associated with a falling-off in the ventricular output, and this ultimately leads to residual dilatation with its attendant train of evils. The treatment of the thyroid cachexia is to be directed to the general condition and not to the heart, although a little digitalis combined with Hoffman's anodyne or valerian may be exhibited to mitigate severe throbbing. General tonics, outdoor air, and good food, supplemented by the use of the thyroid or thymus extract, are recognized at the present time as offering the best hope for a modification of the course of this prolonged and obstinate affection.

Angina Pectoris.—This name was introduced by Heberden in 1768. It was intended to denote a strangulation (*angere*, to bind, to strangle). The affection has been known as stenocardia, sternalgia, and breast-pang. There are two varieties, the true, or angina pectoris proper, and the false, or pseudo-angina. True angina is always attended by structural changes and is no more a neurosis than is atheroma or endocarditis. It is usually treated as a neurosis, however, and this is at least partially justified by the fact that the most formidable and striking manifestation of the trouble undoubtedly has its origin in some of the nerve structures communicating with, or located within, the heart. Many authorities have regarded it as a neuralgia. Pseudo-angina is not attended by organic change and is unequivocally a neurosis. The features of false angina are not usually so severe as those of angina vera, but it is not always possible to separate the two. A fatal issue has undoubtedly resulted from pseudo-angina pectoris in some instances.

Causation.—The structural changes which constitute the basis of true angina pectoris do not occur in early life. Thus the affection seldom makes its appearance before the fortieth year of age. All influences which give rise to sclerosis or atheroma of the arterial coats tend to lay the foundation of this dreadful disease. These are notably alcoholic excess, high living, syphilis, gout, lead-poisoning, and overwork, especially of a mental or sedentary character. Probably ninety per cent. of cases occur in males. It is especially prone to appear in persons in the upper walks of life, those of an intellectual occupation being most liable to it. Physicians, clergymen, lawyers, and writers supply the greater number of victims. Heredity plays a rôle in some instances. The paroxysms may be precipitated by exhaustive work or sudden excitement, anger, fright, etc. A patient under the writer's observation suffering from a double aortic lesion developed attacks of unmistakable angina on two successive occasions when taken before a class of students.

Morbid Changes in True Angina Pectoris.—Atheroma of the coronary arteries is the most constant lesion present. Welch found this change in every one of six fatal cases. Embolism of these vessels may also be present, as pointed out by Cohnheim and Virchow many years ago. The process is apt to begin in the ascending aorta at the point of origin of the left coronary artery. The calibre of this vessel may become entirely occluded, or so contracted that a bristle cannot be passed into it. Various other organic lesions have been observed, notably fatty degeneration of the heart, calcification of the aortic valves, aneurism of the aortic arch, pericarditis, and endocarditis. Not every case of coronary sclerosis is accompanied by angina pectoris. The lumina of the vessels are sometimes left sufficiently patent to admit of enough blood, and in other cases the cardiac circulation is carried on by supplemental coronaries which remain unaffected. It will thus be seen that an imperfect metabolism of the myocardium furnishes the groundwork for angina pectoris. No one has ever seen the heart during a paroxysm of angina, but reasoning from analogy it seems probable that the seizures are precipitated by a sudden ischæmia of the cardiac muscles, the blood supply of the coronaries being for the moment totally, or almost totally, arrested. Pseudo-angina may be produced by any of the causes which have been enumerated as producing other cardiac neuroses. It is in greater or less degree an occasional concomitant of those affections. Probably "angina dyspeptica," a mere gaseous dilatation of the stomach, constitutes a majority of the cases of pseudo-angina pectoris in nervous subjects.

Symptoms.—The paroxysm may set in suddenly without previous warning, although the marks of arterio-sclerosis and general failure of the health and strength may have shown themselves for some years. Cardiac palpitation, arhythmia, etc., may also have been experienced. The attack may come on when the patient is sitting quietly in bed, but is most likely to appear during some exertion, such as climbing a hill, breasting the wind, or straining at stool, or when laboring under some form of excitement. In true angina the seizure is of overwhelming suddenness and of unspeakable intensity. The pain begins in the præcordial region, usually about the mid-sternum, and radiates as a rule upward into the left shoulder and down the left arm, perhaps as far as the finger ends. Sometimes it is propagated in the direction of the right shoulder, or possibly downward toward the leg or the lumbar region. The nervous system is acutely alert. One of the most characteristic features is the feeling of imminent death. Some patients state that the heart feels as though it were being tightly squeezed in the grasp of a gigantic hand. Probably no condition in the entire range of our knowledge entails more dreadful agony than that of a typical attack of true angina. In some cases a sense of coldness, numbness, deadness, creeping, or other vaso-motor disturbance is felt. In others the pain may not be so acute, but takes the form of a dull ache (angina sine dolore) and is overshadowed by the intense anxiety. During the attacks of angina the features are haggard and the face is blanched; the forehead is bedewed with a cold sweat. The respiration, as a rule, is in nowise affected but may be hurried and superficial. The patient is well able to take a deep breath, but is afraid to attempt it. He usually prefers to keep perfectly quiet in a sitting or upright posture for fear of increasing the severity of the paroxysm. A physical examination may or may not show the signs of organic heart disease; the most frequent lesion is aortic stenosis. During the attack the heart's action may be increased or diminished in frequency. In one of the writer's patients it sank below 40 per minute. The blood pressure is always raised at the beginning of the attack. The seizures may last for from a few moments to an hour or more, probably five or ten minutes being about the average. After the attack, which may cease as suddenly as it set in, the patient feels weak and exhausted for a short time. A sensation of numbness and formication in the arms may be experienced. There may be a considerable belching of gas and voiding of a large quantity of urine.

Diagnosis.—This is usually not difficult. No other condition presents the same complex of symptoms—sudden irradiating pain, squeezing, tightening, constriction, overwhelming sense of impending death, etc. As distinguishing between true and false angina, Huchard's aphorisms, while not infallible, are very important: (1) Every angina produced by effort is a true angina. (2) Every angina which occurs spontaneously, without effort, is a false angina. (3) But an angina occurring at night, though independent of effort, is a true angina. Disturbance of the special senses, hysterical manifestations, etc., are far more characteristic of false than of true angina. The absence or presence of signs of organic heart disease is important in the diagnosis. The hysterical pseudo-angina of young women is excluded by the sex and age of the patient, the ability to scream and throw herself about, and the probable presence of anæmia or menstrual disorders. The pseudo-angina of alcohol and tobacco occurs as a rule in young persons and can generally be recognized by the history of the case. The pain here is of subordinate severity. In cardiac asthma dyspnœa is a marked symptom, but is entirely absent in angina pectoris. Then in cardiac asthma are superadded the symptoms of blood stasis in the extremities, cold hands and feet, hyperæmia of the liver, and occasionally the expectoration of blood-stained sputum.

Prognosis.—In true angina the prognosis for complete recovery is hopeless, the anatomical lesions not admitting of restoration. The patient is in constant danger. He may die in the first attack, or after several attacks. Unexpected prolongation of life occurs in some cases. The patient may not have a repetition of the early attacks and may possibly die of other troubles. A case reported by Murrell lasted forty years, and the elder Austin Flint believed that he had seen a case of actual recovery.

Treatment.—This resolves itself into the management, first, of the paroxysm, second, of the interval. When called to see a case during an attack the practitioner should always feel the pulse; this will generally but not invariably be found hard and tense, denoting high arterial pressure, which condition is usually present at the beginning of an attack. The first object of treatment is to lower the blood tension and relieve the strain on the heart. For this purpose the nitrite of amyl is still our most prompt and efficient weapon. This drug may be administered by inhalation, a few drops being placed on a handkerchief and held to the nose, increasing the dose carefully if necessary. Small pearls or capsules may be supplied to the patient with instruction to break and inhale in the event of an attack. Nitroglycerin acts in a similar manner to nitrite of amyl but is much slower. Its effects, however, are more lasting, usually extending over a period of from four to six hours. It is very useful in mild cases of vaso-motor angina and in pseudo-angina. The nitrite of sodium has an effect quite similar to that of nitroglycerin. If the arterial tension be found low and the pain still severe it is better to try the administration of a little chloroform or ether by inhalation, the former being, in the writer's opinion, most trustworthy. These agents must always be used by the physician or a trained attendant. A hypodermic injection of morphine is sometimes demanded. Yeo recommends the continuous current at the inception of a paroxysm. Mild cases may be relieved by antispasmodics and diffusible stimulants such as Hoffman's anodyne, musk, valerian, and assafœtida. Sinapisms or hot-water bags to the surface are sometimes helpful, and the same may be said of the quick application of the ice bag or cold cloths. During the intervals the underlying pathological state—arterio-sclerosis—should be combated by every available means. The iodide of potassium, strychnine, and arsenic form our main reliance. According to Balfour, arsenic is indicated in all heart diseases attended with pain. A most careful and discriminating mode of life is to be observed. All undue exposure and excitement should be avoided and the strictest self-control practised.

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HEART DISEASES: SYPHILIS.—Syphilis of the myocardium manifests itself in the form of a local or diffuse fibroid myocarditis or as a gummatous myocarditis. The interstitial induration is by far the more common process; it may be the result of the direct action of the syphilitic poison or secondary to a syphilitic endarteritis. It occurs in both acquired and congenital syphilis, in the former case as a tertiary lesion. In its earliest stages the interstitial change is found as a cellular infiltration along the small vessels of the intermuscular connective tissue. Fibroblastic proliferation occurs in the latter, leading gradually to the formation of small scattered nodules of granulation tissue, which soon become converted into scar tissue. In the fibroid areas thus formed remains of degenerated muscle, pigment, fat droplets, etc., are found. The newly formed fibrous tissue may undergo caseation or become calcified. The neighboring muscle shows extensive atrophy and fatty degeneration. In other cases the cellular infiltration is in the form of bands or sheets running parallel with the axis of the muscle bundles. These may or may not be sharply defined; usually the new tissue sends out fine prolongations into the neighboring muscle. As the cellular infiltration develops it becomes gradually changed into a coarse hyaline variety of fibrous tissue containing few nuclei. Through atrophy of the intervening muscle the bands of connective tissue gradually coalesce and may come to make up the entire thickness of the heart wall from pericardium to endocardium. The left ventricle, the anterior wall, the apex, and the septum ventriculorum are most commonly affected, the papillary muscles and the walls of the auricles only rarely. In the congenital form the wall of the right ventricle is much more frequently affected than that of the left.

In the majority of cases the fibroid change is directly dependent upon the presence of an obliterating endarteritis in the coronary arteries. Complete occlusion of a terminal branch produces anæmic infarction, the necrosed muscle being replaced by scar tissue; slowly progressive obliteration of the vessels leads to degenerative changes in the muscle with secondary inflammation and proliferation of the interstitial tissue. To the naked eye the fibroid areas appear as white, glistening, tendon-like bands or masses. The weakening of the heart wall, due to the inability of the new connective tissue to contract against the intracardiac pressure, often leads to an aneurismal dilatation at the point of greatest change.

The gummatous form of myocarditis is of much less common occurrence. It is usually associated with a more or less diffuse interstitial change. It is probable that in many cases the fibroid myocarditis found at autopsy is the result of antecedent gummatous processes. Gummata may be found in any part of the heart wall, in the myocardium, or beneath the peri- or endocardium. To the naked eye they appear as yellowish or grayish cheesy nodules, varying in size from a mustard seed to a walnut, and are usually surrounded by a dense fibrous capsule. Fresh gummata are red or pink in color and of a soft homogeneous structure. They do not possess a fibrous capsule and are not sharply outlined from the surrounding tissue. Usually the heart gummata are solitary, but in many cases multiple caseous nodules are found scattered throughout the cardiac wall. When the gumma is located just beneath the peri- or endocardium chronic adhesive pericarditis and sclerosis of the endocardium are usually present. Hypertrophy and dilatation

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