There are cases in which it is often difficult to decide whether malignant endocarditis is present or not. Thus, a patient with aortic valve disease is under treatment for failing compensation and begins to have irregular fever with restlessness and cardiac distress; embolic phenomena may develop—sudden hemiplegia, pain in the region of the spleen, or bloody urine, or perhaps peripheral embolism. There may be a low delirium and the case may run a tolerably acute course; but in other instances the fever subsides and recovery occurs.

In what may be termed the *cerebral group* of cases the clinical picture may simulate a meningitis, either basilar or cerebro-spinal. There may be acute delirium or, as in three of the Montreal cases, the patient may be brought into the hospital unconscious. Heineman reports an instance, with autopsy, in which the clinical picture was that of an acute cerebro-spinal meningitis.

Certain special symptoms may be mentioned. The fever is not always of a remittent type, but may be high and continuous. Petechial rashes are very common and render the similarity very strong to certain cases of typhoid and cerebro-spinal fevers. In one case the disease was thought to be hæmorrhagic small-pox. Erythematous rashes are not uncommon. The sweating may be most profuse, even exceeding that which occurs in phthisis and ague. Diarrhœa is not necessarily associated with embolic lesions in the intestines. Jaundice has been observed and cases are on record which were mistaken for acute yellow atrophy.

The heart symptoms may be entirely latent and are not found unless a careful search be made. Even on examination there may be no murmur present. Instances are recorded by careful observers, in which the examination of the heart has been negative. Cases with chronic valve disease usually present no difficulty in diagnosis.

The course of the disease is varied, depending largely upon the nature of the primary trouble. Except in the disease grafted upon chronic valvulitis the course is rarely extended beyond five or six weeks. As already mentioned, there are instances in which the disease is prolonged for months. The most rapidly fatal case on record is described by Eberth, the duration of which was scarcely two days.

Diagnosis.—In many cases the detection of the disease is very difficult; in others, with marked embolic symptoms, it is easy. From simple endocarditis it is readily distinguished, though confusion occasionally occurs in the transitional stage, when a simple is developing into a malignant form. The constitutional symptoms are of a graver type, the fever is higher, rigors are common, and septic and typhoid symptoms develop. Perhaps a majority of the cases not associated with puerperal processes or bone disease are confounded with typhoid fever. A differential diagnosis may even be impossible, particularly when we consider that in typhoid fever infarctions and parotitis may occur. The diarrhœa and abdominal tenderness may also be present, which with the stupor and progressive asthenia make a picture not to be distinguished from this disease. Points which may guide us are: The more abrupt onset in endocarditis, the absence of any regularity of the pyrexia in the early stage of the disease, and the cardiac pain. Oppression and shortness of breath may be early symptoms in malignant endocarditis. Rigors, too, are not uncommon. Between pyæmia and malignant endocarditis there are practically no differential features, for the disease really constitutes an *arterial pyæmia* (Wilks). In the acute cases resembling malignant fevers, the diagnosis is usually made of typhus, typhoid, cerebro-spinal fever, or even of hæmorrhagic small-pox. The intermittent pyrexia, occurring for weeks or months, has led in some cases to the diagnosis of malaria, but this disease could now be positively excluded by the blood examination.

The cases usually terminate fatally. The instances of recovery are those more subacute forms, the so-called recurring endocarditis developing on old sclerotic valves in cases of chronic heart-disease.

Treatment.—We know no measures by which in rheumatism, chorea, or the eruptive fevers the onset of endocarditis can be prevented. As it is probable that many cases develop, particularly in children, in mild forms of these diseases, it is well to guard the patients against taking cold and insist upon rest and quiet, and to bear in mind that of all complications an acute endocarditis, though in its immediate effects harmless, is perhaps the most serious. This statement is enforced by the observations of Sibson that on a system of absolute rest the proportion of cases of rheumatism attacked by endocarditis was less than of those who were not so treated.

It is doubtful whether the salicylates in rheumatism have an influence in reducing the liability to endocarditis. When the endocarditis is present we know no remedies which will definitely influence the valvular lesions. If there is much vascular excitement aconite may be given and an ice-bag placed over the heart.

The salicylates are strongly advised by some writers and the sulphocarbolates have been recommended by Sansom. In the severer cases of malignant endocarditis the treatment is practically that of septicæmia.

Chronic Endocarditis.

This condition, which is a sclerosis of the valve, may be primary, but is oftener secondary to acute endocarditis, particularly the rheumatic form. It is essentially a slow, insidious process which leads to deformity of the valve segment and is the foundation of chronic valvular disease.

Certain poisons appear capable of initiating the change, such as alcohol, syphilis, and gout, though we are at present ignorant of the way in which they act. A very important factor, particularly in the case of the aortic valves, is the strain of prolonged and heavy muscular exertion. In no other way can be explained the occurrence of so many cases of sclero-

sis of the aortic valves in young and middle-aged men whose occupations necessitate the overuse of the muscles.

Morbid Anatomy.—Vegetations in the form in which they occur in acute endocarditis are not present. In the early stage, which we have frequent opportunities of seeing, the edge of the valve is a little thickened and perhaps presents a few small nodular prominences, which in some cases may represent the healed vegetations of the acute process. In the aortic valves the tissue about the corpora Arantii is first affected, producing a slight thickening with an increase in the size of the nodules. The substance of the valve may lose its translucency, and the only change noticeable is a grayish opacity and a slight loss of its delicate tenuity. In the auriculo-ventricular valves these early changes are seen just within the margin and here it is not uncommon to find swellings of a grayish-red, somewhat infiltrated appearance, almost identical with the similar structures on the intima of the aorta in arterio-sclerosis. Even early there may be seen yellow or opaque-white subintimal fatty areas. As the sclerotic changes increase the fibrous tissue contracts and produces thickening and deformity of the segment, the edges of which become round, curled, and incapable of that delicate apposition necessary for perfect closure. A sigmoid valve, for instance, may be narrowed one fourth or even one third across its face, inducing the most extreme grade of insufficiency without any special deformity and without any definite narrowing of the arterial orifice. In the auriculo-ventricular segments a simple process of thickening and curling of the edges of the valves, inducing a failure to close without forming any obstruction to the normal course of the blood-flow, is less common. Still, we meet with instances at the mitral orifice, particularly in children, in which the edges of the valves are curled and thickened, producing extreme insufficiency without any material narrowing of the orifice. More frequently, as the disease advances, the chordæ tendineæ become thickened, first at the valvular ends and then along their course. The edges of the valves at their angles are gradually drawn together and there is a definite narrowing of the orifice, leading in the aorta to more or less stenosis and in the left auriculo-ventricular orifice—the two most frequently involved—to constriction. Finally, in the sclerotic and necrotic tissues lime salts are deposited and may even reach the deeper structures of the fibrous rings, and the entire valve becomes a dense calcareous mass with scarcely a remnant of normal tissue. The chordæ tendineæ may gradually become shortened, greatly thickened, and in extreme cases the papillary muscles are implanted directly upon the sclerotic and deformed valve. The apices of the papillary muscles usually show marked fibroid change.

In all stages of the process the vegetations of simple endocarditis may be found and upon sclerotic valves we find the severer, ulcerative form of the disease.

Chronic *mural* endocarditis produces cicatricial-like patches of a gray-

ish-white appearance which are sometimes seen on the muscular trabeculæ of the ventricle or in the auricles. It often occurs in association with myocarditis.

The frequency with which chronic endocarditis is met with may be gathered from the following figures: In the statistics, amounting to from 12,000 to 14,000 autopsies, reported from Dresden, Wurzburg, and Prague the percentage ranged from four to nine. The relative frequency of involvement of the various valves is thus given in the collected statistics of Parrot: The mitral orifice was involved in 621, the aortic in 380, the tricuspid in 46, and the pulmonary in 11. This gives 57 instances in the right to 1,001 in the left heart.

The endocarditis of the fœtus is usually of the sclerotic form and involves the valves of the right more frequently than those of the left side.

The effects of sclerotic endocarditis are practically those of chronic valvular disease, and the general influence on the work of the heart may be briefly stated as follows: The sclerosis induces insufficiency or stenosis, which may exist separately or in combination. The narrowing retards in a measure the normal outflow and the insufficiency permits the blood current to take an abnormal course. In both instances the effect is dilatation of a chamber. The result in the former case is an increase in the difficulty which the chamber has in expelling its contents through the narrow orifice; in the other, the overfilling of a chamber by blood flowing into it from an improper source, as, for instance, in mitral insufficiency, when the left auricle receives blood both from the pulmonary veins and from the left ventricle.

The cardiac mechanism is fully prepared to meet ordinary grades of dilatation which constantly occur during sudden exertion. A man, for instance, at the end of a hundred-yard race has his right chambers greatly dilated and his reserve cardiac power worked to its full capacity. The slow progress of the sclerotic changes brings about a gradual, not an abrupt, insufficiency, and the moderate dilatation which follows is at first overcome by the exercise of the ordinary reserve strength of the heart muscles. Gradually a new factor is introduced. The reserve power which is capable of meeting sudden emergencies in such a remarkable manner is unable to cope long with a permanent and perhaps increasing dilatation. More work has to be done and, in accordance with definite physiological laws, more power is given by increase of the muscles. The heart hypertrophies and the effect of the valve lesion becomes, as we say, *compensated*. The equilibrium of the circulation is in this way maintained.