

comes clear that should there arise either an alteration in relative length, as, for instance, in dilatation of the left ventricle, or increased difficulty in impulse passage, in one or more portions, as in toxic heart block or in bundle branch lesions, that the usual balance will be disturbed. What may be expected to follow as the result of such disturbances is an alteration in the relative value in time of the two monophasic waves we have been considering. Should the impulse pathway be altered so that the impulse reaches the second earlier than the first, the inverted spike is the form we should expect.

If the formation and form of the first ventricular waves depend on forces such as have been described, it follows that the actual weight of the muscle which constitutes the walls of the ventricles and enters the septum need not have altered to produce change. But we are not yet in a position finally to adopt this view.

Finally, it may be urged that the fundamental changes in the electrocardiogram have been explained as due to alterations in relative weights on the basis of a crude technic. The theory has been that the muscle which confines each ventricular cavity is an entity. We know, however, from the work of MacCallum and of Mall, that this arrangement does not represent the facts, although for rough purposes it serves in most instances. Therefore an elaboration of the weight theory, rather than the introduction of a new hypothesis, may be required, weight being taken in terms of the layers shown by MacCallum and Mall to exist, rather than weight in terms of the cavities' walls. I have attempted to correlate electrocardiograms with the weights of the layers in such dissections, but on account of the difficulty of the technic have not been able to come to a conclusion of the study.

It appears, then, that the electrocardiogram, usually so constant a physical phenomenon, depends on factors scarcely understood. The form of the curve is explained as depending on the algebraic sum of electrical potentials arising on muscular activity in different portions of the ventricles. These may depend primarily on activity above and below a horizontal line through the heart or to right and left of a vertical one. Both views have been suggested, and these are probably capable of being harmonized. But it is at this point that our difficulties begin. On both sides of either of these lines, what are the factors that determine the value of the potential generated? They may, as we have noticed, be altered by a variety of agents,—by nerve stimulation, by the application of heat and cold and chemicals in experiment, by drugs and means apparently physical in human beings. Are these factors which determine changes, the weight of the muscle, its tension, or its ultimate chemical relations? So far the only investigation attempted relates to muscle weight. Studies in other directions have been suggested, but so far the problem is still open to further study.

ACUTE CARDITIS.

BY FRANK TAYLOR FULTON, M.D., PROVIDENCE, R. I.

IN the consideration of the subject of acute carditis it is my purpose to limit the discussion chiefly to the condition as met with in the course of acute rheumatic fever. Commonly, in referring to an acute infection involving the heart, if there is a friction rub one speaks of acute pericarditis. If, perchance, a murmur and other signs develop, one characterizes the condition as acute endocarditis; but the intimate relation between the pericardium, myocardium, and endocardium is such that there is not likely to be a very active infection of any one of them without, to a certain extent, having involvement of one or both of the others. A consideration of the anatomy and of some of the well-known acute cardiac pathology will further convince us that the heart musculature should receive more attention in acute infection than we have been in the habit of giving it.

As you may know, Aschoff and Tawara,¹ stimulated by the desire to discover the reason for the comparative feebleness of hypertrophied heart muscle, examined a large number of hearts very carefully, the result in the majority of cases being negative. In a certain number of cases of chronic valvular disease supposedly of rheumatic origin, they did discover certain inflammatory changes characterized by the presence of what has been called submiliary myocardial nodules. Coombs² has since, in corroboration, given a particularly thorough and detailed account of these nodules and their distribution. They are inflammatory in nature—rather smaller than a miliary tubercle, with a groundwork of fibrin in which are various types of cells, some of them leukocytes, some of them plasma cells, some of them endothelial cells, but most important and characteristic of all, very large multi-nucleated cells, these latter of varying size and shape. There is no absolute regularity of distribution, but they are found more commonly in the perivascular tissue, especially in the walls of the left ventricle, but there is some evidence that the region about the central fibrous body is very liable to be invaded. The muscular part of the interventricular septum and the papillary muscles are only rarely involved, and the auricular muscle almost never. They are in the subpericardial tissues, and are quite likely to be present in the fibrous tissues of the valves, most often mitral, but sometimes the aortic and tricuspid. They are more abundant in valves that have been vascularized and deformed from former inflammatory attacks. The evidence seems to be that they always arise in the connective tissue, that they are of short duration, that they may come in successive groups, and that they may vanish into scar tissue.

The question has continually arisen as to whether these so-called Aschoff bodies are characteristic of rheumatism or whether they are

present in association with other infections. Without entering into the discussion, I may say that the weight of opinion and of evidence is that they do develop only as a result of the infection which we have long known as acute rheumatic fever. They have been found in association with chorea, a point which is brought forward to prove the intimate relationship believed to exist between chorea and rheumatism.

Another result of the work of Aschoff and Tawara was the demonstration in detail of the conducting system of the heart. As you know, the muscle bundle of His has long been recognized as the path for the conduction of the impulse between the auricle and ventricle. Tawara found that the main trunk of this bundle passed along below the membranous portion of the interventricular septum, and that just above the muscular portion of this septum it divided into two branches, one going to the left ventricle and the other to the right. He further found that these main branches subdivide into small branches, and ultimately into minute ramifications which are distributed throughout the inner surfaces of the right and left ventricle, being continuous with the so-called Purkinje net-work. I mention this conducting system in some detail because it is of fundamental importance in understanding some of the damage which may be done by a myocardial infection. You can easily see by referring to the specimen which I now show you how readily an acute infection involving the muscle of the heart might encroach upon the system at some point or points and interfere materially with its action. In fact, Aschoff³ makes the following statement:

"Now we do find not infrequently, especially in cases of rheumatic myocarditis, actual destruction of the smaller and larger branches of the conducting system, the already described specific rheumatic nodular structures having a special tendency to develop beneath the endocardium and occasionally right in the connective tissue sheaths of the system. Such inflammatory multiplication of cells finally destroy the muscle fibers of the conducting system."

When this inflammatory process is particularly active in the region of the main stem of this bundle, the function of the bundle may be seriously interfered with or, for a time, entirely suspended. Normally there is an appreciable time consumed in the passage of the impulse through the main stem of the bundle. The maximum normal time in the passage is about one-fifth of a second. This is indicated in the difference of time in the contraction of the auricle and the ventricle, and can be very readily measured, either by the polygraph or by the electrocardiograph. Whenever there is an increase in the time interval between the contraction of these two cavities, we know that there is an impairment of the conductivity of the bundle.

Formerly, heart-block was considered chiefly

in connection with chronic heart disease. It is now recognized as one of the important complications of an acute inflammatory condition of the heart. It may occur with many of the acute diseases, and in acute rheumatic fever it is not at all uncommon. Its recognition is of prime importance; for, if we detect its presence, we have then direct evidence that the myocardium is involved. It may occur at any time during the course of acute rheumatic fever, and may be the only evidence which can be obtained that the heart is actually attacked, for the toxic effect of the infection may temporarily give rise to much the same cardiac symptoms as though the myocardium were actually invaded. Moreover, it may be the first sign to suggest that the infection is of the rheumatic type. White⁴ has only recently reported a case of a boy of 18 who came to the out-patient department, apparently suffering from an acute cold of four days' duration, associated with cough and coryza. At that time he had an irregular heart due to acute heart-block, caused by acute myocardial infection. He subsequently ran a typical course of acute polyarticular rheumatism. The writer has recently observed a case in a young physician, an intern in a hospital, who did his hospital work up to the time of the onset of heart-block. The first symptom which he noted that seemed of any consequence was an irregular heart action, associated with a sense of pressure over the precordium. This was found to be due to partial heart-block. The irregularity persisted for one day only. Electrocardiographic records were taken daily and the conduction time, which was at first double that of normal, returned to normal within a week. Rapid heart action with some cardiac enlargement persisted for a good many weeks. There was at no time any fever. Another case observed by the writer was that of a young man of thirty, who three weeks after an acute tonsillitis was admitted to the hospital almost *in extremis*, with acute cardiac dilatation. His heart action was very rapid, but regular. About two weeks afterwards, he developed an irregular pulse which was demonstrated to be heart-block. The irregularity persisted for about two days only. The conduction time was increased for some time. The exact date of its return to normal was not observed, but it was normal three weeks later. The temperature in this case was 104 on admission. It dropped to normal on the following day and was not above normal again, although the heart-block did not develop until two weeks later. In these two cases the diagnosis of rheumatism is not entirely clear. Considering all the evidence, it is fair to conclude that they were caused by the same infecting agent which is the cause of acute rheumatic fever.

So much for the consideration of the effect

of an acute infection upon the conducting mechanism of the heart. There is, however, still remaining a large amount of the myocardium which may be involved without this infection being manifested by any disturbance of the heart rhythm.

Coombs⁵ has written rather extensively on the subject of rheumatism, and divides the cases of rheumatic infection in which there are serious heart complications into four groups. In the first and more important group are those cases in which there is general enlargement of the cardiac area with signs of mitral insufficiency; second, those in which the same signs are present, associated with pericarditis; third, those in which there is associated some evidences of valve deformity, either aortic incompetency or a stenosis of the mitral valve; and, fourth, a group rarely represented, characterized by a malignant endocarditis. His studies had largely to do with children, and of these, about three-quarters of the patients belonged in the first group; that is, those in which there is a marked increase in the area of cardiac dullness, and a well-defined systolic murmur. The evidence, according to him, is that the insufficiency which is present under these circumstances is relative, and is due to the dilatation of the left ventricle; and it is fair to assume that this dilatation is due to the inflammatory reaction in the muscle. It is scarcely conceivable that the small vegetations which are frequently observed at autopsy on mitral valves in such cases can have much, if any, influence upon the formation of such a murmur. In two of Coombs' cases which came to autopsy there was considerable enlargement and dilatation. There was a systolic bruit and a sharp diastolic shock felt over the pulmonic area with the accentuation of the pulmonic second and reduplication of the second sound at the apex. In these, there was not the slightest evidence of endocarditis or pericarditis. Briefly stated, his belief—in which the writer concurs—is that a large percentage of cases have myocarditis without either pericarditis or endocarditis that is *demonstrable*. He believes that the cause of death in rheumatic disease, before the age of sixteen, is more often due to acute inflammation of the myocardium than to anything else, and even above this age, it often plays a very important part.

A word with reference to the late sequelae of an acute cardiac infection. It is a difficult matter to estimate the amount of permanent damage which has been done either to the valves, the heart muscle proper, or to the conducting system. Lewis is of the opinion that an ordinary attack is usually only one of a number of steps which lead to the crippling of the heart, and that it is by oft-repeated damage that the incurable rheumatic hearts are produced. However, there is a good deal of presumptive evidence that, after the valve is once damaged, there is a progressive change which leads gradually in later years to those serious valve de-

formities and insufficiencies which are so commonly met, although there is no question but that hearts with repeated infections are far more likely to fail rapidly. It is certainly true that a valve can never be returned to normal after having become deformed. It is reasonable that the same opinion should be held with regard to the involvement of the conducting system. This involvement may be in the main stem, in either of the two main branches, or in the smaller branches. It may be transient, lasting but a few days, or it may be permanent; it may be a complete destruction of the bundle, or a slight local inflammatory swelling or edema; it may be recognized with fair certainty, on physical examination, by noting the dropped beats if they occur, and with absolute certainty by polygraphic or electrocardiographic tracings. Generally it is rather transient, but its duration may vary a great deal. If produced by some edematous swelling, it is conceivable that it would last only for a day or two; but if produced by inflammatory infiltration, the condition of the block may last for several weeks; or, at any rate, the facility with which the impulse is conducted may be impaired for a considerable period, and possibly permanently.

In acute rheumatism where there is slight impairment of conduction, if digitalis is given, the degree of impairment is likely to be increased and a serious heart-block may occur. On that account one should always be cautious about the use of digitalis in acute rheumatism.

Most cases of auricular fibrillation fall into one of two main groups: one group includes those occurring in elderly people and such cases are of the cardio-sclerotic type; the other group includes those cases which occur in younger people, usually associated with mitral disease, and usually with a history of rheumatism or chorea.

It is well known that the most striking effect of digitalis in slowing the heart rate has been in cases of auricular fibrillation. It is also well known that digitalis reduces the heart rate in these cases by its action upon the conducting system, reducing its conductivity so that fewer impulses are transmitted to the ventricle. It is further known that the bundle once damaged is distinctly more susceptible to the action of digitalis than is the normal bundle, and it is not at all unlikely that in many cases of rheumatic infection, the conductivity of the bundle is permanently impaired. It has been found to be a general rule, however, that the sclerotic cases are not well controlled by digitalis, but that the cases associated with mitral disease with a history of rheumatism or chorea are particularly susceptible to digitalis. It is quite likely that the efficiency of the drug is to some extent due to the previously existing impairment of the conductivity of the bundle.

It is now generally recognized that in acute rheumatic fever there is usually some primary focus of infection aside from that of the joints or the heart. This may

be in the tonsils, in the teeth, or in some other locality. If that focus persists, it is, of course, important to discover it and, if possible, remove it. Whether it is advisable to do this during an acute febrile attack may be questionable, unless there is a prolonged persistence of the symptoms. Absolute rest in bed is imperative. How long the patient should be kept at absolute rest is often very difficult to decide. It is much better to make the mistake of having too much restraint than of giving too much freedom. I think it is quite certain that the majority of cases are allowed to get up too soon. The patient should be confined to bed until there is every evidence that the heart has returned as nearly as possible to its normal condition. During this time the patient should have plenty of fresh air and should be given a well-regulated diet of sufficient caloric value. In no other condition is judgment and tact more needed than in handling these cases. A patient suffering from acute myocardial infection, well treated, may recover so as to live an active, useful life. If badly treated, however, his whole future may be greatly modified by a seriously crippled heart.

REFERENCES.

- ¹ British Medical Journal, Oct. 27, 1906.
- ² Journal of Pathology and Bacteriology, April, 1911.
- ³ *Ibid.*
- ⁴ Amer. Jour. of Medical Sciences, Oct. 1, 1916.
- ⁵ Quarterly Journal of Medicine, October, 1908.

DISCUSSION.

DR. SAMUEL A. LEVINE, New York: My remarks will be necessarily scattered, touching on a point or two brought out in the papers here.

The first thing that occurs to one is, what has electrocardiography taught the medical profession? I think it may be summarized in the statement that it has given us a clearer conception of the heart mechanism. It has taught us a great deal about the irregularities of the heart. We understand them; how often they occur, their clinical significance, and in many instances we know what the future course of events is apt to be, as a result of a certain arrhythmia. That part of the work is almost complete. But the work that Dr. Cohn has mentioned and Dr. Williams has spoken about is a line that may be very fruitful, namely, changes in the form of the curve. The changes in the form of the curve may give us indications as to what is going on in the heart muscle that we cannot get any other way, because, as is needless to say, the electrocardiogram is a delicate representative of the heart mechanism, of something that is going on in the heart, and may be changed by disease. As we will understand the forces that control the form of the curve more thoroughly, we will understand the aberrations of the heart mechanism, and this will help in an early diagnosis of chronic myocarditis.

The recent work done by Oppenheimer and Rothchild of New York is very suggestive, as they have shown that certain changes in the electrocardiogram are frequently present in chronic myocarditis, *i. e.*, the senile heart. So it seems that changes in the

form of the curve may be of increasing significance as time goes on.

Dr. Christian spoke about the importance of the history and symptoms in appraising what a heart is doing, and I just wish to emphasize that point. A systolic murmur with a history of rheumatic fever means a great deal more than a systolic murmur without a history of rheumatic fever. A diastolic murmur, as he said, in civil life is significant. Generally it means organic heart disease. Some French military men have recently found soldiers who have had normal hearts, develop diastolic murmurs in the pulmonary area from the strain of war life, who have not had heart disease, and they attribute it to temporary dilatation of the pulmonary ring. That may be of some importance to those of us who meet soldiers with diastolic murmurs in the pulmonary area and with no other symptoms of heart disease.

Dr. White of the Massachusetts General Hospital has found pulsus alternans with surprising frequency in various heart cases, and we all know that pulsus alternans when present is of very grave prognostic significance. The duration of life, on the average, is from six months to a year. Not infrequently one can detect pulsus alternans while taking the blood pressure. I thought it would be worth while to mention this because we do not all know about taking tracings and we do not all have the means of taking them. While recording the blood pressure as the mercury falls from 180, at 160 only every second beat might come through, that is, the stronger beat of the alternating pulse, and as the mercury falls to 150, the other or weaker beat comes through as well. The difference of 10 millimeters between the strong and the weak beat may become evident in taking the blood pressure and one will be thrown on one's guard as to the presence of alternation in this way. That is particularly true in the cases of angina pectoris, where the main symptom may be pain. I remember a case of that type in a man where the pulsus alternans became evident very readily while taking the blood pressure.

The importance of the increase of the conduction time, that is, so-called P-R interval in electrocardiography, comes out in a series of cases which one might call acute polyserositis. There are quite a few cases of acute rheumatic fever which run a course as follows: First, the synovial membranes of the joints are affected, then the synovial membrane around the heart, and very frequently the pleura as well. One has therefore an acute arthritis, acute pericarditis and acute pleuritis. In fact, if one remembers this symptom-complex, one can frequently tell what has happened and what will happen in some of these cases. You may hear a friction rub over the pericardium; then if you listen below the left scapula, you will find bronchial breathing. Sometimes a few hundred cc. of fluid may be removed from the left pleural cavity. These cases always show a lengthening in the time it takes for the impulse to come from the auricles to the ventricles. Lengthening of the P-R interval is an absolute indication of a toxic process going on in the heart muscle, and that lengthening may persist for two weeks or three weeks after the symptoms of rheumatic fever have disappeared. If one appreciates the significance of this, he can see that such a patient has an acute process of the myocardium and, as Dr. Fulton has emphasized, needs a more careful and more prolonged rest treatment than others