

ELECTROCARDIOGRAPHIC EVIDENCE OF MYOCARDIAL INVOLVEMENT IN RHEUMATIC FEVER.

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PLATES 1 TO 3.

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It was Wells (1), a native of South Carolina, who recorded the circumstances in which rheumatism came to be associated with the development of heart disease. According to Wells, Dr. David Pitcairn was the first to point out this relation. He began to be interested in and to talk about it at St. Bartholomew's Hospital as early as 1788. Baillie (2)¹ appears to have been the first to refer to this teaching of Pitcairn's. Stimulated by Pitcairn's observations and Baillie's reference, Wells published² an account, illustrated by case reports, of rheumatic heart disease which leaves little doubt that he appreciated the connection between the two conditions. Later, after the introduction of the method of auscultation, it became possible during the life of patients to ascertain whether the rheumatic process involved the cardiac valves. It was probably³ Dr. James Hope (3) who realized (1832) this possibility. Among the exciting causes of diseases of the valves, he specified "inflammation of the internal membrane of the heart, resulting from carditis, pericarditis—especially rheumatic, from fever or from any other cause."⁴ That the heart muscle and the pericardium were in all likelihood also part of the process was in general assumed, but it remained for Aschoff and Geipel to demonstrate that rheumatic fever is accom-

¹ Baillie (2), p. 46.

² By this time accounts based on Baillie's report had been published also by Odier and by Dundas.

³ There is an interesting controversy concerning this subject to be found in the 3rd edition of Hope's book, and in Bouillaud's *Traité* (1835).

⁴ Hope (3), p. 319.

panied by a specific lesion and that this lesion is to be found in the heart muscle of patients dying of the disease. In the course of years this association has become so well recognized as to require no further emphasis.

Whether heart disease is likely to become established in an individual suffering from rheumatic fever, is still impossible to say during the course of the acute stage of the infection. There are no criteria for arriving at a judgment, even in the case of apparent affection of the cardiac valves. It often requires, indeed, the passage of long periods of time, sometimes of years, before the issue becomes apparent. The making of prognoses rested on no consideration beyond the state of the valves until 30 years ago, when the functions of the heart muscle came to be analyzed in detail. Since then the number of functions and mechanisms that can be distinguished and recognized has increased and has made possible the description of kinds of derangement not possible at an earlier period. In terms of these more newly described mechanisms observers now recognize injury to the heart not only at earlier stages in the course of several diseases, but have come also to describe types of injury hitherto quite unsuspected.

We have utilized the methods more recently introduced for investigating the effect on the heart of the process of rheumatic fever. The function of the heart to which we paid attention especially during this study is that of conduction between the auricles and the ventricles. But we have also utilized the fact that the general form of the electrocardiogram is, relatively speaking, constant in normal persons over prolonged periods of time. Under these circumstances, the occurrence of deviations from the contour of the usual curve becomes a sign of value. For the inference is clear that if the outline of the curve changes an alteration must have occurred in the heart to bring about this result. These signs, conduction time and curve form, have been employed before now as indications of change in the behavior of the heart, not only in rheumatic fever, but in other conditions as well.

As the result of a number of reports it is now well known that disturbance of conduction, and especially of heart block, occurs in the course of rheumatic fever (4). It may indeed be the first sign to

indicate the presence of the disease (5). Heart block has been observed at various stages in the febrile portion of its course and not uncommonly in the post febrile and convalescent periods. How often transient disturbances of conduction have led to permanent heart block and to heart failure in the later stages of the clinical history is not known. The interval may be brief, for occasionally (6), even in the early period of the rheumatic process, involvement of the heart leads promptly to death.

In the present enquiry we were not concerned with the problems of the late history of cases of cardiac involvement, but rather with the frequency with which the heart shares in the rheumatic process. We have made a systematic study in 37 patients.⁵ The changes we have studied are chiefly, as has been said, of two sorts; first, alterations in the duration of the auriculoventricular conduction interval, and second, alterations in the form of the ventricular portion of the electrocardiogram.⁶ Changes in the mechanism of the heart beat were observed in a number of patients. On these we will comment incidentally.

The cases are divisible into three groups; first, 17 cases preponderantly arthritic in type in the first attack of the disease; second, 12 cases also preponderantly arthritic but which had suffered at least one attack before coming under observation; and 8 cases suffering from the recurrent cardiac type of the affection. Although the last group of cases is classified as cardiac, arthritis was a feature in each case at some time during the period of observation. The changes we describe were not associated with the exhibition of drugs, such as digitalis, salicylates, or neocinchophen, except in those instances to which special reference is made.

The conduction time (auriculoventricular, P-R interval) was taken as normal for each individual when for a number of days in a febrile or

⁵ Curves were taken of the same patient as often as every 24 hours; in some cases at intervals somewhat longer. 2,591 curves were taken in all.

⁶ The events in which we were interested have been charted in a condensed form so that changes in the behavior of these functions might be correlated. Of the data recorded, only the temperature, the rate of the heart beat, and the conduction time are described in this report. The method of charting consists in joining, by vertical lines, the highest and lowest figures for the day. The upper limit of normal temperature is taken to be 99.6°F.

post febrile period, a constant level was maintained or when on subsequent examinations after discharge from hospital, this figure was obtained from electrocardiograms. It is of course not impossible that even with these precautions the normal conduction time for a given person was not obtained; the possibility of doing so may have disappeared with the onset of the disease.

Change in the duration of auriculoventricular conduction was a frequent, almost a constant occurrence; but we have noticed that the alterations, although frequent, did not run a course parallel with or corresponding to the temperature. We noticed in one patient (Text-fig. 1) in whom the conduction time was 0.24 second on the 16th day of disease when the temperature was 103.5°F., that 4 days later, when the temperature had fallen to 100°F., a marked increase in its duration took place to the extent that dropped beats (incomplete heart block) were observed. At that time she was taking neocinchophen. It is inferred that neocinchophen was not responsible for this occurrence because of the fact that later, although the exhibition of the drug was continued, the conduction time fell to limits which, for this patient, were considered normal. Parkinson (4) has made similar analyses of the relation of antirheumatic drugs to cardiac arrhythmias. When the temperature rose subsequently to 101° there was no further alteration in the conduction time. In another patient (Text-fig. 2) on the 6th day of disease, when the temperature was about 103.5°, the conduction time was within limits usual for him. Subsequently (24th day of disease) during a period of falling temperature the conduction time rose to 0.20 second. And later still (43rd day of disease) during an irregular febrile movement the conduction time attained its greatest elevation. Similar relations or lack of correlation were seen in the case of other patients (Text-fig. 3).

A close correspondence between these curves was seen, however, not infrequently. In one patient (Text-fig. 4) even when the elevation in temperature was slight, an increase in the duration of conduction was observed (end of March, beginning of May and June). In the May attack, when the conduction time was the longest recorded, the temperature reached only about 100.2°. At the same time there was, however, a burst of rapid heart action. Two other attacks of tachycardia were seen in this patient, each time associated with a

period of prolonged conduction time. In still another patient (Text-fig. 5) the correspondence in behavior of the two curves was striking, especially for about the first 100 days of the disease. Later, with or without fever, there was more or less continuous prolongation of the conduction rate.

These cases illustrate the changes to which we wish to call attention. In the 37 cases now reported we encountered it 31 times altogether, 24 times when fever was present, 15 times when it was absent. In 5 patients it was found in both associations. In 8 patients alteration in conduction did not end with increase in the P-R interval, but went on to the stage in which ventricular beats occasionally dropped out or to incomplete heart block (Tables I, II, and III).

The alteration which we have described does not include prolongation of conduction in the two main branches of the auriculo-ventricular system or its subdivisions. We do not distinguish between defects in the main divisions (bundle branch) and defects in the terminal arborizations (arborization block). Of such alterations we saw three examples. These were all transient.

The Q R S Period.—We describe next alterations in the ventricular portion of the electrocardiogram. Of these we distinguish three main varieties; first, that involving the Q R S complex; second, that involving the R-T period; and third, that affecting the T wave itself. Of changes which occur with respiration, for example, in the height of the R (or S) wave in Lead III, we have numerous examples. To these we wish merely to refer. They occur naturally when the direction of potential is nearly perpendicular to a lead or when, in the rotation of the heart in the course of contraction, a new electrical potential is projected on the frontal plane. A change of a different nature is seen in another series of curves (Fig. 1). The curve taken on October 3, 1922, may be taken as a control. In the curve taken on June 2, the S wave in Lead III, usually absent, is much increased, while the R wave, usually prominent, is decreased. There is, moreover, a notch in the R waves. Transient changes like this are probably uncommon and are probably significant. We attach no especial importance to changes which affect the amplitude of the waves, for rather striking changes are not infrequent even in quite normal

persons. Apparently when the R waves in Lead III are split, there is the greater tendency to take on these variant forms.

A change in the R waves of much interest is one which we observed in two cases. In one case (Text-fig. 6 and Fig. 2) it consists of an

TABLE I.
Cases in the First Attack of Rheumatic Fever Showing Especially Polyarthritiis.

Case No.	Sex and age. yrs.	Increase in P-R time.*		Change in Ekg.	Irregularities.	Valvular disease.	Pericarditis.†	Subcutaneous nodules.‡
		Febrile.	Non-febrile.					
1†	F. 17	+	0	+	I.H.B.; A.P.C. blocked.§	?	+	0
2	M. 21	+	+	+	V.P.L.	?	0	0
3	F. 30	+	0	+		M.I.**	0	0
4	F. 30	0	0	+	S.I.; V.P.L.	M.I.	0	0
5	M. 31	0	0	0	V.P.L.; S.I.	0	0	0
6	M. 14	0	+	+		M.I.	0	0
7	M. 19	+	+	+		?	0	0
8†	M. 26	+	0	+	V.P.L.	?	0	0
9	F. 30	0	+	+	S.I.; V.P.L.	?	0	0
10	M. 33	+	0	+	A.F.; V.P.L.; V.P.C.L.	M.I.	0	0
11	M. 17	0	+	+	A.P.C.	?	0	0
12†	F. 31	+	0	+	A.F.; A.Fl.; I.H.B.; V.P.L.; V.P.C.R.; I-V Block.	?	+	0
13	M. 23	0	+	+	I.H.B.; A.P.C.	?	0	0
14	M. 14	+	+	+	A.P.C.; V.P.C.R.	M.I.	+	0
15	F. 25	0	+	+		?	0	0
16	M. 19	+	+	+	I.H.B.; P.C.	?	0	0
17	F. 29	+	+	+		M.I.	0	0

* An increase of 0.02 second or more.

† In this and the following two tables + represents the presence of this lesion.

‡ Indicates special charts.

§ The abbreviations in this column are arranged alphabetically. A.F. indicates auricular fibrillation; A.Fl., auricular flutter; A.P.C., auricular premature contractions; I.H.B., incomplete heart block; I-V Block, intraventricular block; P.C., premature contractions; S.I., sinus irregularity; V.P.C.L., ventricular premature contractions left; V.P.C.R., ventricular premature contractions right; V.P.L., ventricular preponderance left.

|| Indicates in this and the two following tables that the presence of valvular disease is doubtful.

** In this and the two following tables M.I. represents mitral insufficiency.

TABLE II.

Cases of Recurrent Attacks of Rheumatic Fever Exhibiting Especially Polyarthritits.

Case No.	Sex and age. <i>yrs.</i>	Increase in P-R time.*		Change in Ekg.	Irregularities.	Valvular disease.	Pericarditis.	Subcutaneous nodules.
		Febrile.	Non-febrile.					
18	M. 14	0	0	+		+† M.I.; M.St.	+	0
19	F. 38	+	0	+	A.F.; A.P.C.; +P-R; I.H.B.; V.P.R.; V.P.C.R.‡	+ M.I.; M.St.; A. rough.	+	0
20	M. 12	+	0	+	S. I.	+ M.I.	0	0
21	F. 19	+	0	+	V.P.C.R.; V.P.L.	+	+	+
22	M. 14	+	+	+	A.P.C.; Atyp. A.P.C.	+ M.I.; A.I.; A. rough.	0	0
23§	M. 8	+	0	+	V.P.C.; I.H.B.; A.P.C.; A- V.P.C.; V.P.C.L.	+ M.I.; M.St.	0	+
24	M. 22	0	0	+	V.P.L.; A.P.C.	+ M.I.; A.I.?	0	0
25	F. 22	0	+	+	V.P.L.? A. Par. Tachy.; S.I.; Atyp. Ekg.; transition to normal.	?	0	0
26§	M. 52	+	0	+	A-V Rhythm; A. Par. Tachy.; V.P.L.	? M.I.	0	0
27	M. 15	+	0	+	V.P.C.R.; V.P.R.	+ M.I.	0	0
28	M. 15	+	+	+	A-V Rhythm; runs of nodal P.C.; A-V.P.C.	+ M.I.	0	0
29	M. 17	+	0	0		+ M.I.; A.I.?	0	0

* An increase of 0.02 second or more.

† In this and the following table M.St. represents mitral stenosis; A. rough., aortic roughening; A.I., aortic insufficiency.

‡ For abbreviations in this column see Table I. Additional ones are given as follows: + P-R, prolonged auriculoventricular interval; A-V.P.C., auriculoventricular premature contractions; A.Par.Tachy., auricular paroxysms of tachycardia; Atyp. Ekg., atypical electrocardiogram; A-V Rhythm, auriculoventricular rhythm; V.P.R., ventricular preponderance right.

§ Indicates special charts.

TABLE III.

Cases of Rheumatic Fever Predominantly Cardiac but Exhibiting Also Arthritic Involvement.

Case No.	Sex and age. <i>yrs.</i>	Increase in P-R time.*		Change in Ekg.	Irregularities.	Valvular disease.	Pericarditis.	Subcutaneous nodules.
		Febrile.	Non-febrile.					
30	M. 13	+		+	V.P.L.; V.P.C.R.; +P-R.; I.H.B.; Atyp. Ekg.†	+ M.I.; M.St.	+	+
31	M. 8	+ 0	+	+	V.P.L.; Tachy.	+ M.I.; A.I.; A. rough.	0	0
32	M. 14	+		+	V.P.L.; I-V Block; A.F.? Tachy.	+ M.I.; A.I.; A. rough.	0	0
33	M. 11	+	0	+	V.P.R.	+ M.I.; A.I.?	+	0
34	M. 7	0	0	+	A.P.C.	+ M.I.; A.I.?	0	0
35	F. 9	0	+	+	V.P.R.; S-A Block; I.H.B.; V.P.C.R.; V.P.C.L.; A.F.	+ M.I.; M.St.	+	+
36‡	F. 17	+		+	+P-R; A-V Rhythm; Atyp. Ekg.	+ M.I.; A.I.; Tr. I.; § Tr. St.	+ M.I.; A.I.; Tr. I.; Tr. St.	0
37	F. 41	0	0	+	A.P.C.; A-V.P.C.; S.I.; A-V Rhythm; I-V Block; Interpolated V.P.C.R.; A-V S-A Rhythm.	+ M.I.; A.I.?	0	0

* An increase of 0.02 second or more.

† For abbreviations in this column see Tables I and II. Additional ones are given as follows: S-A Block, sinoauricular block; A-V S-A rhythm, auriculo-ventricular and sinoauricular rhythm.

‡ Indicates special charts.

§ Tr. I. indicates tricuspid insufficiency; Tr. St., tricuspid stenosis.

|| This measurement did not increase even in an attack of influenza.

interruption of the down stroke of the R wave in Lead I (January 31) and the beginning, immediately at the point of interruption, of a wave convex upward occupying the whole R-T period. In its usual form, the foot points of the R wave are to be found on almost the same abscissa. This deformity persisted for several days; later (March 2) the shape of the wave returned to the initial state. A change of a similar nature was found in the second patient in Lead III, on December 1 and 10 (Fig. 3). In this case, the prominent phase of the Q R S complex was the S wave. The up stroke of this wave overshot the level of the foot point of the R wave, so that the R-T period lies at a level considerably higher than that of the P-R interval or of the diastolic (T-P) period.

The significance of this phenomenon is by no means clear. Similar changes have been noticed in conditions other than rheumatism. It has been described by Pardee (7) as evidence of blocking of a branch of the coronary artery. In other cases of coronary thrombosis Wearn (8) failed to find this sign or a sign which Pardee described as its sequel. It is, however, not remarkable that appearances like this are found. Their discovery should be expected in several circumstances for the form of the curve is probably not characteristic of the nature of the injury, but is the result rather of any injury involving a given area. The contour of the curve probably is an indication of disturbance of certain functions of the muscle, and is perhaps dependent on the site in the heart wall at which the injury has taken place.

It is important to notice that these disturbances are transient and indicate the fleeting nature either of the injury or of its effect. They are entirely comprehensible in cases of rheumatic fever in which the ephemeral character of certain of its manifestations, whether in the joints, the skin, or in the other tissues which are frequently involved, is well known. The inference it seems to us is, therefore, justified that the nodule described by Aschoff, which is its basic lesion in the heart, and perhaps the ischemic areas consequent upon blood vessel involvement, underlie the electrical disturbance to which the injured muscle gives rise. As to how extensive these lesions need be in order to bring about this result or in what situation in the heart they must be located are points on which we can make no

precise report although this subject has been the occasion of much speculation.

Similar alterations though less striking were observed in 15 other patients. In one of them (Text-fig. 7, Rate 85) the change took place in Lead III, at a time when a reduction, in Lead I, of the height of the R wave and a lengthening of the S wave took place.

The R-T Period and T Wave.—Numerous changes were observed in the later portion of the ventricular electrocardiogram, after the termination of the Q R S complex. This period from the end of the R wave or S wave to the end of the T wave may be divided into two portions; the first portion terminates at the onset of the T wave; the second portion is occupied by the T wave itself. Alterations may be distinguished which involve these segments either separately or together.

In the usual approximately normal electrocardiogram, there follows at the conclusion of the R wave or S wave, either a short isoelectric period or perhaps more frequently still, a period in which the curve rises, the rate of rising being very slow. When the direction of this portion of the curve changes and becomes steeper, the R-T period may be said to end and the T wave proper to begin. In a set of curves taken of the same individual, just as is usually the case in respect to other parts of the curve, there is striking uniformity in the general shape not only from day to day but, indeed, over long periods of time. In many of the patients, however, uniformity gave place to diversity; in some cases the level of the R-T period alters in respect either to a line joining the foot points between successive P waves or to the level of the P-R interval, so that this level (of the R-T period) lies either higher or lower than in other curves. There is often an isoelectric period of prolonged duration, or the direction of the curve instead of being horizontal may slope upward or downward, or may slope first in one and then change rapidly and slope in another direction. Alterations so begun may be prolonged beyond the point at which the T wave proper may be expected to begin; that is to say, the T wave may actually be deformed by the process (Text-fig. 8, *a*, Rate 93). In certain of these instances it is possible to show that the T wave itself occupies its anticipated position in the cycle as can be demonstrated by identifying its terminal

portion (Text-fig. 8, *a*, Rate 93). In other cases the R-T period is not strikingly involved in the deformity; the T wave alone undergoes change such as inversion (Text-fig. 9). Alterations in the terminal portion of the T wave probably also occur. From the point of view of the relation of the excitation and contraction processes of the heart, it would be important to know whether there is a parallel alteration in the Q R S complex and the T wave; that is to say, alteration dependent on the order of excitation of the ventricular fractions, and alteration in the end-phase of the T wave, connected with the order of release from the contractile state. The contour of the T wave is, however, not sufficiently complex to afford criteria for judging this matter. There are probably cases in which both processes are involved.

DISCUSSION.

Although increase in the length of auriculoventricular conduction accompanies, or is the result of a variety of processes, it is not a phenomenon found in constant association with other infectious diseases to the extent in which it is in rheumatic fever. In lobar pneumonia, it was found rarely in the course of the febrile movement, with the exception of those cases in which digitalis or an allied drug was administered. In the course of influenza, bronchopneumonia, diphtheria, and typhoid fever, cases of heart block have, however, been published. Increase in the length of the conduction time is not, in short, associated specifically with rheumatic fever.

It has been supposed that increase in the length of the P-R interval was due to elevation in cardiac rate. Waller (9) thought exercise might bring on this result. There is doubt, however, as to the correctness of his observation. In a more exact study of the effect of work and elevated heart rate on auriculoventricular conduction, Lewis and Cotton (10) failed to corroborate his result. The contrary is indeed the fact; the P-R time often shortens just at the cessation of exercise. In further studies Parkinson and Drury (11) found first, shortening, not lengthening, when the rate rose, in cases of soldier's heart even though there was a history of rheumatism; and second, a similar shortening in other men in whom the rate was high at rest and who had taken tincture of digitalis. Fever may

have an effect on rate similar to work, for in lobar pneumonia (12) elevation in rate is almost constantly found, while increase in auriculoventricular conduction is relatively speaking infrequent.

In the measurements made of the length of the auriculoventricular interval, variations were found, as has been said, frequently. The variations occurred in the same individual, not only in the same curve, but also from day to day. The precaution was taken to make the measurements in Lead II. These variations were, we presumed, due to the rheumatic process. What variations to expect in normal persons we did not know. We have accordingly studied the behavior of this portion of the curve in 6 normal persons. Electrocardiograms were made synchronously of the 3 leads, using 3 galvanometers. The respiratory movements were inscribed on the curves

TABLE IV.

*The Duration of the Auriculoventricular Interval in Three Normal Persons in Inspiration and Expiration.**

Case No.	Inspiration.		Expiration.		Difference between average inspiration and expiration.
	Average.	Greatest range.	Average.	Greatest range.	
1	0.1369	0.0203	0.1302	0.0361	0.0067
2	0.1932	0.0214	0.1862	0.0152	0.0070
3	0.1507	0.0674	0.1395	0.0476	0.0112

* The measurements were made by means of a Lucas comparator.

according to a method described elsewhere (13). In the case of these normal persons we obtained data showing the variations which occurred from day to day and also the difference which took place between inspiration and expiration (Table IV).

The range in the first two cases is 0.02 second in inspiration; and 0.03 and 0.015 second in expiration. In Case 3, the range is much greater, 0.0674 second in inspiration and 0.0476 in expiration. This range (Case 3) is wide and far greater than one would expect if one used our entire experience as the basis of judgment. The reason for it must be quite exceptional. On the whole, the average of the inspirations is a trifle greater than that of the expiration. On any given day one measurement (inspiration) may be greater than that of the other or the same. That there is a daily variation is in itself

a matter of interest. These figures furnish information on the degree of fluctuation which the factors that are concerned in the production of these events experience. Among these are such factors as vagus tone, and the presence of an inflammatory lesion. It is not surprising, therefore, in the light of these data, to find differences from day to day in the curves of rheumatic patients. The daily records did not of course include curves of respiration; but even if they had, considerable difference in the same phase of breathing, as we have shown, would have been found. In the light of these facts we are, it seems to us, justified in placing significant changes in conduction at a figure at or above 0.02 second.

It is not the purpose in this paper to discuss the question whether it is possible from studying these curves to conclude which portion of the heart is involved in the rheumatic process; nor whether the nature of the process is toxic or structural; nor whether the alterations are specific for rheumatic fever; nor whether the injury to the heart indicated by the alterations in the curves is transient or permanent. To us it appears that, for the moment, none of these questions is solvable. The alterations found in this series of curves have, however, led us to investigate the constancy of the electrocardiogram in a single person. For this reason we have examined series of 8 to 10 curves taken over periods of about 14 days of the 6 persons already mentioned, with the view to ascertaining how uniform over limited periods of time the electrocardiogram of a single person actually is (13). In each series taken from day to day alterations were in fact found. But they were slight, quantitative in nature, not qualitative such as those one recognizes as quite different and distinct forms.

If the conclusion is correct that the form of the electrocardiogram does not change either in conduction or in contour in normal individuals in the sense in which it does in rheumatic fever, it is probable that a process of some nature is operative in the heart of individuals infected with rheumatic fever. Whether the alteration in the heart to which the rheumatic process gives rise is specific either in the nature of the lesion or process, or in its location is at present a matter of secondary importance to us. It is our purpose now to indicate the frequency of its presence. In other infectious diseases such as lobar

pneumonia it is already known that changes in the curves do not take place; certainly not with the frequency with which they have been found in rheumatic fever. As a result of this study it is possible to emphasize the fact that in rheumatic fever we possess a means of knowing whether, and when, the heart is involved in the process. That the heart may become involved is a fact already well known. Further study will show whether information, obtained by this means represents changes in the heart of significance for prognosis.

TABLE V.
*Summary of Cardiac Irregularities.**

Premature contractions	1
Auricular premature contractions	10
Ventricular premature contractions, right	7
" " " left	3
Auriculoventricular premature contractions	2
Interpolated ventricular contractions	1
Sinoauricular block	1
Auriculoventricular block	8
Intraventricular block	3
Auricular fibrillation	5
" flutter	1
Auriculoventricular rhythm	4
Auricular paroxysms of tachycardia	2

* If the entire material had been searched, a greater number of instances of irregular heart action would undoubtedly have been found. But even this figure would not give an accurate account of the incidence; more than were photographed must have occurred.

It is unnecessary to report in detail the irregularities that were observed in the course of this study. A summary of them is given in Table V. We wish to point out that the number of cases of heart block, that is to say cases of extreme affection of the auriculoventricular system, was relatively speaking infrequent compared with the number of times this structure was found to be affected. Cases of transient auricular fibrillation were also encountered; in one of these we obtained a record of the transition to the normal mechanism.⁷

⁷ Cohn, A. E., *Irregularities*, Nelson, Loose-leaf medicine, New York, 1920, iv, 329, Fig. 102.

Aside from the variety of irregularities, the number of cases in which they were observed, and the frequency with which they occurred in the same case, are striking. We point to these occurrences only to emphasize the fact that they represent contributory evidence of the fact of how often the heart is affected by the rheumatic process.

SUMMARY.

These observations then show that in one way or the other the heart was affected in 35 of 37 cases of rheumatic fever. The evidence was of three sorts; first, the duration of auriculoventricular conduction was increased, though not always, and usually not to the degree of causing heart block; second, alteration in the ventricular complex of the electrocardiogram affecting in certain instances the Q R S complex and in others either the interval between R wave and T wave, or the T wave itself; and third, the occurrence of numerous irregularities in cardiac rhythm.

These signs are characteristic of cardiac involvement, but are not specific for rheumatic fever; that is to say, from their presence rheumatic fever cannot be diagnosed; all of them express merely derangement of the heart. They are signs which are, however, to be taken as evidence that the heart is affected by the rheumatic process even though an inference cannot be drawn as to the nature or the permanence of the injury. This may be slight and altogether transient even if the sign appears to be of advanced degree. The facts brought forward show that it is possible during the course of the disease to know whether the heart is involved in the general process. When sufficient data have been accumulated over a sufficiently long period of time, the usefulness for prognosis of observations like these will become established.

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Explanation of Text-Figs. 1-6.

Each day is represented by one vertical column.

The maximum and minimum temperature for a day is represented by the upper and lower end, respectively, of the line in the temperature chart; hence, the range of temperature for a day is indicated by a single line. All temperatures are taken by rectum, hence 99.6°F. is considered normal.

Range of pulse for a day represented in a similar manner.

Conduction time range represented by a vertical line and the upper point joined by a light line to show the variation from day to day.

Degree of arthritis: Each block represents one joint; a solid block indicates marked signs of inflammation; a dot, slight signs.

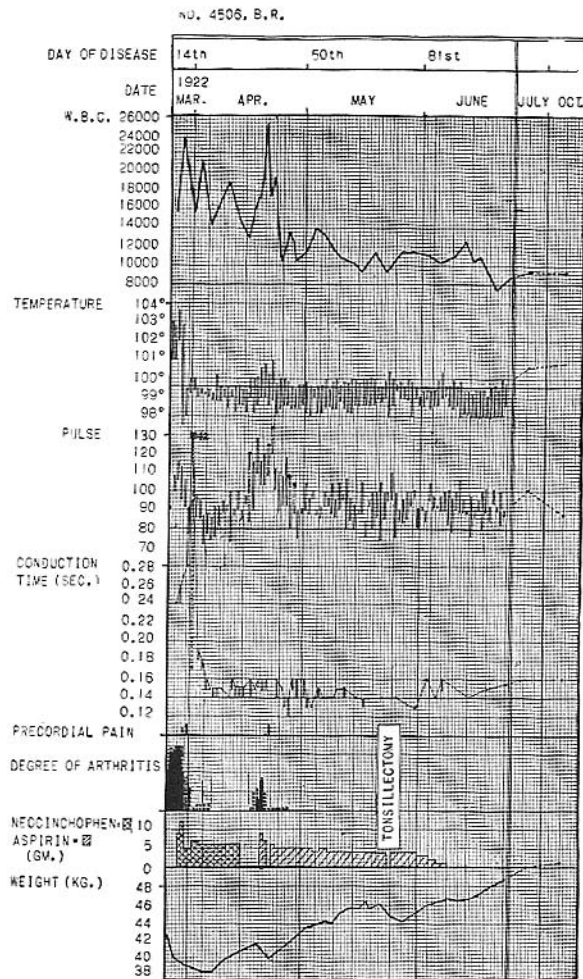
Precordial pain and tenderness: The length of a line indicates the degree of intensity of the respective symptom for 1 day.

Drugs: Sodium salicylate, aspirin, cinchophen or neocinchophen—each small square represents 1 gm. Digitan—each small square represents 0.1 gm.

W. B. C.: Range of leucocytosis.

Weight curve is self-explanatory.

Solid lines indicate observations made while patient was in the wards. Broken line indicates observations made at the time of return follow-up visits.



TEXT-FIG. 1.

No. 4506.—B. R., female, age 17 years.

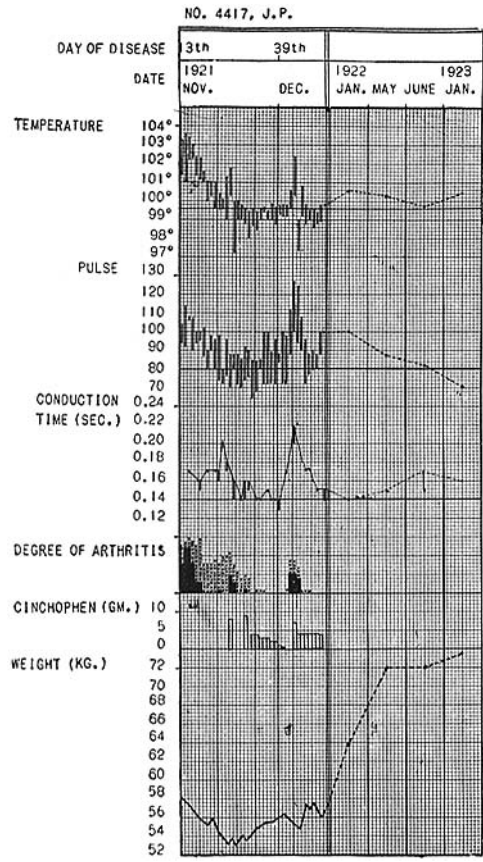
Admitted Mar. 26, 1922. Discharged June 24, 1922.

First attack of rheumatic fever.

Present Illness.—Began 13 days previously with anorexia, malaise, loss of weight, and pain in ankles; 4 days ago arthritis began to migrate to knees; 2 days ago, to arms.

Physical Examination.—Marked, extensive polyarthritis. Heart markedly irregular with groups of coupled and tripled beats. Apical murmur not transmitted.

Course.—(Text-fig. 1.) Day following admission regular cardiac rhythm with marked gallop character; prolongation of P-R time. Spread of polyarthritis and further increase of P-R time. Rapid disappearance of arthritis and fever following the administration of neocinchophen; but P-R time increased; partial heart block developed on the 4th day of administration; a transitory rub over the precordium. Drug continued; conduction time normal on the 25th day (Apr. 6); gallop rhythm disappeared. Continuing leucocytosis indicated a persistence of the infection in spite of lack of symptoms. Neocinchophen discontinued; a definite relapse of polyarthritis, fever, increase of heart rate, and gallop rhythm 2 days afterward. Symptoms and signs disappeared following large doses of aspirin; but the drug was continued for 50 days because of the persistent leucocytosis. Patient recovered with only persistent apical systolic murmur not transmitted; no new murmurs nor signs of cardiac insufficiency.



TEXT-FIG. 2.

No. 4417.—J. P., male, age 26 years.

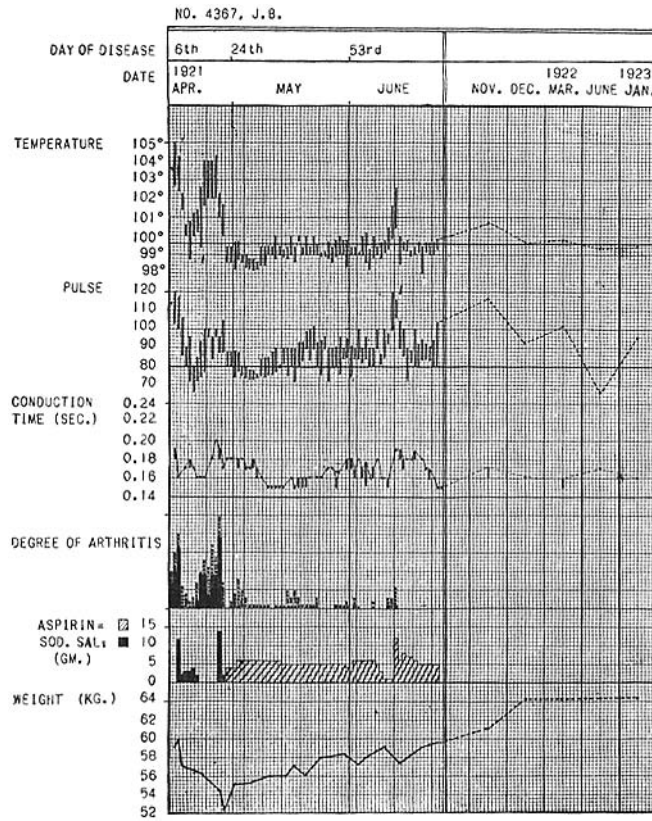
Admitted Nov. 5, 1921. Discharged Dec. 13, 1921.

First attack of rheumatic fever.

Present Illness.—3½ weeks before admission, sore throat, fever, malaise, followed by a cough, for 3 days. 12 days ago, polyarthritis and fever began and lasted 5 days. Following exertion, polyarthritis and fever recurred; 2 days before admission he was somewhat better.

Physical Examination.—Mild pharyngitis, extensive polyarthritis. Heart not enlarged. Soft precordial and apical systolic murmurs not transmitted.

Course.—(Text-fig. 2.) Without drugs, fever, pulse rate, toxicity, and polyarthritis diminished for 9 days; then a relapse occurred. Polyarthritis and fever preceded by an increase in P-R time (Nov. 24). Large doses of cinchophen followed by marked relief. Dose reduced and then discontinued; definite relapse followed consisting of fever, pharyngitis, polyarthritis, and a much higher pulse rate. P-R time again prolonged; precordial burning sensation during one night. Cardiac murmur no longer heard. Fever and symptoms again responded to cinchophen. Murmurs did not reappear. Patient left hospital against advice. Small doses of aspirin at home; no more relapses. No symptoms of cardiac insufficiency nor signs of valvular disease since recovery.



TEXT-FIG. 3.

No. 4367.—J. B., male, age 18 years.

Admitted Apr. 6, 1921. Transferred to Presbyterian Hospital, June 25. Discharged Sept. 11, 1921. Readmitted Apr. 18, 1923.

Previous Rheumatism.—First attack of rheumatic fever with migratory polyarthritis 4 years ago. Second attack 3 years ago; heart involved then. No cardiac insufficiency.

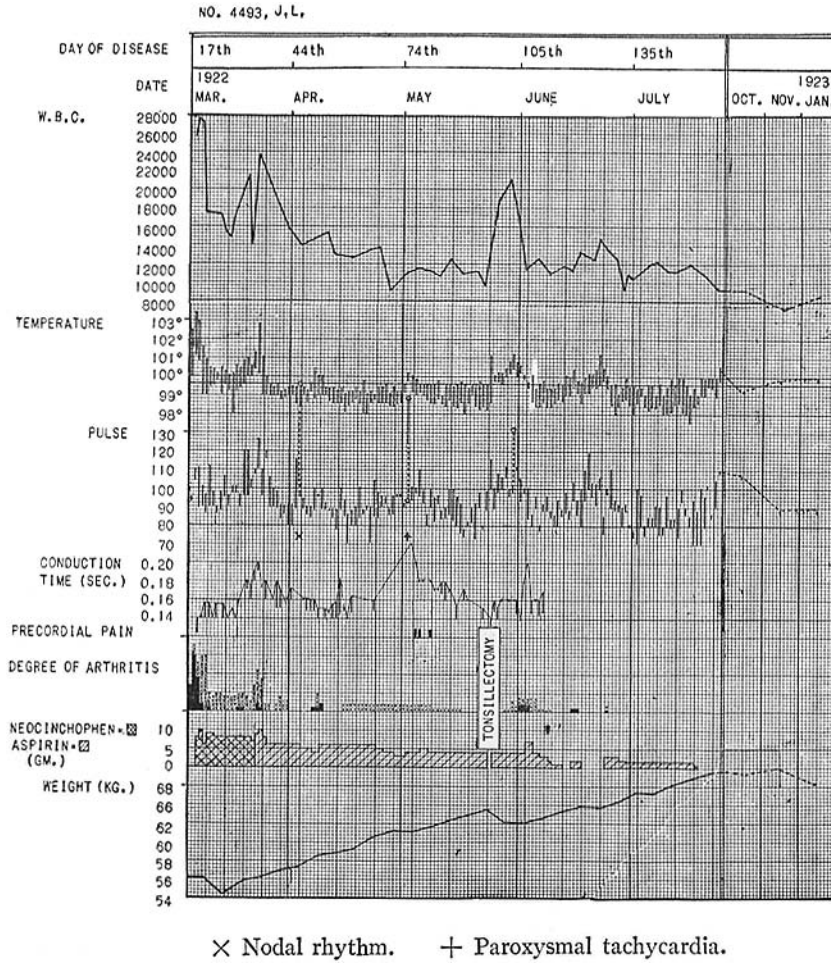
Present Illness.—4 weeks ago sore throat and fever of 104°F. In bed 1 week 2 weeks ago. Slight pain in left ankle. 5 days ago polyarthritis began; now increased in intensity and extent.

Physical Examination.—Pharyngitis, enlarged tonsils, polyarthritis. Heart: mitral stenosis and insufficiency; second pulmonic sound markedly accentuated.

Course.—(Text-fig. 3.) Pyrexia, toxicity, and polyarthritis decreased only during administration of sodium salicylate. Later, 5 gm. of aspirin were given daily. Occasional slight fever, increased pulse rate; mild arthritis. Relapse when aspirin was reduced to 1 gm. daily; disappeared following large doses of the drug. On admission and in relapses, P-R time increased. During June subcutaneous fibroid nodules in tendon sheaths of hands and over left elbow.

Patient transferred to Presbyterian Hospital. During his stay (11 weeks) several bouts of fever, polyarthritis, precordial pain, cardiac arrhythmia, and new subcutaneous nodules. Tonsillectomy.

From Nov., 1921, until Apr., 1923, well, except for signs of mitral disease. Apr. 16, 1923, fourth attack of rheumatic fever; symptoms, signs, prolonged P-R time, and course similar to that above recorded.



TEXT-FIG. 4.

No. 4493.—J. L., male, age 52 years.

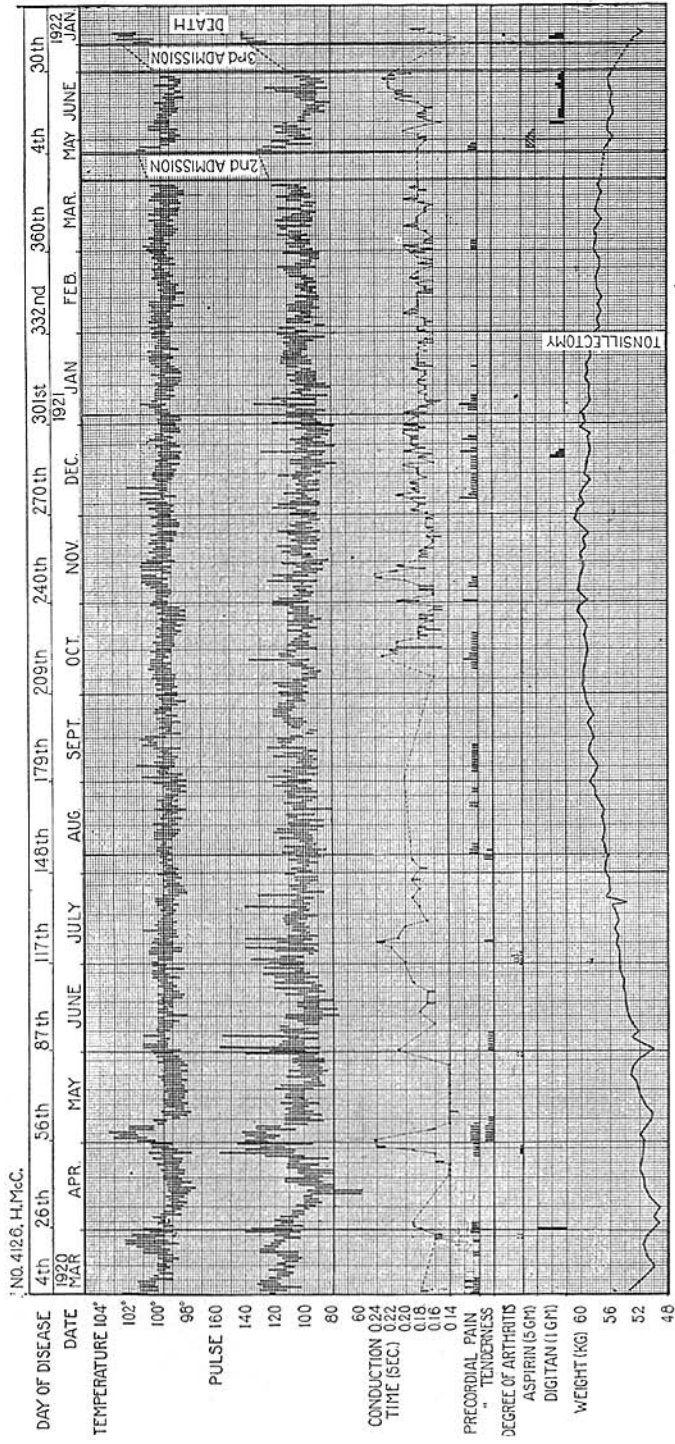
Admitted Mar. 5, 1922. Discharged July 25, 1922.

Previous Rheumatism.—Mild arthritis in ankles 2 years ago; duration 3 days. Sore throat for many years.

Present Illness.—Arthritis in ankles began 16 days ago, migrated to knees and hips; yesterday shoulders, elbows, and wrists were involved.

Physical Examination.—Conjunctivitis, rhinitis, pyorrhea alveolaris, buried tonsils, marked polyarthritis, emphysema. Cardiac apical systolic murmur.

Course.—(Text-fig. 4.) Polyarthritis and fever increased. Marked improvement after large doses of neocinchophen. Leucocytosis, slight fever, increased pulse rate, and stiffness in a few joints persisted. Relapse, accompanied by increased P-R time occurred while taking 8 gm. of neocinchophen a day. Aspirin substituted for neocinchophen with marked benefit. Attack of nodal tachycardia Apr. 3. Leucocytosis, increased pulse rate, and slight stiffness of shoulders persisted. Aspirin decreased; May 2, auricular tachycardia. Afterward increased P-R time, precordial pain, and slight fever. Later, while under influence of aspirin signs of infection except leucocytosis and increased heart rate disappeared. Apical systolic murmur and occasional gallop rhythm persisted. May 24, tonsillectomy. Ekg. during operation normal. After tonsillectomy, arthritis and tachycardia (short attack), followed in 3 and 4 days by increased conduction time. In June, mild relapse with rapid heart action; apical systolic murmur rougher. Joints bared, active and passive motion, and progressive exercise, followed by improvement. Since discharge, no symptoms of cardiac insufficiency; occasional stiffness in joints, persistence of apical systolic murmur, not transmitted, but auscultatory diagnosis obscured by pulmonary emphysema.



TEXT-FIG. 5.

No. 4126.—H. McC., female, age 17 years.

Admitted Mar. 10, 1920. Discharged Mar. 24, 1921. Readmitted May 28, 1921, and discharged June 23, 1921. Readmitted Jan. 20, 1922. Died Jan. 24, 1922.

Previous Rheumatism.—First attack 3 years ago; duration 3 months. Tonsillitis every winter for many years frequently accompanied by quinsy. Tonsillitis 2 months ago. 7 weeks ago told of presence of heart trouble. Since then slight precordial pain on exertion. Light work until 2 weeks ago.

Present Illness.—Began 3 days ago with mild polyarthritis for 1 day. 2 days ago severe precordial pain; now increased with palpitation and dyspnea.

Physical Examination.—Patient showed orthopnea, rapid respirations, distinct pallor, enlarged tonsils, mild pharyngitis. Heart enlarged to right and left. Gallop rhythm, blowing apical systolic murmur, accentuated second pulmonic sound, and pericardial friction rub persisting 7 days.

Course.—(Text-fig. 5.) For 2 months, relapses with fever, high pulse rate, precordial pain, increased P-R time, and mild arthritis. Next 2 months relapses with less fever, but paroxysms of tachycardia, prolonged P-R time, and mild arthritis. Subsequent relapses during 1st year were cardiac, characterized by paroxysms of tachycardia, precordial pain and tenderness, gallop rhythm, and prolonged P-R time. During the first 8 months relapses about every 4 weeks; later more irregular. Aortic diastolic and presystolic apical murmurs sometimes heard. Later (Jan., 1921) constant aortic systolic murmur. Jan. 31, 1921, tonsillectomy. Exercise was started Feb. 15, 1921, and gradually increased. Mar. 1, slight precordial pain lasting 4 days. Aortic diastolic murmur not heard for some time now heard again. Mar. 24, discharged, feeling well although mild fever and some increase in pulse rate were present; also gallop rhythm, apical systolic murmur, aortic systolic murmur, a diastolic murmur over upper sternum, and soft systolic blowing murmur over the lower sternum to the ensiform.

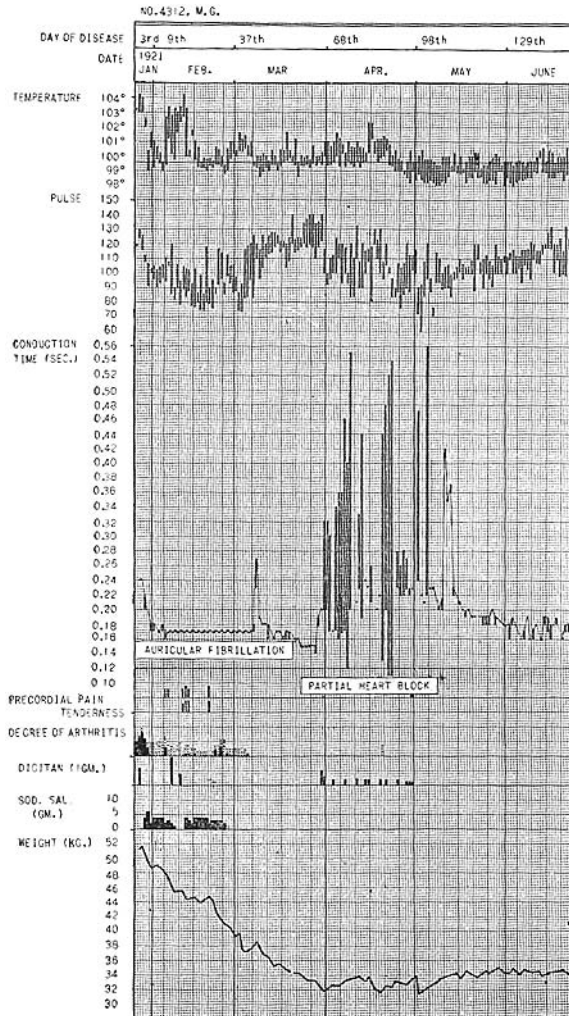
At home Mar. 24 to May 28. Well the 1st week, then mild polyarthritis, "cold" in the chest, and attack of palpitation. Another cold the middle of May; rapid heart and precordial pain; herpes labialis. May 15 to 28, in bed with constant precordial pain and dyspnea.

Second Admission.—May 28 to June 23, 1921. Temperature 101°, pulse rate 120 to 130, precordial pain, gallop rhythm, and apical systolic murmur. June 6, aortic diastolic murmur again heard; digitan June 6; some slowing of pulse. Discharged June 23.

At home June 23, 1921 to Jan. 20, 1922. Aug. 7 to 21, relapse with precordial pain radiating down left arm and into left side of neck; fear of impending death. Until Sept. 20, better and no pain. Sept. 20, gallop rhythm, aortic diastolic and apical systolic murmurs present.

Readmitted Jan. 20, 1922. Marked cardiac decompensation. Edema of extremities, fluid in abdomen and chest, and in knee joints. No precordial pain, tenderness, or hyperalgesia. Apical systolic murmur, gallop rhythm, systolic and diastolic blowing murmurs over the aortic area. Rhythm regular. Liver enlarged. Digitan given without effect. The patient died suddenly Jan. 24, 1922.

MYOCARDIAL INVOLVEMENT IN RHEUMATIC FEVER



TEXT-FIG. 6.

Postmortem Diagnosis.—Adherent pericardium; hypertrophy and dilatation; chronic thickening of the tricuspid, mitral, and aortic valves; verrucous endocarditis of the tricuspid, mitral, and aortic valves. Chronic fibrinous pleurisy; chronic passive congestion of the liver, spleen, and kidneys. Ascites. Edema of legs.

Microscopic Examination.—Numerous Aschoff bodies throughout myocardium, mural endocardium, and pericardium; inflammation of tricuspid, mitral, and aortic valves and of chordæ tendineæ of mitral and tricuspid valves. Endarteritis and thrombosis of fine branches of coronary artery.

No. 4312.—M. G., female, age 31 years.

Admitted Jan. 26, 1921. Discharged June 24, 1921.

First attack of rheumatic fever.

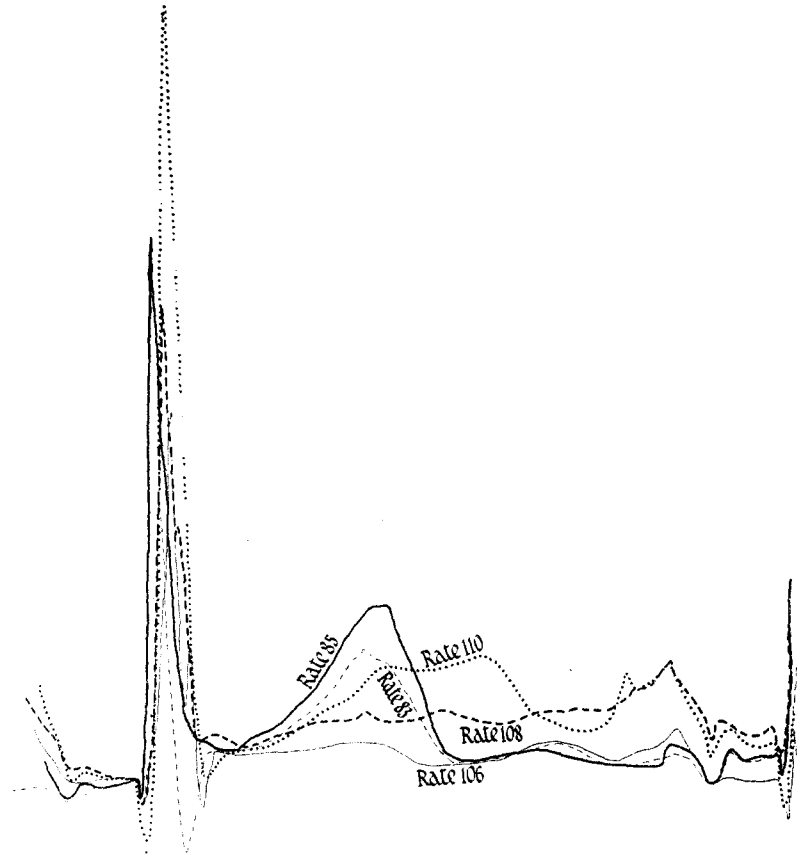
History.—Tonsillitis 1 month ago; in bed 10 days. Following this, loss in weight (20 pounds), occasional pain in ankles, and dyspnea on climbing stairs. 2 days ago chills and fever, 1 day ago, pain in ankles; arthritis spread to knees and lumbar spine the day of admission; *i.e.*, 3rd day of disease.

Physical Examination.—Redness of pharynx, polyarthritis involving joints of leg and lumbar spine. Heart enlarged. Numerous premature contractions. Rough systolic and short diastolic rubbing sound over base of heart. Prolonged P-R time.

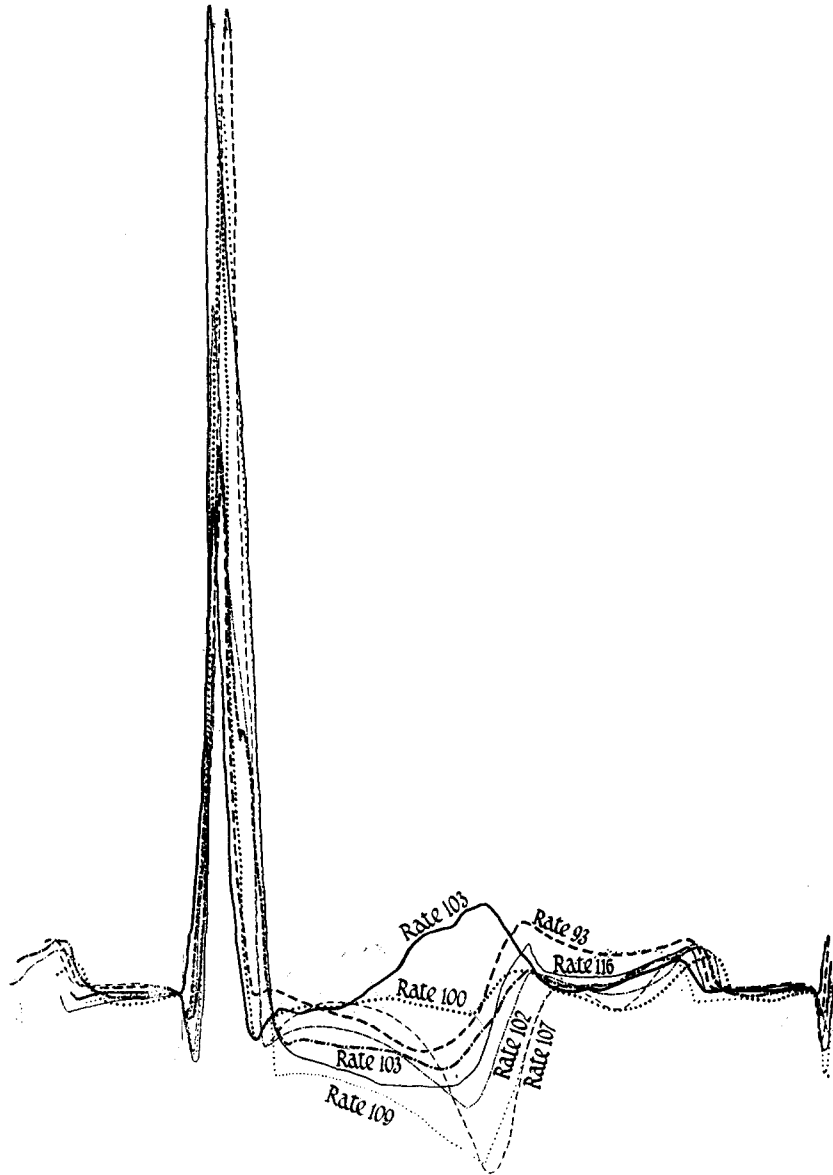
Course.—(Text-fig. 6.) Marked intoxication; spread of arthritis on 4th and 5th days. Extrasystoles disappeared, murmurs changed to rough systolic over base and soft systolic blow at apex; P-R time somewhat shorter on 6th day. Sodium salicylate with marked benefit; arthritis diminished; fever less.

12th day yellowish pus expressed from both tonsils. 13th to 26th day, "to and fro" murmurs heard the 4th and 5th days again present; now have character of pericardial friction. Systolic blowing murmur over aortic area. Dose of sodium salicylate reduced because of nausea and vomiting. 14th day, recurrence of high fever, increase of arthritis, total cardiac irregularity (auricular fibrillation), and marked pulse deficit. 40th and 41st days, auricular flutter. Doses of sodium salicylate were small because of nausea; drug discontinued because of vomiting. 26th day, signs of pericardial effusion; and double pleurisy with effusion from 17th to 35th days. Arthritis disappeared 2 days before cessation of auricular fibrillation. For next 3 weeks rapid heart action with low grade fever; no arthritis; continuous loss of weight due in part to anorexia and nausea. Small doses of digitan given for 5 weeks because of tachycardia caused partial heart block with reduction in heart rate and improvement in general condition. May and June, occasional slight febrile relapse. Anorexia and vomiting troublesome. Short rough systolic murmur almost continuously over base of heart, loudest over sternum, and soft blowing apical systolic murmur from time to time. On discharge there was merely a clicking systolic sound at base to left of sternum.

2 months after discharge gained 11 kilos and had no symptoms of cardiac insufficiency. No murmurs heard. Letter of Mar. 28, 1922, reported improvement, and return to work as housemaid. The only symptoms were occasional attacks of palpitation.

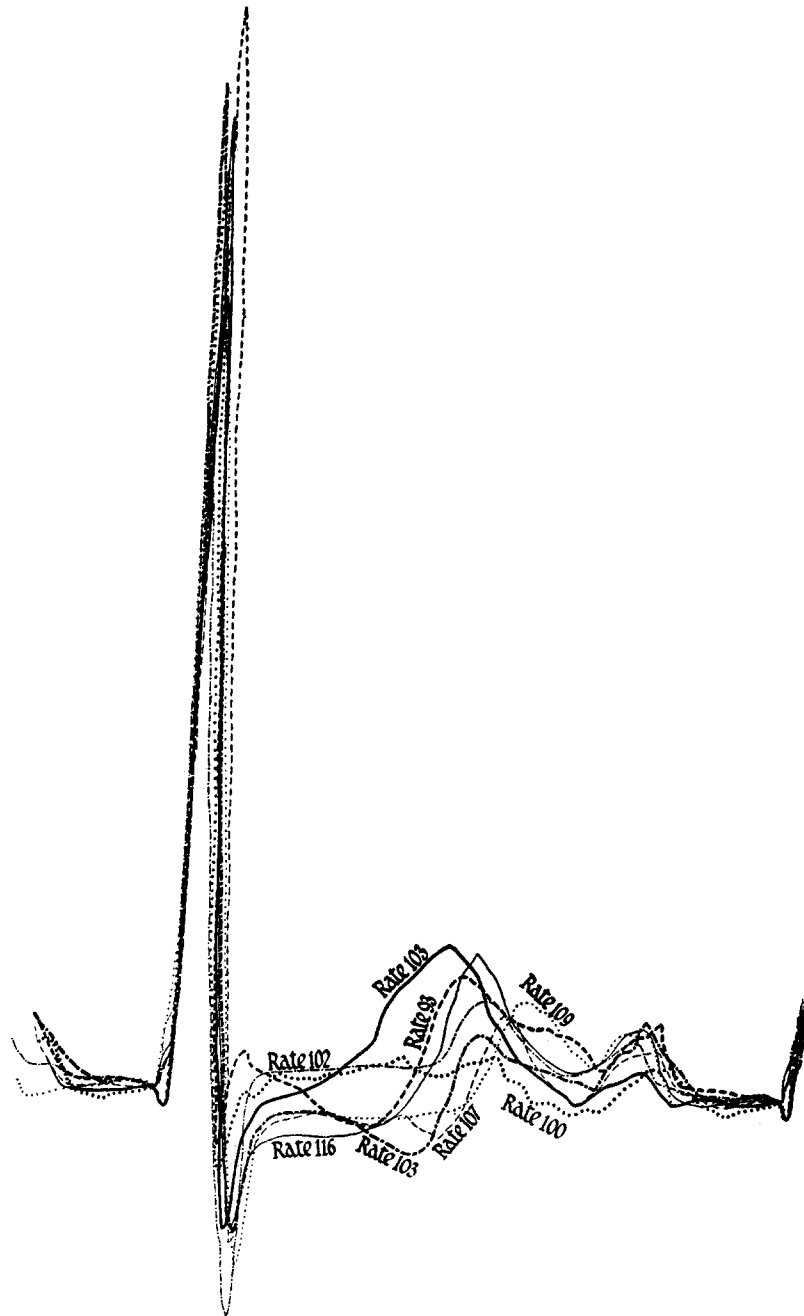


TEXT-FIG. 7. In this figure are reproduced outline drawings of Lead III of 5 electrocardiograms taken from the same patient. Each complex was projected on a screen, the magnification being so arranged that the points of onset either of the Q or R waves of two succeeding cycles were made to coincide. The outline was then drawn. In this way the proportion of the cycle taken by each event is accurately given. The differences in outline, especially in the region of the T waves in the various curves are strikingly exhibited. The influence of the rate of the heart on the relative diastolic time occupied by the T waves is also shown.

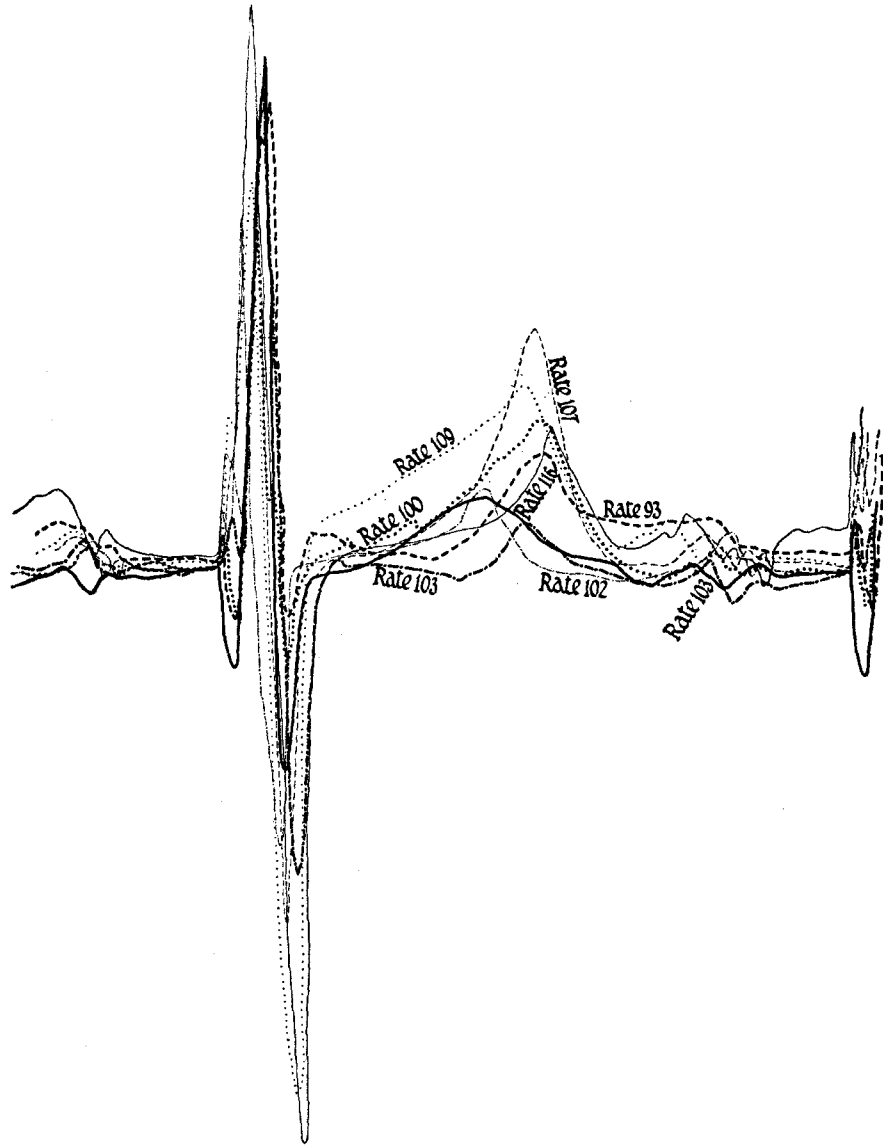


TEXT-FIG. 8, *a*.

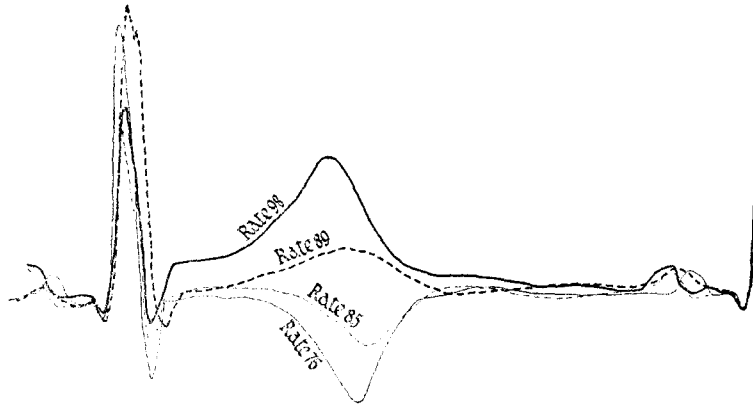
TEXT-FIG. 8, *a, b, c*. Curves constructed in a manner identical with those in Text-fig. 7 are shown. In *a*, drawings of 8 cycles of Lead I are reproduced; in *b*, 8 corresponding cycles in Lead II; in *c*, 8 corresponding cycles in Lead III. The wide variations in form and the influence of rate on form are shown. The change in the level and direction of the curves in the R-T period should be noticed.



TEXT-FIG. 8, *b*.



TEXT-FIG. 8. c.



TEXT-FIG. 9. Cycles in Lead III of 4 electrocardiograms, constructed like those in Text-Fig. 7 are exhibited. The R-T period is seen to be little affected. The chief changes are seen in the T wave itself.

EXPLANATION OF PLATES.

PLATE 1.

FIG. 1. A series is shown of electrocardiograms taken of a single patient (J. L., Case 26). In this and the succeeding electrocardiograms the ordinates are 0.04 apart; the abscissæ, 10^{-4} volts. The mechanism is normal except in the curves of Apr. 3 and May 2; in these two, it may be normal, the P wave being isoelectric or, on the assumption that the P wave cannot be distinguished from the R wave, either the auriculoventricular interval is much prolonged, or nodal rhythm is present. An explanation of the form of the curve of June 2, is given in the text. Changes in the form of the T wave are seen in Lead III.

PLATE 2.

FIG. 2. A series is shown of electrocardiograms taken of a single patient (M. G., Case 12). The mechanism is normal in the curves taken on Jan. 27, 31, Mar. 8, 23, and June 23. Fibrillation of the auricles is seen in those of Feb. 16, 21, Mar. 2, and 7. The curve of Mar. 7, Lead II, exhibits a transition from the fibrillatory to the normal mechanism. In the curve of Mar. 6, flutter of the auricles is seen. Ventricular premature beats are seen in the curve of Mar. 2. Prolonged conduction and varying degrees of block occur in the curves of Mar. 8, Apr. 2, 23, 26, and May 11. Curves of Jan. 31 and Feb. 16 show an alteration in the down stroke of the R wave in Lead I, described in the text.

PLATE 3.

FIG. 3. A series of electrocardiograms is shown of a single patient (H. E., Case 30). The curves of Nov. 24, 26, and Dec. 1 show striking alterations in the T waves in Lead I. These may be compared with the second and third curves in Fig. 2. In the curves of Dec. 1, and Mar. 3, premature contractions of ventricular origin are shown.

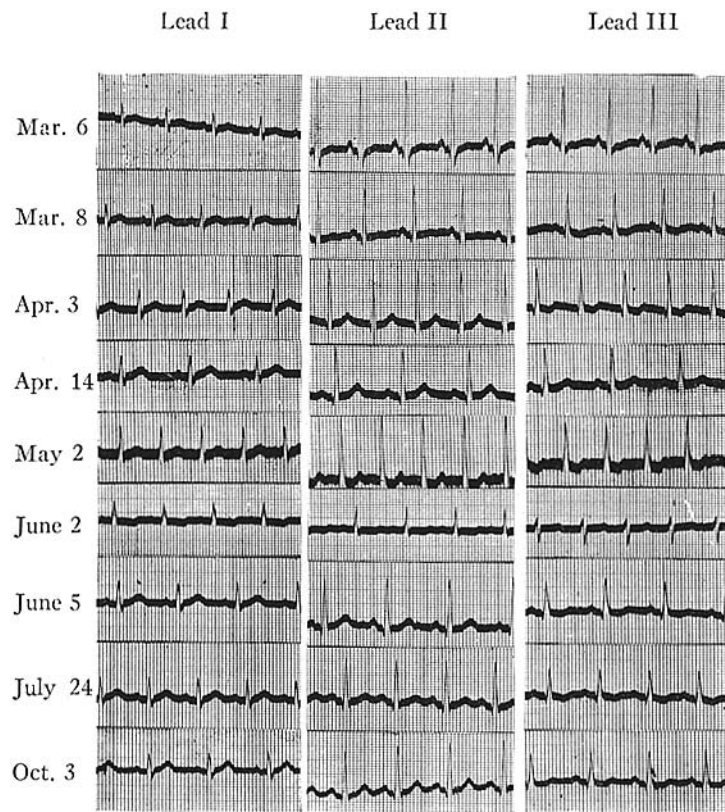


FIG. 1.

(Cohn and Swift: Myocardial involvement in rheumatic fever.)

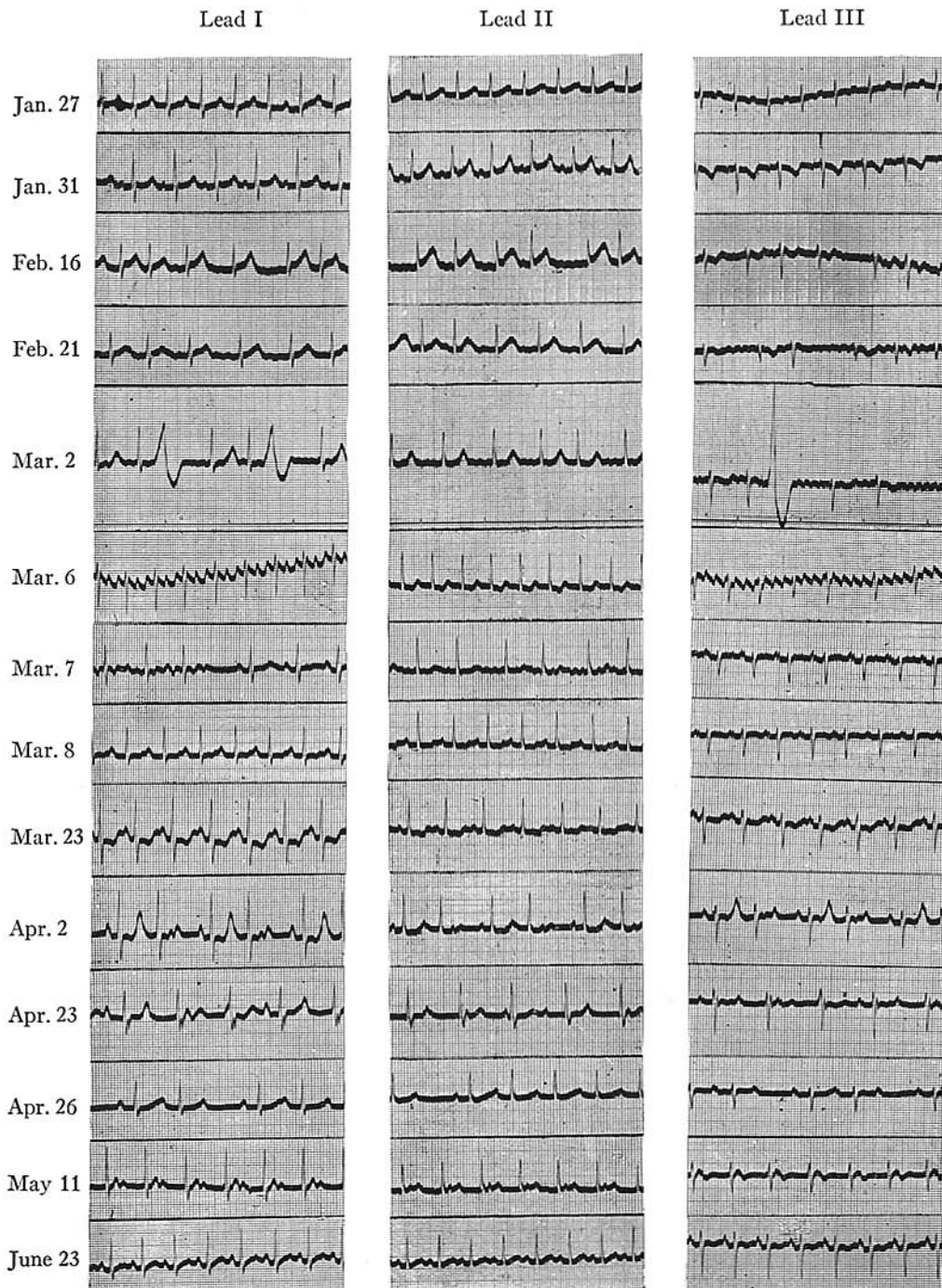


FIG. 2.

(Cohn and Swift: Myocardial involvement in rheumatic fever.)

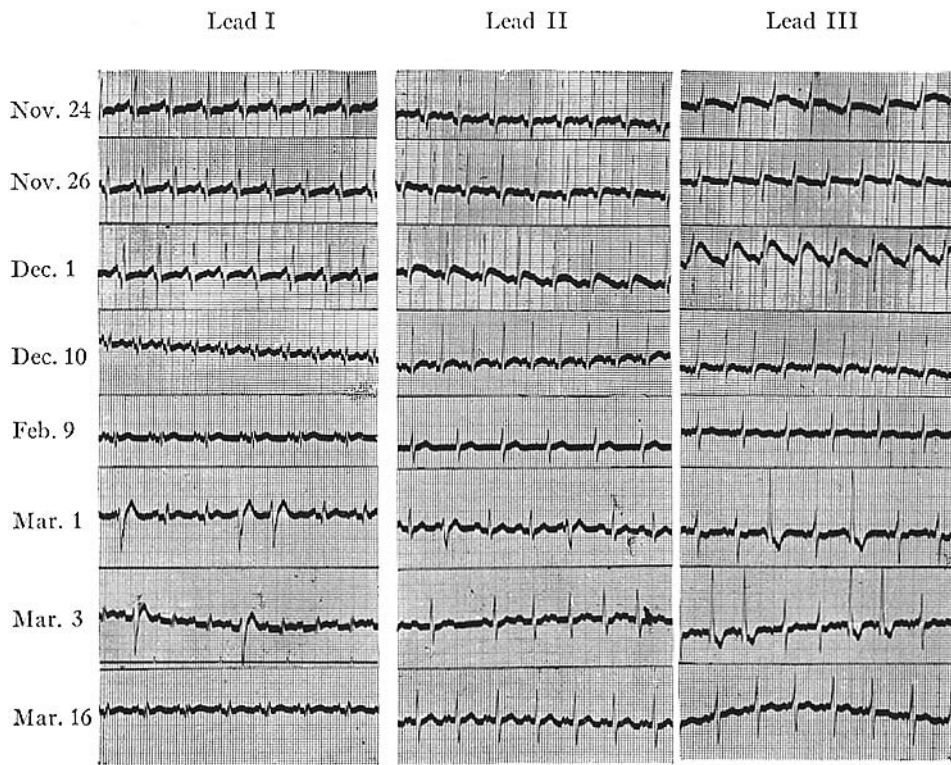


FIG. 3.

(Cohn and Swift: Myocardial involvement in rheumatic fever.)