Atrioventricular conduction in acute rheumatic fever¹

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A new study of normal PR intervals was undertaken for the purposes of analysing atrioventricular conduction. Abnormalities of conduction were identified in 84 per cent of 508 patients with acute rheumatic fever. The PR index was used as a simple, reliable method of showing these changes. Such conduction disturbances occur more commonly than carditis, arthritis, subcutaneous nodules, chorea, or erythema marginatum, the five major features of the disease. Streptococcal infection and acute glomerulonephritis were not associated with similar abnormalities of conduction.

It appears that atrioventricular conduction disturbances that occur after a haemolytic streptococcal infection are relatively specific for rheumatic fever, either with or without carditis. It is suggested that reversible PR prolongation be used as a major criterion, when using Jones' criteria for the diagnosis of rheumatic fever, providing there is proof of a preceding streptococcal infection.

A number of reports of PR prolongation in rheumatic fever studied by various methods have been published (Cohn and Swift, 1924; Keith, 1938; Taran, 1946; Blackman and Hamilton, 1948; Sokolow, 1948; Mirowski, Rosenstein, and Markowitz, 1964). At the Hospital for Sick Children in Toronto, frequent electrocardiograms have been recorded in children with rheumatic fever, making a detailed study of atrioventricular conduction possible. In 508 patients seen between 1948 and 1971, over 2,500 electrocardiograms were available for analysis.

PR intervals in normal children

To evaluate atrioventricular conduction, the normal child must first be studied. The PR interval varies with both age and heart rate (Ziegler, 1951; Sodi-Pallares *et al.*, 1958). To date, a number of published tables of normals exist. Those of Ziegler (1951) do not give the variation for heart rate. Ashman and Hull's (1937) figures were considered inadequate by Alimurung and Massell (1956), but their own figures are unreliable because of the small numbers of patients in many of the groups. Though the latter authors were able to give

detailed figures for the *average* PR intervals, it is the *maximum* normal that is required when studying atrioventricular conduction.

To overcome these objections, a new study of PR intervals in normal children was undertaken. The results are shown in Fig. 1. The PR interval was measured on the electrocardiogram of 672 normal children using the method described below. The results are in essential agreement with the above authors but give more details useful for the purposes of this study of rheumatic fever. The maximum normal PR interval was taken as the 100th percentile for that age and heart rate. Nine normals were excluded from the analysis because their PR intervals were considerably prolonged compared with the others in the group. This confirms the observation of Ziegler (1951), that 2 per cent of normal children have very long PR intervals for no apparent reason. Only those electrocardiograms with heart rates of plus or minus 5 of 60, 90, 110, 140, and 160 were selected in the 6 to 11 years old group (Fig. 1b) to increase the accuracy of the relation. (Numerous electrocardiograms were usually available.) This enabled the results of each section to be plotted as a single point on the graph without further analysis. Similar criteria, but different heart rates, were used for the patients younger (Fig. 1a) and older (Fig. 1c) than this group. The linear relation between PR interval and heart rate shown in our graphs agrees with the findings of Alimurung and Massell (1956). The small-

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FIG. 1 Maximum normal PR interval according to heart rate and age. (a) 3-5 years; (b) 6-11 years; (c) 12-15 years.

est number of patients in a section was 41 and the largest was 59.

Measurement of PR index

The PR index (Mirowski *et al.*, 1964) was calculated by dividing the measured PR interval by the maximum normal value for that age and heart rate. This normal value was read off the appropriate graph to the nearest second decimal place, thus having regard for the degree of accuracy with which the interval itself was measured. A PR index greater than 1.0 indicates delay in atrioventricular conduction.

For example, a 9-year-old patient with a febrile illness had a heart rate to the nearest 5 of 130 a minute and a measured PR interval of 0.12 sec. The maximum normal for that age and rate (Fig. 1b) is 0.14 sec, to the nearest second decimal place. The PR index was:

$$\frac{\text{Measured PR}}{\text{Maximum normal PR}} = \frac{0.12}{0.14} = 0.86.$$

Two weeks later, the same patient had a heart rate of 65 with a PR interval of 0.16 sec. The normal for this rate is 0.18 sec. The PR index at this stage was $\frac{0.16}{0.18} = 0.89$.

There was thus no change in PR index in spite of a wide range of heart rate. The patient was subsequently shown to have rheumatoid arthritis.

Rheumatic fever analysis - materials and methods

The records of 508 patients were studied. Each had electrocardiograms taken in the acute and

convalescent stages; rheumatic fever had been diagnosed according to the modified Jones' criteria (1955, 1965). The PR index as described above was analysed in all cases in sinus rhythm (445 cases), the remaining 63 having various dysrhythmias in which the PR interval could not be reliably assessed.

All electrocardiograms had been recorded using a Sanborn direct-writing machine at a paper speed of 25 mm/sec. Heart rate was estimated (using a standard electrocardiogram ruler) to the nearest 5. The PR intervals were all measured by the same observer using a magnifying glass. This enabled reasonably accurate assessment to the second decimal place: it was possible to distinguish, for example, between a PR interval of 0.14 and one of 0.15 sec. The longest PR interval in any of the standard or praecordial leads was recorded. Frequently, this was found in standard lead II, but occasionally in V1 or V6. Where the PR interval was very prolonged the P wave could be hidden in the T wave of the preceding complex in some leads. The true nature of such a phenomenon was revealed by analysis of the whole electrocardiogram (Fig. 2). The PR interval was measured according to the recommendations of the American Heart Association (1954).

The PR index during the acute stage of rheumatic fever was compared with that in the same patient when convalescent. The interval between the two electrocardiograms varied from 8 days to 3 years, but was usually between 2 to 6 weeks.

Patients were divided into those with and those without evidence of carditis. The criterion for carditis was usually the development of a significant cardiac murmur, though occasionally patients

FIG. 2 The rhythm in lead II appears to be nodal. Analysis of AVR and V6 shows a very long PR interval, with the P wave partially hidden in the T wave of the preceding complex.

were placed in this category on the basis of a pericardial rub alone. Cardiac murmurs were assessed carefully by staff cardiologists so that patients with innocent systolic murmurs were not included in the group with carditis.

Results

The various electrocardiographic abnormalities found in the 508 patients with acute rheumatic fever are summarized in Table 1. The patients were aged 2 to 14 years; the average age was 7.8 years.

Because of method error, a random PR index of up to 1.03 should only be accorded the same suspicion as a high-normal reading.

TABLE 1 Atrioventricular conduction changes in acute rheumatic fever

Rhythm	Carditis	No carditis	Total
PR index > 1 during acute stage	136	187	323
PR index greater in acute stage than in quiescence	16	25	41
Wenckebach second degree AV block	7	5	12
Complete AV block	1	2	3
Junctional rhythm	5	8	13
AV dissociation with accelerated nodal pacemaker	19	16	35
Normal PR index	34	47	81
Totals	218	290	508

Repeated readings add reliability, and subsequent change of PR index is of great value.

PR index greater than 1.0 during acute stage Three-quarters of the patients in sinus rhythm (323 out of 445) had a PR index greater than 1.0 during the acute stage of rheumatic fever (Fig. 3). There was no difference between those with carditis and those with no cardiac involvement (Fig. 4). No patient was receiving digitalis at the time of the initial electrocardiogram. In every patient with an increased PR index this subsequently became smaller when the disease became inactive. In all but 3 per cent it reverted to less than 1.0 (Fig. 3).

PR index greater during acute stage Forty-one patients without obvious atrioventricular conduction delay had a PR index change from the acute stage to the quiescent phase that was 0.14 or greater, though at no time was the PR index greater than 1.0. A sample of serial electrocardiograms in the same number of normal children showed a

FIG. 3 PR index in 445 patients with acute rheumatic fever in sinus rhythm, compared with the same patient when convalescent.

FIG. 4 PR index in the same 445 patients during the acute stage of rheumatic fever. Patients with carditis compared with those with no carditis.

variation of up to 0.08 on sequential tracings. We have accepted this as the upper limits of normal for change. The atrioventricular block may well be uncovered by this means. Such evidence was present in the 41 patients referred to above, bringing the total to 364 out of 445 patients in sinus rhythm (an incidence of 82%).

Second degree heart block Apart from the PR prolongation described above, cardiac dysrhythmias were found in 63 of the 508 patients with acute rheumatic fever (Table 1). Second degree block of the Wenckebach type (Mobitz Type I) (Fig. 5) without progression to complete block occurred in 12 patients.

FIG. 5 Wenckebach phenomenon (Mobitz Type I second degree block).

Mobitz Type II block was not encountered in any patient with rheumatic fever.

Complete heart block Three patients developed complete heart block; one of these suffered repeated Adams-Stokes attacks (Fig. 6) and required temporary transvenous pacing for eight days. In all three patients, there was a progression from a long PR interval to a Wenckebach phenomenon before heart block was established. The reverse sequence of events occurred as the patient reverted to sinus rhythm. When in heart block the idioventricular focus was of a 'high order' narrow QRS type.

Atrioventricular dissociation It is of interest that 35 patients were found to have atrioventricular dissociation associated with acceleration of a nodal pacemaker (Fig. 7). This dysrhythmia had frequently been erroneously reported as 'heart block'. However, in every case the atrioventricular dissociation was only present where the sinus rate was slower than the junctional focus. When the sinus rate increased normal conduction occurred though the PR interval was frequently prolonged. Four patients were given intravenous atropine to increase the sinus rate, thereby confirming this feature. The heart rate at which atrioventricular dissociation occurred in these 35 patients was in the range 70 to 110, the average being 82. Unlike Rodriguez-Coronel and Miller (1969), we did not find this dysrhythmia to be a specific indication of carditis with later valvular involvement.

Junctional rhythm A further 13 patients exhibited junctional rhythm (Fig. 8) during the acute stage of rheumatic fever. Adding this group to those with acceleration of the nodal pacemaker, a total of 48 patients (9.5%) had evidence of an atrioventricular junctional rhythm when the disease was active. The incidence was the same in the groups with and without carditis.

All 63 patients with dysrhythmias reverted to sinus rhythm during convalescence.

Normal PR interval and index The PR index in 81 patients was normal in acute and convalescent stages, and showed no measurable increase during the illness. The majority of these patients only had one electrocardiogram in the acute stage, and a second electrocardiogram within a few days might have shown a change.

Streptococcal infections The PR index was assessed in 51 patients with streptococcal infections. This group included 16 with acute streptococcal glomerulonephritis. No increase in PR index was found either in the acute febrile state or some weeks later (Fig. 9). This evidence refutes those suggestions that fever or streptococcal infections *per se* cause frequent atrioventricular conduction delay (Feinstein, 1966). On the contrary, it implies that PR prolongation after a streptococcal infection is a relatively specific indication of rheumatic fever.

Intracardiac electrocardiogram A 5-year-old girl with acute streptococcal glomerulonephritis was also felt to have acute rheumatic fever. The evidence for this was the appearance of an apical systolic murmur of mitral regurgitation, at a time when the blood sedimentation rate was raised to 85 mm in the first hour and the PR interval was prolonged. Though there was no history of joint pains, a previous streptococcal infection was present as indicated by an antistreptolysin O titre of 800 Todd units.

An intracardiac electrocardiogram to include His bundle potentials was recorded on this girl. The electrode was introduced into the heart from the long saphenous vein under fluoroscopic control. After a recording had been taken, the heart rate was fixed by atrial pacing at a faster rate and atropine 0.3 mg given intravenously. The results are shown in Table 2. Because the PR interval on the intracardiac electrocardiogram is shorter than that measured in the standard leads, we will refer to the former as the P'R interval. The prolonged P'R interval was due to an abnormally long P'H segment, indicating delay in conduction proximal to the bundle of His. The HV interval remained unchanged throughout, and was normal for this age (Roberts and Olley, 1971). Atropine temporarily reversed the P'H prolongation. This effect was not due to any change in heart rate as this was fixed by atrial pacing.

Discussion

The frequent occurrence of PR interval prolongation in acute rheumatic fever has been recognized for over 50 years (Parkinson, Gosse, and Gunson, 1920). By using the more elegant method of measuring the PR index and comparing the acute with the convalescent stages, some atrioventricular conduction abnormality has been shown in 84 per cent of our patients. This is a somewhat higher incidence than previously reported (Parkinson

FIG. 6 Ventricular asystole occurring in a patient with acute rheumatic fever and complete heart block.

et al., 1920; Blackman and Hamilton, 1948; Mirowski *et al.*, 1964). Though the published values for normal children that were available to these and other authors were inadequate, none made a special study to determine the normal maximum PR interval. Our higher incidence is partly due to the assessment of normal children and partly to the acceptance that a variation in PR index reflects a change in atrioventricular conduction. His bundle cardiograms could probably show this change, but the simpler non-invasive technique of measurement of index is adequate in the majority of instances.

One of us has shown that atropine reverses the PR interval prolongation of rheumatic fever (Keith, 1938). Study on one patient by intracardiac electrocardiogram showed that the site of reversible delay was proximal to the bundle of His, as expected from other evidence cited below. The effect of atropine is to block vagal receptors in the heart; the principle of these receptors are the sinoatrial and atrioventricular nodes. It is known that acetylcholine may produce heart block by the action on the AV node. The effect has been localized to the atrial margin fibres of the AV node in animal experiments (Cranefield, Hoffman, and de Carvalho, 1958). Further evidence to implicate a parasympathomimetic aspect in rheumatic fever is the sinus bradycardia that frequently occurs in the mildly ill patients (Keith, 1938). It is not known whether these conduction changes which occur so frequently are mediated by acetylcholine-like substances or by direct vagal action.

This phenomenon of PR prolongation is only one aspect of a cardiac disturbance that occurs in acute rheumatic fever. Where second degree heart block occurred in our

FIG. 7 Atrioventricular dissociation with accelerated nodal rhythm.

patients this was always of the Wenckebach type (Mobitz Type I). Furthermore, in our three patients with complete heart block the pattern was identical. The cardiac rhythm progressed through the stages of PR prolongation and Wenckebach block before complete block occurred. When in heart block, the idioventricular focus was of a 'high order' narrow QRS pattern. His-electrocardiogram studies have shown that these characteristics occur most typically where the block is proximal to the bundle of His (Damató *et al.*, 1969; Narula *et al.*, 1971). Mobitz Type II block, which indicates almost exclusively disease distal to the His bundle (Narula *et al.*, 1971), was not encountered in this series. None of the 508 patients had a left hemiblock or right bundle-branch block, further supporting the evidence that the rheumatic affection of atrioventricular conduction is proximal to the trifascicular system.

Other evidence pointing to the locality of the disorder in rheumatic fever is the incidence of junctional rhythms. Atrioventricular dissociation has been reported to be common (Cristal, Stern, and Gueron, 1971), and more frequent electrocardiogram monitoring in our patients might have uncovered a higher incidence than that found. As it was, a random electrocardiogram recorded some time during the first 7 days of a child's admission to hospital showed an incidence of 9.5 per cent of junctional rhythms and atrioventricular dissociation.

A large international multicentre study of rheumatic fever (*British Medical Journal*, 1955) gave the incidence of the major manifestations of the disease as follows: arthritis (43.3%); nodules (14.3%); chorea (10.9%); erythema marginatum (5.8%); carditis (75.1%). Atrioventricular conduction abnormalities detected by the methods described in this paper are thus much more common (84%) than any of the recognized major manifestations of rheumatic fever.

It has long been established that rheumatic fever is invariably preceded by a Group A β -haemolytic streptococcal infection (Rantz, Boisvert, and Spink, 1945; Rammelkamp and Stolzer, 1954). During epidemics with virulent organisms, about 3 per cent of the population affected by such a streptococcal infection will develop rheumatic fever (Rammelkamp and Stolzer, 1954). Levander-Lindgren (1952) found 3.9 per cent of patients admitted to hospital with scarlet fever had electrocardiographic changes similar to those described above, and it seems probable that this was due to the occurrence of rheumatic activity induced by the streptococcus. Rantz, Spink,

FIG. 8 *Functional rhythm with spontaneous reversion to sinus rhythm at a faster rate.*

and Boisvert reached a similar conclusion in 1946. In confirmation of this, our own series of streptococcal infections (Fig. 9) showed no conduction abnormalities unless rheumatic fever developed. It is clear that detectable abnormality of atrioventricular conduction occurs in a high percentage of patients with rheumatic fever (Keith, 1938; Taran, 1946; Blackman and Hamilton, 1948; Sokolow, 1948; Alimurung and Massell, 1956). The above evidence suggests that if these changes (including increased PR index, second and third degree heart block, atrioventricular dissociation, and junctional rhythms) occur after a haemolytic streptococcal infection, they are specific for rheumatic fever.

The exact mechanism by which the rheumatic process causes this vagal effect is unknown. Though Goldstein, Halpern, and Robert (1967) have shown an immunological relation between the Group A streptococcus and the glycoprotein of cardiac valves, to date no such relation with the glycoproteins of conducting tissue has been described. Furthermore, the atrioventricular node has a very low content of glycoprotein compared with the peripheral conducting system (Gee, 1969). It is possible that glycogen, of which there is an abundance in the AV node (Otsuka and Hara, 1965), is the substance affected by the rheumatic process. That glycogen is involved in atrioventricular conduction is further implicated by the presence of a short PR interval in the cardiac glycogen storage diseases (Ehlers *et al.*, 1962).

TABLE 2 *His-bundle cardiogram in acute rheumatic fever*

	Heart rate	P'R interval	P'H interval	HV interval
Resting sinus rhythm	68	190	155	35
Atrial pacing (continued throughout remainder of test)	133	255	220	35
Before atropine	133	255	220	35
30 sec after atropine	133	205	175	30
60 sec after atropine	133	165	130	35
120 sec after atropine	133	225	190	35

All measurements are in milliseconds.

FIG. 9 PR index in 51 patients with streptococcal infection: acute stage compared with 2 to 6 weeks later.

Studies on the incidence of prolonged atrioventricular conduction times in normal and abnormal children are scanty. A list of conditions that may be associated with PR interval changes is shown in Table 3. If those conditions can be ruled out, and one is dealing with a child who has definite evidence of a recent streptococcal infection (usually a raised antistreptolysin-O titre), the prolonged conduction time becomes highly significant.

TABLE 3 PR prolongation in children

Diagnosis	Per cent incidence of PR prolongation in each condition
L-transposition of great arteries	57
Ebstein's anomaly	50
Atrial septal defect	43
Coarctation of aorta	12
Total anomalous pulmonary venous drainage	8
Ventricular septal defect	6
Persistent ductus arteriosus	3.4
Pulmonary stenosis	3
Tetralogy of Fallot	2
Diphtheria	Common
Normal children	Very rare

Adapted from Keith, Rowe, and Vlad, 1967.

Under such circumstances, it should be considered for inclusion with the major Jones' criteria, with the provision that such significance only applies if the conduction time returns to normal, or significantly lower levels. In the absence of evidence of carditis, PR prolongation does not appear to have a bad long-term prognosis, but may carry the requirement for long-term antistreptococcal prophylaxis. Rheumatic fever can, of course, occur in the above congenital cardiac defects which have permanent prolongation of the PR interval. A further increase of PR index during the acute stage will still have the same significance as in a normal child.

Atrioventricular conduction abnormalities were found in 84 per cent of our patients with acute rheumatic fever, though in some patients they could only be identified in retrospect when the convalescent recording was compared with that taken in the acute stage. Junctional rhythm, sometimes with atrioventricular dissociation, is part of the abnormal response. The changing pattern of acute rheumatic fever can make definite diagnosis difficult (Feinstein and Spagnuolo, 1962; Besterman, 1970). The judicious use of the PR index with comparison of acute and quiescent stages may make the PR prolongation a more important criterion in the diagnosis of acute rheumatic fever.

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