VARIATIONS IN THE FIRST HEART SOUND AND THE AURICULO-VENTRICULAR CONDUCTION TIME IN CHILDREN WITH RHEUMATIC FEVER

BY

J. D. KEITH, M.D.*

(The Birmingham Children's Hospital.)

It is well recognized that a soft first heart sound is frequently found in rheumatic heart disease in children. When this has been marked it has been presumed that a severe degree of carditis existed. It is the purpose of this paper to point out certain relationships regarding the soft first heart sound and the accentuated first sound in rheumatic fever and discuss the reasons for their occurrence.

Coombes³ in his book on 'Rheumatic Heart Disease' says 'In very acute carditis the first sound at the apex may become softened and almost inaudible; this used to be accepted as evidence of pericardial effusion until it was shown that this complication was extremely rare. At the same time it became apparent that enfeeblement of the myocardium was at the bottom of the signs wrongly ascribed to pericardial effusion. It occurs only in the severest types of carditis and is to be interpreted as a direct sign of grave failure of ventricular contractivity.' Cabot² in 'Physical Diagnosis' refers to weakening of the first sound at the apex as occurring in the course of long-continued fevers in which there is degeneration of the heart muscle. He concludes 'All in all however these changes in the first sound are of very little diagnostic use.'

Present investigation

It was noticed that cases of acute rheumatic fever that had softening of the first heart sound at the apex were frequently not of a severe type, and for that reason the question has been investigated.

A case that illustrates this is that of O. O., aged 10 years, who was admitted to the Birmingham Children's Hospital suffering from chorea. There was no evidence of heart disease on admission. During her stay in hospital she developed a transient mild carditis of a degree that presented only the faintest signs. The intensity of her first heart sound became fainter

* Working under an Atholl-Moynihan Scholarship, University of Birmingham, in the Rheumatic Research Department.

as the P-R interval lengthened and improved as it shortened. These findings are recorded in the following table:—

DATE.	SEDIMENTATION RATE.	HEART RATE.	P-R INTERVAL SECONDS.	FIRST SOUND.	MURMURS.
Admission	—	100	0.15	Full. Well heard.	None
10 days	—	94	0.18	Diminished.	None
22 days	5 mm.	92	0.20	Almost inaudible.	Very faint systolic.
1½ months	6 mm.	85	0.18	Diminished.	None
2 months	—	90	0.16	Blunt. Distinctly heard.	None
2½ months	—	90	0.15	Full. Well heard.	None

Blood leucocyte count at 5 weeks—10,800 per c.mm.

The first heart sound has been studied in over two hundred children with chorea, chorea with rheumatic carditis and in cases of uncomplicated rheumatic carditis. In the same series of cases repeated electrocardiographs have been studied and ventricular conduction time has been noted. A group of sixty-two patients of rheumatic heart disease with P-R intervals over 0.18 seconds were investigated in detail. These were divided into two groups, those in their first attack of carditis and those that had previously had attacks.

Group 1. First attack of carditis.—There were thirty-seven cases of rheumatic fever with a first attack of carditis in which the conduction time exceeded 0.18 seconds.

Average age 8.4 years.

Range 3 to 12 years.

Average sedimentation rate 28 mm. per hour.

Range 3-60 mm. per hour.

32 mild cases.

3 moderately severe cases.

2 severe cases.

Four of these children showed mitral diastolic murmurs, in two, such murmurs were temporary and disappeared within two months. The first sound was markedly diminished in intensity, and in many cases was inaudible, while twenty-one had an apparent accentuation of the second sound in the mitral area.

In this first group most of the patients were only mildly ill and the pulse rate fell below 100 to a level practically normal within a few days of admission. The temperature was as a rule elevated only on the day of admission or on some days previously at the onset of their rheumatic attack. The soft first sound usually persisted for one to two months and then became more normal in intensity. The second sound at the apex was in many cases well heard and often sounded accentuated because of the diminution of the first. In this group the heart was rarely enlarged beyond

a minor degree. All had systolic murmurs at the apex and a few of these disappeared during the hospital stay. In two cases the conduction time continued to be 0.2 seconds after the systolic murmur had disappeared and the only clinical evidence of heart disease then was a soft heart sound.

It was found then that many cases of acute rheumatic fever have a conduction time of 0.2 seconds without having the most severe form of carditis. Thirty-two cases out of thirty-seven had a relatively mild lesion, and the softened first sound appeared to be associated only with the lengthened conduction time, rather than myocardial weakness.

Dock⁴ has summarized the experimental evidence on the mode of production of the first sound and conducted some interesting experiments along these lines. Using anaesthetized dogs and recording heart sounds by a modification of the method of Wiggers and Dean he recorded heart sounds (1) with the venous return clamped and (2) with a ligature round the auriculo-ventricular ring. In both instances the heart continued to beat for some time but the heart sounds were practically eliminated. In the first instance there was an average of 95 per cent. reduction in the amplitude of the sound of vibration produced at the beginning of systole, while the ligature around the auriculo-ventricular ring reduced the amplitude by 93 per cent. He says 'Not only was the amplitude diminished but the recorded vibrations were so different in form from those recorded from the hearts before the circulatory condition was altered as to leave no doubt that in clamped off or ligatured hearts the recorded vibrations were inaudible mechanical effects of ventricular movement.' He concludes that the first sound is due to putting under high tension suddenly the previously slack fibres of the auriculo-ventricular valves.

Ruane^t in 1832 first expressed the theory that the sudden tension of the auricular-ventricular valves is an important factor in the production of the first sound. In 1851 Halford⁵ recorded experiments on a dog in which clamping off the venous supply caused the first sound to disappear.

From these experiments it is seen that vigorous systole produces no appreciable sound. Furthermore, that the theory that the first sound is due to the tensing of the heart wall around its incompressible contents is disproved by the fact that little or no sound was produced when the auricular-ventricular ring was ligatured in Dock's experiments. Dock⁴ uses the old example of a handkerchief or string to explain the mode of production of the first sound. When the border of a handkerchief is suddenly drawn taut it produces a sound like that of the first heart sound, and he points out that if a piece of carpet or strips of meat as thick as the left ventricle are used such a sound cannot be artificially produced. In the question of intensity of the first sound the valvular theory suggests that if the auricular-ventricular valves are lax and suddenly drawn taut a sound is produced normally. If, however, they are already closed and part of the tension taken up at the beginning of ventricular systole no sound occurs. Using the analogy of the handkerchief if the slack is taken out quickly a noise is made similar to the first sound. If, however, the slack is taken out gradually at first and then sudden tension produced even with great force no sound results. Applying this to the study of the first sound and the

auricular ventricular conduction time, in the children with a P-R interval of 0.2 seconds the mitral valve would be permitted to close and the tension partly taken up before ventricular systole occurs, and the first sound is soft. Kerkhof⁷ has shown that when patients with mitral stenosis go into auricular fibrillation the cardiac minute volume diminishes about 22 per cent. and concludes that auricular systole adds approximately this much to the cardiac output. In a normal heart the ventricle is partially filled in diastole before auricular systole takes place. If auricular systole then adds 22 per cent. to the ventricular volume the tension is considerably raised and would result in closure of the auricular ventricular valves and put them under a moderate degree of tension. If ventricular systole occurred after this process was complete the auricular-ventricular valves would contribute little to the first heart sound. This appears to be actually what takes place in these children with lengthened P-R intervals and a poor first heart sound results.

In the thirty-seven cases of acute rheumatic fever in the first attack in which the conduction was over 0.18 seconds, all had marked diminution in intensity of the first sound; in many cases the first sound was practically inaudible. The clinical significance is indicated by the fact that in 100 cases of rheumatic heart disease investigated at this hospital it was found that 26 per cent. had P-R intervals of 0.2 seconds or over at some time over a period of two years: 52 per cent. had P-R intervals of 0.18 seconds or over, and it has been found that these conduction changes can be determined with a moderate degree of accuracy with the stethoscope. Heart sounds were studied in many cases that had shorter P-R intervals and the following scheme is recorded to indicate the relationship of the conduction time to the first heart sound. This scheme cannot be applied with mathematical accuracy, but holds true generally.

0.12	0.14	0.16	0.18	0.20
Sharp first sound.	Moderately sharp first sound.	Full or blunt first sound.	Diminished intensity of first sound.	Very faint or inaudible first sound.
First sound greater in intensity than second.		First and second equal.	Second sound greater in intensity than first.	

These variations in the first sound related to the P-R interval are not as useful when the P-R is 0.16 or less. When the conduction time is more than 0.16 seconds, diminution in the intensity of the first sound in cases of rheumatic heart disease in their first attack is almost invariably found provided certain factors are eliminated. It must be emphasized that these factors must be taken into consideration or eliminated before the intensity of the first sound can be evaluated. Thickness of the chest wall, the presence of pericardial or pleural effusion or emphysematous lung tissue,

heart rate and degree of damage of the myocardium are the chief factors in altering the intensity of the first sound other than the conduction time. In children the chest wall is thin and change in the heart sounds can be much more readily discerned than in the adult. In rheumatic fever pericardial effusion is rare. The myocardium is as a rule not so severely damaged in the first attack of rheumatic fever as to produce a resulting myocardial weakness, and does not play a very large part in changing heart sounds at this stage. Increased heart rate will augment the intensity of the first sound and this must be taken into consideration. A heart rate of less than 100 beats per minute will not usually interfere with interpretation of the first sound in relation to the conduction time. In children with mild rheumatic heart disease by far the most important factor in producing softening of the first sound is alteration in the conduction time.

Group 2. One or more attacks of carditis. --Twenty-five cases of rheumatic fever which had previously had one or more attacks of heart disease and which had conduction times over 0.18 seconds.

Average age 10 years.

Range 6 to 13 years.

Average sedimentation rate 38 mm. per hour.

Range 15-60 mm. per hour.

6 mild cases.

7 moderately severe cases.

12 severe cases.

14 cases had mitral diastolic murmurs.

4 cases had accentuation of the first sound, in the rest the first sound was practically inaudible or 'replaced.'

This second group is comprised of more severe cases as would be expected in rheumatic patients who had had more than one attack of carditis. In the majority the hearts were appreciably enlarged and the murmurs indicated damage of sometime standing to the valves. Twelve were severe cases (judged by demonstrable lesions and recent acute infection together), seven moderately severe and six mild cases. Fourteen had mitral diastolic murmurs which appeared to be permanent.

The diagnosis of mitral stenosis is made in a case of rheumatic heart disease on the sum of several pieces of evidence. The most important are accentuated first heart sound, mitral diastolic murmur and forceful apex beat accompanied by thrill. The last is not essential for the diagnosis and most emphasis has been placed on the presence of a mid-diastolic murmur, or an auriculo-systolic murmur. Certain of these diastolic murmurs are recognized by their sound and intensity as characteristic of obstruction at a grossly narrowed valve lumen in the heart, and leave no doubt as to the diagnosis. However, it has been frequently noted by many observers that a mitral diastolic murmur may exist in cases of rheumatic heart disease in which no stenosis of the mitral valve is apparent at post-mortem examination. Further, diastolic murmurs occur sometimes in patients with heart disease of other than rheumatic origin.

The question of mid-diastolic murmurs in rheumatic heart disease has been thoroughly dealt with by Bland, White and Jones¹. From a clinical and post-mortem study of a hundred patients under twenty-one years of age it was shown that in many cases diagnosed clinically as mitral stenosis the diagnosis was not borne out at autopsy. They discuss the significance of a mitral diastolic murmur and pointed out that it could not be used as an infallible sign of mitral stenosis. It was not uncommonly found in the early stages of their cases of rheumatic heart disease (the minimum time for mitral stenosis to develop they found was two years) and often disappeared during convalescence. They believed myocardial weakness and dilatation of the ventricle to be responsible in their cases for its production, for this is generally accepted now as the most probable explanation of this transient diastolic murmur. In the hundred cases studied mitral stenosis was considered to be present clinically on the basis of the mid-diastolic murmur best heard at the apex in sixty-eight cases. It is interesting that they record that of these only twenty-one had anatomical stenosis at post-mortem examination.

Since so many of the signs of mitral stenosis are fallible the value of the accentuation of the first heart sound of the fourteen cases in the present group with mitral diastolic murmurs must be discussed. Only four had an accentuated first sound, several of the others had accentuation of the first sound on admission but when the heart approached a more normal rate the first sound disappeared or became very faint. These probably had no anatomical narrowing of the mitral orifice but undoubtedly had valvular damage. When these hearts are beating rapidly the first sound has exactly the same slapping quality of the first sound in mitral stenosis. However, because of the gross enlargement of these hearts the mitral opening is not infrequently larger than normal. The four cases with an accentuated first sound at all rates of the heart may be diagnosed as having mitral stenosis.

The mechanism of this accentuation in mitral stenosis is closely related to the experiments, mentioned above, by Dock. In mitral stenosis the peak of intra-auricular pressure may occur later to be sustained longer than in normal hearts. When the passage of blood from the auricle to ventricle is impeded and the pressure from the auricle is forcing blood through the narrowed opening, the mitral valve is extended into the ventricle as far as its limited capacity permits and when ventricular systole occurs it is suddenly slapped in the opposite direction. This results in the accentuated first sound, and when mitral stenosis is present one nearly always hears this accentuated first sound. If the P-R interval was sufficiently long the ventricle could be filled and the slack taken out of the valves before the beginning of ventricular systole. However, Wolferth and Margolies⁷ found in cases of mitral stenosis accentuation was still present when the P-R was 0.23 seconds. When complete block supervenes one gets a varying intensity of the first sound depending on the relation of auricular and ventricular pressure at the time of ventricular systole.

In children mitral narrowing is more likely to occur if the valvular damage far outstrips the myocardial damage. In this case the evidence of

mitral narrowing might be a well heard first sound with a moderately prolonged P-R interval, when otherwise a soft or absent first sound would be expected, but since the usual process in severe rheumatic fever in children is for the myocardial damage far to outstrip the valvular and therefore there is a widening of the auricular ventricular opening and not stenosis.

In the twenty-one patients without diastolic murmurs but who had had more than one attack of rheumatic fever the first sound was not heard and these fall into the same category as group 1. These are described as having the first sound replaced by a murmur, but it was found in a number of these cases that the P-R interval at a later date became shortened again. When this occurred the first sound could be readily heard although the murmur remained unchanged. Occasionally these enlarged hearts have the first sound 'replaced' when the P-R interval is only 0.16 seconds. Such cases are difficult to explain adequately, but it is probably due to the degree of dilatation present.

In interpreting these sounds and murmurs it is worth while noting the findings in the second group reported by Bland, White and Jones¹. These were patients who had definite deformity of the mitral valve without anatomical stenosis. They found that the degree of mechanical handicap imposed on the heart appeared to be the same as in the group with mitral stenosis, so from the point of view of prognosis accurate diagnosis of the anatomical narrowing of mitral stenosis is not of prime importance as long as the nature of the lesion is understood.

Summary

The relationship of the intensity of the first heart sound to the P-R interval is discussed and the experimental results of Dock are applied clinically to cases of children with rheumatic heart disease of various degrees of severity. It is found that the P-R interval can be predicted with a fair degree of accuracy by the intensity of the first heart sound. This is of most value in the milder cases. The clinical significance of this is emphasized by the fact that in a hundred cases of rheumatic heart disease investigated at this hospital, it was found 26 per cent. had P-R intervals of 0.2 seconds or over at some time during a period of two years. Fifty-two per cent. had P-R of 0.18 seconds or more over the same period. In the past too much emphasis has been placed on heart murmurs in rheumatic carditis and evidence has been presented in this communication of the definite importance of heart sounds. The accentuated first sound of mitral stenosis is discussed and it is suggested that the presence of this sign in rheumatic children (provided the heart rate is not notably raised) is more valuable in the diagnosis of mitral stenosis than the mid-diastolic murmur.

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