MECHANISM OF THE WOLFF-PARKINSON-WHITE SYNDROME

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The Wolff-Parkinson-White syndrome has been described frequently during the past few years, and there is considerable evidence now that the short P–R interval associated with an aberrant QRS is probably due to short circuiting of an auricular stimulus through one or more accessory bundles.

Hunter, Papp, and Parkinson (1940) came to the conclusion that the syndrome represented a double rhythm by two interfering pacemakers, one near the sinus and the other in one bundle branch, but Wolferth and Wood (1941) raised objection to this hypothesis, and adhered to their previous theory (1933) put forward to explain this type of cardiogram, namely, that the ventricular asynchronism is due to premature stimulation of one ventricle by conduction through the bundle of Kent. Parkinson and others described gradual changes of P coinciding with gradual changes in the ventricular complex, and argued against the bundle of Kent hypothesis. Wolferth and Wood, however, thought there was as much variation between the P waves before aberrant as there was before normal complexes, and quoted a case of Tung (1936) in which the P waves became smaller and the QRS complexes normal twenty minutes after the injection of atropine; thirty minutes after the injection the P waves had returned to their normal size, while the QRS complexes still remained normal. It was suggested that the atropine may be a factor in this change in the character of the P wave. Butterworth and Poindexter (1944) suggested that fusion beats may be the explanation of the cardiographic changes in this syndrome. Rosenbaum, Hecht, Wilson, and Johnston (1945) used unipolar leads from the œsophagus, præcordium, and other parts of the thorax, and gave observations supporting the presence of one or more accessory conducting bundles. Stein (1945) described a case showing normal P–R intervals and complexes alternating with those of the Wolff-Parkinson-White syndrome immediately after cessation of attack of paroxysmal tachycardia, suggesting also a shorter accessory pathway between the auricles and ventricles. Öhnell (1944) in a comprehensive monograph " Pre-excitation—a cardiac abnormality " showed various types of this syndrome. He assumed that there were two excitatory waves, the regular impulse via the bundle of His and an additional premature ventricular spread. These sometimes varied in time in the same case, and he described this as a " concertina effect."

The following (Case 1) showed all the features of this Wolff-Parkinson-White syndrome. A boy, aged 17, was admitted to the Aberdeen Royal Infirmary on June 12, 1942, complaining of palpitation which was found to be due to paroxysmal tachycardia. He had three attacks of palpitation during the previous year, and, except for one fainting attack, after a strenuous game of football, there were no other symptoms. He gave no history of any serious illness. On physical and X-ray examination there was no abnormality of the cardiovascular system. Cardiograms were obtained showing paroxysmal tachycardia (Fig. 1), and others showing the characteristic *Sh. P–R : B.B.Bl.* syndrome—a short P–R interval and the QRS of the bundle branch type (Fig. 2). Since his discharge from the hospital in August 1942 he has had paroxysms of tachycardia, occurring at varying intervals and lasting from a few minutes to several hours. During these he had palpitation and breathlessness, but at other times he felt quite fit.

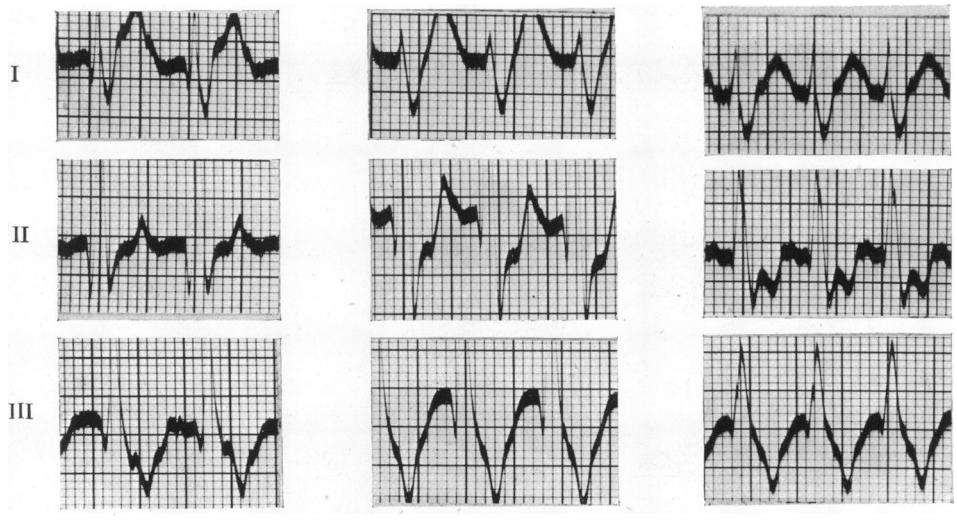

FIG. 1.—Case 1. Paroxysmal tachycardia. (A) 15/6/42. (B) 27/8/42. (C) 30 minutes after atropine.

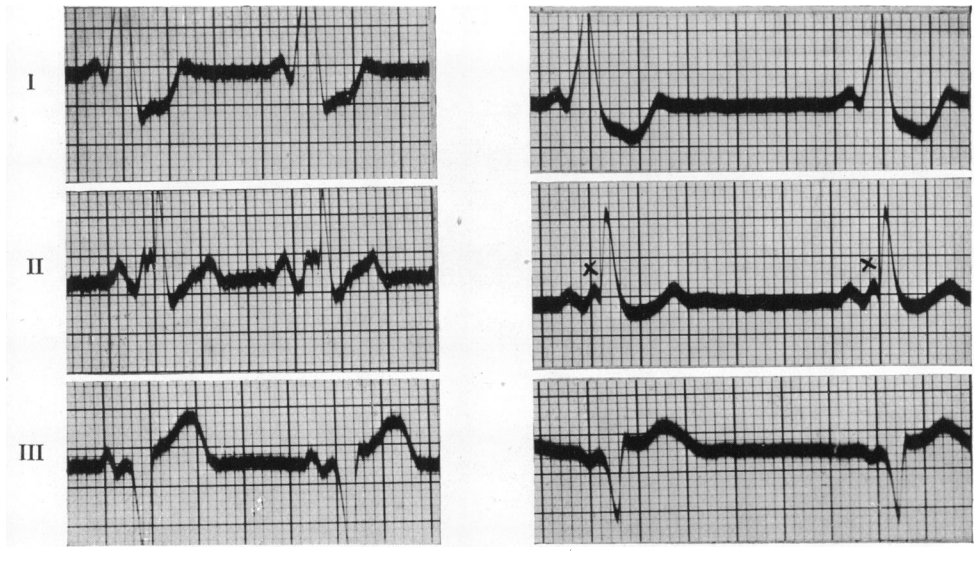

FIG. 2.—Case 1. Showing *Sh. P–R : B.B.Bl.* complexes of two different types.

CARDIOGRAPHIC ANALYSIS

The cardiograms in Fig. 2 and 3A show, especially in leads I and III, the typical short P–R interval and the widened QRS complex, somewhat resembling a left bundle branch block. Fig. 1 shows paroxysmal tachycardia, and other figures illustrate the effect of 1/50 of a grain of atropine given subcutaneously.

The following features are worthy of note.

(1) When the *Sh. P–R : B.B.Bl.* rhythm is present, the complexes are not all similar (Fig. 2 and 3A): there is a variation of P waves, of QRS complexes, and of S–T segments. This indicates variability in the path followed by the impulse over both auricles and ventricles. Öhnell (1944) would regard the differences in the complexes as due to a variation in the interval between the beginning of two assumed ventricular excitation waves.

(2) The second leads in Fig. 2B, Fig. 3A, and Fig. 5B and D show, after each P wave, another upward deflection (afterwards indicated as X) just before the QRS complex.

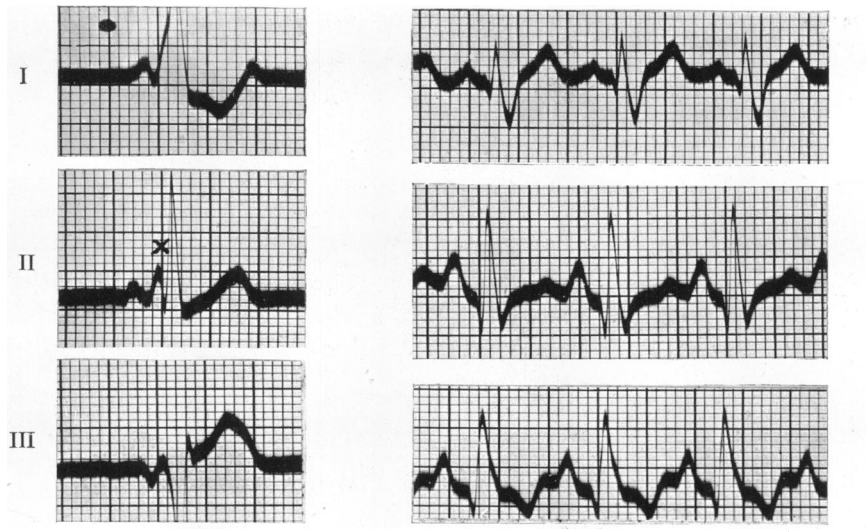

FIG. 3.—Case 1. 2/9/42. (A) *Sh. P–R : B.B.Bl.* complexes immediately before injection of 1/50 grain atropine. (B) 15 minutes after injection of atropine.

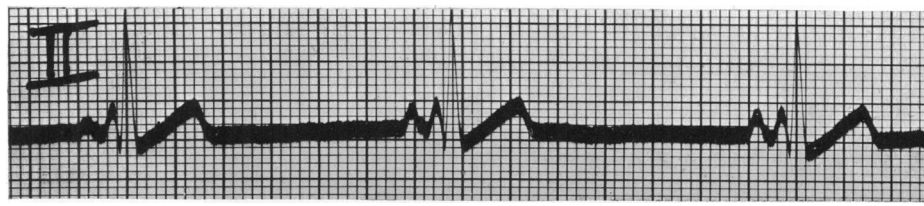

FIG. 4.—Case 1. 2/9/42. After atropine showing variation of P waves.

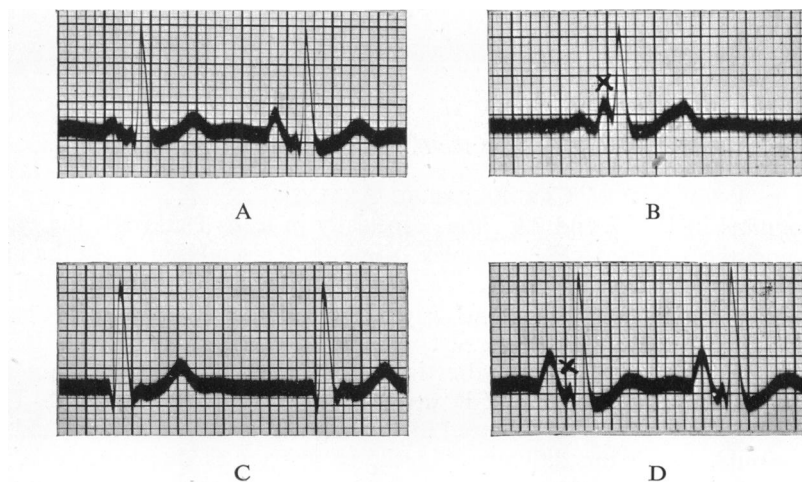

FIG. 5.—Case 1. 2/9/42. After atropine. All lead II. (A) Showing further variation of P waves. (B) Note relative size of P and X waves. (C) Showing change of pacemaker to A-V node. (D) Compare with Fig. (B). Change in size of P and X waves.

(3) The length of the P–Q interval in lead II of Fig. 3A, where the abnormal rhythm is present and the interval includes an X deflection, is equal to that of the P–Q interval in lead II of Fig. 3B. This shows right bundle branch block after atropine, but the P–R and the P–Q intervals may be considered normal. It may be noted that the configuration of QRS is similar to that in the paroxysmal tachycardia in Fig. 1.

(4) Atropine can modify the P waves and QRS complexes in many ways.

(*a*) Fig. 4, and 5A, B, and D, show P waves of varying sizes and shapes, mostly followed by X waves, which also vary.

(*b*) The P–R intervals in Fig. 5A are shorter than the assumed normal interval in Fig. 3B.

(*c*) Fig. 5C shows nodal rhythm with disappearance of P and X.

Discussion

The cardiograms suggest two special comments. The variation in shape of the P waves and the P–R intervals points to changes in the position of the pacemaker. There is room for considerable variation of its position within the sheath of muscle described anew by Glomset and Glomset (1940). Abnormal lability in its position seems to be one feature of the disorder.

Secondly, the shape of the ventricular complex in most cases of the syndrome shows two peculiarities. The first is a thickening and slurring of the initial rise of R with resultant widening of the complex; the second is an abnormality of the S–T segment tending to make the whole complex diphasic but less so than in ordinary bundle branch block. It would appear, therefore, that the excitatory stimulus to the ventricle is abnormal and also premature. This may be explained by assuming an accessory bundle joining the auricles and ventricles. If an excitatory wave from the auricles passes down the main A-V bundle, it cannot stimulate the part of the ventricular muscle that is already responding to the accessory bundle stimulation. Whether the greater or earlier stimulus to the ventricle is through the main bundle or through an accessory bundle probably depends on the position, size, and conducting capacity of the accessory junctional tissue. Assume accessory junctional tissues either on the right or left side of the main bundle (A C or B C in Fig. 6). Suppose the auricular stimulus reaches the A-V node (AV), and another point A, the beginning of the accessory junctional tissue, at the same time; if A–C is shorter or of higher conductivity than AV–C, C may be stimulated through A before the excitatory impulse from A-V arrives. There will, therefore, be a change in the cardiogram during the P–Q interval depending on the distance

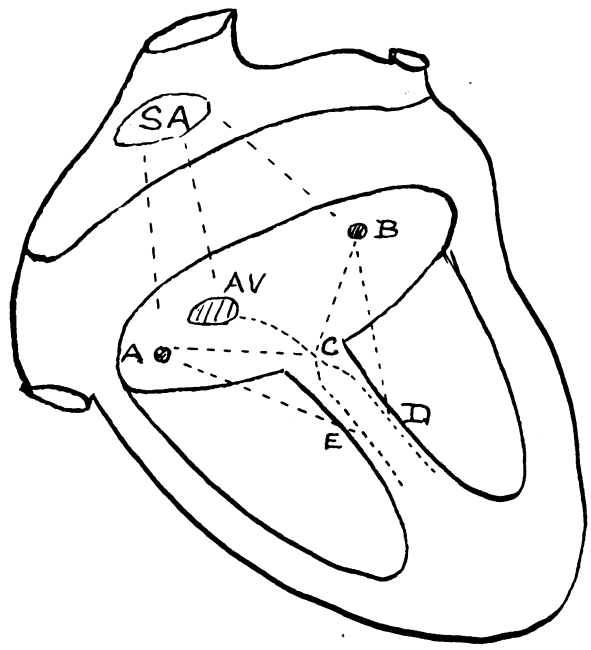

Fig. 6.—Diagram of the heart. See text.

of A and AV from C, and the rates of conduction of A–C, AV–C. The accessory junctional tissues may possibly be longer, A–E or B–D, and there would be short circuit stimulation in the region of one of the main branches, either right or left, depending on the position of the accessory conducting tissue.

The main A-V bundle is normally well insulated from the surrounding muscular tissue and the stimulus is conducted to the branches before there is response of the ventricular muscle. An accessory bundle may not be so completely insulated, resulting in infiltration of the stimulus to the ventricular muscle during the passage from A to C or A to E, and partly explaining the variation of the beginning of the QRS complex.

An accessory bundle (or bundles) between the auricles and ventricles may be the commonest cause of this early excitation of the ventricles, and in support of this theory are the post-mortem results of Öhnell (1944) and Wood, Wolferth, and Geckeler (1943), but there are other possible causes. Anatomical abnormalities occur in all tissues and an accessory branch from the bundle proximal to the main division should be kept in mind as a possible cause of pre-excitation.

In these cases of accessory junctional tissue, the auricular impulse therefore finds the shortest and easiest way to the ventricle and when the sympathetic and vagal mechanisms are stabilized, the auricular stimulation apparently pre-excites the ventricle, but if the vago-sympathetic control is changed by atropine or other drugs, the conduction capacity of junctional tissues is changed. Wilson (1915*a*) showed that even normal rhythm could be converted into auriculo-ventricular with right bundle branch block by stimulation of the vagi, and that by administration of atropine normal rhythm could be restored. He concluded that the vagi were partially responsible both for the change in the location of the pacemaker and for the abnormality of the ventricular complexes. Wilson (1915*b*) also found it possible to produce A-V rhythm in a large proportion of young persons by vagus stimulation during the intermediate period between injection of atropine and the appearance of its maximum effect, and he inferred a selective action of atropine on the vagal endings in the A-V node. The same author (1915*c*) described cases showing changes in the location of the pacemaker associated with respiration. The changes were of three kinds—migration of the pacemaker within the intermediate neighbourhood of the pacemaker, migration to the A-V node, and complete auriculo-ventricular dissociation. In the *Sh. P–R : B.B.Bl.* syndrome there is no doubt that in the majority of cases, including the first case presented above, atropine has this action on the pacemaker. Öhnell (1944) has described various ways of changing the mechanism in these cases—by carotid sinus pressure, by holding the breath, and by change of posture. Quinidine medication has also been shown to change the excitatory mechanism, possibly by its effect on the conducting tissues.

It seems, therefore, that the auricular stimulus in the Wolff-Parkinson-White syndrome is transmitted to the ventricles by the main bundle and an accessory bundle. By decreasing the vagal control, the stimuli through the main bundle are increased, and those through the accessory bundle decreased. This may be due to a relatively increased conduction capacity of the main bundle or due to a change of the position of the original pacemaker nearer the A-V node, favouring increased excitation through the main bundle.

As indicated in Fig. 6 an accessory bundle may be long (A–E) causing premature stimulation of the ventricle some distance from the main bundle and short-circuiting in the region of a branch. This type may show a cardiogram of the typical *Sh. P–R : B.B.Bl.* syndrome, but the accessory tissue may be less extensive (A–C, Fig. 6) and the premature excitation may be only very slight. Another case is given to illustrate this.

A medical man (Case 2), aged 35 years, who was being treated for pulmonary tuberculosis by artificial pneumothorax, had a severe attack of paroxysmal tachycardia in 1945. He gave the history of having similar attacks when he was a student. When he was seen soon after the paroxysm in 1945 there was nothing abnormal found on physical examination in the cardiovascular system, but a cardiogram (2/11/44) (Fig. 7) showed a small wave (X) between P and Q. On 8/11/44 (Fig. 8) the X wave was absent, but there was slurring at base of QRS. The history of attacks of paroxysmal tachycardia and the abnormality in cardiogram suggested pre-excitation of the ventricles. After atropine the slurring of QRS disappeared (Fig. 9). A cardiogram taken on 9/5/34 (Fig. 10) showed a similar abnormality at the base of QRS,

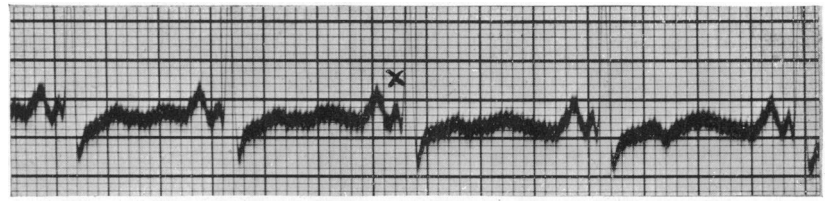

FIG. 7.—Case 2. 2/11/44. After an attack of paroxysmal tachycardia showing small X wave after P.

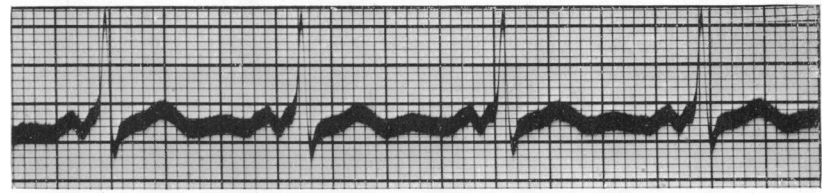

FIG. 8.—Case 2. 8/11/44. Showing slurring at base of QRS.

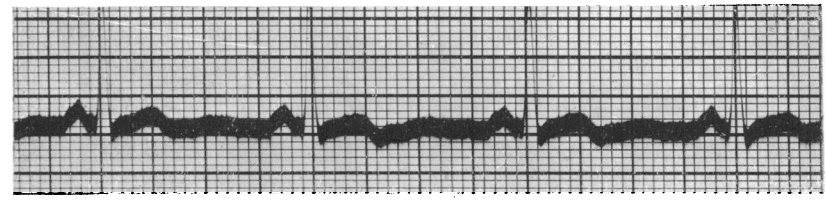

FIG. 9.—Case 2. 8/11/44. Showing disappearance of slurring after atropine 1/50 grain.

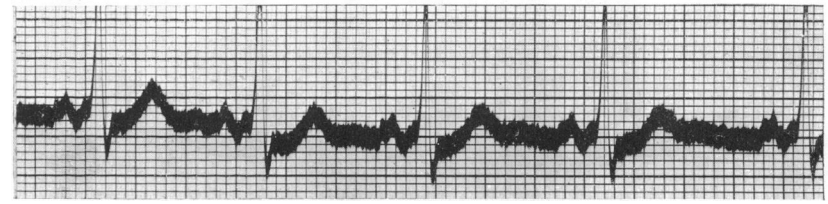

FIG. 10.—Case 2. 9/5/34. Cardiogram taken ten years previously showing abnormality at base of QRS.

showing that this pre-excitation had been present for at least ten years. This case would appear to have a minimal change in the cardiac conducting tissues (as compared with the more marked changes in the first case showing the typical *Sh. P–R : B.B.Bl.* syndrome). Pre-excitation may, therefore, vary in degree.

Doubtful cases showing slurring at the base of QR, especially if there is a history of attacks of paroxysmal tachycardia, should be investigated by means of atropine or by one of the other methods mentioned by Öhnell.

Disease or injury of the main junctional tissues may be a cause of pre-excitation of the ventricles. Accessory bundles could be present without pre-excitation showing, provided the conduction capacity of these is relatively small as compared to that of the main bundle. But if the latter is affected by disease, such as diphtheria, the auricular stimulus will tend to take the next easiest path—the accessory tissue which may be present but normally inactive. Pre-excitation of the ventricles will then result. Cookson (1945) describes a case of diphtheria showing what is almost certainly ventricular pre-excitation. If there had been no accessory junctional tissue, probably complete heart block would have occurred.

There may be little or no change in the cardiogram after the administration of atropine, suggesting that conduction through accessory tissue is more permanent or fixed and not influenced by changes in the vago-sympathetic mechanism. This is illustrated in another patient.

A girl (Case 3) aged 21, a munition worker in Coventry, was seen in the out-patient department of the Aberdeen Royal Infirmary on 6/10/44. She gave a history of attacks suggesting paroxysmal tachycardia and the cardiogram (Fig. 11A) showed a short P–R interval and broadening of the QRS complex, almost certainly due to pre-excitation of the ventricle. The maximum change after atropine is slight (Fig. 11B).

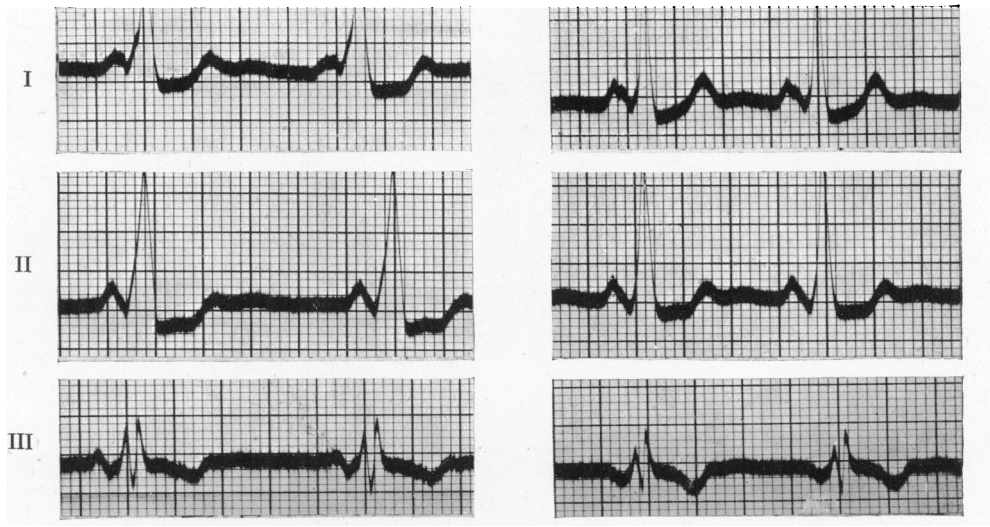

FIG. 11.—Case 3. (A) Cardiogram of girl with a history of attacks of paroxysmal tachycardia showing *Sh. P–R : B.B.Bl.* complexes. (B) Cardiogram showing only slight changes after atropine 1/50 grain.

SUMMARY

A typical case of the Wolff-Parkinson-White syndrome is described and the mechanism discussed. Premature excitation (or pre-excitation) of the ventricle is probably the cause, and published autopsy reports suggest that this is due to accessory conducting bundles between the auricles and ventricles. Other abnormalities, such as an accessory branch of the main bundle, could cause pre-excitation and should be looked for in future autopsies.

Pre-excitation may vary in degree. The degree of short-circuiting of the auriculo-ventricular conduction may depend on the position of the accessory bundle or bundles and generally changes or disappears with variation of the vagal tone.

A case has been described showing slight pre-excitation which has been present for at least ten years.

Pre-excitation is generally demonstrated by changes in the P–Q interval after injection of atropine, but a case with almost fixed pre-excitation is described.

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