CLINICAL PROGRESS

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Syndrome of Short P-R Interval with Abnormal QRS Complexes and Paroxysmal Tachycardia (Wolff-Parkinson-White Syndrome)

By Louis Wolff, M.D.

The syndrome of short P-R interval with abnormal QRS complexes and paroxysmal tachycardia was first described in 1930. Attention was called to its occurrence in young healthy individuals with normal hearts, to the frequency of paroxysmal tachycardia, to the unusually short P-R interval with abnormal QRS complexes, and to the spontaneous or induced change from the abnormal to a normal electrocardiogram, or the reverse. These are now universally recognized as the distinguishing features of this syndrome, which was thus established on a firm clinical and electrocardiographic basis for the first time. However, the nature of the disorder was left in doubt.

Terminology and Description

About 300 articles dealing with this anomaly have been published, and many of the authors have ventured to name the syndrome. The names most commonly used are the Wolff-Parkinson-White syndrome (W-P-W syndrome), anomalous atrioventricular excitation, the syndrome of short P-R interval with abnormal QRS complexes and paroxysmal tachycardia, pre-excitation, and the bundle of Kent syndrome.

The features which distinguish the syndrome are the peculiar electrocardiogram and paroxysmal rapid heart action. The P-R interval is 0.10 second or less in about 85 per cent of the cases, and is rarely greater than 0.12 second. The QRS complex measures 0.11 to 0.12 second in almost half of the cases, but may be as long as 0.20 second, or within normal limits in occasional instances. A characteristic feature of the ventricular complex is heavy slurring of the initial deflection of the QRS group. Gross notching of the QRS complex sometimes occurs; and QS deflections are not infrequently seen in leads II, III, aVF, the superior esophageal, and the right-sided precordial leads. Q waves do not occur in the left-sided precordial leads (fig. 1). An observation noted in the first case studied by us, and one which is a noteworthy feature of the syndrome, is the spontaneous or induced transformation of the electrocardiogram: the P-R interval lengthens but remains within normal limits, and the QRS complex assumes normal proportions and morphology (fig. 2).

The abnormal mechanism is called the anomalous mechanism, or anomalous atrioventricular excitation. When it prevails the short P-R interval is referred to as the anomalous or normal P-R interval; the QRS complex is variously designated as the anomalous, abnormal, or aberrant QRS or ventricular complex; and the heavily slurred deflection which initiates the QRS group is called the anomalous or premature component of QRS. The last has also been called the delta wave. When the normal type of tracing occurs the designations used are the normal P-R interval and normal QRS or ventricular complex, even if abnormalities, other than those inherent in anomalous excitation, are evident.

The paroxysmal rapid heart action is always

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SYNDROME OF SHORT P-R INTERVAL

Fig. 1. Short P-R interval and abnormal QRS complexes in a 20 year old man with a normal heart. The tracing resembles left bundle-branch block in many ways. However, the P-R interval is 0.08 to 0.10 second, and since the P waves are upright in the standard limb leads and inverted in aVR, a normal sinoauricular mechanism is present. The P-J interval is 0.26 second. The premature, or anomalous, component (delta wave) of the QRS complex is best seen in V4 and V1 as the heavily slurred initial part of the ascending limb of the R wave; notching of QRS is seen in leads II, aVR, aVL, and V1. Note the QS deflections in leads V6, V5, and V1, and the absence of Q waves in the left sided precordial leads. The T waves are inverted in leads II, III, aVF, and V6. Precordial leads recorded at half normal standardization.

(Same as figure 102, p. 175, Electrocardiography: Fundamentals and Clinical Application, by Louis Wolff, M.D., Philadelphia, W. B. Saunders Co. Reproduced by courtesy of the Publishers.)

of the supraventricular variety. The most common type resembles paroxysmal auricular tachycardia, but paroxysmal auricular fibrillation and paroxysmal auricular flutter also occur.

ETIOLOGY

The disorder is, in all probability, of congenital origin. The increasing number of cases observed in infants supports this concept. Since the abnormal mechanism may be latent for years, its demonstration, for the first time late in life cannot be considered evidence of its initial appearance. There is no proof that the syndrome is acquired. Recently, however, aberrant ventricular complexes have been noted during and immediately following cardiac catheterization as well as in animal experiments in which subthreshold stimuli were used to enhance the irritability of the ventricular musculature, and their resemblance to the anomalous complexes in cases of the Wolff-Parkinson-White syndrome has been commented upon. It is impossible to differentiate these from ventricular premature beats arising immediately after the onset of auricular systole.

MECHANISM

It is generally agreed that premature activation of a small fraction of ventricular musculature is responsible for both the short P-R interval and the abnormally wide QRS complex. There is still considerable difference of opinion, however, concerning the manner in which pre-excitation is brought about. These opinions embrace two concepts: the one supposing the mechanism to be an anomaly of impulse formation, the other that it is an anomaly of conduction. The former invokes the existence of an irritable center below the A-V node which discharges an impulse immediately after the onset of auricular systole. The latter presupposes that action currents set up in the auricles during auricular systole are conducted, with or without the aid of a structural path-
The first two beats are normal, the remaining ones anomalous. B, Another case with alternate normal and abnormal complexes, actually 2:1 block of the accessory pathway. In the normal beats the P-R and QRS intervals are 0.18 and 0.08 second, respectively, there is a small Q wave followed by an R deflection with a sharp upstroke, and an upright T wave. The corresponding intervals in the abnormal beats are 0.08 and 0.13 second, respectively, the Q wave has disappeared, the R wave is much taller and is initiated by a heavily slurred component (premature or anomalous component, delta wave), and the T wave is inverted. The premature component of the anomalous QRS complex encroaches on the P wave, but the portion of the latter which remains visible is identical to the corresponding part of the auricular deflection of the normal beats. The difference between the normal and abnormal P-R intervals is 0.10 second; anomalous depolarization is so premature that the excitation wave approaching the ventricles along the normal A-V connections everywhere encounters refractory muscle; therefore, the entire ventricular myocardium is depolarized via the anomalous route; the normal P-J interval is 0.26 second, and the anomalous P-J interval is 0.21 second.


way, to the ventricular musculature in accelerated fashion; Kent and others have described accessory A-V connections, and these have been demonstrated at autopsy in some of the patients with the Wolff-Parkinson-White syndrome. In either case, the A-V node, the natural stumbling block to conduction, is circumnavigated, and auriculoventricular conduction time is consequently abbreviated.

The hypothetic pathway of accelerated conduction, or the anomalous center of impulse formation, cannot involve the A-V connecting tissues in their entirety, for there is evidence that these tissues conduct action currents from the auricles to the ventricles in an entirely normal manner at varying intervals after the onset of the premature ventricular depolarization. That is, the ventricles are subjected to a dual innervation, and are invaded by two separate and independent waves of depolarization. The anomalous one starts first, and is followed by the normal wave a fraction of a second later, the precise time depending on the length of the normal P-R interval. The interval separating the onset of ventricular depolarization via these two separate pathways subtracted from the normal P-R interval corresponds to the anomalous P-R interval, and when added to the normal QRS interval corresponds to the anomalous QRS interval. It is for this reason that the P-J intervals (P-R plus QRS intervals) are identical, or nearly so, in both anomalous and normal beats when both are present in the same electrocardiogram. The exceptions are those cases in which anomalous depolarization begins so prematurely and, therefore, involves so much of the ventricular musculature via the anomalous pathway, that the oncoming normal excitation wave everywhere encounters active (refractory) myocardium. Under these circumstances ventricular depolarization takes place in an
entirely anomalous manner. In these cases the anomalous P-J interval is shorter than the normal one.

Whether ventricular depolarization occurs in the one way or the other, its onset invariably is via the anomalous route, and appears to proceed from epicardium to endocardium. This reversal in direction of ventricular activation alters the initial and early resultant cardiac vectors sufficiently to preclude the development of those QRS signs upon which a diagnosis of myocardial infarction is based.  

The anomalous mechanism may remain latent for indefinite periods of time, yet it may be possible to induce the appearance of the abnormal electrocardiogram, or the latter ultimately may emerge spontaneously. The abnormal mechanism may appear and disappear in an unpredictable manner, and rare instances in which normal and anomalous beats alternate have been observed (fig. 2B). The abnormal mechanism can be suppressed with certain drugs or procedures.

Paroxysmal rapid heart action occurs in at least 70 per cent of all recognized cases of the Wolff-Parkinson-White syndrome (the incidence is much higher in infants and young children) and must be considered an integral part of the disorder. In all probability the mechanism which is responsible for the abnormal electrocardiogram is also responsible for the paroxysmal tachycardia. It is not difficult to imagine the inception of circus movement following re-entry of the normal excitation wave by retrograde conduction through an accessory conducting pathway. Actually, paroxysmal tachycardia has been produced experimentally in this manner. On the other hand, an irritable anomalous center of impulse formation is an equally adequate explanation for the ectopic rhythm. Nevertheless, although the abnormal electrocardiogram and the paroxysmal tachycardia may arise from a single mechanism, both need not necessarily be in evidence at the same time. In fact, the ventricular complex during paroxysms of tachycardia is usually normal, and only occasionally is the anomalous form observed with the rapid heart rates.

**The Clinical Picture**

The syndrome is seen in all age groups. It is most frequently recognized in those age groups in which electrocardiograms are most commonly obtained. Because of the growing custom of obtaining electrocardiograms more or less routinely, it is being observed with increasing frequency in infants and young children. Many cases are being discovered in the military services, in the course of insurance and industrial examinations, and in mass health surveys. Seventy per cent of the patients are males.

The most common clinical feature is paroxysmal rapid heart action which occurs in well over 70 per cent of the individuals whose electrocardiograms at some time or other display anomalous atrioventricular excitation. The paroxysms may begin at birth, during infancy, in childhood, or in adult life. Paroxysmal tachycardia may occur for the first time at an advanced age, many years after the peculiar electrocardiogram is first observed. Symptoms associated with paroxysmal tachycardia are usually not severe, but result in invalidism in occasional patients. Sudden death rarely occurs. Some patients become chronic invalids despite normal hearts, as the result of a diagnosis of serious heart disease based on misinterpretation of the anomalous electrocardiogram. In general, however, the health of patients with the Wolff-Parkinson-White syndrome is good, and restriction of activity is not necessary. Strenuous exertion need not be proscribed. Indeed, many of our patients were healthy, vigorous athletes, accustomed to strenuous activity, and some were exposed to and tolerated well the hardships and rigors of military life.

Examination of the heart is not remarkable in most of the individuals whose electrocardiograms display anomalous atrioventricular excitation. The first apical sound is often reduplicated, and benign murmurs occur. The blood pressure is normal. The cardiac rhythm is regular, but when the anomalous mechanism prevails the heart rate is usually slow and sinus arrhythmia is common. Auricular or ventricular premature beats occur now and then, and during paroxysms the features of
paroxysmal auricular tachycardia, auricular fibrillation, or auricular flutter are noted. Visceral congestion, or other evidences of heart failure, never occurs, except in the rare instance of uncontrolled tachycardia persisting for an indefinite length of time.

The signs of congenital heart disease, or other congenital anomalies, are rarely noted; and the signs of acquired heart disease or hypertension may be observed. These findings are coincidental and bear no fundamental relation to the syndrome. The incidence of diseases other than those involving the cardiovascular system appears to be the same as in the general population. Intercurrent disease, pregnancy and childbirth, and surgical procedures are not influenced by the syndrome, nor does the latter increase the incidence of cardiac complications, except for the proneness to paroxysmal tachycardia. The syndrome is not a contraindication to pregnancy or operation.

**Electrocardiographic Features**

The characteristic electrocardiographic features of anomalous auriculoventricular excitation are the unusually short P-R interval and the abnormally wide QRS complex. The short P-R interval is diagnostic only in the presence of normal P waves indicative of sinoauricular rhythm. The P-R interval is 0.10 second or less, and the QRS interval is 0.10 to 0.20 second in most cases. QS deflections may occur in leads II, III, aVF, the superior esophageal, and the right-sided precordial leads; QR deflections do not occur in the left-sided precordial leads. A characteristic morphologic feature is the heavily slurred anomalous component which initiates the QRS group of deflections, and there may be notching of QRS. The instantaneous electrical axis of the anomalous QRS is normal in one-third of the cases, deviated to the left in one-half, and to the right in one-sixth of the cases. The S-T segments are isoelectric or slightly or markedly displaced from the base line, and the T waves vary from normal to deeply inverted. Instability of the S-T segments and T waves is a striking feature (fig. 1).

A noteworthy aspect of the electrocardiogram, and one which is of considerable diagnostic importance, is the shifting, back and forth, from anomalous to normal auriculoventricular excitation. This occurs spontaneously, or can be induced with certain drugs and procedures. The change occurs spontaneously in more than one-half of the cases in an unpredictable manner; normal and abnormal complexes may alternate regularly, or varying combinations of them may occur. Repeated observation over a period of years may disclose only the abnormal type of tracing, or just one of many electrocardiograms at the beginning or end of a long series of graphs may reveal anomalous conduction. Anomalous conduction will be induced in some cases by carotid sinus stimulation, the vagotonic action of atropine, or the administration of digitalis. This effect of digitalis persists several hours at the most, and may become evident only upon carotid sinus stimulation or forced expiration.

The abnormal mechanism can be suppressed by exercise, the vagolytic action of atropine, quinidine, amyl nitrite inhalation, procaine amide, deep inspiration, or various combinations of these. Anomalous conduction is precluded when the pacemaker is displaced to the lower reaches of the A-V node, and by some auricular premature beats.

When the anomalous mechanism is suppressed the P-R interval lengthens and the QRS interval shortens, both attaining normal values. Concomitantly the morphology of the QRS and T deflections reverts to normal, the S-T segments return to the isoelectric level, and normal QR deflections appear, providing the heart is normal, and drugs which alter the electrocardiogram have not been exhibited. The instantaneous electrical axis also changes and is normal in 80 per cent of the cases, and deviated to the left in the rest. If there is complicating heart disease and other electrocardiographic abnormalities, these become evident when the anomalous mechanism is suppressed. Then the characteristic QRS, S-T segment, and T-wave abnormalities of myocardial infarction (fig. 3) or the diagnostic features of left ventricular hypertrophy emerge, or right bundle branch block appears.

(Same as Figure 110, p. 181, Electrocardiography: Fundamentals and Clinical Application, by Louis Wolff, M.D. Philadelphia, W. B. Saunders Co. Reproduced by courtesy of the Publishers.)

PAROXYSMAL RAPID HEART ACTION

Electrocardiograms recorded during paroxysms of rapid heart action reveal the presence of normal ventricular complexes in most instances. The diagnosis of supraventricular tachycardia, auricular fibrillation, or auricular flutter offers no difficulty in these.

A small number of electrocardiograms obtained during paroxysms disclose abnormal ventricular complexes, and most of these have been called paroxysmal ventricular tachycardia by the authors reporting them. However, we have pointed out elsewhere that the abnormal ventricular complexes of the paroxysm are similar to the anomalous beats occurring between paroxysms when a normal sinoauricular rhythm exists, that in some tracings auricular deflections are regularly paired with the ventricular complexes, while in others the basic rhythm is auricular flutter with an established or shifting grade of partial auriculoventricular block. We have also observed the occurrence of more than one type of abnormal ventricular complex in different paroxysms, and have noted the same variety of anomalous complexes between paroxysms when the cardiac pacemaker was in the sinoauricular node. For these reasons we believe that most, if not all, of the cases diagnosed as paroxysmal ventricular tachycardia are in reality supraventricular paroxysms distinguished by the persistence of anomalous auriculoventricular excitation. We have not
seen undoubted examples of paroxysmal ventricular tachycardia in uncomplicated cases of the Wolff-Parkinson-White syndrome.

A typical and not uncommon example of persistence of anomalous excitation during rapid heart action is seen in paroxysmal auricular fibrillation. This is characterized by runs of anomalous ventricular complexes at extremely fast rates replacing or interrupting normal ventricular complexes occurring at a slower rate (fig. 4). Most published examples of this sort have been incorrectly interpreted as paroxysmal ventricular tachycardia. The correct diagnosis can be made if the patient is known to have the Wolff-Parkinson-White syndrome, or should be suspected if the heavily slurred premature component initiating the QRS complex is noted. The diagnosis is certain if the rapid ventricular complexes are morphologically similar to the anomalous beats recorded at other times during sinoauricular rhythm.

As a rule digitalis is ineffective in slowing the rapid anomalous ventricular responses. Indeed, this drug often appears to increase the number and rate of anomalous beats, and to perpetuate the arrhythmia. Quinidine effectively blocks the anomalous pathway, slowing the rate at which anomalous beats are conducted to the ventricles, or interrupting these responses completely.

**Diagnosis**

A mistaken diagnosis of heart disease had been made in more than one-third of our cases. The basis for error was an erroneous appraisal of benign murmurs, interpretation of the split first apical sound as a gallop rhythm or a mitral diastolic murmur, and failure to appreciate the part played by paroxysmal rapid heart action in producing symptoms. By far the most important cause of error, however, was misinterpretation of the electrocardiogram.

Diagnosis of the Wolff-Parkinson-White syndrome cannot be made in the absence of the characteristic electrocardiographic features which distinguish this disorder, namely, the unusually short P-R interval and the abnormal QRS complex. The short P-R interval is diagnostic only when the P waves are normal, and, therefore, indicative of a sinoauricular cardiac mechanism. The diagnosis can be made on the basis of a P-R interval of 0.10 second or less, if normal P waves are present, even if the QRS complex is normal. There is no other condition in which P-R intervals of such brevity exist when the heart
is under sinoauricular control. The same is probably true for P-R intervals up to 0.12 second, but as this level is approached, and certainly when it is exceeded, additional data must be available if the diagnosis is to be made, especially if the QRS complex is normal. In such cases the spontaneous or induced transformation from a normal to an abnormal mechanism, or the reverse, with the consequent characteristic changes in the P-R and QRS intervals, furnishes the necessary diagnostic evidence. Electrocardiograms in which measurements of the various intervals or the morphologic features of the ventricular complex are equivocal are not uncommon.

The diagnosis of the Wolff-Parkinson-White syndrome should be considered under the following circumstances: (1) when the P-R interval is 0.12 second or less and the QRS complex is abnormal, (2) when runs of abnormal ventricular complexes at a fast rate occur during paroxysms of auricular fibrillation, (3) when the tracing suggests bundle branch block, (4) when tracings change spontaneously, particularly if the P-R and QRS intervals change in opposite directions, (5) when the initial component of the QRS group is heavily slurred, and (6) in every patient with paroxysmal tachycardia. It should be remembered that patients seen for the first time during paroxysmal tachycardia may have received quinidine, which may have temporarily suppressed the anomalous mechanism.

The recognition of anomalous auriculoventricular excitation is of considerable importance for three reasons. The first of these is that the tracing, by virtue of its obviously abnormal appearance, frequently leads to an erroneous diagnosis of heart disease. The most common errors of interpretation are myocardial infarction, myocarditis, left or right ventricular hypertrophy, and bundle-branch block. The incorrect diagnosis of myocardial infarction can be avoided if it is remembered that QRS deflections commonly occur in leads II, III, aVF, and the right sided precordial leads, and that the S-T segments and T waves are naturally unstable in the syndrome under discussion, and, therefore, subject to spontaneous, unexplained variations. The P-J interval (sum of P-R and QRS intervals) is 0.26 second or less in most cases of the Wolff-Parkinson-White syndrome, and is greater than 0.26 second in most cases of bundle branch block. Furthermore, right bundle-branch block and anomalous auriculoventricular excitation can be differentiated by noting in the latter late "intrinsicoid" deflections in both the right and left sided precordial leads. Most errors in diagnosis will be avoided if the sound principle of refraining from making an additional electrocardiographic interpretation in tracings displaying anomalous excitation is adhered to. Finally, routine measurement of the P-R interval is the best safeguard against overlooking the syndrome. This should be done in all three standard limb leads, since the anomalous component may be isoelectric in one of them; if the measurements are made in the latter the P-R interval will appear longer, and the QRS interval shorter than they actually are.

The second reason why it is important to recognize anomalous excitation is that it masks various abnormalities. The most serious of these is myocardial infarction. Therefore, this diagnosis should never be excluded in patients with anomalous excitation merely because the electrocardiogram does not display the diagnostic QRS signs of this lesion. Suppression of the anomalous mechanism will unmask the signs of infarction, providing such a lesion exists. The electrocardiographic signs of left and right ventricular hypertrophy and right bundle branch block are also masked by anomalous excitation, and will become evident when the anomalous mechanism is suppressed.

Finally, the recognition of anomalous conduction is important because of the extremely high incidence of associated paroxysmal rapid heart action. This may point the way to the diagnosis of paroxysmal tachycardia in infants, in whom it is often an elusive diagnosis, and not rarely a fatal condition if untreated. Furthermore, in any patient, the diagnosis of anomalous excitation may call attention to the possibility that paroxysmal tachycardia is the explanation of an obscure clinical picture. Lastly, the knowledge
that a given individual has anomalous excitation may bar him from occupations in which the sudden onset of tachycardia might be hazardous.

Prognosis

There are no subjective manifestations associated with anomalous auriculoventricular excitation per se. The disorder is entirely asymptomatic and of no more than academic interest in those who have never had paroxysmal rapid heart action. Symptoms occur exclusively during the progress of paroxysmal tachycardia, or during its onset or offset. Clinical manifestations, when they do occur, are not severe, as a rule, since the heart is normal otherwise in the majority of cases. Most patients are able to live a normal, active, and even strenuous life. Occasionally, however, disability is severe, or activity is limited because of the unpredictability of paroxysmal tachycardia with consequent faintness, diminished mental alertness, loss of consciousness, or other ill effects. The occurrence of sudden death has been reported by several authors. Life Insurance experience indicates that applicants with the Wolff-Parkinson-White syndrome are not standard risks in that their mortality rate is approximately three times the normal.

Management of Patients with the Wolff-Parkinson-White Syndrome

The anomalous electrocardiogram itself is of no clinical significance and attempts to alter it should be made only for diagnostic purposes in questionable cases, or for unmasking other abnormalities. The various types of paroxysmal rapid heart action respond to the standard therapeutic measures in the same manner as in patients who do not have anomalous excitation, with one exception, namely, runs of anomalous beats at extremely rapid rates occurring during the course of paroxysmal auricular fibrillation (fig. 4). Control of the ventricular rate in these cases cannot be achieved with digitalis, and the tendency to give increasing amounts of the drug frequently leads to digitalis intoxication. The most effective treatment is quinidine, which blocks the anomalous mechanism and may, at the same time, abolish the auricular fibrillation.

Prevention of paroxysms is achieved with the same drugs and regimen as in cases of paroxysmal tachycardia not having the Wolff-Parkinson-White syndrome.

Special care during pregnancy or child birth, and special preparation preoperatively are not necessary, except in patients who at other times have frequent attacks of paroxysmal tachycardia. The standard and proved prophylactic measures should be employed in these individuals.

Activity should in no way be curtailed except when it predisposes to paroxysms, but patients should be discouraged from engaging in occupations in which sudden tachycardia might prove dangerous.

Summary

The syndrome of short P-R interval with abnormal QRS complexes and paroxysmal tachycardia occurs in otherwise healthy individuals in all age groups. Its interest and importance are related to our knowledge and concepts of the cardiac mechanism, the prevalence and clinical manifestations of paroxysmal tachycardia, the masking of the electrocardiographic signs of heart disease, and the serious consequences involved in making an incorrect diagnosis of heart disease.

A single mechanism is in all probability responsible for the abnormal electrocardiogram and the paroxysmal tachycardia. Premature activation of a small fraction of ventricular musculature shortens the P-R interval and lengthens the QRS interval, thus accounting for the electrocardiographic peculiarities of the syndrome. Whether an anomaly of impulse formation or an anomaly of conduction is responsible for pre-excitation is not known, and the propensity to paroxysmal tachycardia can be explained on either basis. There is no evidence that hitherto unknown phenomena are responsible for the syndrome. The disorder is probably congenital in nature.

A noteworthy feature is the spontaneous or induced shift, back and forth, from the abnormal to the normal type of electrocardiogram.
gram. Many drugs and procedures are available for this purpose. This is of immeasurable help in establishing the diagnosis of the Wolff-Parkinson-White syndrome in doubtful cases, and in unmasking the many abnormalities which anomalous excitation conceals.

Diagnostic errors are common, and the reasons for these have been discussed. Myocardial infarction, mitral stenosis, congenital heart disease, right and left ventricular hypertrophy, myocarditis, and bundle-branch block are the mistaken diagnoses most commonly made. Myocardial infarction, right and left ventricular hypertrophy, and right bundle branch block are the conditions most commonly concealed by the anomalous electrocardiogram.

The most important problems still requiring elucidation are those related to etiology, and to the mechanisms responsible for the abnormal electrocardiogram and paroxysmal rapid heart action.

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Syndrome of Short P-R Interval with Abnormal QRS Complexes and Paroxysmal Tachycardia (Wolff-Parkinson-White Syndrome)

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