Ventricular Activation in the Pre-Excitation Syndrome (Wolff-Parkinson-White)

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Seventy cases of pre-excitation were studied by vector translating methods. Certain properties of the delta event are described that are useful in the recognition of this syndrome electrocardiographically. In about half the cases a ventricular conduction defect without QRS-interval prolongation was demonstrated to have accompanied the pre-excitation. The type of ventricular conduction defect produced depended upon the particular direction of spread of the pre-excitation wave. This and other findings are incompatible with the hypothesis that pre-excitation is due to accelerated atrioventricular conduction and favor the hypothesis that it is due to bridges, whether morphologic or electronic, between atria and ventricles.

Two syndromes have been described that are characterized by a short P-R interval in the electrocardiogram and a predilection for paroxysmal tachycardia. Both have been called the Wolff-Parkinson-White syndrome. In one, the P-R interval is only apparently shortened, for there is an abnormal deflection immediately preceding and fusing with the QRS complex accounting for the shortened P-R interval. The abnormal deflection, called the "delta wave," is due to premature activation of a region of ventricular myocardium and therefore has become called the "pre-excitation" type of Wolff-Parkinson-White syndrome. In the other, less common type the P-R interval is absolutely shortened, there is no delta wave, and the QRS complex is normal in contour and duration. It may be called the "Lown-Ganong-Levine" type after the authors who most completely described it. The present study is concerned only with the pre-excitation type of this syndrome.

Although many case reports of pre-excitation have been published there has been no systematic, controlled study of the electrocardiographic changes that take place in this syndrome. Seventy cases of pre-excitation have been collected in which tracings had been recorded during normal conduction as well as during pre-excitation. By the use of vector-translating methods to compare the normal and the pre-excitation tracings, it has been possible to study some of the properties of the electric forces responsible for the delta waves and to examine the extent to which excitation of the remainder of the ventricular myocardium is affected by pre-excitation. The findings shed light on the probable mechanism of this syndrome in man.

Material and Methods

Twenty-eight personal cases were augmented by an additional 42 cases from the literature with satisfactory tracings during both pre-excitation and normal conduction. No cases were accepted that showed alternating pre-excitation, since one could not be sure that the abnormal complexes were not simply premature ventricular contractions. The method for plotting spatial vectors from the conventional clinical tracings has been described previously. For each case the frontal plane projections of the QRS loop during normal conduction and during pre-excitation (a delta plus QRS loop) were plotted, the same scale being used for both loops. In addition, the spatial direction of the mean delta vector was plotted from the delta waves in the various leads of the pre-excitation tracing.

The method for studying the extent to which QRS forces had been altered by pre-excitation was as follows. First, several instantaneous vectors were drawn on the QRS loop plotted from

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the tracing of normal conduction (fig. 1). These instantaneous vectors were next drawn on the delta-plus-QRS loop with exactly the same magni-
tudes and directions that they had for the normal loop, the terminus of each instantaneous vector being placed at the appropriate instant on the QRS part of the delta-plus-QRS loop. The vector at a given instant during the writing of the delta-plus-QRS loop is the sum of the delta electric force and the QRS electric force generated at that instant. Therefore, when the instantaneous QRS vectors of the normal loop are translated to the delta-plus-QRS loop, the location of the origin of each vector is also the terminus of a delta vector, if one is being generated at that instant. By drawing a line through the origins of the translated QRS vectors a loop is formed that indicates the variation in magnitude and direction of the electric force generated from instant to instant by the pre-excitation wave. This is called a “delta loop.” Delta loops were plotted by this method in each case. However, they could be considered accurate in a given case only if there had been no change in the direction of the QRS vectors as a result of pre-excitation, i.e., that there was no ventricular conduction defect with the pre-excitation. This could be determined by studying the size and shape of the loop plotted for a given case. There are several reasons for believing that the delta loop is narrow, with little variation in the direction of the electric force from instant to instant during the delta event. In the first place, the portion of the delta loop that precedes the writing of the QRS complex is always narrow. This is well shown in cases demonstrating the “concertina” effect, where there is progressively more delta wave preceding the QRS complex from deflection to deflection or from tracing to tracing in a given patient. Several excellent examples of this have been published in the literature illustrating the essentially monophasic contours of the delta waves on the various leads. In the second place, it has been shown in previous studies that when ventricular excitation travels outside the conduction system of the ventricles, as in right and left bundle-branch block, the portion of the loop written by this phase is always narrow. Finally, there are a number of cases of pre-excitation which, when the pre-excitation tracing is compared with normal conduction tracings, are clearly due to perfectly normal QRS complexes super-imposed on monophasic delta waves, and in these cases the delta loop is always narrow and small; figure 1B is an example. From these considerations it was concluded that when the plot of the delta loop was unusually large or bizarre in contour, it probably was not a true delta loop, and the QRS vectors must have been changed in direction by pre-excitation to account for the bizarre loop. This is, of course, an indirect method for deciding whether or not pre-excitation had caused a ventricular conduction abnormality. Further-
more, slight changes in ventricular conduction sequences cannot be detected by this method. Only those cases where the changes were marked were used for the analysis of the ventricular conduction defect seen with pre-excitation.

The method is illustrated by the cases in figures 1, 2, and 3. In the first case of figure 1, the delta forces are at first perpendicular to the lead I axis then point slightly rightward. When the 4 instantaneous vectors of the normal loop are superimposed on the pre-excitation loop it can be seen that their origins define an exceedingly narrow loop. The second case in this figure is another example where, even without the vector plot, the pre-excitation complexes can be seen to be formed by normal QRS complexes simply superimposed on monophasic delta waves for each lead. The contours of the QRS complexes during pre-excitation so closely resemble the normal complexes that there can have been little alteration in the sequences of ventricular depolarization in these cases and therefore the plot of the delta loop must be relatively accurate in both. Note that in the first case, the instantaneous QRS vectors are displaced from the zero point of the triaxial figure for only the first half of the QRS cycle. This means that the delta event lasted only for the first 0.04 second of the QRS interval. This case illustrates how the duration as well as the resultant magnitude and direction of the delta electric event can be measured in a given case using vector translating methods.

In figure 2 are shown 3 tracings from one subject. The 3-stage sequence illustrated here was encountered in 3 cases of the present series but in none were the tracings available for reproduction, and the deflections in this figure were copied from a case reported by Eichert. When instantaneous vectors of the normal QRS loop are translated to the loop for the second tracing, it can be seen that their origins define a small narrow delta loop indicating that there can have been little alteration in ventricular conduction as a result of this stage of pre-excitation. However, when the instantaneous vectors are translated to the loop for the third tracing, their origins define a large and bizarre pathway (dotted line) that cannot plausibly be considered to be defining delta vectors. Indeed, some parts of this loop are larger than the QRS loop itself. It is concluded that the sequences of ventricular depolarization were altered by this degree of pre-excitation, producing a left axis deviation of the QRS vectors. Figure 3 illustrates the other common type of ventricular conduction defect associated with pre-excitation. When the instantaneous QRS vectors of the normal tracing are translated to the loop drawn for the pre-excitation tracing in this case, their origins define a large and bizarre loop (dotted line) that cannot be a definition of delta vectors because it is so large. Furthermore, it defines rightward vectors, which is an exceedingly improbable direction for the vectors of the delta event. It is concluded that a ventricular conduction defect must have accompanied pre-excitation in this case, resulting in right axis deviation of the QRS vectors during pre-excitation.

RESULTS

The Delta Event

In the 70 cases, the mean vector calculated for the portion of the delta event that preceded the QRS forces had the range in direction shown in figure 4. Thirty-two or nearly half the cases had a mean delta vector that was horizontal or leftward in direction, 16 or less than one fourth were inferior or even rightward in direction, and 22 or slightly more than a fourth had a direction between these 2 extremes. The series is of course a selected one, including only those cases in which tracings with normal conduction were also available, and it may not accurately represent the true incidence of different delta vector directions in pre-excitation.

It might be wondered if this variation in frontal plane direction of the delta vector might not be simply due to different electric...
positions of the heart. This was tested by comparing the direction of the mean QRS vector of the control tracing with the direction of the delta vector during pre-excitation in all cases. There proved to be the same incidence of vertical and horizontal electric positions of the heart among cases with a leftward delta vector as among cases with an inferiorly directed vector. The different directions of the delta vector must be due to differences in direction of spread of the delta event in the ventricles rather than to differences in the electric or anatomic position of the heart.

It will be noted that when the delta vector is leftward in direction it writes a negative deflection on lead III (as in the second tracing of fig. 2) and, if markedly leftward, on lead II as well. Under these circumstances the initial take-off of the delta-plus-QRS complex on leads II and III closely resembled the Q₂ and Q₃ of diaphragmatic infarction, and several cases have in the past been erroneously called diaphragmatic infarction because of this resemblance. Similarly, when the delta vector is rightward and inferior in direction (as in fig. 3), it writes negative deflections on lead I and lead aV₉ which have occasionally been confused with Q₁ and Q₉ of anterolateral infarction.

In the past, pre-excitation has been divided into types A and B depending upon whether V₁ has an initial positive or negative deflection during pre-excitation. In the present series of cases the delta vector was more or less parallel with the frontal plane of the body in 90 per cent of cases, pointing only slightly anteriorly or posteriorly from this direction from case to case. When it pointed slightly posteriorly, the V₁ electrode location was now in the area of negativity for this vector direction and recorded an initial negative deflection. When it was directed slightly anteriorly, V₁ was now in the area of positivity, writing a positive delta wave. Thus, whether or not V₁ had a positive or a negative delta wave depended upon only slight differences in the anteroposterior direction of the delta vector in most cases, and is not a critical factor in differentiating cases of pre-excitation. Much more valuable for cataloging cases of pre-excitation is the direction of the delta vector in the frontal plane, for this appears to determine the type of ventricular conduction defect that might appear.

Two calculations of the magnitude of delta vectors were made for each case. One calculation was made from the portion of the delta vector that preceded QRS events, called the manifest delta vector, and the other was made from the maximal amplitude of the entire delta loop, when this could be reliably plotted, called the mean delta vector. In the frontal plane, the manifest delta vector varied from 2 to 10 mm. in magnitude, and the mean delta vector was as much as 15 mm. in amplitude. This means that the delta wave in a given limb lead may account for as much as 15 mm. of the amplitude of the delta-plus-QRS complex. Anteroposteriorly, its magnitude was often considerably greater. In those cases where the manifest delta vector was anteriorly directed its projection on V₁ was from 4 to 8 mm. and that of the mean delta vector as much as 25 mm. From the practical clinical point of view this means that pre-excitation must be considered whenever the initial R wave at V₁ is unusually tall or broad.

In a very small proportion of cases the delta vector had a different direction from those shown in figure 4. For example, in 3 cases the mean delta vector was directed markedly anteriorly. The delta loop was
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relatively narrow spatially in these cases, but because of the anterior direction its projection on the frontal plane was wide. The case in figure 5 illustrates this point. Here the QRS complexes have smaller amplitude during pre-excitation than during normal conduction, an uncommon occurrence in pre-excitation. The explanation for this lies in the reniform shape of the frontal plane projection of the delta loop, with much of it lying in the region of the triaxial figure, which is negative for the 3 standard limb leads. With the normal instantaneous QRS vectors originating from this region of the triaxial reference figure, the magnitude of positivity they project on the axes for the 3 limb leads is reduced, and this accounts for the reduced amplitude of the limb lead QRS complexes.

The pre-excitation events persist through only the first half of the QRS interval in this case, for the second half of the QRS complex on the various leads (and the second half of the QRS loop) has essentially the same size and contour in the pre-excitation tracing as during normal conduction. The duration of the delta event could be reliably measured only in those cases in which there was no ventricular conduction defect during pre-excitation. In over 80 per cent of these cases the pre-excitation event could be identified during only the first .04 second of the QRS interval. This plus the portion of pre-excitation that preceded the QRS events gave pre-excitation a total duration of .07 to .08 second in most cases. In none could it be shown to persist throughout the QRS interval or to extend beyond the end of the QRS interval. This confirms the generally accepted opinion that the delta event is due to depolarization of ventricular myocardium.

QRS Events

There was an unexpectedly high incidence of cases with little or no detectable change in the direction or duration of QRS forces when pre-excitation developed, 33 of the 70 cases being of this type. That QRS forces were unchanged was often not obvious from a "pattern" point of view. For example, if the delta vectors were inferior in direction, the origin of QRS vectors would now be shifted to an area on the triaxial figure where they would project negatively on lead I and the delta-plus-QRS complex would now be largely a negative deflection on this lead, whereas it was a positive deflection prior to the development of pre-excitation. This is illustrated by the first case in figure 1. Likewise in cases with markedly leftward delta vectors, the zero point for the QRS forces was often shifted to the negative region of the triaxial figure as far as the lead III axis is concerned causing the normally directed QRS vectors to write a more negative QRS complex on this lead during pre-excitation than they did during normal conduction.

Among the cases with changed directions of QRS vectors during pre-excitation the direction of the delta vector appeared to be the principal factor determining whether or not the QRS vector change would take place. For example, in the cases with leftward delta vectors, over two thirds showed changes in the directions of the QRS vectors (i.e., a ventricular conduction defect appeared during the
pre-excitation syndrome in these cases). On the other hand, among the cases in which the delta vector was directed inferiorly and rightward, only a third showed changes in the directions of QRS vectors; and among the cases with an intermediate direction of the delta vector less than one fourth showed a measurable change in direction of QRS vectors with pre-excitation (fig. 4).

Furthermore, the type of directional change which the QRS vectors were caused to take was also a function of the direction of the delta vector. Among the 32 cases with leftward delta vectors, 24 showed QRS vector alterations and 22 of these developed marked left axis deviation of the QRS vectors (third tracing of fig. 2). On the other hand, among the 16 cases with the delta vector directed inferiorly, 6 showed QRS vector changes and 5 of these developed right axis deviation of the QRS vectors (fig. 3). Only 5 of the 22 cases with intermediate delta vector direction had detectable QRS vector alterations; 3 developed some degree of the leftward type of conduction defect and 2 the rightward type.

While 85 per cent of cases with QRS vector changes developed either a marked left or a right axis deviation, other types of QRS alteration were occasionally seen. For example, among the cases where the amplitude of the delta event increased from tracing to tracing (the "concertina effect") the QRS components often became smaller and smaller without change in contour or duration. The increasing amplitude of the delta waves could usually be attributed to the fact that pre-excitation started earlier and earlier after the P wave, permitting a longer period of time for pre-excitation to invade the myocardium. The reduction in magnitude of the QRS forces when this took place is perhaps due to the fact that, as more myocardium was depolarized by the pre-excitation event, less was left for depolarization by the normal atrioventricular impulse. This type of alteration differed from the others in that there was no change in the direction of the instantaneous QRS vectors as they became reduced in magnitude. In certain other cases the directions of QRS vectors for just the first .01 to .02 second of the QRS interval were altered by pre-excitation. This suggests that not infrequently the pre-excitation wave may invade the region of the ventricles that is normally first to be depolarized, the endocardial layers of the septal and paraseptal regions of the left ventricle.

An unexpected finding was the rarity with which prolongation of the QRS part of the delta-plus-QRS interval was encountered. This was surprising in view of the fact that this syndrome was once called the "syndrome of short P-R interval with QRS prolongation." In recent years it has been recognized that much of the apparent prolongation of the QRS interval was due to the delta wave, but the notion that the QRS component is abnormal in contour and duration has persisted to the present day. Among cases of this series where accurate measurement of QRS components was possible, a measurable degree of prolongation of the QRS part of the delta-plus-QRS complex was seen in less than 10 per cent of cases, and in none was it prolonged by more than .03 second. Certain cases showed QRS prolongation in serial tracings of pre-excitation, but it is possible that the administration of quinidine may have been responsible for it.

**Discussion**

Half of the 70 cases of pre-excitation studied in this series showed little or no alteration of the QRS vectors during pre-excitation. This means that pre-excitation cannot have greatly affected the spread of ventricular ac-
tivation in these cases. However, the delta event is due to depolarization of ventricular muscle and the electric energy contained in the delta event therefore must be removed from the QRS forces that follow. Why does this change not substantially alter the magnitudes or direction of the QRS vectors? The answer perhaps lies in the observations of Schaeffer, who showed that no more than 10 per cent of the electric energy generated by ventricular depolarization is manifested in the normal QRS complex, with the other 90 per cent canceled out by simultaneous activation of oppositely located regions of the ventricles. There are probably no oppositely located regions undergoing activation during the preexcitation event and therefore, by analogy, perhaps no more than 10 per cent of the delta wave is removed from the QRS events to follow it, an amount too small to detect with conventional methods of recording. Furthermore, it has been suggested that there is normally no resultant electric activity produced by activation of the subendocardial layers of the left ventricle. The delta vector appears to be in part at least due to activation of subendocardial layers, and while not all workers are in agreement that the subendocardium is electrically "silent," if it were, then preexcitation might not significantly subtract from the manifest QRS forces that follow it.

In fact, since the preexcitation event is due to a relatively small electric force, it is surprising that QRS forces are ever altered in this syndrome. Nevertheless, half the cases of this series showed marked QRS changes during preexcitation. It was found that the particular direction of the delta vector played an important role in determining whether or not QRS changes would take place and the type of QRS change that would be produced. For example, three quarters of the cases with leftward and superiorly directed delta vectors developed QRS changes, while in the cases with leftward and inferiorly directed delta vectors QRS changes were infrequent (fig. 4). Furthermore, among the cases that developed QRS vector changes, when the delta vector was leftward and superiorly directed (left axis deviation of the delta vector), the QRS vectors also showed marked left axis deviation; in contrast, when the delta vector was inferior or rightward in direction, right axis deviation of QRS vectors took place (figs. 2 and 3).

Before an explanation for these observations is suggested, it should be recognized that in most cases the change in the directions of the QRS vectors was unassociated with measurable QRS prolongation. It had formerly been thought that ventricular conduction defects were always associated with prolongation of the QRS interval. However, this is the fourth example so far identified of a ventricular conduction defect in man in which there may be no QRS prolongation. The first to be proved in man was peri-infarction block, where the QRS vectors generated during the second half of the QRS interval are changed in direction by the infarction, and the direction they take is a function of the electric location of the myocardial infarction. The second is the S1S2S3 syndrome associated with myocardial infarction. In this the electric location of the infarct is immaterial; it is seen particularly in patients with somewhat rightward terminal QRS vectors prior to the infarct, as if there were a vulnerability of right ventricular conduction in these patients. The third type is less definitively proved than the other two; however, there is considerable circumstantial evidence that uncomplicated left axis deviation of more than −30 degrees without QRS prolongation is a left ventricular conduction defect, most commonly due to myocardial fibrosis such as is seen in advanced left ventricular hypertrophy, arteriosclerotic heart disease, etc. In support of the concept that these are all ventricular conduction defects, in spite of the normal duration of the QRS interval, is the demonstration that left axis deviation and other QRS complex abnormalities without QRS prolongation can be produced in the dog by lesions in certain regions of the left ventricular conduction system distal to the left main bundle.

The explanation for the normal QRS interval duration in these conduction defects is be-
lieved to be related to the anatomy of the left ventricular conduction system, schematically shown in figure 6. The left bundle divides into 2 major groups of fibers, one traveling superiorly and the other inferiorly, and the 2 divisions join peripherally forming a conduction syncytium. It is suggested that if a lesion involves many fibers of one of these divisions, excitation will spread via the other division with an alteration in the sequences of ventricular depolarization, but little or no prolongation of the QRS interval. According to this hypothesis the QRS interval is prolonged by conduction defects only when there is no alternative route in the ventricular conduction system for excitation to follow, for now excitation must travel by the much slower mechanism of intramyocardial spread. This is believed to be the explanation for the QRS prolongation in right and left bundle-branch block.

When pre-excitation is associated with a change in QRS vector direction, either left axis deviation of −30 to −60 degrees or right axis deviation of +80 to +110 degrees is produced and other directional changes are rarely seen. These same 2 alternative vector directions are seen in the vast majority of cases of peri-infarction block, suggesting that perhaps the 2 divisions of the left bundle play a role in pre-excitation similar to that ascribed to them in peri-infarction block.\textsuperscript{12}

These features of pre-excitation lead to the following hypothesis to explain the ventricular conduction defects in this syndrome. It is postulated that in the majority of cases of pre-excitation a stimulus from the atrium crosses the atrioventricular ring into the ventricular septum giving rise to the pre-excitation event, and this stimulus may enter at any of a number of different points in the septum. If it enters near the diaphragmatic margin of the septum (B of fig. 6), the pre-excitation wave will spread leftward and superiorly, producing a leftward and superiorly directed delta vector. With this direction the wave will soon reach the territory of the inferior division of the left bundle; if it reaches these fibers before the normal atrioventricular impulse is delivered to the ventricular conduction system, the ventricles will be depolarized from below upward via the left ventricular conduction pathway syncytium, producing left axis deviation of the QRS vectors without QRS prolongation. On the other hand, if the pre-excitation starts at the upper margin of the septum (A of fig. 6), its direction of spread will be inferior, producing an inferiorly directed delta vector. It spreads toward the territory of the superior division of the left bundle, and if it reaches these fibers before the normal atrioventricular impulse enters the ventricular conduction system, ventricular depolarization will be from above downward within the conduction pathway syncytium, producing right axis deviation of the QRS vectors without QRS prolongation. Then, if pre-excitation should start at a point in the septum between these 2 extremes it will spread leftward and inferiorly producing a leftward and inferior delta vector; but with this direction it would enter both divisions of

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**Fig. 6.** Left, Schema of the ventricular conduction system. The ventricular heart is shown as viewed frontally, with the free wall of the right ventricle partially removed. Right and left bundles are shown as if the septum were transparent. Right, 2 sites of entrance of the pre-excitation impulse are suggested. A, when pre-excitation enters from a superior region, its wave of depolarization spreads inferiorly soon encountering branches of the superior division of the left bundle. B, when pre-excitation enters from an inferior region of ventricular myocardium, its wave spreads superiorly soon encountering branches of the inferior division of the left bundle.
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The left bundle nearly simultaneously and there would be no change of the QRS vectors. This hypothesis explains why certain delta vector directions are more frequently associated with QRS vector changes than others, and why the directions the QRS vectors take is specific for that delta vector direction; it also explains why, with a relatively small part of the ventricles involved in pre-excitation, QRS vector changes are so common; and it explains why there may be no prolongation of the QRS interval with the QRS vector changes.

It follows from this hypothesis that whether the QRS vectors will be normal or not in a given case of pre-excitation will often depend upon the duration of the P-R interval, for this will determine whether the pre-excitation wave or the normal atrioventricular impulse will be the first to enter the ventricular conduction system. Perhaps this is the explanation for the "concertina" effect that Giraud was able to record in many patients with pre-excitation by taking tracings serially immediately after exercise. With the tachycardia of exercise, the P-R interval is always slightly shortened. Therefore, when patients with pre-excitation are exercised, the normal atrioventricular impulse would tend to reach the ventricular conduction system before the more slowly traveling pre-excitation wave, resulting in normal QRS forces. Then, with rest and slowing of the heart rate, the P-R interval would return to its normal longer duration; as a consequence the pre-excitation wave would reach the ventricular conduction system before the normal atrioventricular impulse, resulting in changes in the directions of QRS forces. This role of the P-R interval duration may also explain why many patients with pre-excitation have normally directed QRS forces during episodes of paroxysmal tachycardia, and why atropinization will often normalize the QRS vectors in patients with pre-excitation. Figure 7 illustrates this when the cause for the slight variation in P-R interval is a marked sinus arrhythmia. With the longer P-R intervals (C complexes), the delta wave is larger in magnitude and deformity of QRS forces greater than with the shorter P-R intervals (A complexes).

These observations also shed light on the probable mechanisms of pre-excitation in man, supporting the theory that it is due to anomalous connections between the atria and the ventricles (whether these connections are morphologic, such as the Bundles of Kent, or electrotonic as Sodi-Pallares suggests) and
tending to be incompatible with the theory of accelerated conduction along fibers of the atrioventricular node. In the first place, the delta wave spreads so slowly in the heart that, however it is initiated, its propagation cannot be along conduction fibers and must be by intramyocardial spread of activation. In the second place, for a leftward delta force to cause left axis deviation of the QRS vectors it must enter the septum low enough preferentially to invade the inferior division of the left bundle, while for an inferiorly directed delta vector to produce right axis deviation it must enter the septum high enough preferentially to invade the superior division. These 2 locations are too far apart anatomically to be attributable to fibers in or adjacent to the atrioventricular node. In fact, the wide variation in direction of the delta vector in the frontal plane in pre-excitation (fig. 4) is itself strong evidence against any theory that postulates the same site of entrance of pre-excitation into the ventricles for all cases. To explain these events by accelerated nodal conduction would require postulating that the atrioventricular impulse could be conducted to widely separated parts of the ventricles where it leaves the conduction pathways only to reenter them again in the appropriate division of the left bundle, a sequence of events which by its very complexity is improbable. Finally, the case shown in figure 8 indicates that under certain circumstances conduction through the atrioventricular node may play no part whatever in the pre-excitation syndrome. In this tracing during normal atrioventricular conduction the P-R interval has a duration of nearly .40 second, while during pre-excitation the QRS vectors are generated only .16 second after the P wave. Other, though less striking, examples have been published in the literature. Ventricular conduction must be entirely due to the pre-excitation wave in this case, for it is unlikely that both abnormally accelerated and abnormally delayed conduction could be present simultaneously in the atrioventricular node. It will be noted that there is a ventricular conduction defect without prolongation of the QRS interval in this patient during pre-excitation, and this is perhaps as nearly proof as one is likely to find in man that in certain patients with pre-excitation the abnormal QRS sequences must be related to the pre-excitation wave itself and not to any aspect of atrioventricular nodal conduction.

Several workers have tried to identify the point of origin of the delta wave, using intracavitary electrocardiographic leads. Unfortunately, it is often overlooked that deflections recorded within the chambers of the heart obey the same laws regarding the distribution of potential in a volume conductor as do deflections recorded outside the heart or on the body surface, and intracavitary deflections have therefore often been overinterpreted. A positive delta wave in the right ventricle, for example, does not necessarily mean the delta vector is pointing toward the right ventricle. It means only that the recording electrode lies in the half of the body toward which the delta vector is directed; the delta vector may have any direction through 180 degrees to write a positive deflection at any given electrode position. Nevertheless the published intracavitary findings are compatible with the hypothesis that leftward delta vectors arise from an inferior region of the ventricular mass while inferiorly directed delta vectors arise from a superior region of ventricular muscle mass.

There are, no doubt, still other mechanisms to account for the QRS changes in the pre-excitation syndrome in addition to that postulated above. For example, it has been repeatedly shown that pre-excitation complexes can be produced during right heart catheterization. In the only study of this phenomena so far published where precordial V leads were taken simultaneously with the intracavitary leads, the delta vector proved to be pointing nearly directly posteriorly (i.e., negative delta waves in all the precordial leads). This is the direction of spread one would expect of an impulse starting from the septal surface of the right ventricle, and is a direction that is rarely seen in spontaneous pre-excitation.
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SUMMARY AND CONCLUSIONS

Seventy cases of pre-excitation with tracings during pre-excitation and during normal atrioventricular conduction have been studied by vector translating methods.

The spatial direction of the mean delta vector was examined. The practice of dividing cases of pre-excitation into types A and B from the direction of the delta wave at $V_1$ proved to depend upon a trivial difference in the direction of the delta vector, for in the vast majority of cases the delta vector was more or less parallel with the frontal plane of the body.

In half of the cases the directions of QRS forces were altered by pre-excitation, indicating that a ventricular conduction defect accompanied pre-excitation in these cases. The conduction defect was unassociated with prolongation of the QRS part of the pre-excitation complex. The type of conduction defect that develops depended upon the direction of spread of the pre-excitation wave in the ventricles. An explanation for the conduction defect is suggested, based upon the bi-divisional architecture of the left ventricular conduction network.

A case of pre-excitation with P-R interval prolongation of normally conducted beats is presented. This case and other findings in the study cannot be explained by the accelerated atrioventricular conduction theory of pre-excitation, and favor the theory of anomalous bridges (morphologic or electrotonic) between atrial and ventricular myocardium.

SUMMARY IN INTERLINGUA

Septanta casos de pre-excitation con registrazionem in pre-excitation e in conduction atrioventricular normal esseva studiate per methodos de traduction vectori.

Le direction spatial del vector delta medie esseva examinate. Esseva constatate que le practica de dividir casos de pre-excitation in typo A e typo B super le base del direction del unda delta in $V_1$ depende de un differentia trivial in le direction del vector delta, proque in le grande majoritate del casos le vector delta esseva plus o minus parallel con le plano frontal del corpore.

In un mediate del casos, le directiones del fortias de QRS esseva alterate per pre-excitation. Isto indica que in tal casos un defecto in le conduction ventricular accompania le pre-excitation. Le defecto de conduction non esseva associate con prolongation del parte QRS del complexo de pre-excitation. Le typo del defecto de conduction le qual se disvelop-pava dependeva del direction del diffusion del unda de pre-excitation in le ventriculos. Es proponite un explication del defecto de conduction, basate super le architecture bipartite del rete de conduction sinistro-ventricular.

Es presentate un caso de pre-excitation con prolongation del intervallo P-R in pulsos a conduction normal. Iste caso, e etiam altere constatationes del presente studio, non es explicable per la theoria de un acceleration del conduction atrioventricular in pre-excitation. Le observationes supporta plus tosto le theoria del exsistentia de pontes anormal (morphologic o electronic) inter le myocardios atrial e ventricular.

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REFERENCES


So Nature being perfect and divine, and making nothing in vain, neither gave a heart to any where there was no need, nor made it before there was any use for it, but by the same degrees in the forming of all animals passing through the constitutions of all creatures (as I may in the egg, Worm, and birth) it acquires its perfection in them all. —William Harvey. *De Motu Cordis*, 1628.
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(Wolff-Parkinson-White)
ROBERT P. GRANT, FRED B. TOMLINSON and JAMES K. VAN BUREN

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