LETTERS TO THE EDITOR

Wolff-Parkinson-White Syndrome

To the Editor:

I read with interest the first published case of conversion of type A to type B of the Wolff-Parkinson-White syndrome by Dr. S. Ramachandran (Circulation 45: 529, 1972). However, there are still some minor discrepancies existing.

It is generally accepted that in the presence of anomalous bypass conduction or preexcitation the QRS complexes are the result of a first component due to anomalous bypass conduction and a second component due to the normal atrioventricular conducted impulse. Vera et al.1 have shown that the participation of each of these two components in building up or fusing together the final QRS complex can vary from 0 to 100%. The concertina effect is one example of these variable fusion beats.

Therefore we believe that the direction and sense of the delta-wave vector and of the main QRS vector in the more or less prolonged QRS complex of the Wolff-Parkinson-White syndrome are two independent factors which may or may not be similar in the electrocardiographic leads.

Most electrocardiographers quote the criteria of Rosenbaum et al.2 in classifying the Wolff-Parkinson-White syndrome in type A: “R is the sole or by far the largest deflection in all of these leads (V1V2)” and in type B: “S or QS is the chief QRS deflection in at least one of them (V1V2).”

All kinds of QRS complexes (R, Rs, QS, qR) have been found in lead V1 with preexcitation and we believe it is more correct to distinguish the A from the B type in the preexcitation syndrome by taking only into consideration the initial delta-wave vector or the 25- or 30-msec vector as is usually done in vectorcardiography.3

Thus a positive delta wave or a negative delta wave in lead V1 means type A or type B, respectively, in the Wolff-Parkinson-White syndrome, regardless of the direction and sense of the main QRS vector. This simplification excludes a rare third or fourth type.

In the same article Rosenbaum et al.2 also showed evidence of their hypothesis that an accessory atrioventricular bundle can hardly transmit the excitatory process from auricles to ventricles when the cardiac rhythm is under control of a center in the lower levels of the atrioventricular node or the anomalous ventricular complex assumes the normal form when the pacemaker shifts to the lower levels of the junctional tissues.

These authors showed several ECGs with the Wolff-Parkinson-White syndrome with changes in the main QRS vector during the transition from sinus rhythm to atrioventricular rhythm. Sherif and James4 stressed the fact of the high frequency of supraventricular ectopic centers in patients with preexcitation syndrome: “Usually these supraventricular ectopic rhythms are faster than the sinus rhythm, actively taking over the pacing of the heart and in most cases producing a concomitant change in QRS pattern.”

When we now look carefully to the ECG (fig. 2) of the case published by Dr. Ramachandran with Wolff-Parkinson-White syndrome, type A, it will be noticed that the initial delta-wave vector not only in V1 but in all precordial leads remained positive and unchanged in the two ECGs presented. In the horizontal plane the delta-wave vector is still anteriorly and of type A. If it is correct that type A has its anomalous bypass conduction in the posterobasal part either of the right or left ventricle and type B its bypass in the anterolateral margin of the A-V sulcus of the right ventricle, it would be hard to accept a change of anatomic site of preexcitation without a change in direction and sense of the initial vector in the horizontal plane except if these two bypass tracts would be anatomically unusually closely situated.

Otherwise the ECG in figure 2 on the second day shows negative P waves in leads III and V1 and flat in aVL which were all positive on the first day. Also the P-R which was long, more than 0.12 on the first day, is now shortened on the second day. This means that an ectopic supraventricular focus is taking over from the sinus node. This lower situated pacemaker could result in fusion complexes of diminished or increased preexcitation and aberrant conduction through the normal atrioventricular pathway.

For these reasons, which are not fully explained in the article by Dr. S. Ramachandran, we are still not convinced that the ECG shown in figure 2 signifies the existence of a type A and a type B pattern in the same patient with Wolff-Parkinson-White syndrome.

Of interest are the facts that in 1945 Rosenbaum et al.2 suggested the existence of one or more additional atrioventricular bundles in the Wolff-Parkinson-White syndrome. Boineau et al.5 have shown earlier electrophysiologically and histologically the existence of two anomalous atrioventricular muscular connections precisely at the locations of the biventricular preexcitation in a squirrel monkey.

As Hecht6 said: “The Wolff Parkinson White syndrome has always been a very interesting syndrome, good for many publications. It always entertained us and will fortunately do so for quite some time more.”

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References

2. ROSENBAUM FF, HECHT HH, WILSON FN, JOHNSTON FD: The potential variations of the thorax and the esophagus in anomalous atrioventricular excitation
(Wolff Parkinson White Syndrome). Amer Heart J 29: 281, 1945


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