Left ventricular hemiblock patterns in ventricular pre-excitation

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In a study of 32 cases of ventricular pre-excitation, 12 of 18 cases of type B and 2 of 14 cases of type A were found to have left axis deviation. In 6 (1 type A and 5 type B) of the 11 (4 type A and 7 type B) who had intermittent pre-excitation, a striking shift to the left occurred in the frontal-plane axis with the transition from normal conduction to pre-excitation; that is 5 of 7 type B and only 1 of 4 type A. Grant's hypothesis that the frequency of left axis deviation may be explained by premature excitation of either division of the main left bundle-branch is therefore not compatible with the currently held belief that type A represents left, and type B right, ventricular pre-excitation.

We have been interested in the association of ventricular pre-excitation and complete bundle-branch block (Robertson *et al.*, 1963) and the clues provided by this association in determining which ventricle is excited early. We now consider that pre-excitation combined with ventricular conduction defects of lesser degree deserve closer attention. Their study is best made either on patients with intermittent ventricular pre-excitation or on those whose pre-excitation is constant except during attacks of supra-ventricular tachycardia in which the delta wave is no longer evident. Others (Grant, Tomlinson, and Van Buren, 1958; Bleifer *et al.*, 1959) have commented on the striking difference between the scalar and vector electrocardiograms during normal ventricular conduction and pre-excitation in such patients. The dissimilarity is difficult to ascribe to the addition of a delta vector to the normal vectorcardiogram, and Bleifer *et al.* (1959) concluded that pre-excitation altered the entire course of the spread of excitation in both ventricles. Grant *et al.* (1958) found that in about half of their cases, when the delta vector is ignored, the change from normal conduction to pre-excitation was accompanied by a striking change of the frontal-plane axis without prolongation of QRS. They attributed this to a left ventricular conduction defect based upon the bi-divisional structure of the left ventricular conduction pathways; in other words, what would now be called left ventricular hemiblock (Rosenbaum, Elizari, and Lazzari, 1967). With this aspect in mind

we have reviewed our series of 32 cases of ventricular pre-excitation with particular reference to the association of left ventricular hemiblock patterns.

Case material

This series of 32 cases (Table 1) showed the usual male predominance and the expected proportion of about 70 per cent of cases with paroxysmal tachydysrhythmia (Chung, Walsh, and Massie, 1965).

The cases were classified in the commonly accepted though arbitrary fashion into type A and type B (Rosenbaum *et al.*, 1945; Bleifer *et al.*, 1959). This is dependent on the configuration of the QRS complexes in the right praecordial leads. In type A they are predominantly positive and in type B predominantly negative; the delta wave is positive in type A but variable in type B. An alternative classification proposed by Tranchesi *et al.* (1959) based solely on the polarity of the delta waves in the right praecordial leads has not come into popular use and we have not used it.

The frontal-plane axis lay between 0° and 90° in 14 cases and in only 4 cases, all type A, was it greater than 90°. In 14 cases (12 of the 18 cases of type B and 2 of the 14 cases of type A) it lay between 0° and -90°.

In 8 cases of intermittent pre-excitation and in 3 others who lost their delta waves during paroxysmal dysrhythmia it was possible to compare the mean frontal-plane axis in the presence and absence of pre-excitation (Tables 2 and 3). In 2 cases of type A and 1 case of type B the axes shifted 18°, 25°, and 47° to the right with the change from normal conduction to pre-excitation. In 2 patients, 1 type A and 1 type B, the axes shifted 15° and 37° to the left. However, in 6 patients (5 type B and 1 type A) there was a striking shift of the axis to the left (55° or more) after the change to pre-excitation (Fig. 1).

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TABLE 1 Frontal axis in patients with ventricular pre-excitation

Case No.	Sex	Age (yr)	Type of pre-excitation	Scalar electrocardiogram (frontal axis)	Dysrhythmia
1	M	12	B	-75°	Supraventricular paroxysmal tachycardia
2	F	9 wk	A	-74°	Supraventricular paroxysmal tachycardia
3	M	28	B	-67°	Paroxysmal atrial fibrillation
4	F	13	B	-45°	—
5	F	40	B	-42°	Paroxysmal atrial fibrillation
6	F	38	B	-39°	Paroxysmal atrial fibrillation
7	M	5	B	-34°	Supraventricular paroxysmal tachycardia
8	M	48	B	-22°	—
9	M	45	B	-22°	Supraventricular paroxysmal tachycardia
10	M	12	B	-21°	Supraventricular paroxysmal tachycardia
11	M	39	B	-16°	Supraventricular paroxysmal tachycardia
12	M	21	B	-13°	—
13	M	60	A	-7°	Supraventricular paroxysmal tachycardia
14	F	7	B	-5°	Supraventricular paroxysmal tachycardia
15	M	5	B	+6°	—
16	F	24	A	+30°	Supraventricular paroxysmal tachycardia
17	F	31	A	+38°	Supraventricular paroxysmal tachycardia
18	M	52	B	+45°	Supraventricular paroxysmal tachycardia
19	F	62	A	+46°	Supraventricular paroxysmal tachycardia
20	M	53	B	+47°	Supraventricular paroxysmal tachycardia
21	F	4	A	+63°	—
22	M	49	B	+66°	—
23	M	50	A	+69°	Supraventricular paroxysmal tachycardia
24	M	38	B	+72°	—
25	F	18	B	+74°	Supraventricular paroxysmal tachycardia
26	M	50	A	+78°	Paroxysmal atrial fibrillation
27	M	15	A	+90°	—
28	M	16	A	+90°	Supraventricular paroxysmal tachycardia
29	M	39	A	+104°	—
30	M	28	A	+105°	Supraventricular paroxysmal tachycardia
31	F	21	A	+110°	Supraventricular paroxysmal tachycardia
32	F	46	A	+141°	Supraventricular paroxysmal tachycardia

TABLE 2 Change in frontal axis in intermittent pre-excitation

Case No.	Type of pre-excitation	Frontal axis in scalar electrocardiogram		Axis shift during pre-excitation
		Normal conduction	Pre-excitation	
21	A	+38°	+63°	25° to right
27	A	+72°	+90°	18° to right
15	B	+21°	+6°	15° to left
14	B	+50°	-5°	55° to left
12	B	+45°	-13°	58° to left
9	B	+60°	-22°	82° to left
7	B	+67°	-34°	101° to left
2	A	+167°	-74°	241° to left

TABLE 3 Change in frontal axis when pre-excitation disappears during paroxysmal dysrhythmia

Case No.	Type of pre-excitation	Frontal axis in scalar electrocardiogram		Axis shift during pre-excitation
		During paroxysm of dysrhythmia with loss of pre-excitation	During pre-excitation	
25	B	+27°	+74°	47° to right
17	A	+75°	+38°	37° to left
6	B	+35°	-39°	74° to left
9	B	+63°	-22°	85° to left
7	B	+82°	-34°	116° to left
2	A	+196°	-74°	270° to left

FIG. 1 Case 7. A boy of 5 years. (a) 11 April 1968: intermittent ventricular pre-excitation, type B. Note the left axis deviation when the limb leads are recorded during pre-excitation. (b) 29 February 1968. In this tracing pre-excitation is only recorded in leads V1, V2, and V5. Note the conspicuous difference in frontal-plane axis as compared with (a). (c) Record of a paroxysm of supraventricular tachycardia. The delta wave is not evident. Note that the frontal-plane axis is conspicuously different from that recorded during pre-excitation and is similar to that recorded during normal conduction. (d) Vectorcardiograms. The upper row shows type B pre-excitation with slowly inscribed delta vector orientated to the left. The bottom row shows normal conduction. Note the change in configuration of the QRS loops in the frontal plane correlating with the differences in frontal axis in the scalar electrocardiograms.

Corresponding changes in the mean QRS horizontal-plane axes were not so striking as those in the frontal plane and were most obvious in cases of type A pre-excitation, with reversed polarity in the right precordial leads (Fig. 2).

Discussion

The title of the classical paper by Wolff, Parkinson, and White (1930) was 'Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia'. When the mechanism of pre-excitation and the genesis of the delta wave

were understood better (Holzmann and Scherf, 1932; Wolfarth and Wood, 1933), opinions on the lengthening of QRS changed and it was then attributed to the addition of a delta wave to a QRS complex of normal duration. It was commonly stated that a small 'pre-excited' area of ventricular muscle was prematurely depolarized and that thereafter depolarization of the rest of the myocardium proceeded in the usual manner by the normal conducting pathways. However, evidence has accumulated over the years indicating that this view of cardiac conduction in pre-excitation is much too simple.

FIG. 2 Case 17. A woman of 31 years. (a) Type A ventricular pre-excitation. (b) Paroxysm of supraventricular tachycardia with loss of delta wave. Note the reversed polarity in lead V1.

All possible combinations of pre-excitation and bundle-branch block in the same ventricular complex have been reported (Robertson *et al.*, 1963; Laham, 1969), i.e. type A (assumed left ventricular) pre-excitation with left or right bundle-branch block and type B (assumed right ventricular) pre-excitation with left or right bundle-branch block. Moreover, ventricular pre-excitation may alternate with bundle-branch block in the ventricular complexes and this has most often been commented upon in type B pre-excitation and right bundle-branch block.

Extreme degrees of axis deviation (chiefly left) have also aroused interest. Bleifer *et al.* (1959) found a high incidence of left axis deviation in type B pre-excitation; of 22 cases, 15 had left axis deviation and 7 a normal axis. By contrast, in 11 cases of type A, 3 had left axis deviation, 5 had normal axis, and 3 right axis deviation. Our series also shows a higher proportion of patients with type B (12 out of 18) than type A (2 out of 14) pre-excitation manifesting left axis deviation. To understand these ratios it is important to recall the work of Grant *et al.* (1958) who studied 28 of their own patients and 42 reported cases, all of whom had intermittent pre-excitation. Of these 70 cases, 35 showed little or no alteration of QRS during episodes of pre-excitation except for the addition of the delta wave and the authors thought that pre-excitation could not have greatly affected the subsequent spread of ventricular excitation. In the other 35, however, the pre-excitation was associated with a striking change in the frontal-plane QRS axis. Of 32 cases with delta

vectors directed to the left and superiorly, 22 showed a conspicuous deviation of the QRS axis to the left during pre-excitation when compared with that during normal conduction, and 2 showed a shift to the right. Of 16 cases with a delta vector directed to the right and inferiorly, only 6 showed a significant shift of the QRS axis during pre-excitation, 5 to the right and 1 to the left. Of 22 cases with delta vectors intermediate between these two extremes, only 5 showed significant shifts of the QRS axis, 3 to the left and 2 to the right.

Grant explained these findings in terms of the bifascicular division of the left bundle-branch. Pre-excitation in the inferior aspect of the interventricular septum would account for a delta vector pointing to the left and superiorly, and earlier excitation of the posteroinferior division of the left bundle-branch would produce left axis deviation as it does with left anterior hemiblock. Similarly, pre-excitation of the superior aspect of the interventricular septum would give an inferiorly directed delta vector, and premature excitation of the anterosuperior division of the left bundle-branch would produce right axis deviation, as it does with posterior hemiblock. Grant's concept is thus in keeping with later work on axis deviation related to the asynchronous excitation of the two major divisions of the left bundle-branch (Rosenbaum *et al.*, 1967).

Our findings (Tables 1-3) are in keeping with those of Grant. When ventricular pre-excitation replaces normal conduction, there is a striking shift in the frontal-plane axis in about half of the cases and it is more often to the left than to the right. In seek-

ing an explanation for this, one should recall that the larger part, about two-thirds, of the left side of the interventricular septum is supplied by the posteroinferior division of the left bundle-branch and only about one-third by the anterosuperior division. If pre-excitation sites on the left side of the interventricular septum are distributed in a random manner, the posteroinferior division of the left bundle-branch should be prematurely excited oftener than the anterosuperior division; this might be the explanation of the greater incidence of left axis deviation. Such an explanation is not, however, compatible with the fact that left axis deviation is characteristic of type B (assumed right ventricular) pre-excitation and not type A (assumed left ventricular) pre-excitation. This paradox emphasizes our ignorance regarding the sites of ventricular pre-excitation and the consequent pathways of ventricular excitation. A rational classification of ventricular pre-excitation must await detailed mapping of ventricular excitation pathways in a large number of cases of ventricular pre-excitation.

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