CASE REPORTS

OCCASIONAL APPEARANCE OF QRS COMPLEXES WITH VENTRICULAR PRE-EXCITATION

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There are infrequent instances when cardiac beats with the electrocardiographic characteristics of the pre-excitation type appear occasionally in an otherwise normal tracing (Sanghvi and Misra, 1958). A recent observation helped us to clarify the problem of diagnosis and interpretation, and we present the following case history because of its practical and theoretical interest.

B. T., a 48-year-old barmaid, complained of amenorrhœa of two years' duration, exertional dyspnoea, and paroxysms of tachycardia, mostly nocturnal and of variable duration.

Electrocardiograms were recorded in August, September, and November 1962.

The first record (August) is illustrated in Fig. 1, and shows sinus rhythm with an average rate of 75 to 80, and the RR intervals range between 0.8 and 0.75 sec. Atrio-ventricular conduction ($PQ = 0.18$ sec.) and QRS duration (0.09 sec.) were both normal. Besides the normally conducted sinus beats, there were occasional beats preceded by P waves of normal configuration similar to the P waves with normally con-

FIG. 1.—Cardiogram (August 1962) showing pre-excitation beats. (A) Continuous strip—lead II; (B) aVF; (C) V₁. (For details, see text.)

FIG. 2.—Cardiogram (September 1962) showing pre-excitation beats. (A) Lead III; (B) continuous strip, lead II. (For details, see text.)

ducted sinus beats. The PQ interval was constantly shorter than the basic one, and was followed by a QRS showing an aberrant morphology characterized by a slow, thick beginning of the R wave (with the "delta" wave aspect), an increase in the QRS duration, and an alteration of the ST segment with an opposite polarity and terminal negativity of the T wave. Such beats appeared over long strips with a certain rhythm, mostly occurring after three normally conducted beats: sometimes the pace lengthened and then they occurred after five beats, or they faded away over strips of variable length. This phenomenon was most evident in the limb leads I, II, III, AVF, and the right præcordial, mainly in V1, where the "delta" wave was positive and sluggish. The characteristic elements of these complexes (short PQ, widened QRS due to "delta" waves, alteration of ST-T segment) showed small variations with an inversely proportional relation between short PQ and aberrant QRST.

The following features should be stressed: (1) even at a slight variation of the basic rate the PPwpw interval was unchanged compared with the PP; (2) the PQ interval of the WPW beats was constantly shorter than the basic PQ (PQ = 0.18 sec., PQwpw = 0.11–0.14 sec., respectively); (3) the aberrant conduction, i.e. the width and the height of the "delta" wave, was greater in the beats with a shorter PQ interval; (4) the PJ interval of the WPW beats was practically the same as that of the normal beats; (5) the RWRw intervals were almost identical, being a multiple of the basic interval and ranging between 0.78 and 0.8 sec.

A second cardiogram (September 1962), illustrated in Fig. 2, failed to reveal in its initial strip any anomaly either of rhythm or conduction. During the recording of lead III (Fig. 2) the patient was invited to carry out a forced inspiration: this induced a moderate stepping up of the rate and a succession of two normally conducted beats with a PP period of 0.75 and 0.7 sec. respectively. The last cycle was followed by a longer pause, with an interval of 1.0 sec. from the last P, at the end of which an anomalous beat appeared. This was represented by an initial sluggish thick portion followed by a faster rise of the positive wave; the second half of the QRS had a reversed polarity with a depressed ST segment and an inverted T wave. After an RR period of 1.04 sec., the first normally conducted post-extrasystolic beat was preceded by a slightly diphasic P wave which had a lower voltage than the positive P waves preceding the extrasystole. The PQ interval was 0.16 sec. The QRS complexes which followed showed aberrant ventricular conduction which was related to

the P wave which precedes them at a distance of 0.10 and 0.12 sec., always shorter than the basic PQ interval.

Following lead III, lead II was recorded continuously (Fig. 2B). Here the P waves followed each other regularly with PP intervals ranging from 0.71 sec. to 0.83 sec. However, the ventricular complexes were slightly aberrant: there was a constant relation with the P wave, though the PQ intervals remained below the basic value and varied from 0.11 to 0.13 sec. except for three complexes where PQ was 0.14 sec. in two, and 0.15 sec. in one respectively. The aberrant ventricular conduction showed itself in a thickened "delta" wave with a minimal upward deflection; the PJ interval calculated on all the complexes in lead II showed only a minimal variation of 0.02 sec., though the last three beats with an aberrant QRS had a PQ conduction time slightly above the ones recorded in similar WPW beats: this always remained below the atrio-ventricular conduction time of normal beats. The width of the QRS in the normally conducted beats varied within the very narrow limits of 0.08–0.10 sec.; in contrast, the QRS of WPW type varied from 0.12 to 0.15 sec.

The WPW beats in Fig. 2B tended to appear at regular intervals. In the recorded sequence (Fig. 2B), beats, anomalously conducted, followed each other exhibiting predominantly 3:1 conduction, occasionally 5:1 conduction and, in one instance, 1:1 conduction. Where the succession occurred with 3:1 conduction, the RR intervals of the WPW complexes were of a duration from 3.25 to 3.28 sec., thus being multiples of a basic value of 0.82 sec.; where the relation was 5:1 the intervals varied from 4.59 to 4.85 sec., thus being multiples of a basic value of 0.79 sec.; where the succession took place with 1:1 conduction the RR distance of the two WPW complexes was equivalent to 1.62 sec., equal to twice the basic value of 0.81 sec. The average RR period of the normally conducted beats was of the same duration in this part of the record. The same mathematical relation existed in other sequences of the record where aberrant conduction occurred.

The cardiogram recorded in November did not exhibit any WPW complexes. There was sinus rhythm throughout with normal A-V conduction and a QRS of 0.08 sec. Nor did the exercise test elicit any anomalously conducted beats though it produced a slight elevation of the J junction with an upward convexity of ST terminating in an inverted and deeper T wave than in the recording at rest.

Discussion

The aberrant complexes cannot be due to the activation of an acquired ventricular extrasystolic focus as postulated by Bandiera and Antognetti (1958). The regular succession of the PP intervals independent of the variations of intraventricular conduction, the delta wave shortening the PQ interval and concomitantly widening QRS, and the constancy of P–J, both in the normally conducted as well as the aberrant beats, suggest that the aberrant QRS complexes are conducted from the atria. The accelerated A–V conduction may be explained by a fast atrio-ventricular bypass (Katz and Pick, 1956), regardless of whether this is produced by an accessory conduction pathway, or by an accelerated conduction in the specific system of Tawara (Prinzmetal *et al.*, 1952), or by a mechanism of electrical atrio-ventricular synchronization (Segers, 1951), permitting an accelerated conduction on the basis of a certain periodicity.

It is difficult to conceive that the functional state of the accessory pathway should be such as to conduct impulses at fixed regular intervals. A state of hyperexcitability, on the other hand, may easily explain the electrocardiographic features of this case.

Summary

In a case of paroxysmal tachycardia QRS complexes of pre-excitation type appeared at fixed intervals in an otherwise normal electrocardiogram. This is contrary to the idea of an accessory pathway and illustrates hyperexcitability as a cause of accelerated conduction.

References

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