Two Active Junctional Pacemakers Manifested by Atrioventricular Dissociation and Junctional Captures*

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A case of atrioventricular dissociation with RP-dependent abrupt prolongation of ventricular cycles is presented. The prolongation was less than expected with concealed junctional captures. It is assumed that resetting of an upper junctional pacemaker allowed impulses from a lower one to escape.

Numerous reports on concealed junctional captures in atrioventricular dissociation have been reviewed recently.1 A sudden prolongation of the R-R interval that is critically dependent on the relative timing of the R and P waves is the distinctive mark of this phenomenon. The long R-R interval due to the resetting of the junctional pacemaker by a sinus impulse equals the sum of the following four components: (1) the R-P interval; (2) the time of conduction from the atria to the junctional pacemaker (A-J); (3) one cycle of the junctional rhythm; and (4) the time of conduction from the junctional pacemaker to the ventricles (J-V). Such a prolonged cycle may be cut short by a subsequent sinus impulse which captures the ventricles before the arrival of the junctional impulse. When the R-R interval is suddenly prolonged but falls short of the expected length in the absence of a ventricular capture, the mechanism requires explanation.

CASE REPORT

A 66-year-old woman with cardiomegaly and the electrocardiographic pattern of incomplete left bundle-branch block was treated with digoxin, furosemide, and quinidine for congestive failure and paroxysmal atrial fibrillation. In the electrocardiogram taken on Feb 1, 1976, at least five different shapes of P waves were discernible, and the atrial rhythm was grossly irregular. Atrioventricular dissociation with single and consecutive ventricular captures was present. Some groups of atrial and ventricular deflections suggested reciprocation in the atrioventricular node (not shown). Occasionally, the P waves were uniform and probably of sinus origin. A somewhat accelerated, fairly regular junctional rhythm with a cycle length of 0.79 to 0.83 second resulted in atrioventricular dissociation; the P waves either slightly preceded or merged with the QRS complexes.

Repeatedly (five times in the long record), slowing of the sinus rhythm caused the P waves to appear after the QRS complex, with the R-P intervals becoming progressively longer; when they reached 0.19 second, ventricular captures with markedly prolonged atrioventricular conduction times appeared, followed by the usual slight shortening of the subsequent R-R interval due to the prolonged J-V conduction of the capture impulse. There was no manifest capture when the R-P interval measured only 0.04 to 0.16 second; instead, the junctional cycles, as represented by the R-R intervals, were suddenly prolonged to 0.92 to 0.95 second. Two consecutive instances of such a progressive prolongation of the R-P interval were selected for presentation (Fig 1).

The arrhythmia was evanescent. Chronic atrial fibrillation supervened. No electrophysiologic studies have been performed; one long tracing from the surface ECG was available for analysis.

DISCUSSION

The sudden prolongation of R-R intervals could have been attributed to a resetting of the junctional pacemaker by a sinus impulse which subsequently underwent extinction before reaching the ventricles; however, such a resetting would result in a considerably longer ventricular pause. The observed lengths could not accommodate the four components listed previously; the observed intervals would provide no margin for A-J and J-V conduction time, even on the improbable assumption that these times were not prolonged. A short-cutting by a ventricular capture was excluded.

We propose to account for the discrepancy by assuming that a lower, and accordingly slower, junctional pacemaker was available but was usually being discharged prematurely by impulses coming from above.
When an occasional decrementally conducted sinus impulse succeeded in discharging the upper pacemaker without reaching the lower one, conditions were created in which the lower focus could escape. Thus, the prolonged R-R interval resulted, which was approximately as long as the cycle of the lower pacemaker and shorter than expected with a simple junctional capture. (While this paper was being prepared, the case of Nordenberg and associates7 came to our attention; in the legend to Figure 1 of their report on sinus nodal dysfunction, they consider the same explanation for a similar phenomenon.)

Alternative explanations of the observed prolongation of the R-R interval come to mind. The prolongation could have been due to chance; since the same arrhythmic pattern repeated itself five times, this assumption seems highly improbable. A possible direct humoral or autonomic depression of both the sinus and the junctional pacemaker could have caused their simultaneous slowing. These two interpretations cannot be ruled out with certainty.

We favor the mechanism diagrammed in Figure 1 for the following reasons: (1) parts of the record which are not shown supply ample evidence for diffuse dysfunction of the conduction system, especially in the atrioventricular junction (as noted above); (2) different levels of impulse penetration in atrioventricular conduction have been documented in animals and in man;8,9 (3) the mechanism of capture was present in this case, as testified to by the manifest ventricular captures; and (4) in the long record, no concomitant fluctuation of P-P and R-R cycles was seen, and abrupt prolongation of R-R intervals occurred only in a setting in which concealed atrioventricular conduction was expected to produce "a disturbance of the impulse formation of the nodal pacemaker" as originally formulated.9 The purpose of our report was to draw attention to a peculiar variant of this disturbance.

REFERENCES