Dissociation of the Atrioventricular Node in Acute Inferior Wall Myocardial Infarction*

2. Longitudinal Dissociation (Dual Atrioventricular Nodal Pathways)

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Four cases of longitudinal dissociation of the atrioventricular node, with dual pathways developing during the acute phase of an inferior wall myocardial infarction (three cases) or during acute ischemia (one case), are presented. In all four cases, two grossly different P-R intervals were recorded, and in two cases, studies of the His bundle confirmed the location of the dissociation within the atrioventricular node. In one case, premature atrial depolarization caused a bidirectional shifting of P-R intervals, while in the remaining three cases, premature ventricular depolarization (spontaneous or pacemaker-induced) was responsible for this phenomenon. In all cases, evidence of longitudinal dissociation of the atrioventricular node appeared during the acute phase of the infarction or ischemia, and in all of them the phenomenon was transient. This favors the assumption that this phenomenon is of a functional nature, most probably related to the ischemic lesion of the atrioventricular node.

Since the early report of Moe et al., there has been increasing evidence of the phenomenon of longitudinal dissociation of the atrioventricular node, with dual pathways showing different functional properties. So far, longitudinal dissociation of the atrioventricular node has been accepted as the mechanism for the occurrence of two grossly different P-R intervals in the same patient.

The clinical significance of this phenomenon has been stressed in recent reports, where the presence of dual atrioventricular nodal pathways has been found to be a common electrophysiologic response to atrial extrastimuli. Moreover, a relationship has been demonstrated between this phenomenon and paroxysmal supraventricular tachycardias, as well as with tachycardias associated with syndromes of preexcitation.

Our purpose is to present four cases of transient longitudinal dissociation of the atrioventricular node in the setting of an acute inferior wall myocardial infarction or ischemia.

CASE REPORTS

CASE 1

A 55-year-old woman was admitted to our coronary care unit following an episode of severe pain in the chest. The electrocardiogram obtained on admission was diagnostic of acute inferior wall myocardial infarction. On the second day of hospitalization, two grossly different P-R intervals were observed. An electrode pacemaker-catheter was located in the right ventricular apex. Short periods of ventricular pacing produced, at will, shifts in the P-R interval. In the upper strip of Figure 1 (lead 2), the first three beats originate from the sinus node and are conducted with a P-R interval of 180 msec; the following six beats were induced by ventricular pacing and continued by the same sinus rhythm, but with a P-R interval of 400 msec. In the lower strip of Figure 1, the ventricular pacing shifted the P-R interval from 400 msec to 180 msec. It is of note that the shift of P-R intervals was clearly dependent on the "R-P" interval. With a long R-P interval (820 msec), the fast pathway was recovered for conduction, and a short P-R interval resulted; but with a short R-P interval (400 msec), the fast pathway was refractory, and the slow pathway was utilized for conduction, resulting in a long P-R interval. The long P-R interval was maintained most probably by repetitive retrograde concealed conduction to the fast pathway.

CASE 2

A 68-year-old man with a past history of old anterior acute myocardial infarction was admitted to our coronary care unit following an episode of severe precordial pain. The ECG obtained on admission was consistent with an acute inferior wall myocardial infarction with right bundle branch block and left anterior hemiblock. On the following day, two grossly different P-R intervals were recorded. An electrode pacemaker-catheter was located in the right ventricular apex.

In Figure 2A the presence of a prolonged P-R interval of 440 msec is observed, interrupted by a pacemaker-induced ventricular extrastimulus and followed by sinus rhythm with a small increase in rate and with a shorter P-R interval of 260 msec; this rhythm is again interrupted by a coupled and short run of ventricular extrastimuli, followed by sinus
rhythm with a prolonged P-R interval of 440 msec. Study of the His bundle (Fig 2B) revealed two different P-R intervals (140 and 400 msec) due to changes in the atrio-His (A-H) interval. The application of ventricular extrastimuli provoked a shifting of P-R intervals. As seen in case 1, the shift of P-R intervals was dependent on R-P intervals; with a long R-P interval, a short P-R interval resulted, while a short R-P interval was followed by a prolonged P-R interval.

Case 3

A 61-year-old woman was admitted to our coronary care unit following a history of severe pains in the chest prior to admission. The ECG and serum levels of cardiac enzymes were typical for acute inferior wall myocardial infarction. On the third day of hospitalization, two grossly different P-R intervals were recorded on the ECG.

A study of the His bundle performed simultaneously (Fig 3) showed that the changing delays in atrioventricular conduction occurred in the atrioventricular node. A shift of P-R interval (A-H interval) from short to long was induced by a premature atrial depolarization with a coupling interval of 400 msec, and a shift of the P-R interval (A-H interval) from long to short was induced by a blocked premature atrial depolarization with a coupling interval of 380 msec. The intravenous administration of atropine did not alter the occurrence and characteristics of the phenomenon. On the following day the phenomenon could not be recorded, despite atrial or ventricular stimulation.

**Figure 2.** A, Electrocardiogram. Upper strip demonstrates prolonged P-R interval interrupted by ventricular extrastimulus (VE) and followed by short P-R interval. Lower strip shows several ventricular extrastimuli shifting P-R interval from short to long. Ladder diagram represents double atrioventricular nodal pathway. AT, Atrium; AV, atrioventricular nodal conduction; V, ventricle; F, fast atrioventricular nodal pathways; and S, slow atrioventricular nodal pathway. B, His bundle electrogram (HBE) reveals two different A-H intervals. Shifting between P-R intervals is induced by ventricular extrastimulus. Measurements are in milliseconds. HV, His-ventricle interval (case 2).
Case 4

A 77-year-old man was admitted to our coronary care unit because of a history of unstable angina with severe pains in the chest. His medical history revealed old anterior and inferior myocardial infarctions. The ECG obtained on admission showed new ischemic changes in the inferior wall. Serum levels of cardiac enzymes remained normal throughout hospitalization. On the first day of hospitalization, while the patient complained of pain in the chest, two different P-R intervals were recorded. Figure 4 shows a bidirectional shifting of the P-R intervals by a premature ventricular depolarization from 200 msec to 400 msec. This was observed to occur spontaneously, and shifting of the P-R intervals was always induced by premature ventricular depolarizations for periods lasting a few hours. On the following day, while the patient was free of pain and the electrocardiographic signs of ischemia disappeared, an electrophysiologic study failed to demonstrate the presence of dual pathways in the atrioventricular node.

Discussion

Recent electrophysiologic studies from several laboratories have suggested that the atrioventricular node can longitudinally dissociate into two pathways with different functional properties. The presence of discontinuous curves of atrioventricular nodal conduction or of two grossly different P-R intervals (or both) has been the strongest suggestion for the existence of longitudinal dissociation with dual pathways. This phenomenon has acquired great clinical significance since it has been related to paroxysmal supraventricular tachycardias and tachycardias occurring in the syndromes of preexcitation. It is suggested that in paroxysmal supraventricular tachycardia the mechanism of reentry is usually characterized by conduction via anterograde slow and retrograde fast pathways, although in a recent report an inverse mechanism has been postulated. In the Wolff-Parkinson-White syndrome, each of the dual pathways could be part of the reentrant loop, causing paroxysmal supraventricular tachycardia inde-
dependent or in association with the anomalous pathway.\textsuperscript{16}

Our four cases meet the criteria for longitudinal dissociation of atrioventricular node; all of the cases had two grossly different P-R intervals, and in two of them, the study of the His bundle demonstrated two different A-H intervals (Fig 2B and 3). The shifting of P-R intervals was induced by a premature atrial depolarization in case 3 and by premature ventricular depolarizations in the other three cases. Both atrial and ventricular premature depolarizations penetrated the atrioventricular node nonhomogeneously and facilitated the longitudinal dissociation. In cases 1 and 2, pacing-induced premature ventricular depolarizations allowed us to cause a shift between these pathways at will (Fig 1 and 2).

One of the main characteristics in our cases of longitudinal dissociation of the atrioventricular node was the transient nature of the phenomenon. In cases 1, 2, and 3, following the acute phase of the myocardial infarction, it was impossible to reproduce this phenomenon. In case 4, the dual pathways could not be demonstrated when the clinical and electrocardiographic signs of acute ischemia disappeared. This supports our assumption that longitudinal dissociation of the atrioventricular node with dual atrioventricular nodal pathways in the setting of acute myocardial infarction is of a functional nature, most probably related to the ischemic process taking place in the atrioventricular node as a consequence of the acute myocardial infarction.

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REFERENCES

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