Ectopic left atrial rhythm that produces QRS changes in absence of Wolff-Parkinson-White syndrome

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ABSTRACT  In an 18-year-old patient without manifest or concealed Wolff-Parkinson-White syndrome, spontaneous and paced left atrial impulses penetrated a left-sided AV nodal input and thereafter activated the ventricles in a normal fashion exclusively through the His-Purkinje system. On the other hand, sinus and paced right atrial impulses entered a right-sided atrioventricular nodal input that was completely dissociated from the left-sided input to subsequently activate the ventricles partly through Mahaim fibers and partly through the His-Purkinje system. The Mahaim fibers, which acted as “bystanders” during episodes of atrioventricular nodal reciprocating tachycardia, seemed to have extended from a “distal,” common (right-sided) intranodal pathway (or “proximal” His bundle) to the right ventricle or, although this is less likely, to the right bundle branch. More studies are necessary to determine whether the association on the surface electrocardiogram of an ectopic slow left atrial rhythm with changes in QRS morphology (but not in QRS duration) always reflects the existence of Mahaim fibers.

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A SPONTANEOUS or pacing-induced change from a right to a left atrial site of impulse initiation that may produce variations in QRS morphology is known to occur in patients with Wolff-Parkinson-White syndrome.1,2 This report, however, deals with the findings in a patient in whom the phenomenon under consideration was due to previously undescribed electrophysiologic mechanisms.

Methods and results

Patient. An 18-year-old male patient with a congenital aneurysm of the posterior (noncoronary) sinus of Valsalva had an 8 year history of recurrent supraventricular tachycardias. Tracings recorded while tachycardias were not present revealed periods of sinus rhythm (rates between 75 and 95 per minute) alternating with runs of ectopic left atrial rhythm3 at similar or slightly slower rates. Echocardiograms showed marked left ventricular hypertrophy and dilatation.

Surface electrocardiograms. Sinus rhythm (figure 1, bottom) was characterized by (1) normal P wave axis, (2) PR interval of 160 msec, (3) QRS axis of about +70 degrees, (4) initial 20 msec vectors that were oriented to the left, as determined from the r or R waves in leads I, aVL and V5, and superiorly, as determined by the q waves in leads II, III, and aVF (the latter two leads not shown), (5) QRS duration of 100 msec, and (6) absence of delta waves.

The main features of the ectopic left atrial rhythm (figure 1, top) were (1) P waves axis pointing superiorly and to the right, (2) dome-and-dart P wave morphology in leads V1 and V2, (3) QRS axis of about 0 degrees, (4) initial 20 msec vectors pointing to the right (evidence of which was the q waves in leads I, aVL, and V6) and inferiorly (as determined by the R waves in leads II, III, and aVF), and (5) QRS duration of 95 msec.

Electrophysiologic study. The patient was studied while in the postabsorptive, nonsedated state by techniques previously described.4 A septapolar catheter electrode was used to pace and/or record from the high right atrium, midright atrium, and right ventricular apex. By means of a quadripolar catheter electrode, stimulation was applied to and recordings were made from the coronary sinus. His bundle electrograms were obtained with a quadripolar catheter electrode positioned over the septal leaflet of the tricuspid valve.4

Ectopic left atrial beats had a distal coronary sinus/proximal coronary sinus/septal right atrial (a deflection of the His bundle electrographic lead) sequence (figure 2, right). The duration of the pertinent intervals was: AH, 75 msec; HV, 50 msec; proximal coronary sinus–H, 95 msec; H–right ventricular apex (RVA), 80 msec. Although the duration of the QRS complexes in the surface leads was 100 msec, total ventricular activation time, measured from the onset of ventricular depolarization (which occurred in the His bundle electrographic lead and surface leads at more or less the same time) to the end of the last deflection (which occurred in the distal coronary sinus lead), was 120 msec.

During programmed premature stimulation from the proximal coronary sinus at a driving cycle length of 600 msec, the A2H2 intervals gradually increased (figure 3, right) until reach-
ing a level of 195 msec; this occurred before the effective refractory period of the corresponding left atrial site was reached at an S₁S₂ interval of 250 msec.

Driven and premature beats and the escape beats depicted in figure 4, _left_ had the same normal HV intervals (50 msec) and QRS complexes. Apparently the left atrial impulses entered the AV node through a left-sided input and, after reaching the His bundle, activated the ventricles in a normal fashion through the His-Purkinje system (figure 5, _right_).⁵⁻⁹

On the other hand, sinus beats had somewhat longer AH intervals (115 msec) and slightly shorter HV and H-RVA intervals (35 and 45 msec, respectively). There were significant differences in the morphologic characteristics and in the direction of the initial forces of the QRS complexes (figure 2, _left_). Although QRS duration in the surface leads was 100 msec, total ventricular activation measured from the onset of ventricular depolarization (which occurred in the His bundle electrographic lead before than in the surface leads) to the last deflection (which occurred in the distal coronary sinus lead) was 140 msec.

![QRS morphology during slow ectopic left atrial rhythm (LAR) and during sinus rhythm (SR). Whereas in the top strip there are q waves in leads I, aVL, and V₆, these deflections are not present in the bottom strip. The changes in QRS contour were not associated with significant variations in QRS duration.](image)

**FIGURE 1.** QRS morphology during slow ectopic left atrial rhythm (LAR) and during sinus rhythm (SR). Whereas in the top strip there are q waves in leads I, aVL, and V₆, these deflections are not present in the bottom strip. The changes in QRS contour were not associated with significant variations in QRS duration.

![Surface and intracardiac leads during sinus and ectopic left atrial rhythm. Total ventricular activation time, measured from onset of the earliest to the end of the ventricular deflection (in whichever lead [intracardiac or surface] they occurred first) was longer in sinus and right atrial paced beats than in left atrial beats (see text for discussion). A = atrial deflection in the His (H) bundle electrographic lead (HBE); HRA = high right atrium; MRA = midright atrium; DCS = distal coronary sinus; PCS = proximal coronary sinus; RVA = right ventricular apex.](image)

**FIGURE 2.** Surface and intracardiac leads during sinus and ectopic left atrial rhythm. Total ventricular activation time, measured from onset of the earliest to the end of the ventricular deflection (in whichever lead [intracardiac or surface] they occurred first) was longer in sinus and right atrial paced beats than in left atrial beats (see text for discussion). A = atrial deflection in the His (H) bundle electrographic lead (HBE); HRA = high right atrium; MRA = midright atrium; DCS = distal coronary sinus; PCS = proximal coronary sinus; RVA = right ventricular apex.
During high right atrial pacing (figure 3, left) the AH intervals of progressively premature beats also increased (to 225 msec) before the effective refractory period was reached at an S1S2 interval of 260 msec. The corresponding HV intervals and QRS complexes were similar to those of sinus beats.

In this patient echoes and AV reciprocating tachycardia (of the long PR and short RP type) were induced by ventricular, but not atrial, stimulation techniques (figure 6). The sequence of retrograde atrial activation was the same (septal right atrium/proximal coronary sinus/distal coronary sinus) in driven and premature beats (figure 6, top). When tachycardia was present, the prolonged AH intervals were followed by HV intervals and QRS complexes similar to those occurring during sinus rhythm and high right atrial pacing.

The events occurring during sinus rhythm, high right atrial pacing, and AV reciprocating tachycardia may be construed to indicate that the corresponding impulses entering the AV node through a right-sided input traversed the “fast” and “slow” AV nodal pathways, thereafter activating the ventricles differently than did the left-sided impulses (as determined by the different QRS morphology and initial ventricular depolarization). Moreover, right-sided impulses reached the right ventricle earlier than left-sided impulses, as determined by the shorter (but still within normal limits) HV and H-RVA intervals. In figure 2, the shorter H-RVA interval in the left panel compared with that in the right panel was a striking finding, indicating that the corresponding impulses reached the right ventricular apical recording electrodes ahead of time as they do in the right-sided preexcitation syndromes.

These findings may be explained by the existence of Mahaim fibers originating in the “final” or “distal” AV nodal pathway and common to both right-sided (slow and fast) AV nodal pathways. However, this distal common pathway must have been inaccessible to the impulses entering the AV node through the left-sided input since they did not follow the same pathway into the ventricles. The Mahaim fibers probably terminated in the right ventricle or, although this is less likely, the right bundle branch (figure 5, left). The longer total activation time illustrated in figure 2, left and figure 3, left suggested that the corresponding QRS complexes were fusion beats resulting from ventricular activation through both Mahaim fibers and the normal His-Purkinje system.

The presence of Mahaim fibers was supported by the existence of those escape beats that had a complete left bundle branch block normal axis morphology (figure 5, right). It is possible that the impulses arose in the distal portions of the Mahaim tract and, from their site of origin, retrogradely activated the His bundle slightly before reaching the right ventricle (antegradely). This explains the short time interval (20 msec) between onset of His bundle and ventricular depolarization which, therefore, did not reflect linear conduction time from His bundle to ventricles, but rather reflected differences in the moment of arrival of excitation at the corresponding sites. Similar phenomena may be observed when impulse formation occurs in the right bundle branch.

In any case, the Mahaim tract was a “bystander” during the
tachycardias since it was not a necessary part of the circuit that involved, anterogradely, the slow AV nodal pathway and, retrogradely, the fast AV nodal pathway.

**Discussion**

**Dual AV nodal inputs.** Several authors have reported the occurrence of shorter AH intervals with coronary sinus pacing as compared with high right atrial stimulation. This finding was explained in the following different ways. (1) Direct stimulation of, or rapid entry into, normal transitional or James fibers. (2) Apparent shortening of the AH interval due to a pacing site-induced alteration of the relationship between the A deflection of the His bundle electrographic lead and the true onset of AV nodal activation, which was not recorded by the His bundle electrographic lead. Thus considered, the interval between A and H deflections did not reflect linear conduction time through the AV node, but differences in the moment of arrival of excitation at the sites from which these deflections were recorded. (3) A real decrease in AV nodal conduction time occurring as a result of the altered direction of the left atrial impulses. This would allow the impulses to penetrate the AV node through an input different than...
that through which high right impulses entered. The latter explanation is in keeping with the results of microelectrode studies describing the existence of a dual input to the AV node in the rabbit heart during sinus rhythm and atrial pacing.8,9

Recently Morady et al.15 made interesting observations in patients with AV reciprocating tachycardias involving the AV node anterogradely and a left free wall accessory pathway retrogradely. They noted that impulses emerging from the accessory pathway could penetrate the AV node (to activate the His bundle) through a left-sided input even when the septal right atrium (given by the A deflection of the His bundle electrographic lead) was (pre)activated by paced impulses emanating from the high right atrium. Although these inputs need not have been anatomically positioned left and right, they were considered to be on the left or right from an electrophysiologic point of view because of their ability to preferentially transmit the impulses from the corresponding atria.15

Electrophysiologic characteristics of the Mahaim fibers. In patients with typical nodoventricular (Mahaim) fibers originating from the more proximal portions of the AV node, right atrial pacing produces, for unexplained reasons, significantly wider QRS complexes in the surface leads than does coronary sinus pacing.16,17 Because this finding is not so apparent in figures 1 through 4, it is probable that Mahaim arose from the distal common pathway or, although this is less likely, from the proximal His bundle. In either case this presupposes the occurrence of longitudinal dissociation of the entire AV node and even of the proximal His bundle.

Organic (congenital or acquired) longitudinal dissociation of AV node and His bundle. The longitudinal dissociation observed in our patient with a congenital cardiac malformation could have an organic basis, as suggested by results of recent histopathologic studies. For example, Brechenmacher et al.18 found, in a patient who had Wolff-Parkinson-White syndrome and dual electrophysiologic AV nodal pathway response, two distinct AV nodal inputs separated by fibrotic tissue. Similarly, Bharati et al.19 studied the heart of a person dying of unknown causes in whom electrophysiologic studies had shown a dual AV nodal pathway response. Among other findings, an accessory AV node in the right AV ring (a potential anatomic substrate for longitudinal dissociation) was observed.

It is also possible for organic longitudinal dissociation to occur only in certain portions of the His bundle. This was observed by Bharati et al.19 in a 13-year-old girl with ventricular tachycardia who died suddenly. Results of postmortem studies were interesting because the authors found a small AV node partly engulfed in the central fibrous body and many connective tissue fibers that completely and longitudinally divided parts of the His bundle. In our patient longitudinal dissociation of the more proximal portions of the His bundle may have reflected an affectation of the "transverse interconnections," as has been reported by several authors.20-25 This could explain the inability of impulses arriving or originating at the left side of the proximal His bundle to reach the right-sided AV nodal input and the capacity of ventricular impulses entering the distal His bundle retrogradely as a different wavefront to activate the right-sided input (figure 5). In addition, it should be pointed out that, according to James et al.,26 abnormalities or lesions of the posterior margin of the noncoronary sinus of Valsalva may cause pathologic changes of the His bundle because of their close proximity to this structure.

Clinical implications. The events in our patient may be construed to indicate that, in the surface electrocardiogram, the coexistence of an ectopic, slow left atrial rhythm with changes in morphologic QRS characteristics (but not in QRS duration) should raise the possibility of the existence of bypass tracts. Although similar phenomena have been reported, mainly in patients with congenital heart disease,3,27-29 intracardiac recordings were not obtained in these patients. However, further prospective studies are required to corroborate our assumption and to determine what type(s) of bypass tracts may be found in these patients.

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