Electrophysiologic Studies during Accelerated Idioventricular Rhythms

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SUMMARY
Accelerated idioventricular rhythms (AIVR) are ectopic ventricular rhythms with rates intermediate between idioventricular escape rhythms (30 to 40/min) and ventricular tachycardia (120 to 180/min). Differentiation of AIVR from supraventricular arrhythmias rests primarily on demonstration of their ventricular origin. His bundle electrograms (HBE) were recorded in four patients during AIVR. HBE verified the idioventricular nature of the ectopic rhythm and excluded supraventricular rhythm with aberration as a cause. In addition, they permitted the recognition of normally conducted sinus beats, fusion beats, and idioventricular beats. The pacemaker site for the AIVR was below the bundle of His. AIVR became manifest when the heart rate was slowed by increasing vagal tone, premature atrial stimulation, and high degree atrioventricular (A-V) block. AIVR could be suppressed and 1:1 A-V conduction established by increasing the atrial rate with atropine or by atrial pacing.

Additional Indexing Words:
His bundle recordings Fusion beats Ectopic ventricular rhythm Atrial pacing
Ectopy Atropine Digitalis Slow ventricular tachycardia

RECENT attention has been directed to the occurrence of ectopic idioventricular rhythms with rates intermediate between idioventricular escape rhythms (30 to 40/min) and ventricular tachycardia (120 to 180/min). Recognition of such accelerated idioventricular rhythms (AIVR) rests primarily on the demonstration of their ventricular origin. This report indicates the use of His bundle recordings to document such an origin in four cases and describes some of the characteristics of these arrhythmias.

Methods
Four patients were studied because of an ectopic rhythm which appeared to be secondary to an accelerated idioventricular focus. The patients were advised of the nature of the study and a signed consent was obtained. Right heart catheterization was performed in the postabsorptive, nonsedated state. Recordings of the His bundle were obtained according to methods previously described. In addition, a quadripolar electrode catheter was introduced into the right antecubital vein and positioned fluoroscopically against the lateral wall of the right atrium. The distal pair of electrodes was used to pace the right atrium above sinus rates using a battery-powered pacemaker (model 5837*) which delivered impulses of 2-msec duration with milliamperage adjusted to approximately twice threshold. The proximal pair was used to record a high right atrial electrogram. A time mark generator (model 184+) was utilized for recording time intervals of 10 and 100 msec. The A-H (atrial to His) interval was taken as a measure of atrioventricular (A-V) nodal conduction time (normal, 60 to 140 msec)

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while the H-V (His to ventricle) interval was taken as a measure of His-Purkinje conduction time (normal, 30 to 55 msec).

All equipment was carefully grounded to avoid introduction of random currents. All patients tolerated the procedure well without complications.

Results

In all four cases, bundle of His recordings verified the idioventricular nature of the ectopic rhythm and permitted the recognition of normally conducted sinus beats, fusion beats, and idioventricular beats. The term “fusion beat” will refer to ventricular depolarization which results in part from a supraventricular impulse propagated through the His bundle and in part from activation spreading from an ectopic ventricular pacemaker. Electrocardiographically, they are characterized by configurations intermediate between normally conducted sinus beats and idioventricular beats and are associated with H-V times of 0 to normal.

Report of Cases

Patient 1 was a 64-year-old man with congestive failure and probable digitalis toxicity. Sinus rhythm was interrupted at frequent intervals by runs of an ectopic rhythm consisting of 3 to 30 beats with a cycle length slightly slower than sinus rhythm (690 msec vs. average sinus cycle of 650 msec). The emergence of the ectopic focus was usually related to sinus slowing. Occasionally, the ectopic focus appeared independent of the sinus rate. The patient was unaware of changing rate or rhythm and, despite lengthy runs of AIVR, had no apparent hemodynamic consequences.

Figure 1, panel A, shows normal sinus rhythm with a cycle length of 960 msec. The A-H interval is 115 msec, and the H-V interval is 55 msec. The remaining panels show incomplete A-V dissociation during idioventricular rhythm in which there are capture and fusion beats. During the idioventricular rhythm, the ectopic ventricular impulse is retrogradely conducted to the bundle of His and is concealed in the A-V node. In panels B to E, failure of the first sinus impulse (A) to capture the ventricles is explained by retrograde concealed conduction. The same phenomenon explains the prolonged A-H interval of the second sinus impulse; the closer the sinus impulse to the preceding ectopic ventricular impulse, the longer the A-V conduction time (A-H interval) of the sinus beat.

The AIVR was suppressed when the sinus rate was increased to 100/min following administration of 1 mg of atropine. The cycle length of the ectopic focus varied within 85 msec (from 650 to 735 msec) and did not act as a parasympathetic focus. The ectopic rhythm resolved after the discontinuation of digitalis therapy.

Patient 2 was a 62-year-old woman with rheumatic heart disease and evidence of mitral, aortic, and tricuspid insufficiency, whose clinical course was strongly suggestive of digitalis toxicity. She was unaware of change in rate or rhythm, and tolerated frequent episodes of AIVR, consisting of 2 to 16 beats, without apparent consequence. AIVR became manifest following ventricular slowing during sinus rhythm, sinus arrhythmia, carotid sinus massage, atrial flutter with high degree A-V block, and premature atrial stimulation, and was suppressed by pacing the atrium slightly faster than the rate of the ectopic focus.

Figure 2 shows one mechanism for the appearance of the ectopic rhythm. Sinus slowing subsequent to a premature atrial beat unmasked the underlying AIVR. At other times, the R-R interval during AIVR frequently increased to approximately twice the basic cycle length, suggesting exit block. No evidence of parasystole was present. After administration of digoxin had been discontinued for 5 days, the AIVR disappeared.

Patient 3 was a 64-year-old man with remote myocardial infarction, anginal syndrome, and compensated congestive heart failure. In the course of maintenance on digitalis and diuretics, arrhythmia and hypokalemia developed. Right heart catheterization revealed normal pressures. An ectopic atrial focus was documented (fig. 3) with 2:1 A-V nodal block. An ectopic ventricular rhythm was also present which appeared at coupling intervals ranging from 480 to 640 msec and persisted for several minutes at cycle lengths of 620 to 700 msec. Fusion beats were present. There was no evidence of parasystole. The atria could be paced to 240/min with persistent 2:1 block. The resultant ventricular response of 120/min suppressed the ectopic ventricular rhythm. Following discontinuation of atrial pacing, both ectopic rhythms resumed. Normal sinus rhythm returned 24 hours after potassium repletion and discontinuation of digitalis therapy.

Patient 4 was a 38-year-old man with a 3-month history of palpitations. Physical examination, chest X-rays, blood chemistry, and electrocardiogram were unrevealing except for the presence of an arrhythmia.

Sinus rhythm (cycle length, 640 to 680 msec) was frequently interrupted by paroxysms of 1 to 17 beats of an ectopic rhythm, appearing late in
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Figure 1

Case 1. Concealed retrograde conduction from the idioventricular focus. (Panels A through E) The top three tracings are simultaneously recorded standard limb leads, and the bottom tracing is a His bundle electrogram (HBE). A = atrial electrogram; H = His bundle electrogram; V = ventricular electrogram; Vf = ventricular fusion beat. R-R represents the interventricular intervals in msec. (Panel A) Sinus rhythm with a cycle length of 960 msec. The A-H interval is 115 msec, and the H-V interval is 55 msec. (Panels B to E) AIVR is present. The ventricular focus is retrogradely concealed in the A-V node. The first sinus impulse in each panel is blocked in the A-V node because it occurs while the latter is still completely refractory. The second sinus impulse in each panel captures the ventricle. The earlier this sinus impulse occurs in relation to the preceding ectopic beat (500 to 350 msec), the more it is delayed in the A-V node (A-H, 125 to 235 msec). (Panels B, C, and E) Capture results in a fusion beat (Vf). The idioventricular beats are not preceded by His deflections. (Abbreviations will remain the same in subsequent figures.)
A-A 900 1030 Vv 900 VA
A-H 150 t H' 101 A-H 150
H-V 45

Figure 2
Case 2. Following the first two sinus beats, in which the A-H and H-V intervals measure 150 and 45 msec, respectively, the region of the coronary sinus (CS) is prematurely stimulated (S). The prematurely stimulated atrial beat is blocked proximal to the bundle of His. The subsequent atrial cycle length slows to 1,270 msec, and the AIVR emerges. The variable lengths of the atrial (A-A) and ventricular (R-R) cycles result in a fusion beat for the first complex of the bottom panel and a capture beat for the last complex of the bottom panel. The A-H interval of the fusion beat is normal while H-V of 10 msec is spuriously short in view of the fact that the ventricle is activated in part by an antegrade sinus impulse, and in part by a ventricular ectopic impulse. (Same abbreviations as in figure 1.)

diastole with a coupling interval of 510 to 550 msec and continuing with a cycle length ranging from 430 to 600 msec (average, 530 msec; fig. 4). Following spontaneous pauses of 760 to 1,080 msec, sinus rhythm would resume. Fusion beats were occasionally seen.

Following the administration of 1 mg of atropine, the sinus rate accelerated sufficiently to suppress the ectopic rhythm. Intravenous administration of 100 mg of lidocaine had no effect, but following the intravenous administration of 100 mg of procainamide, the ectopic rhythm terminated. Because of the complaint of palpitation, the rhythm was kept suppressed with oral doses of procainamide.

Discussion
Ventricular ectopy can be classified into at least three types; extrasystolic, parasystolic, and idioventricular.1, 2

Idioventricular foci have an intrinsic rate of 30 to 40/min and, therefore, are generally latent pacemakers, serving a protective escape function. The rate, however, may become accelerated, and the result is an accelerated idioventricular rhythm (AIVR). The hallmarks of this arrhythmia are its emergence in late diastole or after a pause in the normal rhythm, its lack of protection from antegrade depolarization, and its notably benign course.

AIVR was first noted by Harris,18 after experimental myocardial infarction, and has subsequently been observed and reported with various terminologies.3-5, 14-23 Following
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Figure 3
Case 3. Ectopic atrial tachycardia with 2:1 A-V nodal block and AIVR. The A-H interval of the first two conducted beats is 215 msec, and the H-V interval is prolonged at 60 msec. The third ventricular complex is a fusion beat, and the His deflection occurs simultaneously with the onset of ventricular depolarization. The fourth complex is an idioventricular beat. An antegrade His deflection is seen buried within the ventricular electrogram and is associated with an A-H interval of 215 msec. Note the bigeminal variation of cycle lengths of the ectopic atrial pacemaker (340 to 385 msec). HRA = high right atrium. Time lines (T) appear at 100-msec intervals.

Figure 4
Case 4. AIVR with retrograde atrial capture. The ectopic rhythm appears as a late extrasystole 510 msec after the preceding sinus cycle. Following the second ectopic beat, the sequence of atrial activation is reversed, proceeding from low atrial septum recorded in HBE to the high right atrium (HRA), associated with biphasic P waves in leads II and III. The last three ectopic beats demonstrate progressive slowing of the focus, a phenomenon frequently seen in this patient prior to spontaneous termination of the rhythm.

The primary factor in diagnosis is the establishment of the ventricular origin of the arrhythmia. It is known for example that A-V

the suggestion of Marriott and Menendez, the rhythm has been referred to as "accelerated idioventricular rhythm" in this study.

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junctional beats or rhythms may occur with abnormal QRS configurations which are due to delay or block within the conduction system, or accessory A-V conduction pathways. Conduction disturbances have also been noted in conjunction with slower heart rates and prolonged cycle lengths. The most reliable electrocardiographic indicator of an active ventricular pacemaker has been the presence of fusion beats. However, even the significance of fusion beats has been questioned. By utilizing His bundle recordings, we showed that rhythms which satisfied the criteria for accelerated idioventricular rhythms were of ventricular origin in four instances in this study. The ventricular depolarizations resulting from the ectopic focus were not preceded by a bundle of His deflection in any of these four cases. When fusion beats occurred, as they did in all four cases, His bundle deflections preceded ventricular depolarizations with varying H-V intervals. When the antegrade His deflection appeared after the onset of ventricular depolarization, the sinus impulse appeared to make little or no contribution to ventricular activation. Antegrade bundle of His deflections were observed to fall randomly throughout the ventricular electrogram, making it unlikely that macro-reentry utilizing the bundle of His or bundle branches is responsible for the arrhythmia. Because of the absence of retrograde His deflections and the ability of the His bundle to be depolarized antegrade in a random fashion, an ectopic origin in the most proximal segments of the fascicles of the conduction system is also unlikely.

AIVR became manifest when the heart rate was slowed during sinus rhythm by increasing vagal tone (carotid sinus massage), premature atrial stimulation, and high degree A-V block. The AIVR could be suppressed and 1:1 A-V conduction established by increasing the atrial rate by giving atropine or by atrial pacing.

Concealed retrograde conduction in the A-V node was demonstrated in one instance (patient 1), and retrograde atrial capture occurred in another (patient 4). Retrograde atrial capture is associated with more sustained periods of arrhythmia because the sinus pacemaker comes under the control of the idioventricular pacemaker.

AIVR has been noted in association with acute myocardial infarction and digitalis toxicity, and in a pregnant woman with no other evidence of cardiovascular disease. In this study, three cases appeared related to digitalis toxicity and the remaining patient had no associated findings. The clinical course in all was benign.

In this study, His bundle recordings permitted verification of the idioventricular nature of the ectopic rhythm and the exclusion of supraventricular rhythms with aberration as a cause. In addition they permitted recognition of normally conducted sinus beats, fusion beats, and idioventricular beats.

References

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