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.

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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)

*Medtronic, Inc., Minneapolis, Minnesota.
†Tektronix, Portland, Oregon.
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 complica-
tions.

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 depolar-
ization which results in part from a
supraventricular impulse propagated through
the His bundle and in part from activation
spreading from an ectopic ventricular pace-
maker. 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 inter-
vals 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 inde-
pendent of the sinus rate. The patient was
unaware of changing rate or rhythm and, despite
lengthy runs of AIVR, had no apparent hemody-
namic 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 dissoci-
ation 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 administra-
tion 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 parasystolic focus.
The ectopic rhythm resolved after the discontinue-
tion 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 disap-
ppeared.

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 pac-
ing, 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 examina-
tion, chest X-rays, blood chemistry, and electro-
cardiogram 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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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; $V_f =$ 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 ($V_f$). The idioventricular beats are not preceded by His deflections. (Abbreviations will remain the same in subsequent figures.)
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, parasytolic, 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,13 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 geminal 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 extrastole 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...
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 antegradely 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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