Differentiation of Ventricular Tachycardia from Junctional Tachycardia with Aberrant Conduction

The Use of Competitive Atrial Pacing

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SUMMARY
A case of regular tachycardia with an intraventricular conduction defect and possible retrograde P waves is reported. The condition was interpreted as being either ventricular tachycardia with 1:1 retrograde V-A conduction or an atrioventricular junctional tachycardia with aberrant intraventricular conduction. Atrial pacing resulted in normalization of intraventricular conduction as well as the production of fusion beats and allowed a definitive diagnosis of ventricular tachycardia. The use of atrial pacing as a diagnostic technique has not been previously discussed in the literature.

Additional Indexing Words:
Intra-atrial electrocardiogram Arrhythmias Intraventricular conduction Quinidine therapy

THE DIFFERENTIATION of ventricular tachycardia from atrioventricular (A-V) junctional tachycardia with aberrant conduction has perplexed clinicians for many years. This dilemma has been increased since Kistin described the frequent occurrence of ventricular tachycardias with 1:1 retrograde V-A conduction. Previously described in a review of the literature by Foster and Thayer, the occurrence of 1:1 retrograde V-A conduction has been recently reemphasized by Massumi and associates. Langendor first pointed out, in 1950, the fallacy of relying on atrioventricular dissociation as a criterion for ventricular tachycardia. It is clear, also, that physical signs that reflect atrioventricular dissociation such as asynchronous venous pulsations in the jugular vein, changing intensity of the first heart sound, and irregular pulse pressure are of no value in diagnosing the focus of the ectopic tachycardia.

This is a report of a case of ventricular tachycardia with 1:1 retrograde atrial activation. The ventricular origin of the ectopic rhythm was established with the use of an atrial pacemaker which produced a competing atrial tachycardia. To our knowledge this is the first time an atrial pacing catheter has been used for this diagnostic purpose.

Report of Case
R. H. (RGH no. 23-24-20), a 66-year-old man, was admitted to the Rochester General Hospital on May 5, 1967, with anterior subternal chest pain extending to both arms. The electrocardiogram (fig. 1A) and serum enzyme determinations were diagnostic of an acute anterior myocardial infarction. The patient developed mild congestive heart failure shortly after admission and was treated with morphine, mercurial diuretics, digoxin, and anticoagulants. He made a slow but uneventful recovery and on June 5, 1967, was discharged to his home for further convalescence on a regimen consisting of

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low sodium diet, 0.25 mg of digoxin daily, and anticoagulants.

During the evening of the day after discharge, he noticed the onset of right subscapular pain. He was seen by his private physician who administered 2 cc of meralluride (Mercuhydrin) intramuscularly and an extra oral dose of 0.25 mg of digoxin because of increasing dyspnea associated with the pain. The symptoms were not relieved and he was readmitted to the hospital on June 7.

On admission he appeared anxious and was having some chest pain. Blood pressure was 100/80 mm Hg; pulse rate was 144, and the pulse regular. The neck veins were slightly distended without abnormal pulsation. Moist rales were heard bilaterally in the lower part of the chest. The heart was slightly enlarged to the left to percussion. The heart sounds were regular with no variation in the first sound; an S3 gallop was present, but no murmurs or pericardial rubs were noted. The liver edge was palpated 4 cm below the right costal margin. There was no variation in the peripheral pulse volume or in the systolic blood pressure.

A roentgenogram of the chest showed marked pulmonary congestion consistent with acute left ventricular failure. The electrocardiogram showed evidence of regular tachycardia with an intraventricular conduction defect and possibly retrograde P waves. This was interpreted as either a ventricular tachycardia with 1:1 retrograde V-A conduction (fig. 1B) or an A-V junctional tachycardia with aberration.

After oral treatment with quinidine, 0.4 g every 4 hours, for 12 hours, the rate of the tachycardia slowed slightly to 125. Carotid sinus massage had no effect. No digitalis preparation was given. On June 8, because of the persistent tachycardia and congestive heart failure, an attempt was made to convert the tachycardia. One hundred milligrams of lidocaine was given intravenously in 5 minutes, followed by 200 mg over a 30-minute period. This failed to change the cardiac rhythm and resulted in dysarthria and mental confusion.

A platinum-tipped, Teflon-coated, stainless steel wire* was then advanced transvenously into the right atrium with ECG monitoring, through a large needle placed in an antecubital vein. An intra-atrial electrocardiogram was recorded from the right atrium (fig. 2A). This confirmed the

*No. 12569, Davis and Geck, Division of American Cyanamid Co., Danbury, Connecticut.

Figure 1

Standard electrocardiograms. (A) During first hospitalization demonstrating evidence of an acute anterior myocardial infarction. (B) At time of second hospitalization (June 7, 1967) with QRS of 0.13 sec and possible P wave immediately following QRS, seen best in right precordial leads.
COMPETITIVE ATRIAL PACING

Figure 2

(A) Intra-atrial electrocardiogram showing wide QRS (R) followed by sharp spiking P wave; R-P interval = 0.19 sec. (B) Double standard lead I during atrial pacing. Competition is demonstrated between the ectopic ventricular rhythm and the atrial paced rhythm. The atrial pacemaker stimulus artifact (S) does not produce atrial depolarization until P1. The next stimulus results in P2, which is conducted to the ventricle and participates in the fusion beat (F); the next eight pacemaker-induced P waves are each followed by a normally conducted QRS complex; the ninth fails to depolarize the atrium and the ectopic tachycardia again emerges.

Figure 3

(Upper strip) Standard bipolar limb leads during atrial pacing demonstrating normal atrio-ventricular and intra-ventricular conduction identical to initial electrocardiogram (see fig. 1A). (Lower strip) V, is recorded; there are five beats of the ectopic rhythm. The seventh and eighth atrial pacemaker impulses result in atrial depolarization with partial ventricular conduction expressed as fusion beats (F1 and F2). The rest of the strip demonstrates continuous atrial pacing recorded in V, having the same configuration as in figure 1A.
impression of 1:1 retrograde V-A conduction. An attempt was then made to capture the cardiac rhythm with atrial and ventricular pacing by connecting the electrode to an external battery-powered generator.* Ventricular pacing could not be accomplished. It was possible, however, to effect atrial pacing at a faster rate than the ventricular rate, but each time the pacing rate was slowed the ectopic tachycardia emerged. The electrocardiographic recording during atrial pacing (fig. 2B) demonstrated normal atrioventricular and intra-ventricular conduction identical to the previous electrocardiogram (fig. 1A). In addition, competition between the atrial paced rhythm and the ectopic ventricular tachycardia could be demonstrated along with numerous ventricular fusion beats (figs. 2B and 3). These findings confirmed the diagnosis of ventricular tachycardia.

The patient was maintained on quinidine since he was unable to tolerate even a slow lidocaine drip. His blood pressure fell to 70/40 and a metaraminol drip was started which elevated the blood pressure to 100 mm Hg systolic. Shortly after this, during preparation for d-c cardioversion, the rhythm was noted to have reverted to a sinus rhythm with a normal QRS configuration identical to the previous ECG. Maintenance quinidine therapy was associated with no further difficulty.

Discussion

The problem of differentiating supraventricular tachycardias with aberrant conduction from ventricular tachycardias has been discussed by Kistin.5 He described several features which aid in this difficult problem. The findings which suggest the presence of an A-V junctional tachycardia are as follows: (1) The tachycardia is initiated by an ectopic or a premature P wave. (2) The tachycardia is initiated by a QRS complex with too brief an R-P interval to be compatible with retrograde ventricular-atrial conduction (less than 0.11 sec). (3) The QRS configuration, although abnormal, is unchanged from previous normally conducted rhythms. (4) The rate of the abnormal rhythm is identical with a previous supraventricular rhythm. (5) The P and QRS are so related that it is evident that the QRS depends on a supraventricular focus (that is, the Wenckebach pattern). (6) The arrhythmia can be slowed or terminated by carotid sinus stimulation.

In the case reported, the difficulty in differentiating the A-V junctional arrhythmias from a ventricular tachycardia, in addition to the failure of the arrhythmia to respond to intravenous administration of lidocaine, led us to use atrial pacing in an attempt to capture and slow the rhythm as reported by Kastor and associates.6 Massumi and associates,7 and Durrer and associates8 have recently pointed out the use of atrial pacing to terminate reciprocating tachycardias occurring in the Wolff-Parkinson-White syndrome, presumably by interrupting a circus movement on which the reciprocation depends.

The intra-atrial electrocardiogram (fig. 2A) showed a wide QRS followed by a sharp P spike. The R-P interval of this apparent retrograde P is 0.19 sec, compatible with retrograde ventricular-atrial activation, and somewhat against an A-V junctional tachycardia. However, as pointed out by Kistin, A-V junctional rhythms with retrograde atrial activation may have a prolonged R-P interval.

During atrial pacing, however, we noted several electrocardiographic changes which confirmed our diagnosis of ventricular tachycardia. When atrial pacing at a more rapid rate than the ectopic rhythm was achieved, there was normalization of intraventricular and atrioventricular conduction (fig. 2B) and a return of the ECG to its former pattern in the bipolar limb leads and V1 (fig. 3). In addition, the presence of ventricular fusion beats (figs. 2B and 3) occurring with atrial capture is further evidence of ventricular tachycardia.

Cohn and associates9 have stated that retrograde P waves are usually not seen with clinical ventricular tachycardia. Kistin and associates,10 however, have demonstrated its occurrence with experimentally induced ventricular tachycardia in man during cardiac catheterization, although for short periods. It is of interest that in our patient the retrograde 1:1 conduction was maintained for the duration of the arrhythmia without any evidence of V-A block.

*Model 5800, Medtronics, Inc., Minneapolis, Minnesota.
COMPETITIVE ATRIAL PACING

Intra-atrial electrodes can be passed with great ease and have been widely used for ECG recordings and to initiate atrial and ventricular pacing. This case demonstrates the use of atrial pacing in differentiating A-V junctional tachycardia with bundle-branch block from ventricular tachycardia with retrograde A-V conduction.

References
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