CASE REPORTS

Termination of Ventricular Tachycardia by Carotid Sinus Massage

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SUMMARY Intracardiac electrophysiologic studies were performed in two patients who had recurrent sustained ventricular tachycardia. In both, the tachycardia was repeatedly terminated by carotid massage. In one patient, intracardiac electrophysiologic studies revealed ventricular tachycardia with 2:1 retrograde ventriculoatrial (VA) block. Carotid massage resulted in alternate Wenckebach retrograde VA conduction terminated by ventricular echo beats. When ventricular echo beats occurred at a coupling interval of 340–400 msec, the tachycardia was terminated. Similarly, induced atrial depolarizations (during ventricular tachycardia) produced ventricular capture and terminated the tachycardia when the resultant ventricular coupling interval was 330–395 msec. In the second patient, progressively premature atrial or ventricular depolarizations did not terminate the tachycardia. Carotid massage had no consistent effect on retrograde VA conduction during ventricular tachycardia, but usually resulted in gradual increases in the tachycardia cycle length (50–100 msec) before abrupt termination of the tachycardia.

This is the first report documenting termination of ventricular tachycardia by carotid massage alone (i.e., without prior drug intervention); hence, tachycardia termination by simple carotid sinus massage does not prove a supraventricular origin. The mechanism of tachycardia termination was due to ventricular echo beats from retrograde atrioventricular nodal reentry in one patient and to direct vagal effects on either the ventricular muscle or the ventricular specialized conduction system in the other.

DIFFERENTIATION of ventricular from supraventricular tachycardia with aberrant conduction traditionally includes the application of maneuvers that either enhance vagal or inhibit sympathetic tone. These include the Valsalva maneuver, carotid sinus massage, and the administration of edrophonium hydrochloride or phenylephrine. Termination of a regular wide-QRS-complex tachycardia using these techniques suggests a supraventricular origin. However, ventricular tachycardia can be terminated by either phenylephrine administration or by application of carotid sinus massage after pretreatment with edrophonium hydrochloride. We studied two patients with recurrent ventricular tachycardia who could terminate their wide-complex tachyarrhythmias by carotid sinus massage alone. Documentation of this phenomenon in the absence of pharmacologic manipulations is reported and adds to the growing evidence that changes in autonomic tone can significantly influence ventricular arrhythmias.

Materials and Methods

All studies were performed in a cardiac catheterization laboratory. Both patients gave informed consent. Patient 1 had been taking quinidine, but the medication was discontinued 48 hours before catheterization. During the study, four quadripolar electrode catheters were inserted into the right femoral vein. Catheters were positioned in the high lateral right atrium, across the tricuspid valve, in the apex of the right ventricle, and across a patent foramen ovale into the apex of the left ventricle.

Patient 2 was not receiving medication for the week before study. Three quadripolar electrode catheters were inserted into the right femoral vein and positioned against the high lateral right atrium, across the tricuspid valve, and in the right ventricular apex. Surface leads X, Y and Z of the Frank orthogonal lead system and intracardiac recordings were displayed simultaneously on an oscilloscope (Electronics for Medicine DR-8) and recorded on photographic paper at speeds of 100–200 mm/sec. Cardiac stimulation was performed using a programmable stimulator (Bloom and Associates) with rectangular 1-msec pulses at approximately twice diastolic threshold. In both subjects, atrial and ventricular overdrive pacing was carried out at varying paced rates, beginning at a paced cycle length of 700 msec and decreasing by 50-msec decrements to a minimal paced cycle length of 300 msec. In addition, atrial and ventricular refractory periods were determined using the extrastimulus technique. After induction of the tachycardia, both subjects underwent repeated individual massage of both right and left carotid sinuses. In addition, atrial and ventricular overdrive pacing was performed during tachycardia. Progressively premature atrial and ventricular extrastimuli were induced at 10-msec decrements during the entire atrial or ventricular diastolic cycle during tachycardia.

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Case 1
A 43-year-old man with a history of recurrent episodes of palpitations since the age of 17 years was referred for diagnostic studies. The episodes occurred without apparent precipitating cause. The patient frequently terminated the palpitations by rubbing his neck, standing on his head, or flying upside down in a private airplane. In recent years, these episodes were associated with dyspnea, weakness, and mild tightness of the chest, and preceded syncopal attacks nine times. Recently, the frequency of the palpitations increased to twice weekly and lasted up to 36 hours, forcing the patient to retire from his job as a salesman. Treatment with quinidine and propranolol failed to alter the course of the tachyarrhythmia. Because the patient had frequent recurrences despite multidrug treatment, he was referred for detailed electrophysiologic studies.

Electrophysiologic Studies

Tachycardia Induction
Single atrial premature depolarizations induced 260–340 msec after the last driven atrial complex (drive rate 570 msec) resulted in a sustained wide-complex tachycardia. Similarly, sustained tachycardia was initiated by either atrial or ventricular overdrive pacing at paced cycle lengths less than 560 msec (fig. 1). During the tachycardia, no His bundle depolarization preceded the ventricular complex, and the tachycardia cycle length varied from 370–440 msec. During the tachycardia, 2:1 retrograde ventriculooatrial (VA) conduction was usually present. The QRS morphology during tachycardia was right bundle branch block with a left superior frontal plane axis, and was identical to that during spontaneous tachycardia.

Tachycardia Termination
Carotid sinus massage resulted in alternate retrograde VA Wenckebach conduction terminated by atrioventricular (AV) nodal echo beats. Echo beats occurred when the interval from initial septal ventricular depolarization to that of the septal atrial electrogram exceeded 180 msec. The resultant ventricular echo occurred within 340–400 msec after the preceding ventricular complex and terminated the tachycardia (fig. 2). Similarly, atrial overdrive pacing resulted in tachycardia termination when a fortuitously timed atrial impulse resulted in a ventricular capture within this same range (fig. 3). Induced single atrial depolarizations also terminated the tachycardia whenever these impulses resulted in ventricular depolarization 330–395 msec after the preceding QRS complex. The tachycardia was also terminated by single right ventricular premature depolarizations induced 300–390 msec after the preceding ventricular complex. In all instances, the tachycardia was terminated abruptly without significant changes in the tachycardia cycle length before termination.

Other Observations
Baseline AV nodal conduction time was 60 msec and the infranodal conduction time was 45 msec. Tachycardia induction could be elicited by single

![Figure 1](imageurl)

**Figure 1.** Induction of ventricular tachycardia in patient 1 by rapid atrial (coronary sinus) pacing. Tracings from top to bottom represent recordings from the X, Y and Z leads of the Frank orthogonal lead system, right atrium (RA), coronary sinus (CS), His bundle electrogram (HBE) and right ventricular apex (RV). The distance between time lines (T) is 1 second. During coronary sinus pacing, no electrical activity is recorded from the coronary sinus electrogram. The first QRS complex represents a normal sinus beat. The arrow signifies the first of four pacing stimuli originating from the coronary sinus. Upon discontinuation of atrial pacing, a wide-complex tachycardia (cycle length 390 msec) was recorded. The tachycardia has a right bundle branch block pattern with a superior axis. During tachycardia, there are no His depolarizations present and atrioventricular dissociation is evident.
atrial depolarizations that were not necessarily associated with prolongation of the AH or HQ intervals. Plots of premature atrial depolarizations (A₁A₂) and the corresponding His bundle responses (H₁H₂) revealed a single curve. Overdrive atrial pacing to cycle lengths of 350 msec were conducted without aberrancy, and there was no evidence of antegrade ventricular preexcitation during either right or left atrial overdrive pacing or programmed stimulation. During either tachycardia or overdrive ventricular pacing, the retrograde atrial depolarization sequence showed that the septal atrial electrogram always preceded that from the coronary sinus or right atrium. Endocardial mapping from multiple sites in both right and left ventricles during tachycardia showed the earliest area of activation near the lateral wall of the
left ventricle approximately 1 cm from the apex. The local endocardial electrogram from this area preceded the earliest surface electrogram by 30 msec.

**Management**

After documentation that the tachyarrhythmia was ventricular in origin, treatment with disopyramide, diphenylhydantoin, propranolol, quinidine, digitalis and procainamide in varying doses and combinations had no effect on tachycardia frequency. The patient refused experimental antiarrhythmic or surgical therapy (including a radiofrequency pacemaker) and presently is taking no medications, but continues to complain of frequent episodes of palpitations.

**Case 2**

A 62-year-old man with a 5-year history of episodic palpitations associated with lightheadedness and decreased visual acuity was referred for study. These episodes were usually precipitated by exertion but also occurred while at rest. They were not associated with chest pain, dyspnea or syncope, and the patient was able to terminate these episodes with a Valsalva maneuver or carotid sinus massage. The patient also had a 15-year history of well-controlled hypertension. Continuous cardiac monitoring revealed frequent prolonged episodes of wide-QRS-complex tachycardia without evidence of fusion or capture beats.

**Electrophysiologic Studies**

**Tachycardia Induction**

Atrial overdrive pacing at cycle lengths from 700–400 msec resulted in 1:1 AV conduction without aberration or preexcitation. Atrial pacing at cycle lengths of 340–385 msec resulted in a sustained wide-QRS-complex tachycardia at a cycle length of 375–400 msec (fig. 4).

Right ventricular overdrive pacing at cycle lengths of 290–450 msec also readily resulted in a similar wide-complex tachycardia. During the tachycardia, no His bundle depolarization preceded the ventricular complex, and the tachycardia cycle length varied from 360–400 msec. During the tachycardia, 1:1 retrograde VA conduction was usually present.

**Tachycardia Termination**

Carotid sinus massage resulted in termination of the ventricular tachycardia except in one instance. The usual finding was gradual prolongation of the tachycardia cycle length by 50–100 msec, followed by abrupt cessation of the tachycardia and resumption of sinus rhythm after a pause of 800–1000 msec (fig. 5A). Twice, however, the tachycardia was abruptly terminated without prior cycle length prolongation (fig. 5B). Overdrive atrial pacing resulted in ventricular capture but did not terminate the tachycardia (fig. 6).

Figure 6 also demonstrates dissociation of the His bundle deflection from the ventricular complexes. At an atrial paced cycle length of 335 msec, the AH interval remained constant while an independent ventricular rhythm (cycle length 380 msec) was recorded. The constant association of the His depolarization with that of the atrium at a cycle length shorter than the cycle length of the tachycardia verifies that the atrium, AV node and His bundle are not involved in the tachycardia circuit. Similarly, ventricular overdrive pacing resulted in ventricular capture but did not terminate the tachycardia. Singly induced atrial or ventricular depolarization did not terminate the tachycardia.

**Other Observations**

The AV nodal conduction time was 100 msec and infranodal conduction time was 60 msec at a spontaneous cycle length of 655 msec. Overdrive atrial pacing from either the high right atrium or coronary sinus at cycle lengths of 600–350 msec were conducted without aberrancy or resulted in ventricular tachycardia. There was no evidence of antegrade ventricular preexcitation or of abnormal retrograde ventricular activation during either right ventricular pacing or ventricular tachycardia.
Management

After electrophysiologic studies, the patient was given oral quinidine therapy. Further cardiac monitoring revealed no episodes of wide-QRS-complex tachycardia.

Discussion

To show that carotid massage alone can terminate ventricular tachycardia, it must be proved that the arrhythmias in these patients were, in fact, ventricular in origin and that termination was related to the carotid massage and was not a spontaneous event. In both patients, the following characteristics support a ventricular origin: absence of a His bundle deflection during wide-complex tachycardia when well-defined His bundle deflections were inscribed during sinus rhythm either before induction or after termination of the arrhythmia; characteristic capture or fusion complexes during tachycardia; narrow QRS complexes at paced atrial rates in excess of that during the tachycardia; and no evidence of accessory pathway (especially Mahaim fiber) conduction. Atrial overdrive pacing in patient 2 resulted in AH prolongation and fusion complexes before the initiation of the tachycardia, raising the possibility of participation of a nodoventricular bypass tract (fig. 4). This possibility was excluded because atrial overdrive pacing at varying rates during tachycardia produced a constant AH interval, with the His deflection dissociated from the ventricular complexes (figs. 4 and 6). The exclusion of the atrium, AV node and His bundle as necessary components of the tachycardia circuit effectively excludes participation of either a nodoventricular or a His-ventricular bypass.

That termination of the tachycardia was not spontaneous is suggested by the fact that untreated spontaneous tachycardia usually persisted for hours, but could be terminated by carotid massage within 5–10 seconds of application and that the phenomenon was reproducible in 14 of 17 episodes. Finally, both patients gave a history of tachycardia termination with vagal maneuvers.

Despite occasional reports of determination of pre-
sumed ventricular tachycardia by vagal maneuvers (i.e., retching), interruption of a tachycardia with vagal maneuvers strongly supports a supraventricular origin. However, there is evidence that tachycardias of ventricular origin may be abolished by enhanced vagal tone. Waxman et al. reported five patients who had ventricular tachycardia in whom the tachycardia was terminated by i.v. phenylephrine, 1–3 mg. Right or left carotid sinus massage with and without concomitant administration of i.v. edrophonium hydrochloride, 10 mg, failed to terminate the tachyarrhythmia despite slowing the atrial rate in those with AV dissociation or blocking retrograde conduction in those with AV association. In a later study, Waxman and Wald studied four patients who had recurrent ventricular tachycardia to elucidate the mechanism of phenylephrine termination of ventricular tachycardia. They found that pretreatment with i.v. edrophonium hydrochloride, 15–20 mg, decreased, whereas i.v. atropine, 2.4 mg, increased by a factor of two, the dose of phenylephrine required to abolish ventricular tachycardia. In addition, they demonstrated that although carotid sinus massage alone failed to terminate ventricular tachycardia in any patient, its application after pretreatment with i.v. edrophonium hydrochloride, 15–20 mg, terminated the ventricular tachycardia in all. In two of the four patients, carotid sinus massage after pretreatment with i.v. propranolol, 5 mg, consistently terminated episodes of ventricular tachycardia. However, in no instance was termination of the tachycardia related to sinus capture or ventricular echo beats.

Our report is, therefore, unique in that documented ventricular tachycardia was terminated by carotid sinus massage alone without prior pharmacologic intervention. In addition, in patient 1, the mechanism of tachycardia termination was unique: Carotid sinus massage resulted in retrograde alternate Wenckebach conduction associated with AV nodal reentry that terminated the tachycardia. Wellens and co-workers reported both induction and termination of ventricular tachycardia in man by critically timed atrial premature depolarizations. They also terminated tachycardia by an AV nodal echo beat, a mechanism identical to that proposed for patient 1. They hypothesized that this mechanism involves interruption of a reentrant ventricular tachycardia circuit by a single capture beat.

In the second patient, carotid sinus massage produced variable effects on VA conduction, but termination of the arrhythmia was usually preceded by lengthening of the tachycardia cycle length. This type of termination was reported by Waxman and Wald when carotid sinus massage was coupled with high doses of either edrophonium or propranolol.

Our observations add to a growing body of information emphasizing the importance of vagal tone in ventricular rhythms and show that the considerable vagal tone thought necessary to abolish ventricular tachycardia in man can be accomplished in selected patients by simple carotid massage. Therefore, interruption of a wide-complex tachycardia by carotid sinus massage alone is clearly not absolute proof of a supraventricular origin. Our subsequent experience and that of others suggest that our findings are quite uncommon and, thus, our conclusions are not meant to disparage the importance of carotid sinus massage in attempting to differentiate the origin of wide-complex tachycardias in man.

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