Valsalva Termination of Ventricular Tachycardia

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SUMMARY Nine patients with recurrent ventricular tachycardia (VT) that could be repeatedly terminated by a Valsalva maneuver are described. In two, the tachycardia would cease for only a few seconds and then resume, whereas in seven, the tachycardia could be permanently and reproducibly terminated with a Valsalva maneuver. In all patients the tachycardia ended during the strain phase of the Valsalva maneuver, when blood pressure and radiographic measurement indicated that cardiac dimensions had been reduced dramatically. The speed with which the Valsalva maneuver terminated VT increased in direct proportion to the strain pressure. Manoeuvres such as standing or nitroglycerin, which independently reduce cardiac dimensions, enhanced the potency of the Valsalva maneuvers. Pretreatment with atropine or propranolol in four patients did not alter the response of VT to the Valsalva maneuver. Thus, it appears that a strong Valsalva maneuver can terminate some forms of VT, most likely related to an abrupt reduction in cardiac dimensions.

PATIENTS WHO SUFFER from tachycardia are often instructed to perform a Valsalva maneuver in an effort to modify or terminate their rhythm disturbance.1,2 A regular tachycardia that breaks with a Valsalva maneuver is presumed to be paroxysmal supraventricular tachycardia (PSVT)3 because it usually involves the atrioventricular (AV) node,4,5 which is vagally responsive. A Valsalva maneuver is one way to promote a strong increase in cardiac vagal traffic,6 which in turn can depress AV nodal conduction and terminate PSVT.3 Three patients were referred to us because of tachycardias that were difficult to manage. Each patient was thought to be suffering from PSVT with aberrant ventricular conduction because they were able to terminate their arrhythmias at least intermittently with a Valsalva maneuver. Careful analysis of these records proved the presence of ventricular tachycardia (VT). We systematically studied the effect of a Valsalva maneuver in 15 selected cases of VT. In this report we describe nine patients who could repeatedly terminate VT by performing a Valsalva maneuver.

Materials and Methods

Patient Population

The effect of the Valsalva maneuver on VT was explored in 15 patients selected from 123 consecutive patients with VT seen by one of the authors from 1973-1978. The cardiac diagnoses of these patients are summarized in table 1. Patients with coronary artery disease and heart failure were excluded from the study. VT was recurrent and sustained in only 32 of the remaining patients. Fifteen of these 32 patients in whom the Valsalva maneuver was attempted met the following additional criteria: (1) They suffered from recurrent, morphologically identical episodes of VT for more than 1 year. (2) They tolerated the episodes well and exhibited no hypotension, heart failure or signs of myocardial ischemia during VT. (3) They were capable of executing a proper Valsalva maneuver. Nine of the 15 patients could terminate episodes of VT with the Valsalva maneuver. In these nine patients, all attacks consistently broke when a Valsalva maneuver of sufficient intensity and duration was performed, and attacks of VT could be easily induced, thereby allowing multiple observations on the effects of the Valsalva maneuver.

Physiologic Studies

Valsalva maneuvers were performed with the patients lying on an electric bed whose position could be accurately controlled. The Valsalva maneuver was performed in two ways. In the first, quantitation was not attempted, and the patients simply inspired to a moderate volume, closed their glottis and raised intrathoracic pressure for as long as 15 seconds. When quantitation of the intrathoracic pressure was desired, the patient respired through a mouthpiece with a unidirectional valve. The inspiratory side of the valve was open to room air and the expiratory side was connected to a manometer and a Statham P23Db strain-gauge transducer. The manometer was in full view of the patient. To perform the Valsalva maneuver, the patient again inspired to a moderate intrathoracic volume and expired against the manometer in such a way as to hold a constant pressure. To prevent the patient from achieving pressure by closing the glottis and generating pressure in the mouth only, a small leak was introduced in the system to prevent closure of the glottis.7 Thus, the recorded pressure reflected the intrathoracic pressure. The transducer signal was conditioned by a Hewlett-Packard preamplifier (model 8805C). The ECG, beat-to-beat heart rate, blood pressure, intrathoracic pressure and chest wall movement were simultaneously recorded on heat-sensitive paper using a Hewlett-Packard recorder (model 7758). Episodes

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of VT were reinduced during these studies by right ventricular stimulation using an endocardial electrode. Valsalva maneuvers were performed in horizontal (0°), upright (+60°) and dependent (−40°) positions. The maneuvers were also repeated 10 minutes after 0.3–0.6 mg of sublingual nitroglycerin. In each case, the Valsalva pressure and time required to terminate the VT were monitored.

In four patients, the effects of Valsalva maneuvers were measured during control conditions and after 1.2–2.4 mg of i.v. atropine and 0.15 mg/kg of i.v. propranolol. To assess the adequacy of vagal blockade, the effect of carotid sinus massage on sinus rate and on AV conduction time after termination of the tachycardia was determined after administration of atropine. Right- or left-sided carotid sinus massage failed to slow the sinus rate or prolong or block AV conduction. Further, carotid sinus massage during VT after administration of atropine did not slow the dissociated atrial activity in two patients or prolong or block the associated atrial activity in two other patients. The adequacy of β-blockade after propranolol was also assessed after termination of the tachycardia. Administration of a 5-µ intravenous bolus of isoproterenol did not increase the sinus rate.

To estimate cardiac dimensions at the time of VT termination, roentgenographic images of the heart were obtained using R-wave synchronized exposures during full inspiration both before and during the strain phase of a Valsalva maneuver that resulted in termination of the tachycardia.

**Intracardiac Electrophysiologic Studies**

Using multipolar electrode catheters, we recorded electrograms from the high right atrium, the His bundle region and the right ventricle. The right atrium or right ventricle was stimulated through adjacent poles on the same catheter. Antegrade conduction intervals were studied during sinus rhythm by incremental atrial pacing to the point of AV Wenckebach conduction. Antegrade refactoriness was determined by introduction of atrial premature complexes. Antegrade conduction was also studied in the presence of vagal stimulation produced by carotid sinus massage during constant atrial pacing at a cycle length of 500 msec. Retrograde conduction was studied during right ventricular pacing. His bundle escape beats were produced by abrupt cessation of rapid atrial pacing or by vagal stimulation using carotid sinus massage during conditions of sinus rhythm or a combination of both. Tachycardia was induced by driving the right ventricle at a rate just above the spontaneous sinus rate and introducing one or two premature complexes after 10 basic cycles. The premature complexes were delivered at 1.5 times the late diastolic threshold, and scanned the entire cardiac cycle at 5–10-msec intervals until the tachycardia was initiated. When a single premature complex failed to initiate the tachycardia, this stimulus was kept fixed at the earliest point in the cardiac cycle that produced a propagated response and a second premature ventricular complex was then introduced. The coupling interval of the second premature stimulus was shortened until the tachycardia was initiated. Tachycardia initiation was also tested by trains of right ventricular pacing at cycle lengths of 350–250 msec. Once the tachycardia was initiated and shown to be sustained, the response to ventricular stimulation was tested. Single ventricular premature stimuli were introduced after every 10 cycles of the tachycardia, and the entire cycle was scanned at 5–10-msec intervals until the tachycardia stopped. Short trains of rapid ventricular pacing at rates 10–50% above the tachycardia rate were also applied to terminate the tachycardia. After each

### Table 1. Clinical Data in 12 Patients with Ventricular Tachycardia

<table>
<thead>
<tr>
<th>Coronary artery disease</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute myocardial infarction (≤ 4 weeks)</td>
<td>17</td>
</tr>
<tr>
<td>Postmyocardial infarction (≥ 4 weeks)</td>
<td>32</td>
</tr>
<tr>
<td>Postmyocardial infarction + LV aneurysm</td>
<td>15</td>
</tr>
<tr>
<td>Angina pectoris without infarction</td>
<td>7</td>
</tr>
<tr>
<td><strong>Subtotal</strong></td>
<td><strong>71</strong></td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td></td>
</tr>
<tr>
<td>LVH</td>
<td>3 (1)</td>
</tr>
<tr>
<td>LVH + congestive heart failure</td>
<td>3</td>
</tr>
<tr>
<td><strong>Subtotal</strong></td>
<td><strong>6</strong></td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td></td>
</tr>
<tr>
<td>Aortic valve disease</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Mitral valve disease</td>
<td>3 (1)</td>
</tr>
<tr>
<td>Combined aortic and mitral valve disease</td>
<td>3 (1)</td>
</tr>
<tr>
<td><strong>Subtotal</strong></td>
<td><strong>8</strong></td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
</tr>
<tr>
<td>Digitalis intoxication</td>
<td>2</td>
</tr>
<tr>
<td>Obstructive pulmonary disease</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Malignant hyperthermia</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
<td>3 (1)</td>
</tr>
<tr>
<td>Calcified cardiac cyst</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Normal</td>
<td>15 (6)</td>
</tr>
<tr>
<td><strong>Subtotal</strong></td>
<td><strong>25</strong></td>
</tr>
</tbody>
</table>

The numbers in parentheses indicate the source and the number of cases of ventricular tachycardia in whom a Valsalva maneuver was attempted.

Abbreviations: LV = left ventricular; LVH = LV hypertrophy.
study, antegrade conduction was restudied after the administration of 1.2-2.4 mg of i.v. atropine. The dose chosen was sufficient to permit 1:1 conduction to the ventricles at a rate at least 10% above the tachycardia rate. Intracavitary electrograms and surface ECG signals were recorded on photographic paper using an Electronics for Medicine recorder (model DR12). Timing of administered pulses was achieved by a Digitimer (model 4020). The timer was coupled to a Grass stimulator (model S88) that delivered pulses to the heart through Grass stimulation isolation units (model SIU5). The purpose of the study was carefully explained, and verbal and written consent were obtained from each patient.

**Results**

Clinical data and characteristics of the VT in each patient are summarized in table 2. In each patient, the VT had a left bundle branch block morphology. The Valsalva maneuver produced permanent conversion of the VT in seven patients and transient conversion in two.

**Electrophysiologic Studies During Sinus Rhythm and Atrial and Ventricular Pacing (table 3)**

In all nine patients the PR interval, AV nodal conduction time (AH), His-Purkinje conduction time (HV) and QRS morphology and duration were normal during sinus rhythm. During atrial pacing or introduction of premature atrial complexes, the AH interval increased inversely with the cycle length, but the HV interval and QRS duration and morphology did not change from control. Early premature atrial complexes and rapid atrial pacing caused progressive AH delay and eventual AV block proximal to the His bun-

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**Table 2. Clinical Patient Data and Characteristics of Ventricular Tachycardia**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Cardiac status</th>
<th>VT rate</th>
<th>VT morphology</th>
<th>AV relationship</th>
<th>Termination by Valsalva</th>
</tr>
</thead>
<tbody>
<tr>
<td>MP</td>
<td>F</td>
<td>26</td>
<td>Calcified cardiac cyst</td>
<td>175</td>
<td>LBBB</td>
<td>AVD</td>
<td>Permanent</td>
</tr>
<tr>
<td>AN</td>
<td>M</td>
<td>26</td>
<td>Normal</td>
<td>180</td>
<td>LBBB</td>
<td>AVA</td>
<td>Transient</td>
</tr>
<tr>
<td>DC</td>
<td>M</td>
<td>18</td>
<td>Malignant hyperthermia</td>
<td>165</td>
<td>LBBB</td>
<td>AVD</td>
<td>Permanent</td>
</tr>
<tr>
<td>RE</td>
<td>M</td>
<td>11</td>
<td>Normal</td>
<td>170</td>
<td>LBBB</td>
<td>AVD</td>
<td>Permanent</td>
</tr>
<tr>
<td>OD</td>
<td>M</td>
<td>63</td>
<td>COPD</td>
<td>160</td>
<td>LBBB</td>
<td>AVD</td>
<td>Permanent</td>
</tr>
<tr>
<td>DC</td>
<td>M</td>
<td>36</td>
<td>Normal</td>
<td>175</td>
<td>LBBB</td>
<td>AVA</td>
<td>Permanent</td>
</tr>
<tr>
<td>MO</td>
<td>F</td>
<td>49</td>
<td>Mitral stenosis</td>
<td>190</td>
<td>LBBB</td>
<td>AVD</td>
<td>Permanent</td>
</tr>
<tr>
<td>TB</td>
<td>F</td>
<td>40</td>
<td>Aortic insufficiency</td>
<td>180</td>
<td>LBBB</td>
<td>AVD</td>
<td>Permanent</td>
</tr>
<tr>
<td>MB</td>
<td>M</td>
<td>24</td>
<td>Normal</td>
<td>170</td>
<td>LBBB</td>
<td>AVD</td>
<td>Permanent</td>
</tr>
</tbody>
</table>

Abbreviations: COPD = chronic obstructive pulmonary disease; VT = ventricular tachycardia; LBBB = left bundle branch block pattern; AV = atrioventricular; AVD = AV dissociation; AVA = AV association.

**Table 3. Electrophysiologic Data**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Normal sinus rhythm</th>
<th>RA pacing</th>
<th>Tachycardia</th>
<th>Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PR AH HV QRS</td>
<td>CL for Wenekebach</td>
<td>Before Wenekebach</td>
<td>Initiation RV pacing</td>
</tr>
<tr>
<td>MP</td>
<td>160 100 40 80</td>
<td>400</td>
<td>240 40 80</td>
<td>+1 (+1)</td>
</tr>
<tr>
<td>AN</td>
<td>170 100 45 80</td>
<td>350</td>
<td>260 45 80</td>
<td>-</td>
</tr>
<tr>
<td>DC</td>
<td>160 100 40 90</td>
<td>400</td>
<td>290 50 90</td>
<td>-</td>
</tr>
<tr>
<td>RE</td>
<td>150 95 40 80</td>
<td>420</td>
<td>300 55 80</td>
<td>+2 (+1)</td>
</tr>
<tr>
<td>OD</td>
<td>180 100 40 90</td>
<td>400</td>
<td>270 40 90</td>
<td>+2 (+1)</td>
</tr>
<tr>
<td>DC</td>
<td>170 110 45 80</td>
<td>390</td>
<td>280 45 80</td>
<td>+2 (+2)</td>
</tr>
<tr>
<td>MO</td>
<td>180 110 50 90</td>
<td>410</td>
<td>280 50 90</td>
<td>+1 (+1)</td>
</tr>
<tr>
<td>TB</td>
<td>180 120 45 80</td>
<td>370</td>
<td>270 45 80</td>
<td>+1 (+1)</td>
</tr>
<tr>
<td>MB</td>
<td>160 100 40 80</td>
<td>380</td>
<td>290 40 80</td>
<td>+1 (+1)</td>
</tr>
</tbody>
</table>

All intervals and durations are in msec. All numbers in brackets denote the number of PVCs needed to initiate or terminate the tachycardia.

Abbreviations: AH = atrioventricular node conduction interval; HV = His-Purkinje conduction interval; CL = cycle length; PVC = premature ventricular complex (paced); RV = right ventricular; + = successful induction or termination of tachycardia; - = tachycardia started spontaneously.
dle in each patient. Vagal stimulation accomplished by carotid sinus massage during constant atrial pacing at a cycle length of 500 msec produced AH interval prolongation in all patients and second-degree AV block proximal to the His bundle in five, without change in the HV interval or QRS morphology. Atrial pacing at rates in excess of the tachycardia was accomplished in each case after pretreatment with atropine; in no patient did the QRS morphology or HV interval change from control.

Attempts to stimulate the His bundle directly failed in all nine patients. His bundle escape beats were induced in four patients and had an HV interval and QRS morphology identical to those under control conditions. Ventricular pacing produced ventriculoatrial (VA) conduction in only two of the nine patients, who also had retrograde conduction during the tachycardia. In the two patients with intact VA conduction, rapid pacing produced incremental VA conduction delay and eventual VA block. Similarly, VA delay and block could be produced by vagal stimulation using carotid sinus massage during constant ventricular stimulation at a cycle length of 500 msec.

**Induction and Termination of VT (table 3)**

In two patients, VT repeatedly resumed spontaneously after termination, and induction of VT could not be studied in them. In these two patients, VT was permanently converted to sinus rhythm by i.v. administration of 500 mg of procaineamide, after which VT could not be evoked with electrical stimulation. VT could be induced repeatedly by introduction of a single early premature ventricular stimulus in four patients and by introduction of two consecutive early stimuli in three patients. These premature complexes were introduced during constant ventricular pacing at cycle lengths 20% shorter than the control sinus rate. Also, short trains of rapid ventricular pacing at cycle lengths of 350–250 msec started VT in all seven patients so tested. In all nine patients, VT could be terminated by premature ventricular stimuli or bursts of rapid ventricular pacing at cycle lengths of 380–250 msec. VT was terminated by a single early premature ventricular complex in five patients, by two early premature ventricular complexes in three patients and by three early premature ventricular complexes in one patient. Induced VT had the same morphology as spontaneous VT.

**Electrophysiologic Studies During VT**

The tachycardia in all nine cases met the following criteria for VT: (1) The QRS morphology during the tachycardia differed from that during sinus conducted beats and the QRS was 120 msec or longer in all patients. (2) AV dissociation was present in seven of the nine patients; in the remaining two patients with AV association, VA block could be induced by carotid sinus massage without altering the tachycardia. (3) Fusion beats and normal capture beats were observed spontaneously in six patients, and could be induced by atrial pacing at rates in excess of the tachycardia after atropine in the remaining three. (4) Atrial pacing at rates in excess of the tachycardia did not produce aberrant ventricular conduction in any of the patients. (5) There was no His bundle potential preceding the QRS complex during the tachycardia, and a clear His potential with a normal HV interval (40–50 msec) was seen during sinus rhythm without catheter repositioning (fig. 1).

**Phase of VT Termination**

At least four consecutive episodes of VT in each patient were terminated by a Valsalva maneuver. In all patients, the VT terminated during the strain phase (phase II) of the Valsalva maneuver. In no instance did the VT break during phase I, III or IV (fig. 2). Phase II of the Valsalva maneuver is characterized by a reduction in cardiac size. Chest roentgenograms were obtained to verify that the VT terminated when the cardiac size decreased (fig. 3).

**Time Required to Break VT Related to the Intensity of the Valsalva Maneuver, Body Position and Nitroglycerin**

The intensity of the Valsalva maneuver was inversely related to the time required to terminate the VT. Figure 4 shows two episodes of VT termination in

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**Figure 1.** Simultaneous recordings of lead 2 and His bundle electrogram (HBE) during ventricular tachycardia (VT) in patient A.N. During the strain phase of the Valsalva maneuver, the tachycardia terminated transiently and a sinus beat was noted. A clear His bundle potential (arrow) preceded this QRS complex with an HV interval of 40 msec.
the same patient at different Valsalva pressures. A small but distinct difference in the time required to terminate the VT was observed in these two instances. This relationship in another patient is plotted in figure 5. The VT broke sooner at higher Valsalva pressures. This relationship was studied in horizontal, upright and dependent positions as well as after nitroglycerin. The upright position and nitroglycerin both greatly reduced the time required to terminate VT at any Valsalva pressure, while the dependent position prolonged the time for termination of VT.

Effects of Atropine and Propranolol on Termination of VT

Pretreatment with atropine alone, propranolol alone or a combination of these agents did not alter Valsalva termination of VT (figs. 6 and 7).

Discussion

The nine patients described fulfilled criteria for VT. Supraventricular tachycardia with aberrant ventricular conduction was ruled out because the atria could be paced at rates in excess of the tachycardia and gain capture of the ventricles with a normal QRS morphology and AH and HV intervals that demonstrated normal AV conduction during this intervention. By contrast, during tachycardia the QRS complexes were wide, had a left bundle branch block morphology and were not preceded by a His bundle potential. Moreover, atrial activity during the tachycardia was dissociated in seven patients and in the two patients in whom it was associated, VA conduction could be blocked without affecting the tachycardia. There was no evidence of an accessory AV muscle bundle (Kent bundle) bridging the atria and

![Figure 2](http://circ.ahajournals.org/figure/2)

**Figure 2.** Simultaneous recordings of surface ECG leads 1, 2, 3, bipolar right atrial electrogram (BAE) and aortic blood pressure (BP) in patient DC. Atrioventricular dissociation was present during ventricular tachycardia (VT). During the initial phase of the Valsalva maneuver (phase I), the BP rose transiently. After several beats of the strain phase (phase II), the BP amplitude decreased (arrow) and VT terminated. When the BP returned toward control several seconds later, the tachycardia resumed.

![Figure 3](http://circ.ahajournals.org/figure/3)

**Figure 3.** Patient DC. The roentgenographic exposure on the upper left was obtained during VT after a deep inspiration (INSP). The exposure on the upper right was obtained after identical inspiration followed by a strong Valsalva maneuver and was obtained during the last VT beat (corresponding to the arrow in figure 2). The bottom diagram is a superimposition of the cardiac silhouettes in the two roentgenograms at the top. The interrupted line denotes the Valsalva exposure, and the solid line represents the control inspiratory film. The heart size was considerably reduced during the Valsalva maneuver.
ventricles, because the QRS morphology and the conduction always proceeded over normal AV connections. The possibility of a reciprocating supraventricular tachycardia proceeding antegrade over such a bypass tract is excluded by the presence of AV dissociation during the tachycardia. The possibility of an accessory nodoventricular or fasciculoventricular accessory connection seems unlikely for the above-stated reasons and because the His bundle escape beats were not aberrant and had normal HV intervals.

VT could be terminated in nine of 15 patients. Thus, we wondered whether these patients differ significantly from those described by other investigators. The series of 123 patients with VT from which our 15 cases were drawn had cardiac diagnosis similar to those in series reported from other laboratories. Despite this, the true incidence of Valsalva termination of VT was not tested in this study because of the rigorous selection criteria. The 15 patients who met the criteria for study, including the nine who successfully terminated VT with a Valsalva maneuver, were unique in that none had coronary artery disease or heart failure and all had stable, well-tolerated VT. The 108 other cases of VT were not tested. We suspect that most other investigators have not endeavored to terminate VT with Valsalva maneuvers in the majority of patients they examined. Thus, we cannot determine how unusual the observed response really is. VT had a left bundle branch block morphology in the 15 patients in this study. The significance of this is not known, but it may point to a special locus of these tachycardias. Induction and termination of VT by programmed electrical stimulation was similar to that described in other reports.

Figure 4. Simultaneous recordings of lead I, beat-to-beat heart rate (HR), blood pressure and chest wall movement (RESP) in patient TB. (left) Termination of ventricular tachycardia (VT) during a Valsalva strain of 50 mm Hg. (right) Termination of VT during a Valsalva strain of 30 mm Hg. In both cases, VT terminated as soon as the blood pressure decreased (arrows), but VT terminated sooner at the higher pressure. Both terminations shown here were permanent. VP = Valsalva pressure.

Figure 5. Patient MB. Time required for ventricular tachycardia (VT) to break plotted against the Valsalva pressure generated. The Valsalva maneuvers were performed in the horizontal (0°) dependent (-40°) and upright (+60°) positions as well as after nitroglycerin. VT terminated sooner with a stronger Valsalva strain. The upright position and nitroglycerin increased the Valsalva potency, whereas the dependent position reduced the Valsalva potency.
Valsalva maneuvers produce pronounced changes in autonomic tone. The Valsalva termination of VT in our cases did not appear to be mediated through a vagal mechanism because pretreatment with atropine did not modify the response. Similarly, the failure of propranolol to alter the response suggests that alterations in sympathetic tone did not play a prominent role. These observations do not completely exclude a partial role of changing autonomic tone. Atropine and propranolol are competitive receptor-blocking drugs, and, at the dosages used, some effects from vagal and sympathetic tone change might still have occurred during the Valsalva maneuver. The completeness of cardiac vagal or sympathetic blockade was tested by observing attenuation in the response of supraventricular structures, such as the sinus node rate or AV nodal conduction, to autonomic stimulation. Figure 6 illustrates a Valsalva maneuver terminating VT after atropine pretreatment had prevented vagally induced retrograde block in conduc-

**Figure 6.** Recordings of lead 3 during ventricular tachycardia (VT) in patient AN. Two applications of a strong Valsalva maneuver before and after atropine broke the tachycardia transiently. In the top strip there is 1:1 ventriculoatrial (VA) conduction. During the initial Valsalva maneuver, VA block developed. After atropine, VA block was prevented yet the VT was interrupted. The arrows denote retrograde activity.

**Figure 7.** Recordings of lead 1, beat-to-beat heart rate (HR), blood pressure (BP) and chest wall movement (RESP) in patient TB, illustrating Valsalva termination of ventricular tachycardia (VT) after pretreatment with atropine and propranolol. VT terminated when blood pressure began to decrease during the strain phase (arrow). VP = Valsalva pressure.
tion to the atria. However, there are no simple markers for judging the adequacy of vagal or sympathetic blockade of ventricular tissue.

Although the mechanism of Valsalva termination remains uncertain, an acute reduction in heart size probably played a central role. VT always broke during the strain phase at a time when a decreasing blood pressure and a reduction in the size of the x-ray silhouette indicated that cardiac dimensions were greatly reduced. The relationship between various body positions and nitroglycerin and Valsalva potency supports the role of cardiac size. Body position and nitroglycerin have known effects on cardiac size, and predictably influenced the potency of the Valsalva maneuver; specifically, reduction in cardiac size induces by upright position or by nitroglycerin enhanced, while a dependent position reduced, the potency of the Valsalva maneuver.

A reduction in cardiac size might have a specific action in cases where VT is initiated and maintained by stretch, whereas in other cases a reduction in cardiac size may not be related in any specific way to the mechanism of spontaneous initiation or maintenance of VT. Insufficient information on the mode of spontaneous onset of VT in these cases is available. The observations made in the two patients in whom the Valsalva maneuver only transiently stopped VT are perhaps pertinent. In both patients VT resumed several seconds later, when blood pressure returned to control (fig. 2). This probably reflects a return of cardiac dimensions to their original size. Similarly, a reduction in cardiac size by digitalis, bed rest, diuretics or afterload reduction may often contribute to termination of a ventricular tachyarrhythmia. A reduction in the frequency of premature ventricular complexes has been reported with the use of digitalis. The hemodynamic role of digitalis in producing this effect has not been assessed. Acute cardiac stretch is often important in generating experimental dysrhythmias. The results of programmed stimulation suggest that reentry is the most likely mechanism, as the tachycardias could be reliably started and stopped by electrical stimulation. Cardiac stretch can enhance Purkinje fiber automaticity, which can lead to a loss of membrane potential and reduce conduction velocity. The latter property is essential in the initiation and maintenance of reentry. An abrupt reduction in cardiac dimensions might reduce Purkinje fiber stretch, lessen automaticity and improve conduction velocity. Such changes could cause a reentrant arrhythmia to stop. A conclusive arrhythmogenic mechanism cannot be offered, because recent work has shown that electrical stimulation can elicit or stop automatic activity in a variety of cardiac tissues.

Our observations are clinically significant. First, Valsalva termination of an unknown tachycardia can no longer be assumed to indicate that the tachycardia is supraventricular in origin. All cases of VT terminated during phase II of the Valsalva maneuver, whereas a supraventricular tachycardia would be expected to slow or stop during phase IV, when a reflex increase in vagal drive is induced by a rise in arterial pressure. Second, blood pressure, roentgenographic and body position data all suggest that a reduction in cardiac size is central to termination. This has many implications because many drugs or other interventions can affect cardiac size and hence facilitate or retard VT termination. Third, the capacity of Valsalva to terminate VT may explain many episodes of spontaneous termination when the patient performs a Valsalva maneuver unconsciously or unnoticed. Two cases of VT were recently described in which sinus rhythm was restored by retching. It would seem reasonable that retching could produce cardiovascular actions similar to those seen during the strain phase of a Valsalva maneuver.

Future studies should endeavor to use more refined methods, such as two-dimensional ultrasound, to quantitate the degree of volume change in each cardiac chamber. It would be of interest to examine cases of VT that do not break with the Valsalva maneuver to see if an insufficient ventricular volume change explains the failure of the tachycardia to break. The overall incidence of Valsalva termination of VT as well as the usefulness of our observations will depend on testing the Valsalva maneuver on a much wider variety of cases of VT. If observations and experiments firmly establish that volume reduction is the central mechanism of Valsalva termination of VT, we will have identified a specific clinical physiologic instrument with great potential.

Addendum

Since submission of this manuscript, a case of a 64-year-old man with severe hypertensive and ischemic heart disease was reported in whom 33 episodes of VT were converted by repeated forceful coughs. Several brief coughs in close succession were needed to terminate the tachycardia. The hemodynamic effects of coughing closely resemble those of a Valsalva maneuver. Thus, this cough-terminated VT may have occurred through a mechanism similar to that in our report.

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