Effects of Respiration and Posture on Paroxysmal Supraventricular Tachycardia

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SUMMARY The capacity of deep inspiration and the dependent body position to terminate episodes of tachycardia was studied in 11 patients with recurrent paroxysmal supraventricular tachycardia (PSVT). In eight patients, a deep inspiration and a dependent position repeatedly terminated episodes of PSVT. Reasons for failure were found in the other three patients. A deep inspiration or assumption of a dependent position dramatically raised arterial blood pressure and terminated episodes of PSVT by reflexly increasing vagal drive. The magnitude of the rise in blood pressure was directly proportional to the depth of the inspired volume and to the extent of body dependency. The upright position attenuated the respiratory-induced increase in blood pressure and blocked PSVT termination. Likewise, vagal blockade with atropine did not affect the effects of respiration or dependent position on blood pressure but prevented termination of PSVT.

PATIENTS with paroxysmal supraventricular tachycardia (PSVT) are encouraged to explore techniques to effect self-termination of the tachycardia. Among the most common maneuvers are carotid sinus massage and the Valsalva maneuver. These methods are not always successful or especially easy to perform properly. Because some patients who suffer from PSVT report tachycardia termination from simpler maneuvers such as deep inspiration or a dependent body position we decided to investigate how these maneuvers might terminate PSVT. Our purpose was to define the mechanisms of action of these maneuvers and to delineate conditions in which they would be successful and those in which they might fail.

Materials and Methods

Eleven consecutive patients who suffered from PSVT for many years were selected for this study (table 1). All had undergone extensive unsuccessful drug trials over a period of years. Characteristically, these patients had episodes of tachycardia that lasted for many hours without spontaneous termination and required multiple hospital visits for electroversion or treatment with potent vagal maneuvers. Although PSVT in all cases could be terminated by vagal techniques, none of these patients had ever tried to terminate PSVT by deep inspiration or by assuming a dependent body position. All patients were referred because they had requested a permanent atrial radio frequency pacemaker for self-termination of the arrhythmias. In this context, we extensively explored the actions of respiration and body position on the tachycardia process.

The techniques used to explore the electrophysiologic character of PSVT and the suitability of a permanent radio frequency pacemaker to effect self-conversion have been extensively described. Stimulating electrodes were placed in the right atrium and/or the right ventricle to initiate tachycardia. Aortic blood pressure, pulmonary artery wedge or left atrial pressure and right atrial pressures were measured continuously. Catheter lumens were coated with heparin and flushed every 5 minutes with a diluted heparin solution. The pressures were sensed by transducers (Statham P23Db), conditioned and amplified (Hewlett-Packard Model 8805C). Chest wall movement was continuously recorded using bipolar electrodes and an impedance amplifier (Gould Model 11-4307-06). Respired volume was measured by a spirometer equipped with an electronic output. The bed used for these studies was an electrically driven circular bed. The position was monitored by a hanging rod that was pivoted around the turning axis of a potentiometer mounted on the bed frame. A constant current was delivered to the potentiometer. As the bed rotated, the rod moved around its axis, thereby turning the potentiometer and changing the resistance. This produced a voltage deflection proportional to the new resistance, reflecting the position of the bed. The output of the surface ECG signal was fed into a rate computer (Hewlett-Packard Model 8812A) that provided a beat-to-beat display of heart rate. All signals were recorded on a strip-chart recorder (Hewlett-Packard Model 7758) and inscribed on thermal paper. In addition, all signals were recorded on a magnetic FM tape recorder (Phillips Analog 14).

PSVT was initiated in one of several ways. (1) Single premature atrial complexes were electrically introduced into the cardiac cycle during sinus rhythm. The entire cardiac cycle was carefully scanned at 5-msec intervals until PSVT could be consistently started. (2) When this failed, a second, third or fourth atrial premature complex was introduced. (3) In other cases, right ventricular stimulation and a similar protocol were used. (4) In some cases, a short fast burst of asynchronous atrial or ventricular pacing was used. Timing of the stimuli was controlled by a quartz-
crystal timing circuit (Digitimer Model 4020). The timer activated a pulse generator (Grass Model S88) that provided pulses of adjustable voltage and duration. Stimuli were delivered through a stimulation isolation unit (Grass Model SIU5). When premature pulses were applied during specific periods of the cardiac cycle, a Schmidt counter allowed these pulses to be introduced after a preselected number of spontaneous or paced complexes. To identify the tachycardia circuit, we measured the coupling interval and AV nodal delay required to initiate the tachycardia; the retrograde atrial electrogram sequence during the tachycardia using recordings from the left atrium or distal coronary sinus, the atrial septum (His bundle recording) and the low and high lateral right atrium; and changes in rate and ventriculoatrial conduction time with the spontaneous appearance or disappearance of functional bundle branch block during episodes of tachycardia.9, 10

Study Protocol

First, repeated episodes of PSVT were induced as outlined above and it was shown that all episodes were stable and exhibited no tendency to terminate spontaneously. All subsequent studies were performed during PSVT after a sufficient time had elapsed from its induction to allow stabilization of its rate and the blood pressure. Second, the response of PSVT rate to elevations in blood pressure induced by bolus administration of i.v. phenylephrine (0.1 mg initially with 0.1-mg increments) was determined. The maximal dose was that which terminated two consecutive episodes of tachycardia. These observations included the minimal elevation of blood pressure required to terminate the tachycardia. Third, the effect of incremental inspiratory volumes (measured from end-expiration) on the tachycardia rate was determined. These observations included the measurement of the minimal inspiratory volume required to terminate two consecutive episodes of tachycardia. These observations were repeated at various body positions (−20° to +60°). Fourth, the effect of various dependent body positions (0° to −60°) on the tachycardia rate during normal respiration was determined. These observations included the minimal dependent body position required to terminate the tachycardia during two consecutive episodes of tachycardia. These maneuvers were repeated after the subjects had been pretreated with 0.01–0.03 mg/kg of i.v. atropine.

The purpose of the study was carefully explained and verbal and written consent were obtained from each patient.

Results

Criteria for PSVT

The following criteria were used to diagnose PSVT. (1) The tachycardia was regular, sudden in onset and offset and the heart rate was 140–220 beats/min with 1:1 atrioventricular (AV) response. (2) QRS complexes were normal or aberrant in a rate-related
manner identical to that during atrial pacing at rates equal to or in excess of the PSVT rate. (3) The tachycardia was consistently terminated with vagal maneuvers (carotid sinus massage, i.v. edrophonium HCl or i.v. phenylephrine). (4) The tachycardia could be started and terminated by critically timed single premature atrial stimuli.

The criteria for AV reciprocation involving an accessory bypass tract were as follows. (1) The atrial activation sequence during the tachycardia proceeded from the atrium on the side of the bypass tract to the atrial septum and to the contralateral atrium. (2) During established tachycardia, spontaneous disappearance of bundle branch block ipsilateral to the bypass tract coincided with acceleration of the tachycardia rate, due to shortening of the RP interval.

The criteria for reciprocation confined to the AV junction included: (1) atrial activation sequence proceeding from the low atrial septum to both atria symmetrically, (2) no acceleration in the tachycardia rate with spontaneous disappearance of bundle branch block, (3) consistent prolongation of the PR interval before the initiation of the tachycardia by premature atrial complexes; and (4) ventriculoatrial conduction time less than or equal to 0 msec during PSVT.

Table 1 is a summary of the case material, the tachycardia circuit, the rate range at 0º position, and the response of the tachycardia in each patient to phenylephrine, deep inspiration and the dependent body position. The tachycardia terminated in response to i.v. phenylephrine in all 11 patients and in response to deep inspiration and dependent body position in eight. In three patients, deep inspiration and dependent body position slowed but did not terminate PSVT.

**Termination of PSVT by Phenylephrine**

The tachycardia slowed in all 11 cases in relation to the dose of phenylephrine and the blood pressure elevation produced. PSVT was terminated in each patient when the blood pressure was sufficiently elevated (table 1, fig. 1). In nine patients, a pressor response of 60 mm Hg or less was sufficient to terminate PSVT. In patients HD and HF, blood pressure elevations of 80 mm Hg and 100 mm Hg, respectively, were required to terminate PSVT. These two patients were among the three who could not terminate PSVT by deep inspiration or the dependent body position. Pretreatment of the patients with atropine did not interfere with the blood pressure response to phenylephrine, but it prevented slowing or tachycardia termination.

 Among the nine patients whose tachycardia circuits incorporated a bypass tract, slowing after phenylephrine administration was exclusively related to prolongation in antegrade conduction across the AV node (P'R), while retrograde conduction across the bypass tract (RP') remained constant. In these cases, the tachycardia always ended as a result of antegrade block in the AV node. In the two patients whose tachycardias were confined to the AV junction, slowing was due to greater prolongation in conduction in the antegrade compared with the retrograde limb. Termination was due to block in either the antegrade or retrograde limb, but block in the antegrade direction was more prevalent. The responses when the tachycardias were slowed and terminated by deep respiration or dependent body position were identical to those in response to phenylephrine.

**Respiratory Termination of PSVT**

In eight of 11 patients, a deep breath consistently terminated PSVT abruptly when the patients were horizontal (table 1, fig. 2). When a deep breath was taken, blood pressure rose in direct proportion to the tidal volume. Although there was a small initial decrease in blood pressure at the onset of inspiration, the middle and latter phases of inspiration produced a significant rise in blood pressure. The tachycardia rate began to slow coincident with the increase in blood pressure. The rise in blood pressure was maximal during the expiratory phase, when PSVT terminated. The inspired volume that resulted in termination of tachycardia was identical during two consecutive episodes in each patient. When a breath was not deep
Figure 2. Patient MW. Simultaneous recordings of atrial stimulus code marker, lead V1, beat-to-beat heart rate (HR), blood pressure (BP) and respiratory activity (RESP). A short burst of rapid atrial (RA) pacing induced an episode of paroxysmal supraventricular tachycardia (PSVT). At the onset of the tachycardia, there was considerable decrease in blood pressure, which spontaneously rose to a higher stable level in several seconds. During PSVT, a deep breath elevated blood pressure and the tachycardia slowed and terminated. The pressure rise began during the midportion of inspiration and the tachycardia ended during the expiratory phase. During sinus rhythm, deep breaths reduced blood pressure during inspiration, and blood pressure returned to the control level during expiration.

Figure 3. Patient MG. Simultaneous recordings of tidal volume (TV), lead V1, left atrial (LA) pressure, heart rate (HR), blood pressure (BP) and respiratory activity (RESP). During control conditions a deep breath increased the blood pressure and terminated paroxysmal supraventricular tachycardia. After 0.6 mg of i.v. atropine, three deep breaths each raised the blood pressure and produced minor rate slowing, but the tachycardia persisted.

enough to terminate PSVT, we observed a transient increase in blood pressure and slowing of the tachycardia followed by a return to control values. Blood pressure increased with a deep breath only during the tachycardia and did not occur during sinus rhythm (fig. 2). Pretreatment with atropine did not alter the rise in blood pressure with a deep breath but did prevent PSVT termination (fig. 3).
Termination of PSVT by Dependent Body Position

The eight cases of PSVT that terminated with a deep breath also terminated whenever the body was rotated into a dependent position (table 1, fig. 4). As the body was rotated to a dependent position, blood pressure increased, heart rate decreased and the tachycardia ended. The degree of body dependency required to terminate two consecutive episodes of tachycardia was identical in all cases. The extent of blood pressure elevation was proportional to the dependent position. Pretreatment with atropine did not attenuate the increase in blood pressure but prevented termination of tachycardia in the dependent position.

Interaction Between Respiration and Body Position

The capacity of a deep breath to break PSVT was greatly reduced by the upright position (fig. 5), whereas the horizontal and dependent positions facilitated respiratory termination of PSVT. The effect of tidal volume on the blood pressure rise during the tachycardia was highly dependent on body posi-
tion (figs. 6 and 7). In upright positions the augmentation in blood pressure by any tidal volume was reduced, whereas the dependent position increased the blood pressure augmentation achieved by any tidal volume. In addition, the blood pressure augmentation required to terminate PSVT in the upright position was greatly increased while the dependent position reduced the pressure required for termination of tachycardia. Thus, even when pressure elevations equivalent to or greater than those generated in the horizontal or dependent positions were produced by a deep breath in the upright position, PSVT often failed to terminate.

**Comparative Effects of Phenylephrine, Deep Respiration and Dependent Body Position on PSVT**

In eight patients in whom PSVT terminated with a deep breath and the dependent position, PSVT also terminated with an intravenous bolus of phenylephrine. All three maneuvers produced an increase in blood pressure, and the pressor response...
Effects of Respiration and Posture on PSVT

First, testing the effects of respiration and posture on patients with paroxysmal supraventricular tachycardia (PSVT). Figure 8 illustrates the very minimal response of the blood pressure and tachycardia rate after deep respiration and dependent body position in patient HL. This patient had gross tricuspid insufficiency. Failure of the tachycardia to terminate in two other patients in response to a deep breath or the dependent body position could be attributed to two reasons. First, testing with phenylephrine showed that these patients required an increase in blood pressure of 80–100 mm Hg to terminate PSVT (table 1). Second, both patients had a reduced lung capacity and were unable to generate tidal volumes greater than 2100 ml and 2600 ml, respectively. This limited the peak blood pressure augmentation by respiration to 30 mm Hg and 40 mm Hg, respectively. The increases in peak pressure with dependent body position of −60° were 50 mm Hg and 45 mm Hg, respectively. Thus, the peak pressure rise generated by either a maximal breath or body dependency was considerably less than the pressor requirements for termination of PSVT with phenylephrine.

Follow-up Observations

The eight patients who were able to terminate episodes of PSVT are being followed without special interventions. These patients are endeavoring to break all episodes of tachycardia with a deep breath or the dependent body position. Of the three patients who are unable to terminate episodes of PSVT, two had permanent atrial radio frequency pacemakers installed with which they electrically terminate episodes of tachycardia. The third patient is being managed with drugs.

Discussion

These studies show how simple maneuvers such as a deep breath or the dependent body position can terminate episodes of PSVT. This is especially significant because eight of 11 consecutive patients with longstanding refractory PSVT were consistently able to terminate all episodes of tachycardia. These eight
patients are managing their tachycardia by means of respiratory and positional maneuvers.

The mechanism underlying termination by phenylephrine, deep breathing and dependent position appears to be the same. All three maneuvers increase blood pressure, which stimulates baroreceptor activity, promoting increased vagal tone and termination of the tachycardia. Atropine's ability to prevent termination without interfering with the blood pressure response provided evidence that vagal tone was the critical determinant of termination.14

Increased vagal tone slowed and terminated the tachycardias by its action on the AV node. In the patients whose tachycardia incorporated a bypass tract, slowing and termination of the tachycardia was related to incremental slowing and eventually blocking of antegrade conduction in the AV node. In PSVT confined to the AV junction, slowing and termination of the tachycardia resulted from conduction depression in antegrade and retrograde directions, but depression in the antegrade direction was dominant. At any point in the study, termination of the tachycardia by any of the interventions used was highly reproducible. Moreover, the blood pressure increments required to terminate the tachycardia was similar with all three techniques (table 1). Although there were some fluctuations in the control PSVT rate at 0° these were generally very minor at any particular time during the study (table 1).

Termination of PSVT uniformly appeared to coincide with the expiratory phase of respiration. Two factors may have contributed to this. First, inspiration operating on pulmonary stretch receptors causes a reduction in efferent vagal tone.15 Expiration reverses the process and enhances efferent vagal tone. This is the classic explanation for phasic sinus arrhythmia.16, 17 One might therefore expect that similar phase changes in vagal tone occur with each breath during PSVT and hence vagal tone would be highest during expiration. Second, the elevation of blood pressure after a deep breath reached its maximum during expiration; this effect operating on the baroreceptors would also accentuate vagal tone. Thus, the expiratory phase might augment vagal tone both through pulmonary reflexes and by the baroreceptors and the two effects combine to raise vagal tone to the level needed to terminate PSVT. Although we do not know the relative contributions by pulmonary stretch receptors and the baroreceptors on the elevated vagal tone, we believe that the respiratory contribution alone is not enough to break the tachycardia. This is nicely shown by patient HL (fig. 9), in whom deep respiration or body dependency failed to raise the blood pressure because of marked tricuspid insufficiency and never terminated.

**FIGURE 9.** Patient HL. Simultaneous recording of bed position, lead 1, pulmonary artery wedge pressure (PAW), heart rate (HR), blood pressure (BP) and respiratory activity (RESP) during paroxysmal supraventricular tachycardia (PSVT). Two deep breaths (left) failed to elevate blood pressure and did not terminate PSVT. Turning the body to a dependent position of -40° (right) failed to elevate blood pressure and the tachycardia did not terminate.
the tachycardia. Also, in the eight patients whose tachycardia could be terminated by a deep breath at 0°, the upright position limited and ultimately prevented termination. The failure to terminate PSVT was clearly not due to a limitation in the inspired volume, but rather to the attenuated blood pressure rise caused by the upright position. The reason for the maximum rise in blood pressure during expiration is not clear, but may be due in part to a phase lag in reflex and hemodynamic changes elicited by a deep inspiration. Such a phase lag effect could be studied in future by having patients hold their breath in a full inspiratory position and observing the time course of blood pressure changes. This would allow one to observe whether there is a phase lag in the blood pressure response and whether expiration is a necessary prerequisite for termination of PSVT.

The mechanism responsible for blood pressure elevation by a deep breath or a dependent position remains to be fully elucidated. Both maneuvers might increase venous return to the heart and thus increase the arterial pressure. The failure of blood pressure to increase during deep breathing in the patient with marked tricuspid insufficiency and the attenuated pressure rise when deep breathing was performed in the upright position support the idea that increased venous return played a central role in the blood pressure rise. The independent capacity of the dependent position to generate blood pressure elevations further supports the role played by venous return.

The following points require further clarification: (1) Why does a deep breath raise the blood pressure considerably during PSVT, whereas during sinus rhythm inspiration normally produces a small decrease in blood pressure? (2) Why does a dependent position produce such a large rise in blood pressure during PSVT compared with sinus rhythm? The answer to both questions is unclear, but may be in the reflex responses to PSVT. When the tachycardia starts, blood pressure decreases significantly, which promotes a great increase in sympathetic tone, decreasing the venous and arterial vascular volumes. The reflex increase in sympathetic tone is responsible for elevating the blood pressure after its initial decrease at the onset of PSVT. The filling pressure in the heart increases considerably during the tachycardia (figs. 3 and 9). Deeper inspiration and the dependent position both enhance the capacity of the venous system to transfer blood to the heart. This process is actually enhanced by the elevated venous filling pressure, which prevents large veins from collapsing during a deep inspiration. During sinus rhythm, when atrial filling pressures are normal, a deep inspiration may be limited in its pumping action as the veins collapse at the point of entry into the thorax. During PSVT, the filling pressures are high and venous collapse does not occur despite deep inspiratory efforts; coupled with the fast rate, this may quickly transfer to the left ventricle the effects of an increase in venous return. Once the left ventricle acquires an increased venous return it may generate an exaggerated blood pressure for any given stroke volume because the arterial tree is constricted. Future studies will have to be undertaken to examine these issues.

The two patients who failed to terminate PSVT because of an inability to take a deep enough breath and because of the high blood pressure elevations required to effect termination are interesting. Both patients had a reduced vital capacity, one because of obesity and the other because of heavy smoking. The high blood pressures required to effect PSVT termination may relate to at least two factors. First, the baroreceptor sensitivity to blood pressure elevation may have been attenuated and the resultant increase in vagal tone blunted. Second, conditions within the AV node itself may have contributed to a reduced sensitivity to augmented vagal tone. Studies directed at defining the precise level of baroreceptor input required to produce a given vagal end point would be of great interest in patients with PSVT.

The interrelationship between respiration and body position has clinical as well as physiologic significance. When patients were upright two things occurred to make respiratory termination of PSVT difficult. First, the upright position decreased blood pressure, which reflexly increased sympathetic tone and reduced vagal tone and resulted in acceleration of the PSVT rate. This would act to oppose any effective increase in vagal tone generated through an increase in blood pressure. Second, the upright position greatly reduced the capacity of a deep breath to raise blood pressure. This likely resulted from gravitational effects on venous return.

Finally, the tidal volumes and body positions required to terminate PSVT in our patients are not unrealistic. A patient with a good vital capacity should be able to take a large enough breath to drive the blood pressure to a successful end point. This would be greatly enhanced by a dependent position. The simplicity of these methods and the security felt by patients suffering from PSVT who understand their use make these techniques worthy of a clinical trial.

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