Acknowledgment

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The Hemodynamic Effects of Induced Supraventricular Tachycardia in Man

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SUMMARY The circulatory effects of supraventricular tachycardia (SVT) were studied in eight patients who reported disabling symptoms during paroxysms of the arrhythmia. Supraventricular tachycardia was induced in each patient by rapid atrial pacing or with atrial premature stimuli. Hemodynamic parameters in sinus rhythm and following the initiation of SVT were recorded and compared.

The following mean values were observed in sinus rhythm (SR) and SVT. Heart rate (beats/min): SR 79, SVT 183; P-R interval (msec): during SR, 154; during SVT, 256; ratio of mean P-R intervals to mean R-R cycle lengths: SR 20%, SVT 76%; brachial artery pressures (mm Hg): SR 141, SVT 99; cardiac index (l/min/m²); SR 3.6, SVT 2.2; pulmonary artery pressures (mm Hg): SR 18/7, SVT 26/15; peak right atrial pressures (mm Hg): SR 4, SVT 17. Large waves appeared in the right atrium during SVT due to atrial contraction against closed tricuspid valves. Pulsus alternans were observed in each case during SVT. Despite the presence of chest pain during SVT, the coronary arteries were normally patent in four patients who underwent coronary arteriography.

SUPRAVENTRICULAR TACHYCARDIA (SVT) may severely disable afflicted patients who are otherwise in good health and without apparent cardiac disease. Dyspnea, weakness, lightheadedness, and even syncope may be produced by SVT at heart rates generally well tolerated when the rhythm is of sinus origin. The hemodynamic basis of these symptoms during SVT has never been fully explained.

Previous hemodynamic observations during SVT have been obtained fortuitously when the arrhythmia developed during cardiac catheterization.4 4 Since the usual mechanism of SVT is atrioventricular (A-V) nodal re-entry it may be predictably initiated and terminated by atrial stimulation.4 7 As a result, comparative evaluation of the hemodynamic consequences of SVT may be assessed with the patient in sinus rhythm serving as his own control. We report here a combined electrophysiologic and hemodynamic study in eight highly symptomatic patients with A-V nodal re-entrant SVT. Our findings help to explain why this arrhythmia produces such discomfort in affected patients.

Methods

Eight patients with a clinical history consistent with the presence of recurrent paroxysmal supraventricular tachycardia were evaluated in the Cardiac Clinical Electrophysiology Laboratory (table 1). Each patient gave informed consent to the performance of both hemodynamic and electrophysiologic studies. The following venous catheters were inserted percutaneously or by cut-down through the right or left basilic veins or the right femoral vein:
TABLE 1. Clinical Data on Eight Patients with Recurrent Paroxysmal Supraventricular Tachycardia

<table>
<thead>
<tr>
<th>Pt/Age/Sex</th>
<th>Symptoms during SVT</th>
<th>ECG</th>
<th>Chest X-ray</th>
<th>Clinical diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>S.L./22/F</td>
<td>Dyspnea, weakness, near syncope</td>
<td>WNL</td>
<td>WNL</td>
<td>NHD</td>
</tr>
<tr>
<td>P.A./28/F</td>
<td>Dyspnea, fatigue, chest pain</td>
<td>WNL</td>
<td>WNL</td>
<td>NHD</td>
</tr>
<tr>
<td>G.R./45/F</td>
<td>Chest pain, weakness, left-sided facial weakness</td>
<td>ST &amp; TWA</td>
<td>WNL</td>
<td>UHD</td>
</tr>
<tr>
<td>M.C./46/F</td>
<td>Fatigue, dyspnea, dizziness</td>
<td>ST &amp; TWA</td>
<td>LVE</td>
<td>Mild HHD</td>
</tr>
<tr>
<td>I.M./49/F</td>
<td>Staggering gait, chest pain</td>
<td>ST &amp; TWA</td>
<td>WNL</td>
<td>Bicuspid valve</td>
</tr>
<tr>
<td>M.S./50/F</td>
<td>Chest pain, syncope</td>
<td>ST &amp; TWA</td>
<td>WNL</td>
<td>Late systolic murm</td>
</tr>
<tr>
<td>J.A./54/M</td>
<td>Chest pain, near syncope, weakness</td>
<td>WNL</td>
<td>WNL</td>
<td>Mild hypertension</td>
</tr>
<tr>
<td>A.E./61/F</td>
<td>Extreme weakness, near syncope</td>
<td>ST &amp; TWA</td>
<td>WNL</td>
<td>UHD</td>
</tr>
</tbody>
</table>

Abbreviations: F = female; M = male; HHD = hypertensive heart disease; LVE = left ventricular enlargement; NHD = no heart disease; ST & TWA = ST and T wave abnormalities; UHD = unknown heart disease; WNL = within normal limits.

Goodale-Lubin catheter with the tip in the pulmonary artery for the measurement of pressure and the injection of dye in determination of the cardiac output; quadripolar electrode catheter without lumen in the right atrium for sensing and pacing; a tripolar electrode catheter positioned across the tricuspid valve for recording of the His bundle electrogram. The left brachial artery was cannulated to record arterial pressure and for sampling of dye. In four patients left heart catheterization was performed in addition: for evaluation of a heart murmur (patients M.C. and M.S.) and for chest pain (patients P.A., G.R., and M.S.). Left heart catheterization was conducted with the Sones technique through the right brachial artery.

Electrophysiologic signals were fed through a specially constructed junction box and lead selector to Model EEP amplifiers in an Electronics-for-Medicine DR16 recorder. Signals above 500 Hz and below 40 Hz were filtered out. Multiple surface electrocardiographic leads were also simultaneously recorded (usually leads I, aVF, and V1).

The hemodynamic information was processed through Statham transducers and Model SGM amplifiers in the Electronics-for-Medicine recorder. The cardiac outputs were performed in duplicate with injection of indocyanine green dye in the pulmonary artery and sampling at the brachial artery. The blood was withdrawn by a Harvard pump, the green dye detected with cuvette, and the signal processed through Model EEP amplifier in the Electronics-for-Medicine recorder.

All surface electrocardiographic, intracardio graphic, electrophysiologic, and hemodynamic signals were tape recorded on a Hewlett-Packard 14 channel instrumentation recorder. Signals were then retrieved after completion of the study on the Electronics-for-Medicine recorder and the information displayed on photographic paper at various speeds.

In each patient baseline electrocardiographic, electrophysiologic, and hemodynamic records were made during sinus rhythm. Re-entrant supraventricular tachycardia was then induced with either rapid atrial pacing or premature atrial depolarizations. Continuous recordings of hemodynamic and electrophysiologic events were performed during the course of the tachycardia, which lasted for varying periods of time in the different patients. If the tachycardia did not remit spontaneously, conversion was performed by carotid sinus pressure, rapid atrial pacing, or by the introduction of single atrial premature depolarizations.

Results

Changes in Heart Rates and P–R Intervals

Supraventricular tachycardia (SVT) was easily induced in each of the eight patients by the introduction of premature atrial depolarizations or by rapid atrial pacing (table 2). The ventricular rates during sinus rhythm (SR) which ranged from 58–103 beats/min (mean 79) increased to 130–210 (mean 183) during SVT (fig. 1, panel A). R–R cycle lengths in SR were 583–1034 msec (mean 786 msec) and in SVT were 286–462 (mean 338 msec). The P–R intervals were 130–180 msec (mean 154 msec) in SR and were 180–390 msec (mean 256 msec) in SVT (fig. 1, panel B). The ratios of mean P–R intervals to mean R–R cycle lengths were 20% in SR and 76% in SVT.

Changes in Brachial Arterial Pressure and Cardiac Index

The mean systolic brachial arterial pressure dropped from 141 mm Hg in SR to 99 mm Hg in SVT (fig. 1, panel C). These measurements were taken 60–90 sec after SVT.

The effects observed at the onset of SVT are illustrated for patient J.A. in whom re-entrant SVT was induced at a rate of 208/min (fig. 2). For three seconds no pulsatile flow was recorded and then a small pulse pressure appeared. Within a minute arterial pressure had risen to a peak systolic value of 93 mm Hg compared with 136 mm Hg during SR.

The mean cardiac index for the eight patients decreased from 3.6 L/min/m² in SR to 2.2 L/min/m² in SVT (fig. 1, panel D). The average percent decrease in cardiac index was 37.5%.

Changes in Pulmonary Artery and Right Atrial Pressures

Pulmonary artery systolic pressure in SR ranged from 10 to 25 mm Hg (mean 18) and rose during SVT to a range of 13 to 46 mm Hg (mean 26). Pulmonary artery diastolic pressures in SR ranged from 3 to 11 mm Hg (mean 7) and rose during SVT to a range of 7 to 23 mm Hg (mean 15) (fig. 1, panel E).

Right atrial pressures were recorded in five of the eight patients studied. In SR the peak pressures of the A waves ranged from 4 mm to 7 mm Hg (mean 5), the V waves from 1 to 8 mm Hg (mean 4) and the mean right atrial pressures from 1 to 7 mm Hg (mean 3). During SVT the A and V waves could no longer be separated from each other. The peak pressures of these combined "AV" waves ranged from
7 to 25 mm Hg (mean 17 mm) and the mean right atrial pressures from 5 to 15 mm Hg (mean 10). Comparison of the peak A wave pressures in SR versus the peak “AV” wave pressures in SVT are illustrated in figure 1, panel F. An individual example of the development of the large AV waves during SVT is shown in figure 3.

**Ventricular Pressures**

Catheters were placed in the left ventricles (LV) in four cases. During SVT it was not possible to accurately gauge LV end-diastolic pressures because of the rapid rate and short diastole.

**Pulsus Alternans**

This finding was observed to varying degrees in each of our eight patients. It occurred at the onset of SVT and decreased or disappeared shortly thereafter (fig. 4).

**Coronary Arteriography**

The coronary vasculature was normally patent in each of

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**Table 2. Comparative Electrophysiologic and Hemodynamic Data on Eight Patients in Sinus Rhythm and Supraventricular Tachycardia**

<table>
<thead>
<tr>
<th>Patients</th>
<th>Ventricular rate (beats/min)</th>
<th>P-R interval (msec)</th>
<th>Brachial artery (mm Hg)</th>
<th>Pulmonary artery (mm Hg)</th>
<th>Right atrium (mm Hg)</th>
<th>Cardiac index (L/min/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SR</td>
<td>SVT</td>
<td>SR</td>
<td>SVT</td>
<td>SR</td>
<td>SVT</td>
</tr>
<tr>
<td>S.L.</td>
<td>103</td>
<td>148</td>
<td>130</td>
<td>180</td>
<td>115/62</td>
<td>105/60</td>
</tr>
<tr>
<td>P.A.</td>
<td>66</td>
<td>188</td>
<td>180</td>
<td>244</td>
<td>118/67</td>
<td>98/67</td>
</tr>
<tr>
<td>G.R.</td>
<td>78</td>
<td>198</td>
<td>130</td>
<td>260</td>
<td>110/69</td>
<td>85/65</td>
</tr>
<tr>
<td>M.C.</td>
<td>84</td>
<td>188</td>
<td>150</td>
<td>240</td>
<td>180/87</td>
<td>87/65</td>
</tr>
<tr>
<td>I.M.</td>
<td>66</td>
<td>210</td>
<td>140</td>
<td>220</td>
<td>138/64</td>
<td>96/72</td>
</tr>
<tr>
<td>M.S.</td>
<td>81</td>
<td>188</td>
<td>180</td>
<td>280</td>
<td>170/104</td>
<td>109/75</td>
</tr>
<tr>
<td>J.A.</td>
<td>95</td>
<td>208</td>
<td>140</td>
<td>230</td>
<td>136/78</td>
<td>93/81</td>
</tr>
<tr>
<td>A.E.</td>
<td>58</td>
<td>130</td>
<td>180</td>
<td>390</td>
<td>157/82</td>
<td>119/93</td>
</tr>
</tbody>
</table>

Abbreviations: SR = sinus rhythm; SVT = supraventricular tachycardia; A = A waves; V = V waves; M = mean pressure.
the four patients in whom this procedure was performed. Three of them (F.A., G.R., and M.S.) had chest pain during bouts of SVT.

Ventricular Angiography

Right ventriculography during SVT was performed in one patient, while in another SVT spontaneously developed during a direct LV angiogram. In neither patient was A-V valve regurgitation seen.

Discussion

Each of the eight patients in this study had a typical clinical history of re-entrant supraventricular tachycardia; characteristic electrocardiographic and electrophysiologic features of this arrhythmia were demonstrated in each case. In addition, all eight patients reported debilitating symptoms during episodes of SVT (table 1). Although weakness and fatigue were most frequently reported (6/8), five actually observed neurological malfunction. Chest pain accompanied SVT in five and three noted marked dyspnea. Because of the severity of these complaints, the patients agreed to undergo study in order to confirm the diagnosis of SVT and to determine if the arrhythmia alone accounted for the symptom.

The average age of the patients (44 years) raises the possibility that some may have had heart disease other than the electrical disorder. However, there was no evidence for more than slight hypertension, mild valve disease, or non-specific ST and T wave abnormalities. None had angina, or had had a myocardial infarction. Furthermore, during sinus rhythm all were in New York Heart Association Class I status, and their hemodynamic measurements at rest were normal. Thus it appears highly likely that most, if not all, of the patients were made uncomfortable during SVT by the effects of the arrhythmia alone in the presence of nearly normal myocardial and valvular function.

Onset of SVT

The data obtained on our patients illustrate why disabling symptoms appear when SVT begins. The ventricle empties rapidly and cannot fill since diastole is so dramatically curtailed at these rates. The arterial pressure drops precipitously and almost no pulsatile flow appears for several beats. At this time dizziness and near-syncpe from cerebral hypoperfusion are reported. Pulmonary artery diastolic pressures (and presumably LA pressures), characteristically elevated during SVT, are not yet raised during these first few seconds. Thus ventricular filling, already compromised by the rapid rate, is not increased by higher mean atrial pressures. Fortunately, compensatory mechanisms appear to develop quickly or syncpe would surely result. Increased systemic vascular resistance probably raises arterial pressure and relieves, to some extent, the hypotension induced by the miniscule stroke volume of SVT at its onset.

Low Cardiac Output

Despite compensation which raises blood pressure, the cardiac output during SVT decreased markedly in most of our patients. The characteristic symptoms of weakness, fatigue and weariness which affect patients during the arrhythmia can be easily understood in light of such changes. Of course, the volume of blood ejected/beat (stroke volume) is even more markedly decreased (fig. 5).

Why does this marked decrease in cardiac output occur? In sinus tachycardia produced by exercise, the cardiac output rises as cardiac contractility and venous return increase.
HEMODYNAMIC EFFECTS OF SVT/Goldreyer, Kastor, Kershbaum

Figure 3. The changes in the right atrial (RA) pressure are illustrated for patient M.S. during sinus rhythm (first two beats, top panel A) and following the induction of supraventricular tachycardia (SVT). Also shown are electrocardiographic leads I, II, and III. As the tachycardia proceeds the combined “A-V” waves grow to peak values of nearly 30 mm Hg. This gradual growth of the right atrial waves would be unlikely if due to tricuspid regurgitation, although in this patient that possibility was not ruled out by right ventriculography. Thirty seconds elapsed between the end of panel A and the beginning of panel B.

At peak exercise in healthy people the heart rate may exceed 180 beats/minute and thus equal the rates observed in our patients with SVT. Consequently, there must be specific alterations other than rate which account for the marked hemodynamic deterioration during SVT. Among those previously suggested have been: 1) decrease in ventricular

Figure 4. Characteristic electrophysiological and hemodynamic findings during supraventricular tachycardia (SVT) are illustrated in this recording from patient J.A. Shown are the intra-atrial electrogram labeled “A,” ECG leads I, II, and III, plus the brachial artery (BA) and pulmonary artery (PA) pressure tracings. Observations in normal sinus rhythm (NSR) at a cycle length of 540 msec are contrasted with supraventricular tachycardia at cycle length of 280 msec. Note during SVT the presence of pulsus alternans in the BA trace and the coincidence of atrial and ventricular depolarisation from the atrial electrogram and surface ECG. Without the electrogram, atrial activation cannot be identified during SVT.
filling because of the shortening of diastole; 2) A-V valve regurgitation; 3) changes in atrioventricular contraction relationships.

The decrease in diastole relative to systole as rate increases occurs whether sinus tachycardia or SVT is present. We do not have comparative data at the same rate in both rhythms, but there is little reason to suspect that an important difference is present.

Whether or not significant mitral or tricuspid regurgitation develop during SVT remains unsettled. However, in two of our patients neither tricuspid nor mitral regurgitation was revealed during right and left ventriculography performed during SVT. The principal argument in favor of A-V valve regurgitation is based on the large atrial waves which form during SVT. They are systolic in timing, so are they the "systolic waves" of regurgitation? If so, the signs of regurgitation should develop as soon as the SVT begins. However, this is not the case (fig. 3). In each of our patients, these characteristic waves in the right atrial tracing gradually increased within the first minute after the onset of SVT. They grew as the brachial artery pressure rose.

**A-V Contraction Relationships**

The large atrial waves have, we suggest, another etiology which can now be appreciated in terms of our current understanding of the electrophysiologic events during SVT. Most paroxysmal atrial tachycardias are initiated and sustained by A-V nodal re-entry. Under this circumstance the temporal relationships of atrial and ventricular activation are such that atrial depolarization usually occurs slightly after ventricular activation begins. Thus the P wave, if it can be seen on the ECG, or the A wave, observed in the atrial electrogram, appears within or shortly after the QRS complex. Consequently, the P-R interval is relatively long compared with the R-R interval. For example, during SVT in patient I.M., the P-R interval was 220 msec, and the R-R interval was 286 msec (rate of 210 beats/min.). The P-R/R-R ratio was 77%. During sinus rhythm in the same patient the ratio was 15% (P-R interval = 140 msec; R-R interval = 909 msec). In the group of eight patients the average P-R/R-R ratio during SVT was 76%.

The P-R interval and hemodynamic changes during SVT are similar to those which occur during atrial pacing at rest. As the paced heart rate is increased the P-R interval lengthens as A-V nodal conductivity diminishes at faster rates. During rapid atrial pacing parasympathetic tone does not decrease nor sympathetic tone increase to the degree that occurs during exercise, when the P-R interval shortens as the rate increases. These factors and others lead to a decrease in the cardiac output during rapid atrial pacing, whereas the output rises during exercise at similar rates.

To establish the relative hemodynamic relationships among SVT, atrial pacing, and exercise, comparative measurements in each patient would be required. We were unable to do this. During rapid regular atrial pacing SVT was always induced. It was not practical during each study to exercise the patients sufficiently to raise the heart rate toward the SVT rates and measure intracardiac pressures and cardiac outputs.

During re-entrant SVT the atria usually contract while ventricular contraction simultaneously occurs. The A-V valves are closed, and the volume of atrial systole is discharged into the pulmonary veins and vena cavae. Giant A waves observed in the neck veins and recorded from hemodynamic catheters in the atria result. The veins are deprived of the contribution of atrial filling when the heart rate is fast and diastole is short. The A waves themselves grow in size after SVT begins as the cardiac output quickly falls and may gradually reach pressures as high as those illustrated in figure 3. These tall A waves probably account for the sensation of "pounding in the neck" which many patients observe during a paroxysm of SVT. This symptom cannot arise from the carotid arteries since both the blood pressure and the stroke volume in them is decreased.

These considerations help to explain why patients do not suffer such hemodynamic embarrassment during sinus tachycardia at similar rates. The P-R interval shortens during physiological rate increases thanks to changes in autonomic tone. Atrial systole contributes its share to ventricular filling, thus augmenting stroke volume and cardiac output.

**Chest Pain**

Myocardial ischemia has frequently been suggested as the cause for this symptom, reported by five of our eight patients during SVT. The commonly observed "tachycardia-induced" ST-segment depressions are taken as support for this explanation. With the increase in oxygen consumption caused by the high rates plus the decrease in coronary perfusion associated with the drop in arterial pressure, ischemia would be expected. Significant coronary arteriosclerosis need not be present as suggested by the youth of many patients with SVT-induced chest pain. Three of our patients with chest pain who were evaluated angiographically had normally patent coronary arteries.

Myocardial infarction probably occurs infrequently during episodes of SVT even though creatine-phosphokinase is often observed. The elevated values actually reflect discharge of the enzyme from hypoperfused skeletal muscle as documented recently by measurement of the iso-

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**Figure 5.** The inverse relationship of stroke volume (SV) to heart rate (HR) is displayed for each of the eight patients during normal sinus rhythm (dots) and supraventricular tachycardia (squares).
enzymes of CPK in patients during bouts of SVT. Although the ischemia concept is attractive, the cause of chest pain induced by SVT has not been fully established.

Correlations with Previous Studies

We have found reports in the English literature of eight patients whose hemodynamics were evaluated during atrial tachycardia; two by Ferrer et al. in 1949, three by Saunders and Ord in 1962, one by Benchimol et al. in 1965, and two by McIntosh and Morris in 1966. General reviews on the effects of cardiac arrhythmias on hemodynamics have also appeared in recent years. In the case reports the data usually became available when the arrhythmia appeared fortuitously before or during hemodynamic catheterization. In two of the cases of Saunders and Ord, for example, SVT was induced by “intra-atrial stimulation with the catheter tip.” Because the information had to be caught “on the run” while the arrhythmias were present, a full evaluation in each case was not possible.

The initial dramatic fall of brachial artery pressure with partial compensation in time and the occurrence of pulsum alternans were documented by Saunders and Ord. They also noted the characteristic decrease in stroke volume, although the cardiac output was seen to rise in two of the cases where it could be measured.

In the other five previously reported cases where cardiac outputs were measured, an increase was observed in one, a decrease in three, and no change in one. We observed a fall in the cardiac index in seven of eight cases. The differences between the studies may be due in part to the time when the outputs were measured, but no other obvious explanation for the discrepancies is present.

Saunders and Ord observed, as we did, an increase in the intracardiac pressures, particularly in the left atrium (pulmonary capillary wedge) and to a lesser extent in the pulmonary artery and right atrium. The mean pulmonary capillary wedge pressures, however, did not exceed 15 mm Hg. This value is lower than the pulmonary artery diastolic pressure observed in some of our patients. Again, however, the absence of marked elevations in the pulmonary diastolic pressures is confirmed with regard to the assumed explanation for the commonly observed symptom of dyspnea.

Saunders and Ord and Ferrer et al. discussed at some length the appearance of the large waves recorded from the right atrium during SVT. The explanations of tricuspid regurgitation or atrial contraction against closed A-V valves received the most attention. These investigators, however, were unaware when their studies were performed that re-entrant SVT is usually associated with long P–R intervals compared to R–R intervals. We have suggested that the gradual growth of the large A-V waves after the onset of SVT favors atrial contraction against closed A-V valves over A-V valve regurgitation as an explanation for the findings. Saunders and Ord also made this observation and raised the possibility that the force of atrial contraction progressively rises as venous return increases, “analogous to the application of the Frank-Starling law to the strength of ventricular contraction.” This property of the human atria has been documented.

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