

## Asymptomatic Recurrent Supraventricular Tachycardia

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*A case of recurrent asymptomatic supraventricular tachycardia in a young transport pilot without any underlying cardiovascular disorder is presented with a brief review of supraventricular tachycardia induced hemodynamic changes, and management strategies.*

### Part I : Initial clinical evaluation

**A** Transport Pilot aged 39 years, was detected to have short burst of supraventricular tachycardia (SVT) recorded in post exercise period following Master's two step exercise test. The patient remained asymptomatic and SVT spontaneously subsided at three minutes in the post exercise period. He was referred for further evaluation. He did not give history of angina, dyspnoea, cough, expectoration or palpitation, and was undertaking regular walking exercises. He has been smoking 10-12 cigarettes per day for past 12 years. Perusal of annual medical evaluation records revealed stable cardiovascular system. Earlier ECGs were normal.

Clinically: Pulse, temperature and respiration were normal; BP was 126/82 mmHg, and general and systemic examination revealed no abnormality. Routine investigations and biochemical parameters including lipid profile were normal. On treadmill test (TMT), he exercised for seven minutes (9.5 METS) with heart rate 150 per minute, normal BP response; Rate pressure product (Heart rate X systolic BP) was  $24 \times 10^3$ . At this stage, he suddenly developed SVT (rate 187/mt) with altered P wave morphology (inverted in lead II, aVF and V5 and flat in V1), while the QRS morphology retained the base line pattern. The exercise was terminated because of exercise induced supraventricular tachycardia (SVT). He remained stable and asymptomatic, but SVT persisted. After 1.7 minutes in the post exercise period, the SVT reverted spontaneously to sinus rhythm with a pause (Fig. -1), the rate abruptly dropping to

100/mt, and P becoming upright in II, aVF and V5 and biphasic in V1 the configuration recorded prior to commencing exercise. He was diagnosed to have exercise induced SVT (EX-SVT).

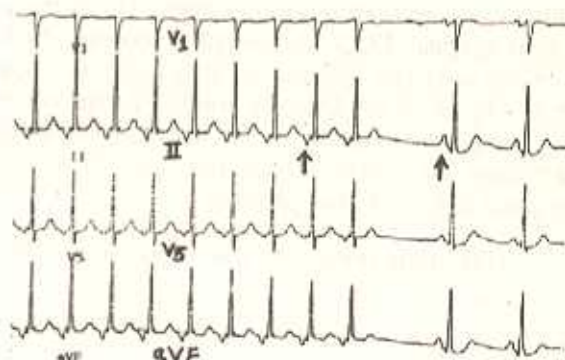


Fig 1

**Question No.1** : What further investigations would you undertake at this time ?

### Part II : Further Evaluation.

The following day, he was subjected to Holter monitoring which revealed frequent bursts of SVT and the length of SVT episodes ranged from 6 beats to 514 beats. The heart rate during SVT varied from 133-187 per minute and figure-2 shows SVT rate of 166/minute with spontaneous abrupt termination. All the other episodes also

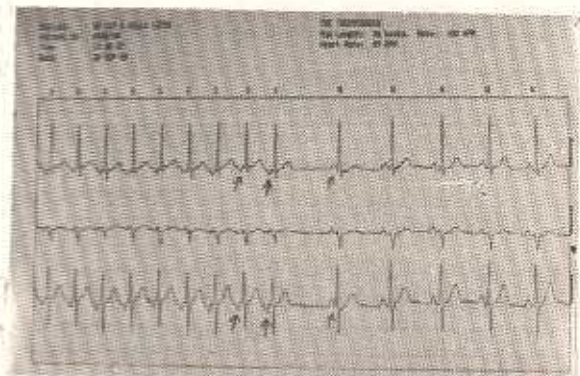


Fig 2

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spontaneously terminated to normal sinus rhythm. The SVT episodes occurred during rest, exercise and sleep without symptom correlation. The 187/minute beat SVT occurred at 7.45 PM. When he was taking bath. No ischaemic ST shift was detected, and no blocks or other arrhythmias were observed. In view of frequent episodes of SVT on Holter study both at rest and during exercise, the diagnosis of EX-SVT was changed to asymptomatic recurrent SVT and was placed on Verapamil 40 mg 8 hourly with the advice to stop smoking and continue regular walking exercises.

Electrophysiological studies (EPS) revealed PA-30 msec, AH=80 msec, HV=40 msec, sinus node recovery time (SNRT)=1110-1230 msec with corrected SNRT=300-410 msec and there were no intraventricular blocks. Antegrade atrioventricular conduction studies were normal. Premature stimulus study failed to induce SVT or VT and atrial stimulation only induced nonsustained self terminating short episodes of atrial fibrillation, a nonspecific response. The EPS studies were normal. Echocardiography revealed normal left ventricular functions and normal Doppler and Colour flow studies.

**Question No.2 :** Will this aircrew be fit to fly again ?

#### **Part III : Aeromedical disposal.**

Without any underlying cardiovascular disorder, normal exercise tolerance, normal noninvasive investigations and EPS studies, the recurrent and hemodynamically inconsequential SVT episodes were considered benign. It is planned to reinduct him to restricted flying with periodic reviews, and finally to full flying status. The maintenance treatment with verapamil is to be withdrawn in due course.

**Question No. 3 :** What are the indications and methods of treating SVT?

#### **Part IV : Reentrant or Ectopic Supraventricular Tachycardia.**

The SVTs could be due to reentry or of ectopic origin<sup>1,2</sup>. The present case shows the features of reentrant SVT as evidenced by : (i) Sudden onset and offset, (ii) No "Warm up"

phenomenon, ie. gradual increase in tachycardia rate, (iii) being low intra- atrial or junctional reentry phenomenon, the P wave bears constant relation to QRS, and precedes it with a negative P in II, aVF and V5 while biphasic in V1 ( the site close to the low atrial reentry focus) and (iv) P waves are of same morphology in ectopic SVT while in reentrant SVT the P waves are of different morphology due to retrograde atrial stimulation and hence the inverted P in II, aVF, V5 in the present case. A timely supraventricular ectopic (SVE) normally terminates such a reentry SVT but in our case, the SVT episodes spontaneously subsided without SVE, probably due to altered conduction in the two limbs of reentry circuit caused by autonomic influence. The termination of SVT is followed by pause each time and this is because of sinus node recovery time after rapid atrial stimulation by reentrant SVT which suppresses sinus node automaticity. The SVT can be tolerated by healthy hearts even for days together, however, it may induce congestive heart failure in some cases. The diseased hearts tolerate SVT poorly especially with diastolic dysfunction (DD) and this is due to loss of atrial impetus to ventricular filling which normally accounts for 15% of left ventricular end diastolic volume (LVEDV) and which may be much more with DD. Two-thirds of reentrant SVTs are due to AV nodal reentry and rest are due to intra atrial, SA nodal or preexcitation pathway reentry. The paroxysms of hemodynamically inconsequential SVTs with normal hearts are compatible with long term good prognosis. If SVTs are hemodynamically compromising and EPS studies demonstrate bypass tracts, surgical or catheter ablation therapy may cure the SVT. The patients may be treated with verapamil, beta-blockers, or digoxin but success may be elusive. If preexcitation pathway is excluded (as in this case), the patients may be returned to normal activity and kept under periodic evaluation. The EX-SVT may occur in 0.02% of cases subjected to exercise tests. However, it is usually free of morbidity<sup>5</sup> and mortality unlike exercise induced ventricular tachycardia<sup>6,6</sup>. For termination of SVT, the procedures commonly adopted are vagal manoeuvres, intravenous verapamil or digoxin and if SVT is hemodynamically compromising and

not responding to usual therapy, DC cardioversion may be done<sup>1,3,4</sup>.

**Question No. 4 :** What are the exercise induced arrhythmias, their relative frequency of occurrence and prognostic significance?

**Part V : Exercise testing and complications.**

The treadmill exercise testing is not without complications. The accepted mortality is one per 10,000 TMTs, and is attributed to ventricular arrhythmias, acute myocardial infarction and asystole (Cardiac arrest) especially with underlying high degree atrioventricular (A-V) block, or alternating bifascicular block. The other arrhythmias which commonly account for exercise induced morbidity are supraventricular tachycardia, junctional tachycardia, atrial flutter, atrial fibrillation and ventricular tachycardia<sup>6</sup> and conduction disorders including bundle branch block. The overall morbidity is 5.2 per 10,000 treadmill tests done<sup>5</sup>, and this can be substantially reduced with careful selection of cases, strictly following the safety measures and clinical examination before commencing TMT. And thus, the importance of most crucial factor, the operator experience in conducting and interpreting TMT. Other complications like hypotension (fall in

systolic BP by more than 25 mmHg), acute left ventricular failure (Ischaemia induced raised left ventricular diastolic pressure) and altered contractility causing "flash pulmonary edema", and cerebrovascular accidents occur in minority of cases. Adequate preparedness to be able to deal with these complications is mandatory.

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