

Close Look at ECG Tracing *after* Termination of Supraventricular Tachycardia with Adenosine

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Key words: electrocardiography, supraventricular tachycardia, adenosine

IMAJ 2006;8:361–362

Figure 1 shows the ECG surface leads during an episode of paroxysmal supraventricular tachycardia in a 69 year old man without obvious heart disease. Figure 2 shows continuous ECG tracing recorded upon termination of PSVT with an intravenous bolus of 6 mg adenosine. How do the ECG findings in Figure 2 help in the diagnosis of the mechanism of PSVT?

Tracing analysis

Figure 1 shows a regular, narrow SVT at a rate of 150/minute. As it is difficult to ascertain the presence of P waves on the tracing, all the following mechanisms may be discussed: a) "typical" AV nodal reentry tachycardia; b) AV reentrant tachycardia involving an accessory pathway in the retrograde direction; c) atrial tachycardia with 1:1 AV relationship; d) atrial flutter at a rate of 300/min with 2:1 AV conduction.

Figure 2 shows sudden termination of the SVT after slight rate slowing to 120/min. Such mode of termination is most consistent with a tachycardia mechanism involving the AV node (AVNRT and AVRT) and excludes the diagnosis of atrial flutter. Analysis of the tracing after resumption of sinus rhythm provides further insight into the possible diagnosis of the tachycardia. The first three QRS complexes (# 1–3) following tachycardia termination have a slow rate and a wide QRS configuration that is markedly different to that of the narrow QRS complexes present during tachycardia; in addition, they are preceded by P waves with varying coupling intervals. Therefore, these complexes have a ventricular origin. In contrast, all subsequent QRS complexes (# 4–11) have a supraventricular origin and are preceded by sinus P waves (arrows). The PR interval preceding complexes # 4–7 is 0.40

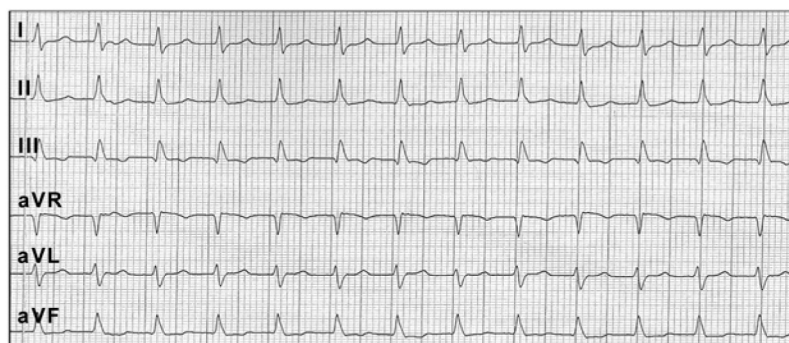


Figure 1. Before IV adenosine 6 mg

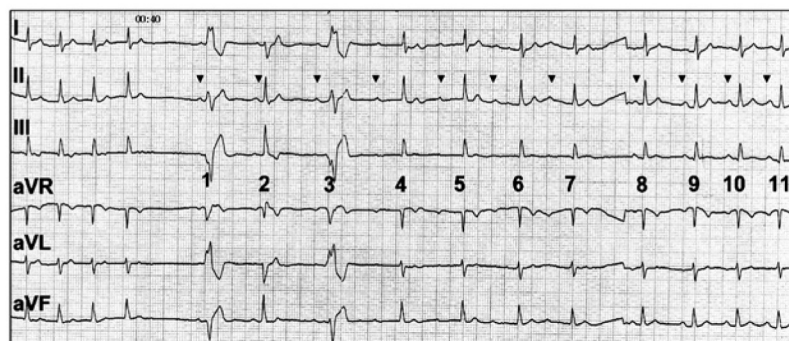


Figure 2. After IV adenosine

seconds. The following QRS complexes are, however, preceded by sinus P waves with shorter and constant PR intervals (0.19 sec).

The sudden shortening of PR interval from 0.40 sec (# 7) to 0.19 sec (# 8) is best explained by the existence of dual AV node physiology: the long PR interval reflecting block in a fast pathway and conduction over a slow pathway (before QRS complexes # 4–7), while normal PR interval reflects antegrade conduction over a fast pathway (before complexes # 8–11). An AV nodal echo was suspected at the end of QRS # 7 due to a marked resetting of the following sinus P wave. In summary, the documentation of dual AV node physiology after termination of PSVT with

PSVT = paroxysmal supraventricular tachycardia
AVNRT = atrioventricular nodal reentry tachycardia

adenosine does suggest (albeit not prove) that the mechanism of the tachycardia is actually AVNRT. In our patient, the latter mechanism was confirmed at electrophysiologic study.

Comment

Adenosine compounds have been shown to be very effective in terminating slow/fast AVNRT, almost always by blocking conduction in the slow pathway [1]. In addition, these agents have also been shown to be very useful for diagnostic purposes after injection in sinus rhythm [2], especially in revealing dual AV nodal pathways in patients with AVNRT [3]. We previously reported [4] a high incidence of dual AV node physiology (36.5%) upon termination of AVNRT with adenosine triphosphate, contrasting with its rare occurrence (5.5%) in a control group of patients with AVRT and no electrophysiologic evidence of dual AV node physiology. The present case confirms these observations and suggests that one should closely look at ECG tracings *after termination* of PSVT with adenosine.

References

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