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 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...
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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**Capsule**

**Gliding chromosomes**

During cell division, chromosomes must establish connections to the opposing spindle poles and become positioned at the spindle equator. Uncorrected errors in this biorientation inevitably lead to aneuploidy and are associated with cell transformation and cancers. How chromosomes attach properly to the mitotic apparatus is not understood. Kapoor et al. used live-cell two-color fluorescence, correlative light and electron microscopy, as well as chemical biology, to demonstrate surprisingly that chromosomes can congress to the spindle equator before they become bioriented. During congression, the leading kinetochore glides alongside kinetochore fibers of other already bioriented chromosomes toward microtubule plus ends. The gliding is mediated by the kinetochore-associated motor protein. Thus, cells possess a mechanism for repositioning mono-oriented chromosomes from the periphery to central areas of the spindle where they can establish connections to the other spindle pole.

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**Capsule**

**IL-2 and T cells**

The cytokine interleukin-2 (IL-2) facilitates proliferation of T cells, but several studies have shown that antibodies that bind IL-2, which at first glance should be inhibitory, can promote the expansion of subsets of memory CD8 T cells. Thus, IL-2 somehow might inhibit suppressive T cell populations that would otherwise prevent memory CD8 T cell expansion. Boyman et al. show that instead, binding of antibodies to IL-2 augments the direct activity of the cytokine on memory CD8 T cells themselves. Immune complexes form that focus local levels of IL-2 through presentation by Fc receptors. These observations could be important to consider in therapies that involve the manipulation of IL-2 and other cytokines, such as bone marrow transplantation and tumor immunotherapy.

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