MECHANISMS OF SUPRAVENTRICULAR TACHYCARDIAS

There are two principal mechanisms of supraventricular tachycardias: (1) increased automaticity, and (2) reentry. With ***increased automaticity,*** an automatic atrial focus begins to discharge rapidly on its own and usurps the pacemaking function from the SA node. This is the usual mechanism of atrial tachycardia that results from digitalis toxicity. Because the AV node is not involved in perpetuation of the arrhythmia, therapeutic measures designed to decrease AV nodal conduction will not terminate the tachycardia (although they may slow the ventricular response and/or increase the degree of AV block). Instead, treatment of atrial tachycardia must be directed toward correcting the underlying cause of the increased atrial automaticity (i.e., withholding digitalis).

Reentry is the other major mechanism of supraventricular tachycardia. Reentry may occur in a number of sites including the SA node, the atria, the AV node, and between the atria and ventricles via an accessory pathway. Sinoatrial reentry and intra-atrial reentry are both difficult to recognize electrocardiographically (without the benefit of electrophysiologic study), as well as being distinctly uncommon in adults. As a result, we do not address them further in our discussion.

Practically speaking, reentry involving the AV node is *by far* the most common mechanism of supraventricular tachycardia that the emergency care provider will encounter in adults.

Excerpted from pp 567-574 of Grauer K, Cavallaro D: ACLS: Comprehensive Review (Vol. 2) - 3rd Edition, Mosby Lifeline, St. Louis - 1993.

The Mechanism of Reentry

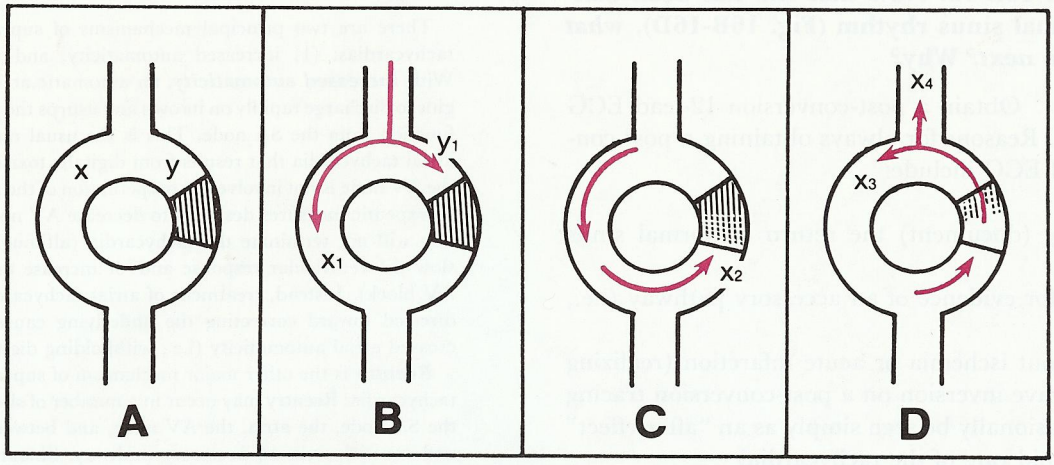

Figure 16B-19A. Theoretical illustration of a reentry circuit. The three necessary components are: (1) two separate pathways (labeled **x** and **y**); (2) unidirectional block in one of these pathways (represented by the lined area in pathway **y**); and (3) slow conduction along the other pathway.

Three conditions must be present for there to be a reentry circuit:

i. The existence of *two separate pathways* that join to form a closed circuit
ii. Unidirectional block in one of these pathways
iii. Slow conduction along the unblocked pathway

These conditions are illustrated in *Figure 16B-19A*. The two pathways are **x** and **y,** and they meet to form a closed loop (Panel A in Figure). Conduction is slowed down pathway **x.** A zone of *unidirectional* block exists along pathway **y** (lined area in the Figure).

When an impulse reaches the circuit, it begins to travel down both pathways. Because of the area of block along the faster pathway, conduction of the impulse is unable to complete the circuit (it is stopped at point $\mathbf{y_1}$ in Panel B). However, conduction of the impulse continues along the unblocked pathway (point $\mathbf{x_1}$ in Panel B). In order for the reentry circuit to be established, conduction must be *slow enough* along pathway **x** for the zone of block to have recovered its ability to conduct by the time the impulse labeled $\mathbf{x_2}$ arrives (Panel C). If this

Reentry in PSVT

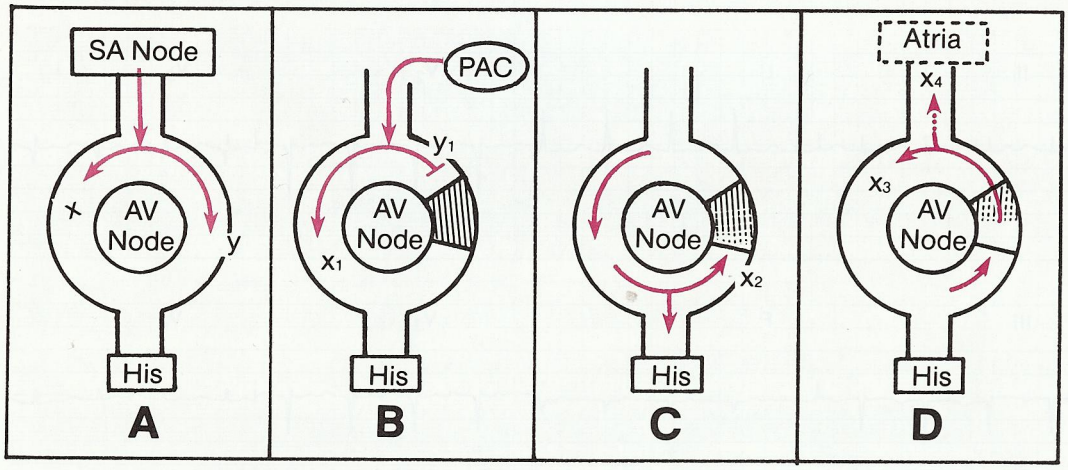

Figure 16B-19B. The theoretical illustration of a reentry circuit that was shown in Fig. 16B-19A is felt to apply to conditions existing at the AV node.

is the case, the impulse can be conducted through this zone, after which it is able to begin traveling around the circuit again (point x_3 in Panel D). Along the way, it may give off a retrograde impulse (x_4 in Panel D).

The three conditions described above that are necessary for reentry are felt to exist at the level of the AV node *(Figure 16B-19B)*. Thus, conduction through the AV node is *not* homogeneous (via a single pathway) as one might imagine. Instead there appears to be a *longitudinal dissociation* of conduction fibers, which results in a functional division into two separate conduction pathways. One of these pathways has a long refractory period and conducts *rapidly* (**y** in Panel A of Fig. 16B-19B). The other has a short refractory period and conducts *slowly* (**x** in Panel A).

Under normal conditions (as one might expect), the impulse is transmitted to the bundle of His by conduction along the faster pathway (Panel A in Fig. 16B-19B).

In Panel B a premature impulse (PAC) occurs. Due to the relatively long refractory period of the fast pathway, the premature impulse is likely to find conduction still blocked down this route when it arrives at the AV node (i.e., it is unable to travel beyond point y_1 in Panel B). However, it is likely that the shorter refractory period of the slow pathway will be over, allowing conduction to proceed via this route toward the bundle of His (x_1 in Panel B).

If conditions are just right, conduction down the slow pathway will outlast the refractory period of the fast pathway. In this case, by the time the impulse reaches point x_2 (Panel C), the fast pathway will have recovered enough to allow *retrograde* conduction. The reentry loop may then be perpetuated if at the time the impulse reaches point x_3, it is able to be conducted again down the slow pathway (Panel D). Along the way an impulse may be conducted (returned) to the atria (Point x_4), producing a *retrograde* P wave on the electrocardiogram (an *echo* beat).

PROBLEM **Consider the ECG shown in *Figure 16B-20.* Look closely at the QRS complex *during* the tachycardia. Is it different at all from the QRS complex during sinus rhythm (beats #1-3)?**

ANSWER TO FIGURE 16B-20 This tracing provides a nice example of how a laddergram may be used to illustrate the mechanism of an arrhythmia. Following three beats of sinus rhythm, a PAC occurs (notching the T wave that precedes beat #4). This first PAC conducts normally to the ventricles. However, a second PAC (that notches the T wave preceding beat #5) apparently sets up the conditions necessary for reentry to occur, and precipitates a run of PSVT at a rate of 180 beats/min.

Close scrutiny of the QRS complex *during* the tachycardia reveals that its appearance is *not* the same as during normal sinus rhythm (beats #1-3). Instead, a distinct notch is present during the tachycardia at the junction of the end of the QRS complex and the beginning of the ST segment (arrow). This notch is the result of retrograde conduction to the atria during the reentry tachycardia (and corresponds to point x_4 in Panel D of Fig. 16B-19B).

Several points may be made about this tracing. Note first how the reentry cycle is initiated by a PAC (corresponding to Panel B in Fig. 16B-19B). Note also that the PR interval of this PAC is *prolonged* compared to the PR interval of the normal sinus beats! This reflects the path of this impulse, which is being conducted down the *slow* pathway (corresponding to point x_1 in Panel B of Fig. 16B-19B). It is then conducted to the ventricles at the same time as it returns up the fast pathway (Panel C). Because conduction goes down the *slow* pathway and back up the fast pathway, this type of PSVT is known as the ***slow-fast form.*** It is by far the most common type in adults. (As we will see momentarily, there is also a *fast-slow* form of PSVT in which conduction first goes down the fast pathway and comes back up the slow pathway.)

ATRIAL ACTIVITY IN PSVT

The presence of atrial activity, its polarity, and its location relative to the QRS complex provide important clues to the mechanism of PSVT *(Figure 16B-21)*. With normal sinus

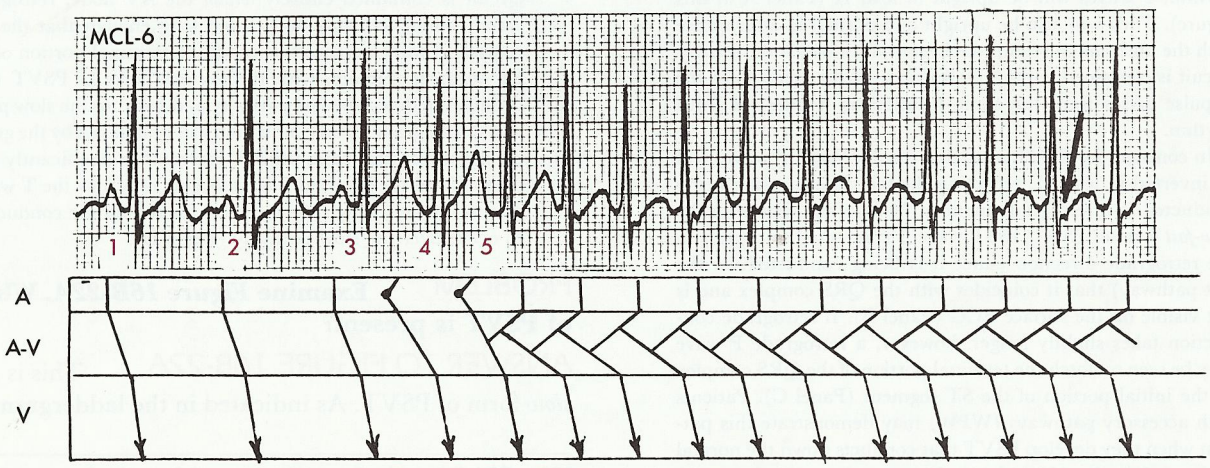

Figure 16B-20. *Is there evidence of atrial activity during the supraventricular tachyarrhythmia that begins after beat #5?*

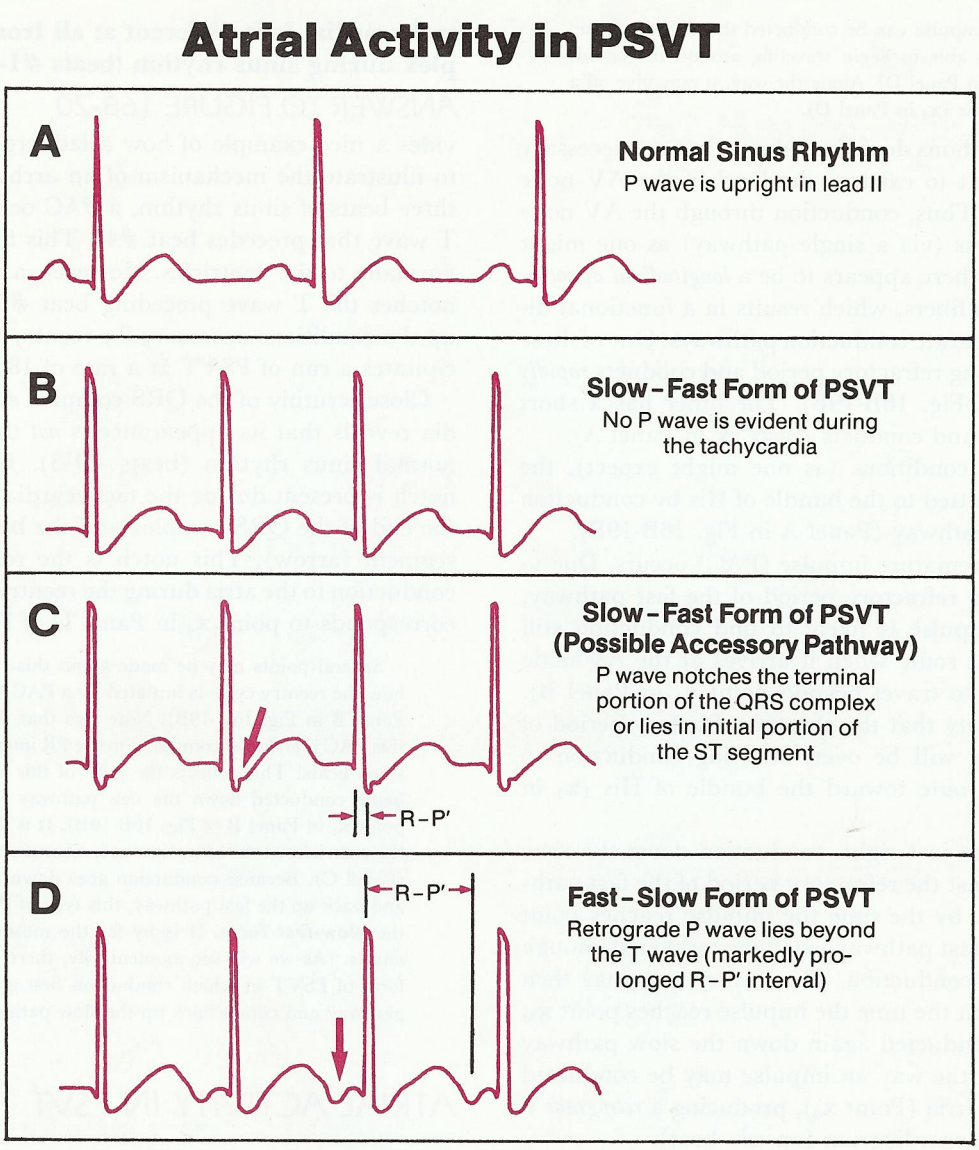

Figure 16B-21. Atrial activity in PSVT.

rhythm, P waves will be upright in lead II (Panel A in this Figure). They will also be upright and normal in appearance with the uncommon reentry tachycardias in which the reentry circuit is contained within the SA node or the atria, since the impulse arises from the same location as it does for sinus rhythm.

In contrast, for PSVT involving the AV node, P waves will be inverted in lead II, reflecting the fact that they are being conducted in a retrograde manner. Most of the time with the *slow-fast* form of PSVT, the P wave is conducted so rapidly in the retrograde direction (since it is being conducted via the fast pathway) that it coincides with the QRS complex and is not visible on the surface ECG (Panel B). If retrograde conduction takes slightly longer, however, a retrograde P wave may be seen to notch the terminal portion of the QRS complex or the initial portion of the ST segment (Panel C). Patients with accessory pathways (WPW) may demonstrate this pattern when they develop PSVT that conducts down the normal pathway (via the AV node) and retrograde up the accessory pathway. Because the reentry circuit of patients with an accessory pathway is longer than that for patients whose reentry circuit is contained entirely *within* the AV node, retrograde atrial conduction may be delayed long enough that the retrograde P wave notches the middle or terminal portion of the ST segment. Finally, with the *fast-slow* form of PSVT (i.e., conduction down the fast pathway and back up the slow pathway), retrograde atrial conduction will be delayed by the greatest degree. In this case, the RP′ interval* is significantly prolonged (and the inverted P wave occurs only *after* the T wave) due to the very long time required for retrograde conduction to occur over the slow pathway (Panel D).

PROBLEM **Examine *Figure 16B-22A*. What type of PSVT is present?**

ANSWER TO FIGURE 16B-22A This is the *fast-slow* form of PSVT. As indicated in the laddergram shown

*The ***RP′ interval*** refers to the interval from the R wave of the QRS complex until the electrocardiographic marker (P′) of retrograde atrial activity.

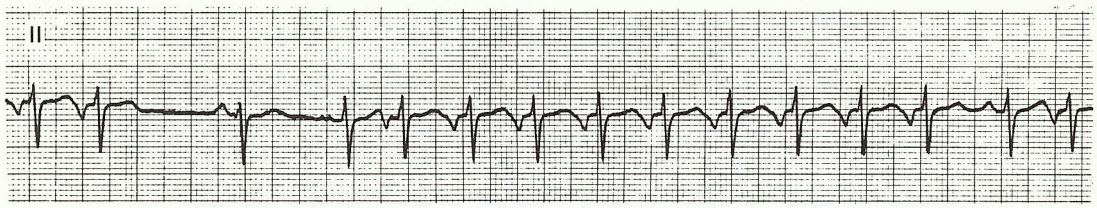

Figure 16B-22A. *What type of PSVT is present?*

in Figure 16B-22B, beat #3 is sinus conducted. Beat #4 is conducted by a P wave with a different morphology (i.e., an ectopic P wave). Following this, the tachycardia begins. As can be seen, the RP′ interval is greatly prolonged, reflecting the *delay* in retrograde transmission of the impulse over the slow pathway.

It is of interest that the tachycardia in Fig. 16B-22B is not only initiated by a PAC, but it is also terminated by a PAC (that produces a slight peak in the T wave of the third to last beat). Because of the reentry nature of the arrhythmia, premature impulses are likely to alter conduction properties of at least a portion of the reentry circuit and abruptly (i.e., *paroxysmally*) either initiate or terminate the cycle.

Clinically, the reason it may be important to differentiate between the fast-slow form of PSVT (seen in Fig. 16B-22A) and the much more common slow-fast form (seen in Fig. 16B-20) is the different response to therapy. In general, the less common fast-slow form of PSVT is much more difficult to treat. Although similar antiarrhythmic medications are used in long-term management, recurrences are often so frequent with the fast-slow form that this arrhythmia is sometimes called an "incessant" tachycardia.

The "incessant" tachycardia (fast-slow form) of PSVT is seen much more often in children than in adults.

PROBLEM **Return one last time to the case of the middle-aged woman whose presenting ECG was shown in Fig. 16B-15 (on page 565). *Do you now see a clue on this tracing that suggests the mechanism of the arrhythmia?***

ANSWER Review of the tachyarrhythmia shown in Fig. 16B-15 reveals that there *is* evidence of retrograde atrial activity during the tachycardia. Although admittedly subtle, a notch is present in the terminal portion of the QRS complex in leads II, III, and aVF on this tracing that is *not* present on the post-conversion tracing shown in Fig. 16B-18. This identification of retrograde atrial activity virtually confirms the reentry nature of the tachyarrhythmia, and supports the use of management measures predicated on interrupting AV nodal reentry (i.e., vagal maneuvers, adenosine, verapamil, digoxin, and/or β-blockers).

Lest there be any doubt about the presence of retrograde atrial activity during the tachyarrhythmia shown in Fig. 16B-15, we provide blow-up pictures of several key leads from the 12-lead ECGs during the tachycardia and after conversion to normal sinus rhythm. Thus, *Figure 16B-23A* is a blow-up picture of leads II, III, aVL, and aVF from the post-conversion tracing shown previously in Fig. 16B-18. The arrow in lead II documents return of normal sinus activity. Similarly, *Figure 16B-23B* is a blow-up picture of these same four leads from the 12-lead tracing during the tachycardia (i.e., from Fig. 16B-15). Arrows in Fig. 16B-23B clearly demonstrate notching in the terminal portion of the QRS complex *during* the tachycardia that is *not* present in Fig. 16B-23A after conversion to sinus rhythm. This notching represents retrograde atrial activity.

Our reason for focusing on such subtle evidence of atrial activity during the run of PSVT is threefold:

i. It makes analyzing these tachyarrhythmias a much more interesting task.

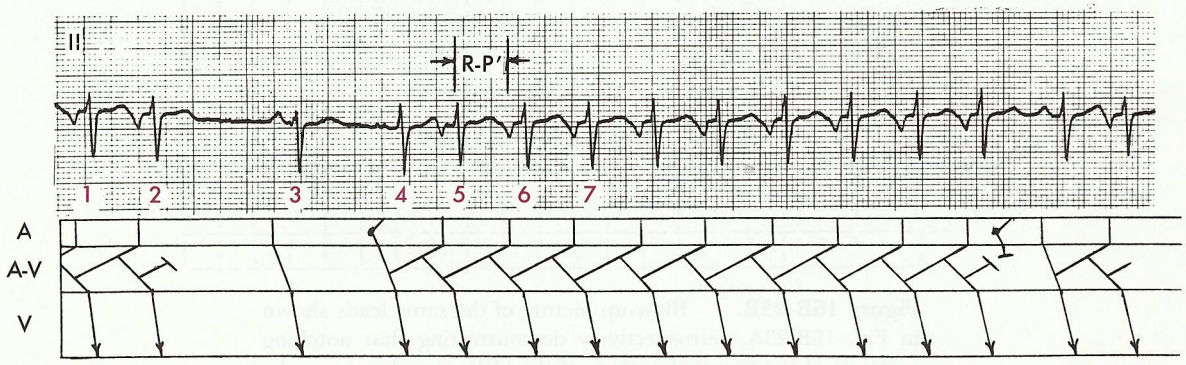

Figure 16B-22B. Laddergram demonstrating a reentry tachycardia that is precipitated by an ectopic P wave (beat #4), and which manifests a prolonged R-P′ interval (suggestive of the fast-slow mechanism form of PSVT).

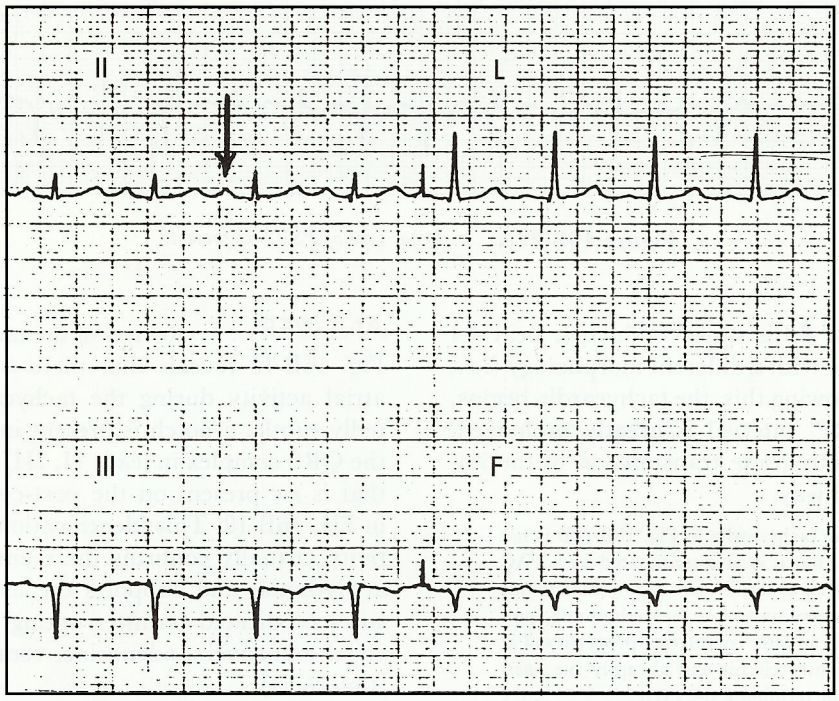

Figure 16B-23A. Blow-up picture of leads II, III, aVL, and aVF of the post-conversion tracing (shown in Fig. 16B-18). The arrow in lead II documents return of normal sinus activity.

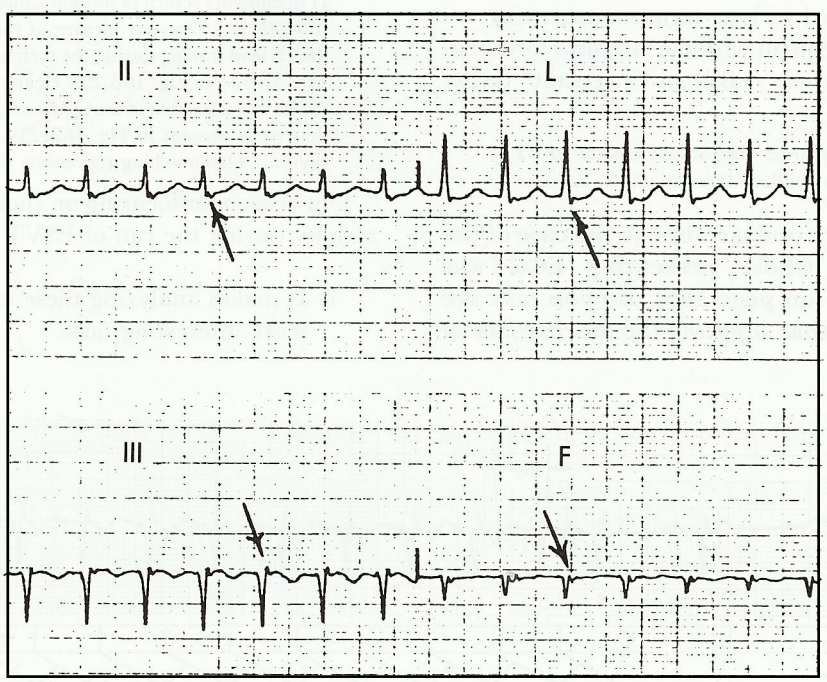

Figure 16B-23B. Blow-up picture of the same leads shown in Fig. 16B-23A, retrospectively demonstrating that notching (arrows) of the terminal portion of the QRS complex *during* the tachycardia (in Fig. 16B-15) is the result of retrograde atrial activity.

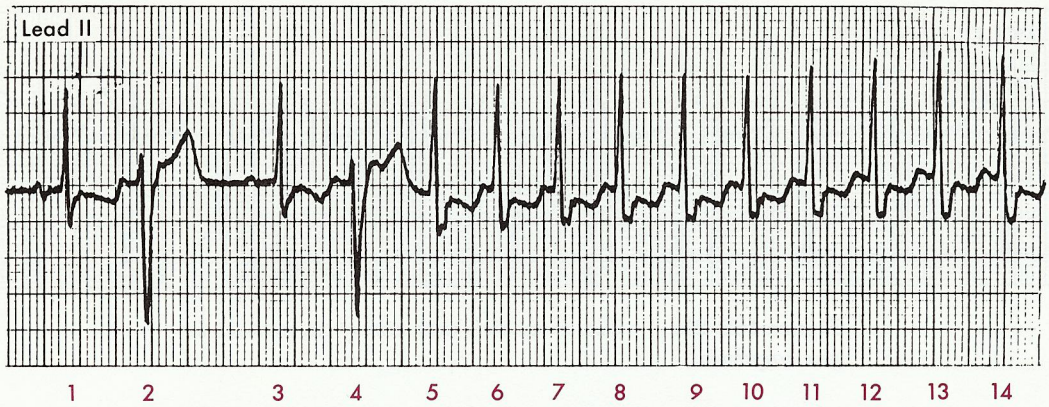

Figure 16B-24A. *Is there a clue to the mechanism of the run of PSVT (beats #5-14)?*

ii. Detection of retrograde atrial activity during the tachycardia provides an extremely helpful clue to the mechanism of the arrhythmia (and therefore to its likely response to therapy).

iii. As we will see in Section C of this chapter, the presence and location of retrograde atrial activity during the run of PSVT also provides a helpful clue to the likelihood of the existence of an accessory pathway.

PROBLEM **Try one final example of PSVT. Is there a clue to the mechanism of the run of PSVT (beats #5-14) in *Figure 16B-24A? Might the second PVC (beat #4) in this tracing be playing a role in initiation of the reentry tachycardia?***

HINT: Is there evidence of atrial activity associated with this second PVC?

HINT TO THE HINT: Is the T wave of the second PVC (the T wave of beat #4) different than the T wave of the first PVC (the T wave of beat #2)?

ANSWER TO FIGURE 16B-24A The rhythm begins with a sinus-conducted beat (#1), followed by a PVC (beat #2), another sinus-conducted beat (#3), and a second PVC (#4)—after which the run of PSVT begins. Note that the S wave of the QRS complex *during* the tachycardia (beats #5-14) is *wider* than the S wave of the two sinus-conducted beats (#1 and #3). This S wave widening probably reflects *retrograde atrial activity* (back up the fast pathway), and suggests that beats #5-14 represent the slow-fast form of PSVT.

The laddergram shown in *Figure 16B-24B* provides insight to the onset of the tachycardia. Note the negative deflection that deforms the T wave of the second PVC, after which the run of PSVT begins. This negative deflection is probably the result of *retrograde* atrial conduction, which then initiates the reentry tachycardia.

Although far from being a simple tracing, Fig. 16B-24B illustrates that reentry tachycardia may be initiated by PVCs as well as by PACs.

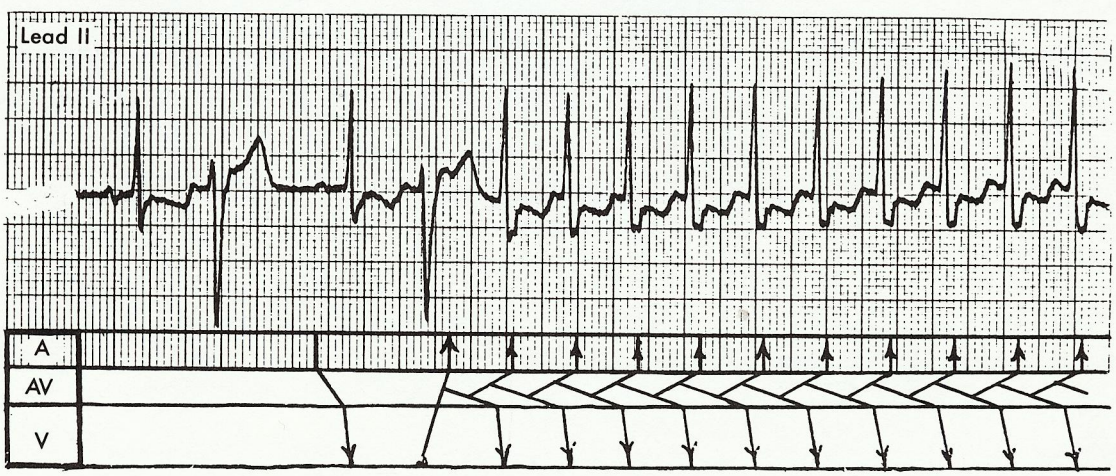

Figure 16B-24B. Laddergram of the arrhythmia shown in Fig. 16B-24A, suggesting that retrograde atrial conduction from the second PVC initiates the run of reentry tachycardia.

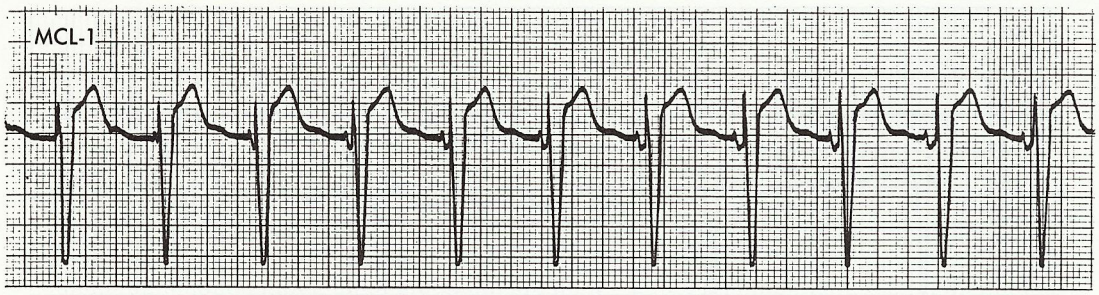

Figure 16B-25. *What is the likely mechanism of this narrow complex arrhythmia?*

PROBLEM **We conclude this section with the narrow complex arrhythmia shown in *Figure 16B-25.* What is the likely mechanism of this rhythm? *What clinical conditions are most commonly associated with this arrhythmia*?**

ANSWER TO FIGURE 16B-25 The rhythm becomes regular after the first complex at a rate of 95 beats/min. The QRS is 0.10 second in duration, or at the upper limit of normal. Atrial activity is evident; however, the PR interval constantly varies! At least in the initial part of the tracing the PR interval is far too short to allow normal conduction. Therefore, *AV dissociation is present.* The underlying rhythm is *junctional tachycardia.* This rhythm most often occurs in the setting of digitalis toxicity, but it may also be seen in association with acute inferior infarction, or after open heart surgery.

The mechanism of junctional tachycardia is *increased automaticity.* Because retrograde block in the AV node is frequently seen with this arrhythmia, the SA node is able to continue firing at its own inherent rate. AV dissociation is the usual result. ***The presence of AV dissociation per se effectively rules out any type of reentry tachycardia!***

Strictly speaking, it would be more correct to refer to the arrhythmia shown in Fig. 16B-25 as an *accelerated* junctional rhythm rather than junctional tachycardia since the heart rate is less than 100 beats/min. Clinical implications of an accelerated junctional rhythm and junctional tachycardia are the same.

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