Atrioventricular nodal reentrant tachycardia (AVNRT) is the most frequent paroxysmal supraventricular tachycardia (SVT) with an incidence in the general population of approximately 5/1000. AVNRT is regarded as a benign arrhythmia although hemodynamic compromise (e.g., dizziness or syncope) can occur. It is usually not associated with structural heart disease. AVNRT can be more frequent in women (~60%) with a peak incidence in our patient population at a mean age of 50 years [9], but AVNRT can be encountered at any age, even in children. AVNRT may cause a significant worsening of the quality of life especially with frequent recurrence. The development of catheter ablation has provided the possibility to definitely cure this arrhythmia.

5.1 Mechanism

AVNRT has traditionally been thought to be due to a reentrant mechanism. The AV node in affected patients is characterized by a so-called longitudinal dissociation due to the existence of fibers with differences in conduction velocity and refractory periods. The part of the AV node called the “fast pathway” has a faster conduction time and a longer refractory period, while the “slow pathway” has a long conduction time and a shorter refractory period. Initially, dual AV nodal physiology was thought to be exclusively present in patients with AVNRT and rare in patients without this arrhythmia. However, studies demonstrated that this pattern can be found in up to 10% of normal subjects who have never suffered from an AVNRT [5]. Subsequent observations suggested that fast and slow pathway represent conduction over different atrionodal connections. Sung et al. [4] first demonstrated that the two pathways actually have two anatomically distinct atrial insertions. During ventricular pacing in patients with dual ventriculoatrial conduction, these authors were able to demonstrate a different retrograde atrial activation sequence depending on whether conduction utilizes the fast or the slow pathway. The retrograde atrial exit of the fast pathway was localized in the anterior part of the septum near the His bundle recording site, while the retrograde atrial exit of the slow pathway was located in the posterior part of the septum near the coronary sinus ostium (CS os), i.e., inferior and posterior to the exit of the fast pathway.

During sinus rhythm, an atrial impulse traverses the fast AV nodal conduction pathway and activates the ventricles via the His bundle. The impulse simultaneously conducts down the slow AV pathway, reaching the His bundle shortly after it has been depolarized via the fast pathway and therefore is refractory (Figure 5.1a).

When an atrial premature depolarization occurs, the impulse may not conduct over the fast AV pathway due to its longer refractory period, and proceeds slowly down the slow AV pathway. This leads to a sudden prolongation of the AH interval and to a corresponding prolonged PR interval. A beat-to-beat change of the AH interval during programmed atrial pacing is a marker for dual pathway physiology. The phenomenon of a sudden prolongation (≥50 ms) of the AH interval (see also Chapter 1, Figure 1.14) during decremental programmed stimulation (with a 10 ms decrement in coupling interval) is called a “jump” (Figure 5.1b).

If conduction down the slow pathway is sufficiently delayed to allow the fast pathway to recover from refractoriness, retrograde conduction over the fast pathway leads to an early retrograde atrial activation. This early retrograde atrial activation is referred to as an “AV nodal echo beat” (Figure 5.1b).

Both phenomena, “jump” and “echo” are often associated and can initiate AVNRT. Some-
Fig. 5.1. The mechanism of AVNRT. (a) During sinus rhythm an atrial impulse antegradeley penetrates both the fast and slow pathways simultaneously. Because the impulse reaches the His bundle via the fast pathway, the PR interval is normal. (b) The effective refractory period of the fast pathway is longer than that of the slow pathway. Conduction over the fast pathway is blocked by an atrial premature beat and conducts down the slow pathway to activate the His bundle. Therefore the PR interval is prolonged. Because the antegrade conduction via the slow pathway is slow, the fast pathway has enough time to regain its excitability. The impulse that was conducted down the slow pathway may therefore reexcite the fast pathway retrogradely, resulting in an AV nodal echo beat. (c) Conduction over the fast pathway is blocked by a short coupling atrial premature beat and conducts down the slow pathway very slowly. This provides time for the fast pathway to recover to allow retrograde conduc-
tion. The slow pathway now also has had time to recover to allow repetitive antegrade reentrance. The continuation of this process leads to the development of typical slow-fast AVNRT. (d) Fast-slow AVNRT. In this uncommon type of AVNRT, antegrade conduction occurs over the fast pathway while the retrograde conduction is over the slow pathway with a short AH interval and a long HA interval. (e) Slow-slow AVNRT. In this type of tachycardia, the slow pathway is used for antegrade conduction and retrograde conduction is over a different slow pathway. It is characterized by relatively long and similar HA and AH intervals.

At times the following phenomenon is observed with programmed atrial stimulation: Decreasing the coupling interval in 10 ms steps, a jump and an echo can be observed, but only after further decremental pacing tachycardia is initiated. An explanation for this phenomenon might be that retrograde fast pathway excitation is only possible if an earlier atrial premature beat (blocked in the fast pathway) conducts more slowly down the slow pathway and arrives later at the fast pathway, when the latter is again excitable. Because of the longer overall conduction time from the first atrial premature beat until atrial reexcitation via the retrograde fast pathway, the slow pathway has had more time to recover excitability and sustained tachycardia results (Figure 5.1 c).

Individuals may demonstrate “dual AV nodal physiology” but will not have spontaneous or inducible AVNRT. It is important to distinguish patients with dual AV nodal physiology but no inducible AVNRT from those with inducible AVNRT. Isoproterenol infusion may be required for initiation of the tachycardia and should be