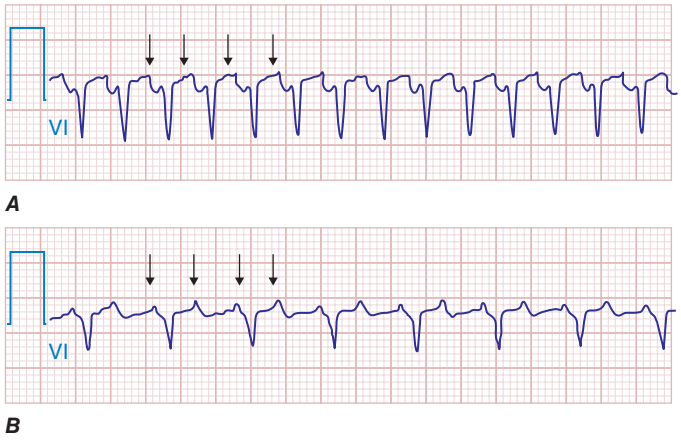

FIGURE 276-4 Atrial tachycardia (AT) with 1:1 and 2:1 atrioventricular (AV) conduction. *Arrows* indicate p waves. ***A.*** AT with 1:1 AV relationship and R-P > P-R. ***B.*** Same AT with 2:1 AV relationship after AV nodal–blocking agent administered. *(Adapted from F Marchlinski: The tachyarrhythmias. In Longo DL et al [eds]: Harrison's Principles of Internal Medicine, 18th ed. New York, McGraw-Hill, 2012, pp 1878–1900.)*

Acute management of sudden-onset, sustained AT is the same as for PSVT (see below), but the response to pharmacologic therapy is variable, likely depending on the mechanism. For AT due to reentry, administration of adenosine or vagal maneuvers may transiently increase AV block without terminating tachycardia. Some ATs terminate with a sufficient dose of adenosine, consistent with triggered activity as the mechanism. Cardioversion can be effective in some, but fails in others, suggesting automaticity as the mechanism. Beta blockers and calcium channel blockers may slow the ventricular rate by increasing AV block, which can improve tolerance of the arrhythmias. Potential precipitating factors and intercurrent illness should be sought and corrected. Underlying heart disease should be considered and excluded.

For patients with recurrent episodes, beta blockers, the calcium channel blockers diltiazem or verapamil, and the antiarrhythmic drugs flecainide, propafenone, disopyramide, sotalol, and amiodarone can be effective, but potential toxicities and adverse effects often warrant avoiding these agents (Tables 276-3, 276-4, and 276-5). Catheter ablation targeting the AT focus is effective in more than 80% of patients and is recommended for recurrent symptomatic AT when drugs fail or are not desired or for incessant AT causing tachycardia-induced cardiomyopathy.

Atrioventricular Nodal Reentry Tachycardia AV nodal reentry tachycardia (AVNRT) is the most common form of PSVT, representing approximately 60% of cases referred for catheter ablation. It most commonly manifests in the second to fourth decades of life, often in women. It is often well tolerated, but rapid tachycardia, particularly in the elderly, may cause angina, pulmonary edema, hypotension, or syncope. It is not usually associated with structural heart disease.

The mechanism is reentry involving the AV node and likely the perinodal atrium, made possible by the existence of multiple pathways for conduction from the atrium into the AV node (Fig. 276-5). In the most common form, a slowly conducting AV nodal pathway extends from the compact AV node near the bundle of His, inferiorly along the tricuspid annulus, adjacent to the coronary sinus os. The reentry wavefront propagates up this slow pathway to the compact AV node and then exits from the fast pathway at the top of the AV node. The path back to the slow pathway to complete the circuit is not defined. The conduction time from the compact AV node region to the atrium is similar to that from the compact node to the His bundle and ventricles, such that atrial activation occurs at about the same time as ventricular activation. The p wave is therefore inscribed during, slightly before, or slightly after the QRS and can be difficult to discern. Often the P wave is seen at the end of the QRS complex as a pseudo-r′ in lead V_1 and pseudo-S waves in leads II, III, and aVF (Fig. 276-5*A*). The rate can vary with sympathetic tone. Simultaneous atrial and ventricular contraction results in atrial contraction against a closed tricuspid valve that produces cannon a waves visible in the jugular venous pulse and that the patient often perceives as a fluttering sensation in the neck. Elevated venous pressures may also lead to release of natriuretic peptides that cause posttachycardia diuresis. Less frequently, the AV nodal reentry circuit revolves in the opposite direction and gives rise to a tachycardia with an R-P interval longer than the P-R interval, similar to AT. The p wave will have the morphology noted above, and in contrast to ATs, maneuvers or medications that produce AV block terminate the arrhythmia.

Acute treatment is the same as for PSVT (discussed below). Whether ongoing therapy is warranted depends on the severity of symptoms and frequency of episodes. Reassurance and instruction as to performance of the Valsalva maneuver to terminate episodes are sufficient for many patients. Administration of an oral beta blocker, verapamil, or diltiazem at the onset of an episode has been used to

TABLE 276-3 COMMONLY USED ANTIARRHYTHMIC AGENTS—INTRAVENOUS DOSE RANGE/PRIMARY INDICATION

Drug	Loading	Maintenance	Primary Indication	Class[a]
Adenosine	6–18 mg (rapid bolus)	N/A	Terminate reentrant SVT involving AV node	—
Amiodarone	15 mg/min for 10 min, 1 mg/min for 6 h	0.5–1 mg/min	AF, AFL, SVT, VT/VF	III
Digoxin	0.25 mg q2h until 1 mg total	0.125–0.25 mg/d	AF/AFL rate control	—
Diltiazem	0.25 mg/kg over 3–5 min (max 20 mg)	5–15 mg/h	SVT, AF/AFL rate control	IV
Esmolol	500 μg/kg over 1 min	50 μg/kg per min	AF/AFL rate control	II
Ibutilide	1 mg over 10 min if over 60 kg	N/A	Terminate AF/AFL	III
Lidocaine	1–3 mg/kg at 20–50 mg/min	1–4 mg/min	VT	IB
Metoprolol	5 mg over 3–5 min × 3 doses	1.25–5 mg q6h	SVT, AF rate control; exercise-induced VT; long QT	II
Procainamide	15 mg/kg over 60 min	1–4 mg/min	Convert/prevent AF/VT	IA
Quinidine	6–10 mg/kg at 0.3–0.5 mg/kg per min	N/A	Convert/prevent AF/VT	IA
Verapamil	5–10 mg over 3–5 min	2.5–10 mg/h	SVT, AF rate control	IV

[a]Classification of antiarrhythmic drugs: class I—agents that primarily block inward sodium current; class IA agents also prolong action potential duration; class II—antisympathetic agents; class III—agents that primarily prolong action potential duration; class IV—calcium channel–blocking agents.

Abbreviations: AF, atrial fibrillation; AFL, atrial flutter; AV, atrioventricular; SVT, supraventricular tachycardia; VF, ventricular fibrillation; VT, ventricular tachycardia.