establish the diagnosis. AV block with only transient slowing of tachycardia may expose ongoing p waves, indicating AT or atrial flutter as the mechanism.

Carotid sinus massage is reasonable provided the risk of carotid vascular disease is low, as indicated by absence of carotid bruits and no prior history of stroke. A Valsalva maneuver should be attempted in cooperative individuals, and if effective, the patient can be taught to perform this maneuver as needed. If vagal maneuvers fail or cannot be performed, intravenous adenosine will terminate the vast majority of PSVT by transiently blocking conduction in the AV node. Adenosine may produce transient chest pain, dyspnea, and anxiety. It is contraindicated in patients with prior cardiac transplantation due to potential hypersensitivity. It can theoretically aggravate bronchospasm. Adenosine precipitates atrial fibrillation, which is usually brief, in up to 15% of patients, so it should be used cautiously in patients with WPW syndrome in whom AF may produce hemodynamic instability. Intravenous beta blockers and calcium channel blockers (verapamil or diltiazem) are also effective but may cause hypotension before and after arrhythmia termination and have a longer duration of action. These agents can also be given orally and can be taken by the patient on an as-needed basis to slow ventricular rate and facilitate termination by Valsalva maneuver.

The differential diagnosis of wide-complex tachycardia includes ventricular tachycardia **(Chap. 277)**, PSVT with bundle branch block aberrancy, and preexcited tachycardia (see above). In general, these should be managed as ventricular tachycardia until proven otherwise. If the tachycardia is regular and the patient is stable, a trial of intravenous adenosine is reasonable. Very irregular wide-complex tachycardia should be managed with cardioversion, intravenous procainamide, or ibutilide, which presumes preexcited atrial fibrillation or flutter (see above). If the diagnosis of PSVT with aberrancy is unequivocal, as may be the case in patients with prior episodes, treatment for PSVT is reasonable. In all cases, continuous ECG monitoring should be implemented, and emergency cardioversion and defibrillation should be available.

COMMON ATRIAL FLUTTER AND MACROREENTRANT ATRIAL TACHYCARDIAS

Macrorrentrant atrial tachycardia is due to a large reentry circuit, often associated with areas of scar in the atria. *Common or typical right atrial flutter* is due to a circuit that revolves around the tricuspid valve annulus, bounded anteriorly by the annulus and posteriorly by functional conduction block in the crista terminalis. The wavefront passes through an isthmus between the inferior vena cava and the tricuspid valve annulus, known as the sub-Eustachian or cavotricuspid isthmus, where it is susceptible to interruption by catheter ablation. Thus, common atrial flutter is *cavotricuspid isthmus-dependent atrial flutter*. This circuit most commonly revolves in a counterclockwise direction (as viewed looking toward the tricuspid annulus from the ventricular aspect), which produces the characteristic negative sawtooth flutter waves in leads II, III, and aVF and positive P waves in lead V_1 **(Fig. 276-10)**. When the direction is reversed, clockwise rotation produces the opposite P-wave vector in those leads. The atrial rate is typically 240–300 beats/min but may be slower in the presence of atrial disease or antiarrhythmic drugs. It often conducts to the ventricles with 2:1 AV block, creating a regular tachycardia at 150 beats/min, with p waves that may be difficult to discern. Maneuvers that increase AV nodal block will typically expose flutter waves, allowing diagnosis.

Common right atrial flutter often occurs in association with atrial fibrillation and with atrial scar from senescence or prior cardiac surgery. Some patients with atrial fibrillation that is treated with an antiarrhythmic drug, particularly flecainide, propafenone, or amiodarone, will present with atrial flutter rather than fibrillation.

Macroreentrant ATs that are not dependent on conduction through the cavotricuspid isthmus are referred to as *atypical atrial flutters*. They can occur in either atrium and are usually associated with areas of scar. Left atrial flutter and perimitral left atrial flutter are commonly seen after extensive left atrial ablation for atrial fibrillation or atrial surgery. The clinical presentation is similar to common atrial flutter, but with different P-wave morphologies. They can be difficult to distinguish from focal AT, and in most cases, the mechanism can only be confirmed by an electrophysiology study.

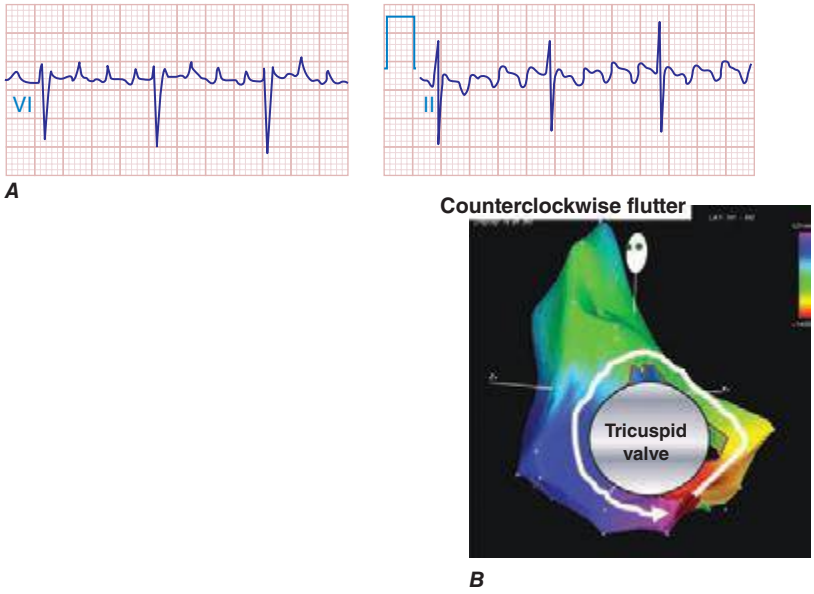

FIGURE 276-10 ***A.*** Common right atrial flutter, also known as cavotricuspid isthmus flutter, showing positive P waves in lead V_1 and negative "sawtooth" pattern in lead II typical of counterclockwise rotation relative to the tricuspid valve annulus. *(Adapted from F Marchlinski: The tachyarrhythmias. In Longo DL et al [eds]: Harrison's Principles of Internal Medicine, 18th ed. New York, McGraw-Hill, 2012, pp 1878–1900.)* ***B.*** A right atrial map of common counterclockwise flutter is shown. Colors indicate activation time, progressing from red to yellow to green, blue, and purple. The reentry path parallels the tricuspid annulus.