Macro-reentrant atrial arrhythmias are a consequence of stable reentrant circuits, which encompass large portions of the atria. All such circuits require a central obstacle and a region of slowed atrial conduction related to atrial dilation or fibrosis. The most common of these arrhythmias is *typical atrial flutter,* which is mediated by right atrial reentry around normal anatomic obstacles. In addition to typical flutter, reentry may occur around acquired obstacles, most commonly scars resulting from prior cardiac surgery or ablation involving the atria. Reentrant arrhythmias tend to manifest clinically as paroxysmal sustained or persistent arrhythmias. Although they may be self-terminating and episodic, individual episodes tend to be protracted.

The final mechanism of atrial arrhythmia is AF. This arrhythmia involves components of focal automatic mechanisms and reentry. The major advances made in the understanding and management of this common arrhythmia are reviewed in the following sections.

Focal Atrial Tachycardia

Focal atrial tachycardia also is referred to as ectopic atrial tachycardia and automatic atrial tachycardia. These terms describe a characteristic clinical pattern that usually manifests as runs of unifocal PACs lasting for seconds or minutes, usually followed by spontaneous termination and subsequent spontaneous reinitiation of additional salvos of tachycardia (see Fig. 9-6A). This arrhythmia less commonly manifests as a paroxysmal sustained tachycardia. When mapped in the electrophysiologic laboratory, these arrhythmias have a focal origin, and although they are sometimes triggered by rapid pacing, suggesting triggered activity, they appear to be automatic rather than a reentrant mechanism.

The electrocardiographic features are characteristic and usually permit accurate diagnosis. Because the arrhythmia is focal and automatic, the morphology of the first PAC of the run is identical to the subsequent PACs. Cycle length tends to vary between and within runs, and tachycardia is unaffected by intermittent AV block, which may occur during the runs. The same focus often fires erratically between runs, resulting in frequent atrial ectopy that is morphologically similar to the P wave observed during the runs.

The arrhythmia appears to be caused by intracellular calcium overload and resultant triggered activity related to delayed afterdepolarizations, making it responsive to calcium-channel blockers and β-blockers. The paroxysmal sustained form of this arrhythmia is also adenosine responsive, giving the false impression of dependence on AV conduction. The use of digoxin may exacerbate triggered causes of atrial tachycardia. Class IC agents, such as flecainide and propafenone, may be useful in patients without structural heart disease or coronary artery disease. Amiodarone can also be used in these patients for rhythm control. The arrhythmia is readily amenable to catheter ablation if ectopy occurs frequently enough to permit mapping.

Typical Atrial Flutter

Atrial flutter is a persistent atrial arrhythmia with an atrial rate of at least 250 beats per minute (see Fig. 9-6C). Because the normal AV node cannot conduct 1 : 1 at these rates, this arrhythmia characteristically manifests with 2 : 1 conduction and a ventricular response of about 140 to −150 beats per minute. During 2 : 1 conduction, the difficulty in perceiving flutter waves may lead to diagnostic confusion. Typical atrial flutter is the most common form of this arrhythmia, and it is mediated by macro-reentry restricted to the right atrium. The central obstacles in this circuit consist of normal anatomic structures, accounting for its stereotyped pattern.

Typical atrial flutter is mediated by counterclockwise reentry around the tricuspid valve as viewed from the ventricle. The valve prevents anterior collapse of the circuit, and posteriorly a long ridge in the atrial wall (i.e., crista terminalis) forms a functional line of block, preventing the circuit from collapsing posteriorly. Because the normal obstacles already exist, flutter development results from the abnormally slowed conduction related to atrial enlargement, fibrosis, or edema, which sometimes is combined with shortened atrial refractory periods due to catecholamine stress. Typical counterclockwise atrial flutter demonstrates a deeply negative F wave in leads II, III, and aVF; a sharply positive F wave in V_1; and a negative F wave in V_6.

A less common reversed form of this arrhythmia is caused by clockwise reentry around the tricuspid valve. It demonstrates an ECG exactly opposite to the counterclockwise form, with a strongly positive F wave in leads II, III, and aVF; a sharply negative F wave in V_1; and a positive F wave in V_6. In both cases, the F waves are often difficult to perceive because of 2 : 1 conduction. If the unusual F-wave vector is not recognized, the ECG may be misinterpreted as sinus tachycardia. Clues to identification of atrial flutter are persistent, unexplained heart rates of about 150 beats per minute with a variation of only a few beats per minute over time and the finding of a negative P wave in the inferior leads, which is expected to be positive in sinus rhythm.

The most fruitful method of diagnosis is the provocation of transient AV block with carotid sinus massage or adenosine infusion. This transiently exposes the underlying flutter waves but does not terminate the arrhythmia.

Although acute therapy involves rate control or cardioversion if drugs are poorly tolerated, long-term rate control for this arrhythmia is difficult. Drug doses that result in acceptable block at rest often fail to control exercise rates, and doses that result in exercise rate control often provoke bradycardia at rest. Early restoration of sinus rhythm is preferred for this arrhythmia.

Atrial flutter is a common transient arrhythmia in acute care hospital settings. The right atrial wall is thin, and pericarditis resulting from cardiac or thoracic surgery results in atrial edema and inflammation that may permit adequate slowing and promote transient atrial flutter. Acute pulmonary decompensation may result in right heart failure and may promote transient atrial flutter. In all of these settings, endogenous or pharmacologic catecholamine stimulation exacerbates the arrhythmia. Transient therapy for up to a month is appropriate in these settings.

When atrial flutter occurs in the absence of an acute precipitant, long-term therapy is required. Given the difficulty of achieving rate control in atrial flutter and the need for antiarrhythmic agents with associated potential morbidity to maintain sinus rhythm, catheter ablation has become the primary means of treating this arrhythmia. Antiarrhythmic therapy for atrial flutter is similar to that for AF (discussed later). Antiarrhythmic drug