therapy should be reserved for temporary treatment of likely transient flutter or for patients who are not suitable candidates for invasive management. Catheter ablation of typical atrial flutter is a low-risk procedure with a long-term success rate exceeding 90% in experienced centers.

Atypical Atrial Flutter and Macro-reentrant Atrial Tachycardia

In addition to the typical atrial flutter circulating around normal anatomic obstacles, atrial disease with associated fibrosis or, more commonly, atrial scars created at the time of cardiac surgery for valvular or congenital heart disease may create alternative substrates for intra-atrial reentry. Common to these arrhythmias is a significant region of scar with a channel of surviving myocardium bridging the scar or between the scar and a normal anatomic obstacle. Within the channel, conduction is slow and electrocardiographically silent, resulting in an isoelectric PP interval. Because the circuit is different from that of typical atrial flutter, the P-wave morphology is atypical.

When the rate is 250 beats per minute or greater, the arrhythmia is arbitrarily classified as atypical atrial flutter, and when the rate is less than 250 beats per minute, it is arbitrarily classified as atrial tachycardia. Like typical atrial flutter, these arrhythmias are paroxysmal sustained or persistent arrhythmias, and when manifesting with 2 : 1 conduction, they may be misdiagnosed as sinus tachycardia if the abnormal P-wave vector and fixed heart rate over time are not recognized. Therapy and prognosis are otherwise similar to those for typical atrial flutter.

Atrial Fibrillation

Overview and Classification

AF is a chaotic atrial rhythm related to continuous and variable activation of the atria. There are no distinct P waves or periods of atrial quiescence. It is characterized electrocardiographically by a wavering baseline associated with an irregular ventricular response (see Fig. 9-6D).

AF is the most common clinically significant arrhythmia. It affects 2.2 million people in the United States. Its prevalence is between 0.4% and 1% in the general population, and it increases with age, reaching 8% in those older than 80 years. Patients with AF have a higher risk of stroke, heart failure, and mortality. However, the role of AF as an independent determinant of mortality is uncertain because it commonly coexists with other important conditions. Patients with lone AF do not have an increased mortality rate, and carefully designed trials exploring the benefit of maintenance of sinus rhythm over rate control show no survival benefit for sinus rhythm. Whether AF is merely a marker for increased mortality or a mechanism remains uncertain.

AF is often classified by its clinical presentation and pattern. When AF is first detected, it is called *new onset*, and its ultimate pattern is initially undetermined. When AF relapses during follow-up, it is called *recurrent* and classified by its clinical pattern. If AF terminates spontaneously, it is called *paroxysmal* AF. Although episodes lasting up to 7 days are defined as paroxysmal, most episodes of paroxysmal AF terminate within the first 24 hours and many terminate within minutes or hours of onset. When AF lasts longer than 7 days, it is designated as *persistent*. AF that persists for a long interval, typically more than a year, without return of an interim period of sinus rhythm (spontaneously or as a result of medical intervention such as cardioversion) is often called *permanent* AF.

Mechanisms of Atrial Fibrillation

Because of its chaotic nature, it has been difficult to study AF, and its mechanisms remain incompletely understood. The initiation of spontaneous AF is a consequence of rapid electrical firing from preferential focal sites of origin. The most common site of focal origin is from left atrial muscle sleeves extending along the outer surface of the pulmonary veins. When firing does not originate from a pulmonary vein, it is commonly from the left atrial tissue immediately adjacent to one of the veins or occasionally from one of the other thoracic veins such as the ostium of the superior vena cava or the ostium of the coronary sinus. Atrial rates recorded in and around the pulmonary veins are significantly higher than at other atrial sites, suggesting that activity in the region of the veins is important in perpetuating AF after initiation.

These insights have produced highly effective techniques for the cure of AF. Ablation techniques designed to isolate these trigger sites from the atrium have success rates of 70% to 80% for the cure of paroxysmal AF and somewhat lower rates for the cure of persistent AF. Ablation restricted to the region of the pulmonary veins and adjacent left atrium is curative in most patients with AF, implying that most cases of AF are arrhythmias entirely contained within and maintained by the left atrium and connecting veins. In the same way that typical atrial flutter is the characteristic arrhythmia of the right atrium, AF is the characteristic arrhythmia of the left atrium.

Anticoagulation and Atrial Fibrillation

During AF (and to some extent, atrial flutter), the atria have incomplete and ineffective contractions. Blood stasis occurs and may result in the formation of intracardiac thrombus, which may lead to thromboembolism and stroke. The overall risk of stroke in patients with AF is 5% per year. Certain risk factors may adjust this risk, including age, gender, rheumatic heart disease, prior stroke, left ventricular dysfunction, left atrial enlargement, hypertension, and diabetes.

Scoring systems have been developed to estimate a patient's AF-related stroke risk based on his or her constellation of risk factors. The most used system is the $CHADS_2$ score (*c*ardiac failure, *h*ypertension, *a*ge ≥75 years, *d*iabetes mellitus, and prior *s*troke). This system has been well validated in assessing the stroke risk of patients with AF. It assigns a single point for age of 75 years or older, diabetes, history of heart failure, and hypertension. It assigns two points for a history of stroke or transient ischemic attack. A score of 0 correlates with a relatively low risk of stroke at 1.9% per year, a score of 1 has a stroke risk of 2.8% per year, a score of 2 has a risk of 4.0% per year, and a score of 3 or higher has a stroke risk of more than 5.9% per year.

The $CHADS_2$ underwent further refinement to increase the granularity of stroke risk stratification with the creation of the CHA_2DS_2-VASc (*v*ascular disease, *a*ge, and *s*ex) scoring system. In this system, congestive heart failure, hypertension, diabetes