The supraventricular (common narrow complex) tachycardias discussed so far have organized atrial activity (manifest by discrete P waves) and, with some notable exceptions, 1:1 atrioventricular (AV) conduction.* Atrial fibrillation (AF) and atrial flutter (AFL) are two related (and sometimes missed or mistaken) arrhythmias with very rapid atrial rates that greatly exceed the ventricular rate (Fig. 15-1). This finding implies that some degree of physiologic (functional) AV block† is almost always present. Furthermore, both arrhythmias involve reentrant mechanisms with impulses rapidly and continuously spinning around, “chasing their tails,” in the atrial muscle itself (see Fig. 15-1). Therefore, instead of true P waves, one sees continuous F (flutter) or f (fibrillatory) waves.

**ATRIAL FLUTTER**

**Location of Conduction Pathways**

The reentrant circuit of “typical” atrial flutter revolves around the tricuspid valve in the right atrium. Like reentrant supraventricular arrhythmias in general, it is initiated by a premature atrial complex (PAC) that blocks in one direction, while propagating in another. The signal then keeps circling in the same trajectory over and over again. In atrial flutter the typical frequency is about 300 cycles per minute (which is determined by the atrial size and conduction velocity) producing identical F waves.

The “bottleneck” where the signal usually blocks is the relatively narrow area between the tricuspid valve and inferior vena cava at the bottom of the right atrium (cavo-tricuspid isthmus). Conduction of the initiating PAC is blocked from propagating from the middle area of the right atrium to its lateral wall through this narrow isthmus. However, the electrical signal can propagate through the rest of the atrium and reach the isthmus from the other side. By this time the isthmus tissue has already recovered electrically and is ready to conduct. The signal goes through the isthmus in a counterclockwise direction, and the large (macro-) reentrant cycle starts again.

The classic “sawtooth” pattern of F waves that are predominantly negative in leads II, III, and aVF and positive in V₁ with a very regular ventricular (QRS) rate of about 150/min (functional 2:1 AV block) is suggestive of the counterclockwise (common) type of typical right atrial flutter (Fig. 15-2A). Less frequently the same circuit gets initiated in the opposite direction, producing “clockwise” flutter. The polarity of the F waves will then be reversed: positive in leads II, III, and aVF, and negative in lead V₁ (Fig. 15-2B). Clockwise and counterclockwise flutter can occur in the same patient and both are usually isthmus-dependent.‡ Clinically, the development of atrial flutter most often indicates the presence of underlying structural/electrical atrial disease.

†The cavo-tricuspid isthmus is the most common area around which atrial flutter develops. However, it can develop around other atrial obstacles such as scars after cardiac surgery, or areas of fibrosis following pericarditis or even following ablation procedures in the left atrium for atrial fibrillation.
Typical Atrial Flutter Variants

**Figure 15-2.** A, Typical atrial flutter most commonly involves a reentrant (“merry-go-round”-like) circuit in the right atrium, proceeding in a highly consistent, counterclockwise pathway. The cycle length (rotation time) is about 200 msec, corresponding to an atrial rate of 300/min. Note that the “sawtooth” flutter (F) waves (arrows) are negative in the inferior leads (II, III, and aVF) and V6, but positive in V1. In the absence of drugs or atrioventricular node disease, the ventricular response is often exactly half the atrial rate (i.e., 150 beats/min). B, With the “clockwise” variant, the flutter (F) waves are positive in the inferior leads and V6, and negative in V1. These variants have the same clinical implications.

**Figure 15-1.** Diagram comparing mechanisms of atrial flutter and atrial fibrillation (AF). Atrial flutter is typically due to a large reentrant wave, originating in the right atrium by a premature atrial complex. With the common type of typical atrial flutter, the wave spreads in counterclockwise direction, involving the area near the tricuspid valve and inferior vena cava (cavo-tricuspid isthmus). In contrast, AF is sustained by multiple reentrant wavelets, not a single one, and often initiated by increased impulse formation in the area of the pulmonary veins in the left atrium. LV, left ventricle; RV, right ventricle, SA, sinoatrial.
Conduction to the Ventricles

The atrial rate during typical atrial flutter, as noted, is around 300 cycles/min (range usually between about 240 to 330 cycles/min). Slower rates can be due to drugs that slow atrial conduction. Fortunately, the AV node cannot conduct electrical signals at that rate to the ventricles—although a bypass tract in the Wolff-Parkinson-White (WPW) syndrome (see Chapter 12) can! Thus, with atrial flutter, physiologic AV block develops (usually with a 2:1 A/V ratio) (Figs. 15-2 and 15-3). In the presence of high vagal tone, AV nodal disease, or AV nodal blocking drugs (e.g., beta blockers, digoxin, and certain calcium channel antagonists) higher degrees of AV block can be seen, for example with a 4:1 conduction ratio (Figs. 15-3 and 15-4).

Often the AV nodal conduction shows more complex patterns and the degree of AV block varies in a periodic way, producing flutter/QRS ratios with repeating patterns (Fig. 15-4) of RR intervals (group beating). This phenomenon is believed to be due to multiple levels of block within the conduction system. Variable AV block may be due to other mechanisms (e.g., AV Wenckebach) and produce noninteger ratios of F waves to QRS complexes (Fig. 15-5).

On the other hand, atrial flutter with 1:1 AV conduction (Fig. 15-6), although uncommon, can occur in three major settings:

- In high catecholamine states (strenuous physical activity, infection, with high fever, shock, etc.)
- With certain antiarrhythmic medications (such as flecainide) that slow down the flutter rate (for example, from 300 to 250/min or less) to the point where 1:1 conduction through AV node becomes possible
- In the presence of a bypass tract (WPW pattern) capable of rapid conduction (short refractory period)

Atrial flutter with sustained 1:1 AV conduction represents an emergency situation, requiring consideration of immediate synchronized electrical cardioversion because of the dangerously rapid ventricular rate.

**Figure 15-3.** A, Notice the variable appearance of flutter waves in different leads. In lead I, the waves are barely apparent, whereas in leads II and III, the classic “sawtooth” waves appear. The ventricular rate is about 160 beats/min, and the flutter rate is about 320 beats/min; thus 2:1 AV conduction is present. B, Carotid sinus massage produces marked slowing of the ventricular rate by increasing vagal tone.
Atrial Flutter with Variable AV Block

Figure 15-4. Atrial flutter from different patients (A through E) showing variable patterns of conduction (block). As shown, the block may alternate between two values. In other cases it is more variable.

Atrial Flutter with Variable AV Block

Figure 15-5. With atrial flutter, the ventricular response may be variable, but not always a simple fraction (\(\frac{1}{2}\), \(\frac{1}{3}\), \(\frac{1}{4}\)) of the atrial rate. Even in these cases, the response usually shows some underlying patterns, in contrast to the random-appearing ventricular rate in atrial fibrillation.

Atrial Flutter with 2:1 and 1:1 AV Conduction

Figure 15-6. Atrial flutter with 2:1 AV conduction (A) compared with 1:1 (one-to-one) AV conduction (B) in the same patient. In the latter case, the flutter waves are hard to locate. Owing to the very rapid ventricular response (about 300 beats/min), atrial flutter is a medical emergency, often necessitating direct current (DC) cardioversion (see later discussion).
Unlike atrial flutter, the reentrant waves of atrial fibrillation (AF) cannot be localized to any repetitive and stable circuit in the atria. Most cases of AF are thought to originate in the area of pulmonary vein–left atrial junctions. With time, more and more of the atrial tissue becomes involved in the active maintenance of the arrhythmia, associated with the simultaneous formation of multiple unstable reentrant circuits throughout the atria (see Fig 15-1).

Atrial electrical activity on the ECG appears as irregular \( f \) (fibrillatory) waves, varying continuously in amplitude, polarity (reversing from positive or negative orientation in same lead), and frequency (changing cycle length, measured as the very brief interval from one \( f \) wave to the next).

Milder degrees of atrial activity “disorganization” or drugs that slow atrial activation may produce coarse AF with high amplitude \( f \) waves resembling atrial flutter (Fig. 15-7).

**Key Point**

Usually, the single best lead to identify the diagnostic irregular atrial activity of AF is lead \( V_1 \), where irregular \( f \) waves are likely to be most clearly seen (Fig. 15-8).

Severe atrial abnormalities (due to atrial dilation, fibrosis, or long-standing fibrillation, or drugs like digoxin) often result in fine AF with almost isoelectric (flat), very fast fibrillatory waves that can be confused with atrial asystole. Sometimes both fine and coarse \( f \) waves can appear in the same ECG.

**Conduction Properties**

In AF, the AV node gets bombarded with highly disorganized impulses of different amplitude at rates of up to 400 to 600/min. Most of the signals are blocked in the node and only a fraction conduct to the ventricles (see Figs. 15-7, B, and 15-8). Still, in the absence of AV nodal disease or certain drugs, the ventricular heart rate in AF is much higher than with sinus rhythm (e.g., usually the mean QRS rate in AF is over 100 beats/min at rest, with inappropriate increases during exercise).

Due to random penetration of the impulses through the AV node, the RR intervals in AF are haphazardly irregular. However, when the ventricular rate gets very fast, this RR irregularity may become more difficult to appreciate; sometimes the rhythm appears regular and may be confused with other tachyarrhythmias such as PSVT (Figs. 15-9 and 15-10).

AF or flutter can occur with complete heart block, in which case the ECG will show a regular very slow ventricular response (Fig. 15-11).

Clinicians should also be aware that AF can be also masked by a ventricular pacemaker (see Chapter 21).

**ATRIAL FIBRILLATION VS. ATRIAL FLUTTER: DIFFERENTIAL DIAGNOSIS**

Although atrial flutter almost always occurs in the setting of structural heart disease, AF can develop in normal hearts. However, patients presenting with
Atrial Fibrillation with a Slow Ventricular Response

Figure 15-8. Atrial fibrillation (not flutter) is present with a very slow ventricular response. The fibrillation waves are best seen in lead V1. There is an atypical left bundle branch block (LBBB) pattern (see Chapter 7). The rsR’ in lateral leads (e.g., V6 here) is highly suggestive of prior myocardial infarction (MI). A QR (or rsR’) complex is also present in leads I and aVL, also consistent with underlying MI. Left axis deviation and a long QT interval are noted as well. The patient had chronic heart failure due to severe coronary artery disease with prior “silent” MIs. The slow ventricular response raises the question of drug effect or excess (e.g., digoxin) or intrinsic atrioventricular (AV) node disease (see Chapter 17).

Atrial Fibrillation with Rapid Ventricular Response

Figure 15-9. This patient has hyperthyroidism. (Note: the commonly used term rapid atrial fibrillation is actually a misnomer, because “rapid” is intended to refer to the ventricular rate rather than the atrial rate. The same is true for the term slow atrial fibrillation.) The atrial fibrillation waves here have a “coarse” appearance.

Atrial Fibrillation with Rapid Ventricular Response (Not PSVT)

Figure 15-10. Atrial fibrillation with a rapid ventricular response. At rapid rates, the RR interval variability may be more subtle, leading to a mistaken diagnosis of paroxysmal supraventricular tachycardia (PSVT). See also Figure 15-9.
PART II  Cardiac Rhythm Disturbances

Atrial Fibrillation with Complete AV Heart Block

Figure 15-11. Complete atrioventricular (AV) heart block (see Chapter 17) can occur with underlying atrial fibrillation (or flutter); the ventricular response will be very slow, usually 50 beats/min or less, and regular. In this case, the narrow QRS complex indicates that the escape rhythm is in the nodal area. Such patients usually require both permanent pacing and anticoagulation.

| TABLE 15-1  Differential Diagnosis of Atrial Fibrillation versus Atrial Flutter |
| --- | --- | --- |
| Feature | Atrial Flutter | Atrial Fibrillation |
| Atrial wave morphology | Identical from one F wave to another | f waves varying in shape and polarity |
| Atrial wave timing | Identical, i.e., “maps out” throughout the tracing | Variable, i.e., does not map out |
| Atrial wave cycle length | F-F intervals ≥180 msec (4.5 small boxes) | Variable f-f intervals can be shorter than 180 msec (4.5 small boxes) |
| Ventricular (QRS) response | Constant (2:1, 4:1) F/R ratio or shows group beating due to patterned response (2:1-2:1-4:1) or Wenckebach conduction | Completely irregular (no pattern) unless complete heart block or ventricular pacing is present |

atrial flutter have an increased chance of showing AF on follow-up. AF and flutter can occur in the same patient, transitioning from one to the other. In such case it is often said that AF “organizes” into atrial flutter or atrial flutter “degenerates” into AF. However, at any given time there is usually one or the other (but not both) rhythms present. Although electrocardiographically these rhythms can appear quite similar, it is important to differentiate between them because of the differences in management.

At any given time there is usually one or the other (but not both) rhythms present. Although electrocardiographically these rhythms can appear quite similar, it is important to differentiate between them because of the differences in management.

The differential diagnosis of AF and flutter is based on the differences in their mechanisms (Table 15-1). The distinction is important because of the different therapeutic implications of the two arrhythmias, particularly the consideration of radiofrequency (RF) ablation as first-line therapy in atrial flutter.

- Atrial flutter has a single, stable reentrant pathway. Therefore, all flutter (F) waves look exactly the same in both shape and duration. A simple, accurate way—the “caliper (calipers) test”—to check for this finding is to measure the interval containing several consecutive clearly visible atrial waves with ECG calipers and move it along the tracing. In case of atrial flutter, the subsequent F wave intervals will “map out” perfectly. In AF, the shape and polarity of f waves often vary over the length of the tracing. Even if the shape of f waves appears similar, their timing will be “off” (Fig. 15-12).

- The propagation velocity of the signal through atrial tissue is limited and it takes a certain amount of time, termed the “cycle length” (usually at least 180 msec, equivalent to 4.5 small “boxes”), for the flutter signal to make a full circle through the atrium. Therefore, atrial waves due to flutter generally cannot appear closer than 4 boxes apart. If they do—it suggests AF. But “coarse” AF can be present with cycle lengths of 180 msec or greater.

- In atrial flutter there is usually either a fixed ratio of F/QRS waves (2:1, 4:1) or a group beating due to a “patterned” ventricular response (e.g., 2:1-4:1). In AF, the QRS interbeat intervals are completely erratic. But be careful: if the ventricular rate is fast, this variability may be subtle.

ATRIAL FIBRILLATION AND FLUTTER: OVERVIEW OF MAJOR CLINICAL CONSIDERATIONS

AF is the most common arrhythmia causing hospital admissions. Over 2 million Americans have intermittent or chronic AF, and the incidence rises with age. Nearly 10% of individuals 80 years or older develop AF.

In some patients, AF occurs paroxysmally and may last only minutes or less, hours, or days. Some patients may experience only one episode or occasional episodes, whereas others have multiple
Atrial Fibrillation and Flutter: Overview of Major Clinical Considerations

recurrences. In some patients, AF is more persistent and may become permanent (chronic), lasting indefinitely (Table 15-2).

During the episodes, some patients are quite symptomatic (typically complaining of palpitations, fatigue, dyspnea, lightheadedness, or chest pain), whereas others have no specific complaints. Syncope can occur, usually as the result of the spontaneous postconversion pauses upon arrhythmia termination (see “tachy-brady” syndrome, Chapter 13).

In the asymptomatic patient, AF may first be discovered during a routine examination or when the patient presents with heart failure or stroke. AF can occur in people with no detectable heart disease and in patients with a wide variety of cardiac diseases.

The term lone atrial fibrillation is sometimes used to describe recurrent or chronic AF in patients without clinical evidence of heart disease. Paroxysmal AF may occur spontaneously, or it may be associated with excessive alcohol consumption in otherwise healthy individuals (holiday heart syndrome). In such cases, the arrhythmia often spontaneously reverts to normal sinus rhythm or is converted easily with pharmacologic therapy alone.

Changes in autonomic tone may provoke AF in susceptible individuals. Sometimes the arrhythmia is related to increased sympathetic tone (e.g., occurring

### TABLE 15-2

<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
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<tbody>
<tr>
<td>Paroxysmal</td>
<td>Recurrent AF (≥2 episodes) that terminates spontaneously in less than 7 days (usually less than 48 hours)</td>
</tr>
<tr>
<td>Persistent</td>
<td>AF that is sustained beyond 7 days, or lasting less than 7 days but necessitating pharmacologic or electrical cardioversion</td>
</tr>
<tr>
<td>Long-standing persistent</td>
<td>Continuous AF present for longer than 1 year</td>
</tr>
<tr>
<td>Permanent</td>
<td>AF lasting for more than 1 year in a patient in whom the decision has been made not to pursue restoration of sinus rhythm by any means</td>
</tr>
</tbody>
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Figure 15-12. Coarse atrial fibrillation and atrial flutter may look very similar. A useful test (the “calipers test”) is described in the text. It is based on the fact that in atrial fibrillation (top panel), the atrial (f) waves (remember these are not P waves) vary in timing and morphology, while in atrial flutter, the atrial (F) waves are identical. Thus, if you fix your calipers (or line up the distance with a 3 × 5 card) between two atrial waves and then “march” that interval forward or backward, the calipers will always hit the same point on F waves (bottom panel) but different ones on f waves (top panel).
during exercise or with emotional excitement). At other times, AF may occur in the context of high vagal tone (e.g., with sinus bradycardia during sleep).

AF is also one of the most frequently observed arrhythmias in patients with organic (structural) heart disease. The prevalence of this arrhythmia rises with advancing age. Common pathologic substrates include coronary artery disease, hypertensive heart disease, and valvular heart disease. Patients with coronary artery disease may experience AF for the first time during an acute myocardial infarction (MI) or, more commonly, as a consequence of chronic ischemic myocardial disease, possibly because of associated atrial dilation or fibrosis. Hypertensive heart disease is often associated with left atrial enlargement. AF is also commonly caused by valvular heart disease, particularly when the mitral valve is involved. For example, severe rheumatic mitral stenosis or mitral regurgitation (of any cause) produces marked left atrial enlargement, a major predisposing factor for atrial tachyarrhythmias.

Numerous other conditions can also lead to AF. For example, patients with thyrotoxicosis (hyperthyroidism) may develop AF. The arrhythmia (or atrial flutter) is quite common after cardiac surgery. It may also occur with pericardial disease (especially recurrent or chronic), chronic lung disease, pulmonary emboli, cardiomyopathy, congenital heart disease (e.g., atrial septal defect), and other forms of heart disease. Obstructive sleep apnea (OSA) increases the risk of AF—it should always be suspected when diagnosis of AF is first made. Often, patients have more than one predisposing factor (e.g., hypertension, sleep apnea, and advanced age).

Chapter 24 includes a summary of important causes and contributors to AF (and flutter).

AF and flutter have two major clinical implications:

1. First and foremost is the increase in thromboembolic risk (most importantly, stroke). Therefore, whenever AF or flutter is present on the ECG, the anticoagulation status of the patient should be reviewed and appropriate treatment promptly initiated. Anticoagulation should not be delayed pending rate control. Remember that recurrent paroxysmal arrhythmia does not decrease thromboembolic risk compared to its persistent or chronic form.

Stroke risk in AF/flutter depends on certain comorbid conditions. The most common factors that increase thromboembolic risk include a history of hypertension, older age (≥75 years; or ≥65 years according to some authorities), history of stroke symptoms or of TIA, history of diabetes mellitus, and history of chronic heart failure, in addition, of course, to evidence of rheumatic heart disease or a mechanical prosthetic heart valve.

Depending on the estimated risk, oral anticoagulation regimens may include just aspirin (e.g., younger patients without other risk factors), warfarin, or one of the newer agents such as dabigatran, a direct thrombin inhibitor. Readers are referred to the Bibliography for additional clinical details and evolving guidelines.

The increase in stroke risk in AF/flutter is related to left atrial appendage thrombi formation caused by the loss of atrial contraction and stagnation of blood flow. It usually takes at least 48 hours of arrhythmia for the thrombi to start developing.

2. The second important clinical implication is the development of chronic heart failure. It can occur immediately due to decreased cardiac output from lack of atrial contraction, and the rapid rate that may be associated with ventricular ischemia. These pathophysiologic events can produce severe shortness of breath and even acute pulmonary edema. Furthermore, long-term (weeks to months) continuation of a rapid uncontrolled ventricular rate can lead to development of a tachycardia-induced cardiomyopathy with ventricular dilatation and decrease in the systolic function.

**TREATMENT OF ATRIAL FIBRILLATION/FLUTTER: ACUTE AND LONG-TERM CONSIDERATIONS**

The first two priorities in the acute treatment of AF and flutter are appropriate anticoagulation and rate control. Potentially reversible causes and risk factors should be reviewed (in particular, OSA and thyroid disease).

There are, in turn, two general treatment strategies for long-term management of AF and flutter: rate control and rhythm control.

**Rate Control**

Rate control centers on limiting the ventricular response, without attempts at restoring sinus rhythm. Rate control can be achieved by using AV nodal blocking agents (beta blockers, calcium channel blockers, digoxin) or AV junctional (AVJ)
Ablation. The criteria of “optimal” rate control are currently under investigation.

Rate control is usually the preferred treatment option in patients with the following:
- **Permanent AF**
- **New-onset AF within the first 24 hours** (which has approximately 50% chance of terminating spontaneously)
- **Reversible acute illness** when achievement and maintenance of sinus rhythm are unlikely until the cause is corrected (e.g., hyperthyroidism, metabolic abnormalities, especially hypokalemia, alcohol withdrawal, acute infection)
- **Asymptomatic patients** who can tolerate a lifetime of anticoagulation

AVJ ablation (with pacemaker implantation) can be used in patients whose rate cannot be effectively controlled with medications. It is a percutaneous procedure electrically “disconnecting” the atria from the ventricles and achieving excellent rate control without any further need for AV nodal blocking agents. The downside of AVJ ablation is that the patient becomes largely pacemaker-dependent. As with any of the other rate-controlling options, anticoagulation has to be continued indefinitely.

**Rhythm Control**

Rhythm control strategy consists of two phases: (1) sinus rhythm restoration (cardioversion) and (2) sinus rhythm maintenance.

Cardioversion can be achieved by using antiarrhythmic medications (chemical cardioversion), electrical cardioversion with direct current shock, or an ablation procedure. With any type of cardioversion, thromboembolic risk and anticoagulation history of the patient should be reviewed because of increase in risk of thromboembolism at the moment of transition to sinus rhythm.

Pharmacologic cardioversion in AF is of limited value. The rate of conversion to sinus rhythm with most antiarrhythmic medications is low. Intravenous _ibutilide_ can convert up to 50% of cases of recent onset AF and up to 70% of cases of atrial flutter. However, the drug can cause significant QT prolongation and sometimes torsades de pointes. Therefore, very careful, continuous ECG monitoring is required during its administration.

Direct current (electrical) cardioversion (DCCV) is a safe and reliable method of restoring sinus rhythm in AF and flutter. A properly timed direct current shock administered through pads on anterior and posterior chest (Fig. 15-13) depolarizes the whole heart, disrupting reentrant circuits and allowing the sinus node to regain control of the atria. It is important to “synchronize” the shock with ventricular depolarization (R wave on the ECG). Unsyncronized shocks if delivered in the ventricular vulnerable period can induce ventricular fibrillation.

Sinus rhythm maintenance can sometimes be achieved with antiarrhythmic drugs (see Chapter 10); drugs most often used are class I agents flecainide and propafenone, or class III agents sotalol, amiodarone, dofetilide, and dronedarone. Unfortunately, antiarrhythmic drugs are only modestly effective in maintaining sinus rhythm. Furthermore, most require monitoring for ECG changes that may forecast electrical instability: for example, QRS interval widening (flecainide, propafenone) and QT(U) interval prolongation with the risk of torsades de pointes (Chapter 16; sotalol, dofetilide, amiodarone, dronedarone, dofetilide). Another major limitation is that none maintains sinus rhythm reliably enough to discontinue anticoagulation. Dronedarone is contraindicated with advanced heart failure.

Ablation procedures using RF catheter–based technologies have become increasingly used both in sinus rhythm restoration and maintenance in AF and, especially, atrial flutter. **RF ablation is highly efficacious in treating typical atrial flutter, with close to a 90% long-term success rate.** A linear lesion in the cavo-tricuspid isthmus using a percutaneously inserted ablation catheter is usually curative because it interrupts the underlying flutter pathway.

In contrast, ablation procedures for AF involve catheter access to the left atrium via a transseptal approach from the right atrium. The mainstay of current AF ablation is electrical disconnection of the pulmonary veins from the atrial tissue by RF energy. In paroxysmal AF, encircling all (normally four) pulmonary veins with ablation lesions is usually sufficient for arrhythmia control. In persistent AF, additional lines are created inside the left atrium. The long-term success rate of left atrial ablation for AF is highly variable (average about 70%), which is higher than that of antiarrhythmic medications. However, repeat procedures are often necessary.

Choosing between rate and rhythm control and medication options, along with making decisions about the indications for and timing of AF ablation, needs to be personalized. This field represents one of the most active areas of contemporary cardiology research.
Figure 15-13. Direct current cardioversion (DCCV) of atrial fibrillation to sinus rhythm. With external DCCV, an electric shock is administered to the heart via special electrode paddles placed on the chest wall. In the case depicted here, one electrode is placed on the anterior chest wall, to the left of the sternum; the other (indicated by dashed lines) is placed on the back, under the left scapula. The shock must be synchronized with the peak of the QRS complex to avoid inducing ventricular fibrillation, which may occur if the stimulus is delivered at the peak of the T wave.