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Ablation of Nonisthmus-Dependent Flutters and Atrial Macroreentry

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KEY POINTS

- Atypical or nonisthmus-dependent atrial flutter and atrial macroreentry requires fixed or functional barriers and regions of slow conduction.
- Atypical flutters are often recognized after catheter ablation or surgical treatment of atrial fibrillation (AF) or after surgery that involves right or left atriotomies.
- Activation mapping is used to demonstrate reentrant circuits, middiastolic potentials, fractionated potentials, and double potentials.
- A multipolar electrode catheter is useful to map atypical flutter in either atrium.
- An electroanatomic mapping system is useful in most cases.
- An irrigated-tip or large-tip (8 mm) ablation catheter is usually employed.
- Sources of difficulty include defining complex reentrant circuits, particularly those with irregular oscillations in atrial cycle length; achieving durable lines of conduction block; and spontaneous conversion of atypical flutter to atrial fibrillation or other arrhythmias.

INTRODUCTION

Atypical atrial flutters refer to a spectrum of arrhythmias that vary in location and circuit dimensions. In many cases these arrhythmias may also coexist with atrial fibrillation (AF) or play transitional roles in the initiation or termination of AF, or in the transformation to typical atrial flutter. Atypical flutters are increasingly recognized after catheter ablation or surgical treatment of AF or after surgery that involves right or left atriotomies.

Historically there has been confusion over the use of the terms *atrial flutter* and *atrial tachycardia* (AT). It has been proposed that these terms be applied based on rate or the presence of isoelectric intervals on the electrocardiogram. The term *atrial flutter* is often used to refer to regular tachycardias with rates of 240 beats per minute or higher and lacking an isoelectric interval between deflections, whereas *atrial tachycardia* refers to arrhythmias with isoelectric intervals between P waves and/or slower cycle lengths. However, these characteristics are not specific for the mechanism of tachycardia.[1,2] In this chapter, the term *atypical atrial flutter* will be used preferentially to refer to reentrant circuits that are not dependent on the cavotricuspid isthmus (CTI). Atypical flutters may be classified based on their chamber of origin, site within a given atrium, and size of the reentrant circuit (Table 12.1).

ANATOMY

The specific configuration of a reentrant circuit is dependent on the local or global anatomy of the atrium, as well as conduction and refractory properties of the atrial myocardium. As a general rule, reentry requires the presence of two limbs, which are anatomically and/or functionally contiguous but dissociated. These limbs are dependent on the presence of an inexcitable central barrier (i.e., atrioventricular annulus, venous ostium, or scar) or a functional line of block. In the right atrium (RA), natural barriers to conduction are the tricuspid annulus, inferior vena cava (IVC), superior vena cava (SVC), crista terminalis, Eustachian ridge, and fossa ovalis. In the left atrium (LA), the mitral annulus and pulmonary venous ostia serve as critical conduction barriers, along with electrically silent areas that may be found in myopathic atria or result from prior ablation or surgery.

PATHOPHYSIOLOGY

Although atypical atrial flutters can arise in structurally normal hearts, these arrhythmias predominantly occur in patients with organic heart disease, after cardiac surgery, or AF ablation. In patients with organic heart disease, the pathogenesis is thought to involve elevated atrial pressure, which causes interstitial fibrosis resulting in conduction slowing and block. LA reentry that arises de novo in the atria frequently involves regions of patchy scar, which presumably occurs as the result of atrial myopathy.[3] These areas of patchy scar can be identified as electrically silent areas during electroanatomic mapping. Other important determinants are slowing of atrial conduction velocity, heterogeneity of atrial refractoriness, and initiating or triggering foci.

In response to premature stimuli, arcs of functional block develop and—if of sufficient length—initiate and support reentry. Functional lines of block develop in structures such as the crista terminalis and the Eustachian ridge, both of which play a critical role in the formation of CTI-dependent atrial flutter.[4] Gaps in these functional lines of block, as in the crista terminalis, permit formation of atypical circuits. Functional lines of block may develop in many other locations, including variable sites in the LA. A combination of fixed and functional block has been demonstrated in animal models of lesional tachycardia, in which a line of functional block develops as an extension to a fixed anatomic lesion.[5] In this case, the anatomic lesion might not be large enough to support reentry, but the combined fixed and functional barrier provides the critical path length needed to maintain reentry. Formation and breakdown of these arcs of block are responsible for interconversion of flutter circuits with each other and the transition to AF.

With the growth of ablation procedures for AF, atypical flutters have become increasingly frequent after both catheter and surgical ablation. Discontinuities in ablation lines can result in

TABLE 12.1 Types of Nonisthmus-Dependent Atrial Flutters

Circuit	Boundaries	Clinical Scenarios
RIGHT ATRIUM		
Upper-loop reentry	SVC and crista terminalis	De novo or with CTI-dependent flutter
Right atrial free-wall reentry	Right atrial scar, lateral tricuspid annulus	Prior right atriotomy, atrial myopathy
Dual-loop reentry (combined lower loop, upper loop, free wall, and/or tricuspid valve)	Combinations of right atrial scar, SVC, tricuspid annulus, with or without CTI	De novo or with CTI-dependent flutter, prior right atriotomy, atrial myopathy
LEFT ATRIUM		
Perimitral annular reentry	Mitral annulus, left atrial isthmus	Following left atrial ablation for AF, after maze surgery, de novo
Roof-dependent reentry	Linear ablation lines around left and/or right pulmonary veins, possibly electrically active pulmonary vein tissue	Following left atrial ablation for atrial fibrillation with encircling lesions, after maze surgery, de novo
Periseptal tachycardia	Right pulmonary veins and mitral annulus or fossa ovalis with right pulmonary veins or mitral annulus	Following atriotomy, left atrial ablation for atrial fibrillation, de novo
MISCELLANEOUS		
Lesional tachycardias	Surgical scars	Following left atriotomy, post maze surgery
Microreentry or localized reentry	Variable	Following left atrial ablation for AF, after maze surgery, atrial myopathy
Coronary sinus mediated reentry	Coronary sinus with left or right atrial myocardium	De novo

AF, Atrial fibrillation; *CTI,* cavotricuspid isthmus; *SVC,* superior vena cava.

unidirectional rate-dependent block or conduction slowing.[6] Furthermore, areas of excluded myocardium, such as those occurring with wide encircling pulmonary vein isolation, give rise to large central barriers that permit circus movement reentry. Many reentrant ATs are complex circuits that involve dual loops (figure-of-eight reentry) or less commonly triple loops. In multiple loop tachycardias, conduction times are nearly equal around the individual loops, but sometimes a dominant circuit exists as a *driver,* which entrains other loops.

Small reentrant circuits, known as *localized reentry* or *microreentry*, can occur in a variety of clinical circumstances, such as catheter ablation of AF, surgical maze procedures, and surgical repair of congenital heart disease. These small circuits can also occur de novo in diseased atria.[7] A unifying condition in cases of localized reentry is the presence of an atrial scar, and these circuits usually localize near prior ablation lines or surgical incisions. Disruption of the myocardial architecture may give rise to markedly slow conduction, anisotropic differences in conduction velocity, and unidirectional block, which allow small circuits to develop and perpetuate.

Cardiac surgery that involves atrial incisions is an important cause of atypical flutter. The location and extent of an incision, in addition to intrinsic disease of the atria, influence the likelihood of developing atypical flutter. Patients who have right atrial surgery are also at risk of developing atypical flutter involving the surgical scar in the lateral RA, whereas those who have a left atriotomy may develop atypical flutter in the LA.[8] In addition, patients who have right or left atriotomies are also prone to develop CTI-dependent flutter.

DIAGNOSIS

Macroreentrant Versus Focal Atrial Tachycardia

A fundamental consideration in evaluating atypical flutter is to establish if the arrhythmia is macroreentrant or focal in origin (Box 12.1). In general, P wave durations are shorter and surface isoelectric intervals are longer with focal tachycardias than with macroreentry.[9] A study of 75 patients with atypical flutters after AF ablation found that a P wave duration of less than 185 ms had a sensitivity of 85% and specificity of 97% in identifying a small reentrant circuit.[10] However, it is possible for a macroreentrant tachycardia to have an isoelectric interval if part of circuit consists of a slowly conducting isthmus generating little electric force.[2] When the tachycardia cycle length varies by 15% or more, a focal mechanism is suggested, but a regular cycle length can occur with both focal and macroreentrant atrial tachycardias (ATs).[11]

The electrocardiographic (ECG) morphology of atypical flutters is highly variable, and substantial overlaps exists between flutter morphologies arising from different anatomic circuits, so that localization of atrial flutters from the surface ECG has limited utility. However, some broad generalizations can be made that distinguish CTI-dependent from non-CTI-dependent flutters, as well as right from left atrial tachycardias.[12] CTI-dependent flutters often show characteristic morphologies, and recognizing deviation from these patterns allows one to conclude that a tachycardia might be atypical flutter. Typical counterclockwise CTI-dependent flutter often has a superiorly directed flutter wave axis with a small terminal positive deflection in the inferior leads (so called *sawtooth* pattern). Lead V_1 has an overall positive deflection with an initial isoelectric or inverted component, and the flutter wave transitions from positive to negative across the precordium. Clockwise CTI-dependent flutter is more variable, but it characteristically shows positive, notched flutter waves in the inferior leads, and lead V_1 usually shows negative flutter waves. It should be emphasized that even CTI-dependent flutters can show variability in ECG morphology, and the *typical* patterns described above might also arise from non-CTI-dependent mechanisms. Typical CTI-dependent flutter that occurs after ablation for AF may also present with atypical ECG morphologies.[13]

Generally flutters that are predominately negative in V_1 arise from the RA, whereas those that show broad positive waves in V_1 (without isoelectric or negative initial components) usually arise from the LA (Fig. 12.1). Left atrial flutters usually demonstrate either positive flutter waves in the inferior leads or low-amplitude/isoelectric signals in the limb and other precordial leads.[12,14] Intermediate patterns that do not fit these various descriptions are common and can be difficult to localize. Regardless of the morphology, confirmation of the tachycardia origin with endocardial mapping is required.

Evidence for a macroreentrant mechanism can be obtained through manifest entrainment, entrainment with concealed fusion (concealed entrainment), or electroanatomic mapping (see *Mapping* section). Manifest entrainment is recognized as fixed fusion at any given paced cycle length and by progressive fusion with progressively rapid

BOX 12.1 Diagnostic Criteria

Macroreentrant Tachycardias

- Entrainment with fusion (with last beat entrained but not fused)
- Electroanatomic mapping of >90% of tachycardia cycle length with adjacent early and late areas of activation
- Insensitivity to adenosine (in dose sufficient to cause AV block)

Microreentrant Tachycardias or Localized Reentry

- Fragmented electrograms that encompass a large proportion of the tachycardia cycle length on single electrode or multiple closely spaced electrodes
- Entrainment with progressively longer postpacing intervals at increasing distances from the apparent source
- Insensitivity to adenosine (in dose sufficient to cause AV block)

Left Atrial Tachycardias

- Passive conduction in the right atrium, with early septal activation and fusion of wave fronts in the RA lateral wall
- Absence of RA activation during long segments of the cycle length (mapping <50% of tachycardia cycle length)
- Large variations in the RA cycle length with a relatively fixed cycle length in the LA
- Entrainment pacing at multiple sites in the RA yielding postpacing intervals >30 ms

AV, Atrioventricular; *LA,* left atrium; *RA,* right atrium.

overdrive pacing.[15] Because the surface flutter wave may not be visible or may be obscured by ventricular depolarization or repolarization, intracardiac electrograms provide a surrogate marker for orthodromic and antidromic capture and the degree of fusion. Manifest entrainment with progressive fusion implies a dimensionality to the tachycardia circuit that includes a separate entrance and exit site, a condition that does not exist for focal tachycardia and is unlikely to be demonstrated for microreentry. The pacing site is considered to be within the tachycardia circuit when the postpacing interval after entrainment is 30 ms (or less) greater than the tachycardia cycle length (Fig. 12.2). Entrainment from two opposite quadrants (such as septal and lateral LA, or anterior and posterior LA), each with return cycle lengths within 30 ms of the tachycardia cycle length, indicates the presence of a macroreentrant circuit.[11] Thus *in-circuit* entrainment from the septal and lateral LA often indicates perimitral reentry; *in-circuit* entrainment from the anterior and posterior LA is consistent with a roof-dependent LA flutter. Entrainment may also give indirect information about the distance of a pacing site from the reentrant circuit, with shorter postpacing intervals indicating closer proximity to the circuit.

The response to adenosine is useful in distinguishing reentrant from focal ATs, in that it exerts mechanistic-specific effects on atrial arrhythmias.[16] With rare exceptions, adenosine does not terminate the vast majority of reentrant atrial arrhythmias, whereas it either terminates focal ATs attributed to triggered activity or transiently suppresses focal AT as a result of enhanced automaticity (Fig. 12.3). Adenosine-insensitive ATs with apparently focal activation patterns show characteristics of localized reentrant circuits, including low-amplitude, long-duration fractionated electrograms, and can be entrained.[7] This simple tool provides a reliable means for establishing a tachycardia mechanism before mapping (Fig. 12.4).

Localization of Reentry to the Right Atrium or Left Atrium

The activation pattern of the coronary sinus (CS) is useful in localizing ATs.[17] RA tachycardias typically exhibit proximal-to-distal activation

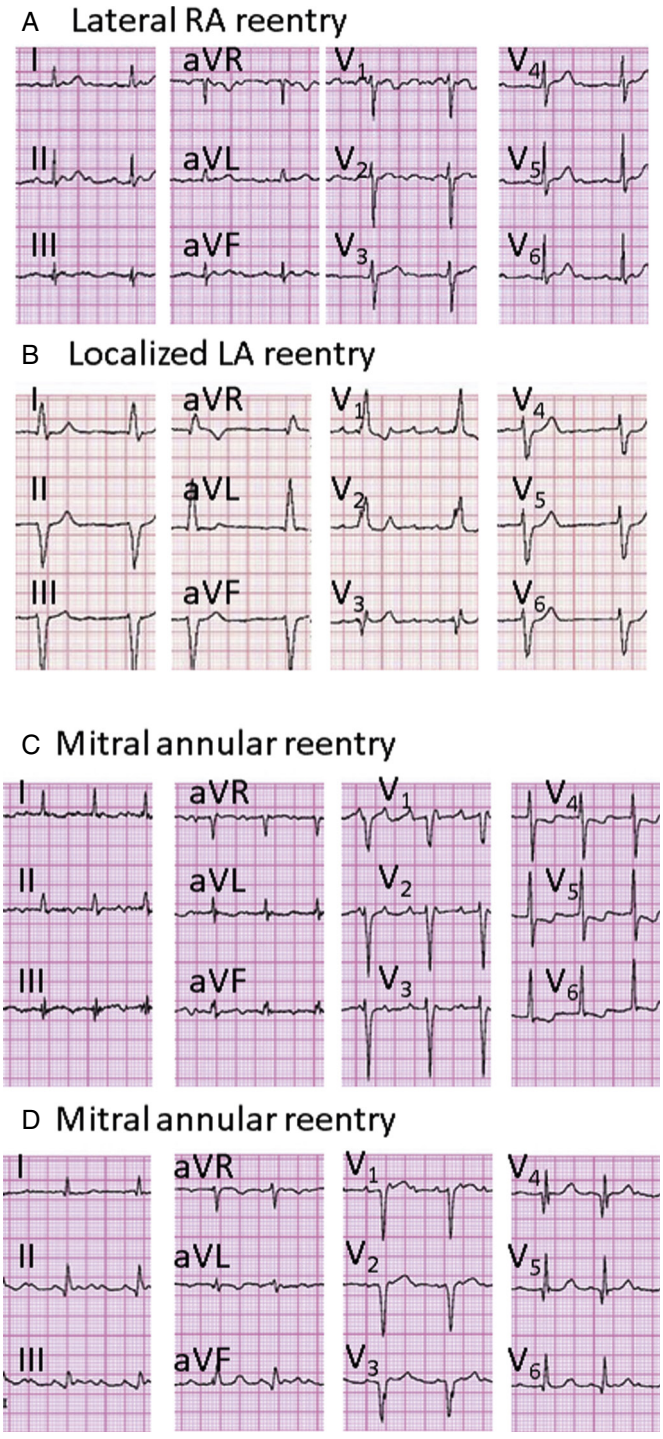


Fig. 12.1 Representative examples of atypical flutters. A, Lateral right atrial (RA) reentry in a patient with prior mitral valve repair with a right atriotomy. In this case, flutter waves are negative in V_1 with an inferior axis. B, Localized reentry from the anterior wall of the left atrium (LA) shows positive flutter waves in V_1 with low-amplitude signals in the limb and lateral precordial leads. C and D, Two examples of perimitral annular reentry in a patient with prior mitral valve repair and a patient with ischemic cardiomyopathy. These show positive flutter waves in V_1 with different axes in the inferior leads. Note the isoelectric interval, illustrating that isoelectric intervals are not specific for focal tachycardia.

in the CS, unless activation proceeds predominantly over Bachmann bundle (or the catheter is positioned very distally in the CS), in which case the distal CS may be activated earlier. LA tachycardias show a

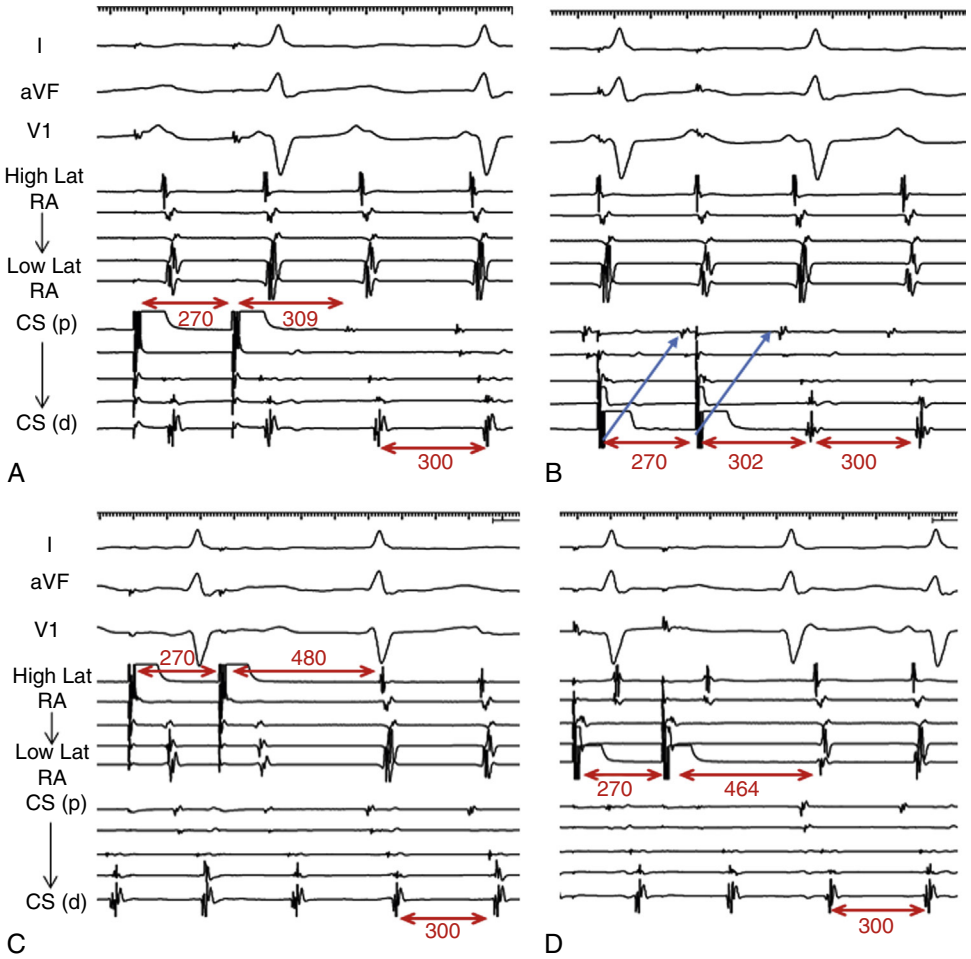


Fig. 12.2 Entrainment from multipolar catheters localizes a left atrial tachycardia. Activation of the coronary sinus (CS) is proximal to distal. Entrainment from the proximal and distal poles of the CS catheter (A and B) results in postpacing intervals nearly identical to the tachycardia cycle length. Panel B shows the phenomenon of downstream overdrive pacing.[83] Entrainment from the distal electrode of the CS catheters (downstream from the proximal electrodes) results in orthodromic capture of the proximal poles after conduction delay. Panels C and D show entrainment from the high and low right atrium (RA), respectively, both of which show long postpacing intervals (as well as fusion of intracardiac electrograms during pacing), indicating that they remote from the tachycardia circuit. These responses are consistent with a left atrial tachycardia, which was perimitral reentry.

variety of activation patterns depending on the particular reentrant circuit (Fig. 12.5). Some LA tachycardias, such as clockwise perimitral annular reentry and tachycardias originating near the lateral LA (by the left pulmonary veins or appendage), exhibit distal-to-proximal CS activation. However, a proximal-to-distal sequence does not necessarily localize the arrhythmia to the RA and may be seen with counterclockwise perimitral reentry or tachycardias involving the septum or right pulmonary veins. *Chevron* and *reverse chevron* activation patterns occur with roof-dependent LA macroreentry. The chevron pattern occurs when a wave front descends the posterior wall and activates the midposterior mitral annulus, and then propagates in both septal and lateral directions along the inferior LA. The reverse chevron pattern occurs when a wave front descends the anterior LA wall and then fuses in the posterior mitral annulus. Focal arrhythmias in the posterior or anterior LA may also produce chevron or reverse chevron patterns. It should be recognized that activation of the CS may be dissociated from the LA endocardium because of a muscular sleeve, which envelops the CS and is attached to the LA through discrete connections. Electrograms recorded in the CS may show disparate activation patterns, and careful analysis may show that the endocardial LA is activated in a pattern different from the CS.[18] Macroreentry involving the CS musculature or the ligament of Marshall as a critical part of the circuit has also been described.[19,20]

It is possible to establish a diagnosis of LA tachycardia during mapping in the RA and thus identify the need for transseptal catheterization and LA mapping.[21,22] Criteria for identifying an LA origin through intracardiac mapping include the following:

1. Passive conduction into the RA, which may be demonstrated as fused wave fronts in the lateral wall of the RA.
2. Earliest RA activation in the septum, typically in the region of Bachmann bundle or the CS ostium, especially if areas of early activation encompass a large area.
3. Large variations in the RA cycle length with a relatively fixed cycle length in the LA, implying LA/RA dissociation or conduction block.
4. Entrainment pacing at multiple sites in the RA (including the CTI and the RA free wall) yielding postpacing intervals greater than 30 ms.

Although fusion of wave fronts in the lateral wall of the RA is common during LA tachycardias, it is possible to record a single wave front

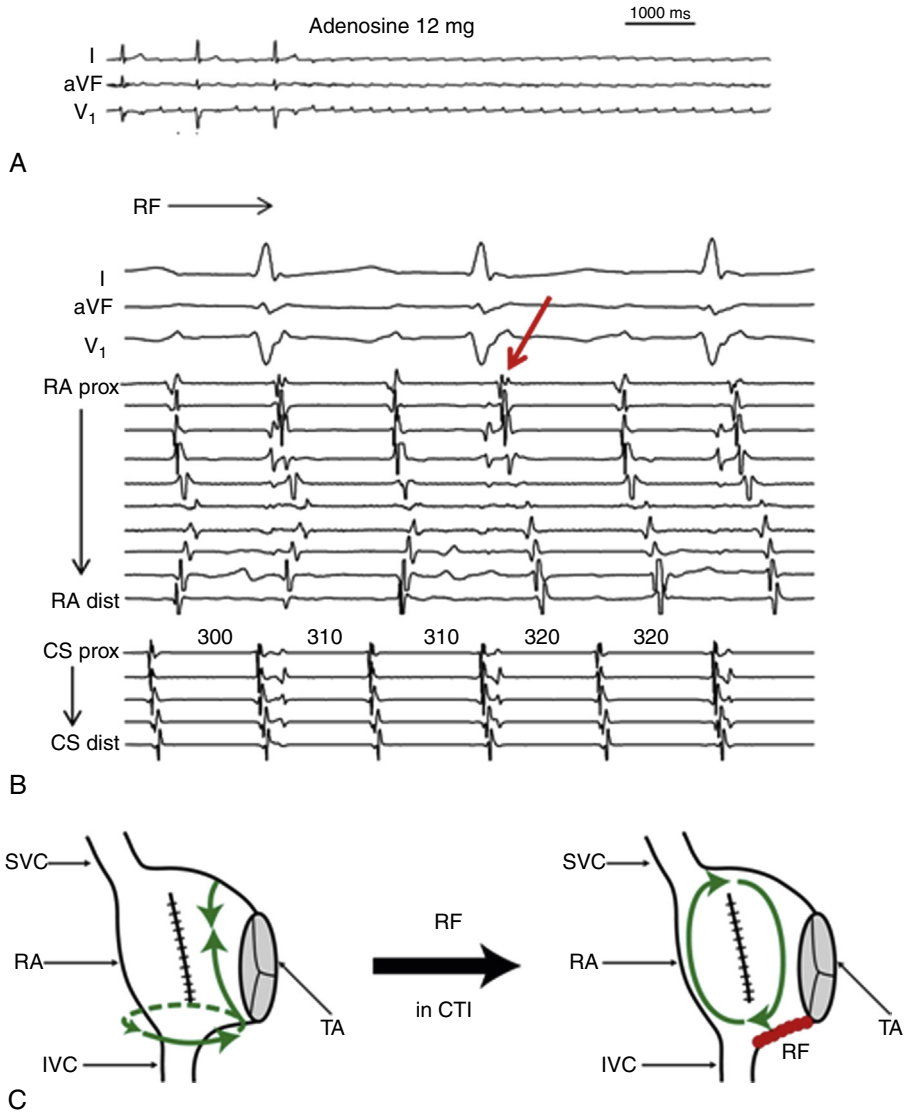


Fig. 12.3 Reentrant atrial tachycardia in a patient with a prior lateral right atriotomy. A, The tachycardia is insensitive to adenosine, which is consistent with a reentrant mechanism. B, A duo-decapolar catheter in the lateral right atrium (RA) demonstrates fusion of wave fronts in the lateral wall, in this case consistent with lower-loop reentry, with a circuit around the inferior vena cava (IVC). During ablation in the cavotricuspid isthmus, the tachycardia cycle length prolongs and activation in the lateral right atrium changes *(arrow),* which is explained by interruption of lower-loop reentry and the existence of a second circuit involving the lateral right atriotomy. C, The change in the tachycardia circuit is illustrated graphically. *CS,* Coronary sinus; *CTI,* cavotricuspid isthmus; *RF,* radiofrequency; *SVC,* superior vena cava; *TA,* tricuspid annulus.

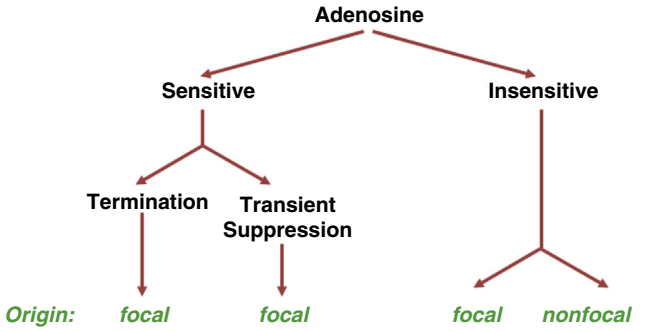
mimicking counterclockwise or clockwise atrial flutter. This situation depends on (1) the location of the multipolar catheter in the lateral wall, (2) the location of conduction breakthrough from the LA (i.e., preferential conduction over Bachmann bundle or the CS), and (3) the presence of conduction block in the CTI. Typically, activation time in the RA is substantially less than the tachycardia cycle length. Exceptions occur when the tachycardia cycle length is short or if conduction is substantially slowed in the RA, incorrectly implying the presence of a RA tachycardia.

MAPPING

Activation Mapping

Conventional activation mapping with multielectrode catheters is usually employed to define the mechanisms of atypical flutters. In the RA, a multielectrode catheter positioned around the tricuspid annulus can provide information suggestive about the tachycardia mechanism. For example, counterclockwise lower-loop reentry reveals lateral-to-septal activation in the CTI and areas of breakthrough in the lateral RA, which can cause fusion of wave fronts or an ascending wave front in the lateral wall (see Fig. 12.3).[23]

Double potentials, which usually signify lines of block, may be identified through conventional activation mapping. If reentry proceeds around a line of block, double potentials are widely split in the middle of the line, and they progressively narrow toward the end of the line where the wave front pivots.[24] An example of this may be found in RA free-wall macroreentry (Fig. 12.6). Middiastolic and fragmented potentials can be recorded within critical zones of slow conduction, but verification of participation in the tachycardia circuit is desirable through other means, such as entrainment maneuvers.



Induction by PES	+	–	+	+
Termination by PES	+	–	+	+
Entrainment	–	–	+	+
Electrogram duration	↓	↓	↑	↑/↓
Catecholamine facilitation	+	+	+/–	+/–

Fig. 12.4 Differentiating mechanisms of atrial tachycardia using adenosine and electrophysiological characteristics. Adenosine-sensitive atrial tachycardia is typically focal in origin caused by triggered activity or, far less commonly, automaticity. Adenosine-insensitive atrial tachycardia is either macroreentrant or microreentrant, depending on circuit size and the resolution of the mapping system. Entrainment with the postpacing interval nearly equal to the atrial tachycardia cycle length is typical for macroreentrant or microreentrant tachycardias. Prolonged electrogram durations may be recorded at early sites in microreentrant tachycardias but are not typical at the origin of triggered and automatic rhythms. Electrogram durations at sites around a macroreentrant circuit might vary depending on local conduction characteristics. *PES,* Programmed electrical stimulation. (Modified from Markowitz SM, Nemirovksy D, Stein KM, et al. Adenosine-insensitive focal atrial tachycardia: evidence for de novo micro-re-entry in the human atrium. *J Am Coll Cardiol.* 2007;49:1324-1333. With permission.)

Activation mapping in the LA relative to a fixed reference can identify or exclude mitral reentry or roof-dependent flutter.[11] Opposite activation sequences in the superior and inferior mitral annulus (e.g., lateral-to-septal activation along the superior annulus, and septal-to-lateral activation along the inferior annulus) identify perimitral reentry. By contrast, similar directions of activation (e.g., lateral-to-septal) in both the superior and inferior annulus exclude perimitral reentry. Opposite activation along the anterior and posterior walls is seen with roof-dependent reentry.

Entrainment Mapping

Concealed entrainment is an essential tool for identifying sites that participate in a reentrant arrhythmia.[25] Entrainment criteria for identifying appropriate sites for ablation are intended to localize protected zones within a circuit. These include (1) concealed entrainment (with P wave and intracardiac activation sequences resembling those in tachycardia), (2) a postpacing interval within 30 ms of the tachycardia cycle recorded at the pacing site, and (3) a stimulus to P wave interval during pacing equal to the electrogram to P wave interval during tachycardia. Because the P wave may not be visible in many cases of macroreentrant AT, identifying the initial inscription of the P wave may be arbitrary, and an intracardiac reference is often used as a surrogate.

Limitations or pitfalls in using concealed entrainment must be recognized: (1) rate-related conduction slowing may occur, and therefore the postpacing interval might exceed the tachycardia cycle length; (2) failure to capture might occur in some critical regions of a reentrant circuit; (3) acceleration or termination of AT with pacing may occur.[26] Inadvertent termination or slowing can be minimized by pacing 20 ms or less shorter than the tachycardia cycle length.

Electroanatomic Mapping

Electroanatomic mapping provides direct visualization of a reentrant circuit, which is defined as the shortest distance of continuous activation comprising the tachycardia cycle length. A hallmark of macroreentrant arrhythmias is the presence of areas of early activation adjacent to late regions, with intermediate values connecting these two regions (see Figs. 12.5 and 12.6). In practice, it is necessary to account for 90% or more of the tachycardia cycle length to visualize a reentrant circuit. Displaying activation information as an isochronal map may clearly demonstrate the direction of wave-front propagation, which is perpendicular to the isochronal steps (Fig. 12.7). Ultrahigh-resolution mapping uses proprietary multipolar mapping catheters to collect a large number of activation points and can identify critical isthmuses of reentrant circuits that are suitable for ablation.[27]

A substantial percent of the tachycardia cycle length may be occupied by conduction through a diseased segment with low-amplitude and fractionated electrograms. If a large part of the cycle length is not accounted for in the map, a careful search for such electrograms should be undertaken to identify a gap in the reentrant circuit; such sites are often characterized by fragmented, low-amplitude electrograms and are appropriate targets for ablation (Fig.12.8). The specific choice of assigning activation times to long duration or split electrograms can greatly affect the appearance of an electroanatomic map and may potentially obscure the delineation of a reentrant circuit. New

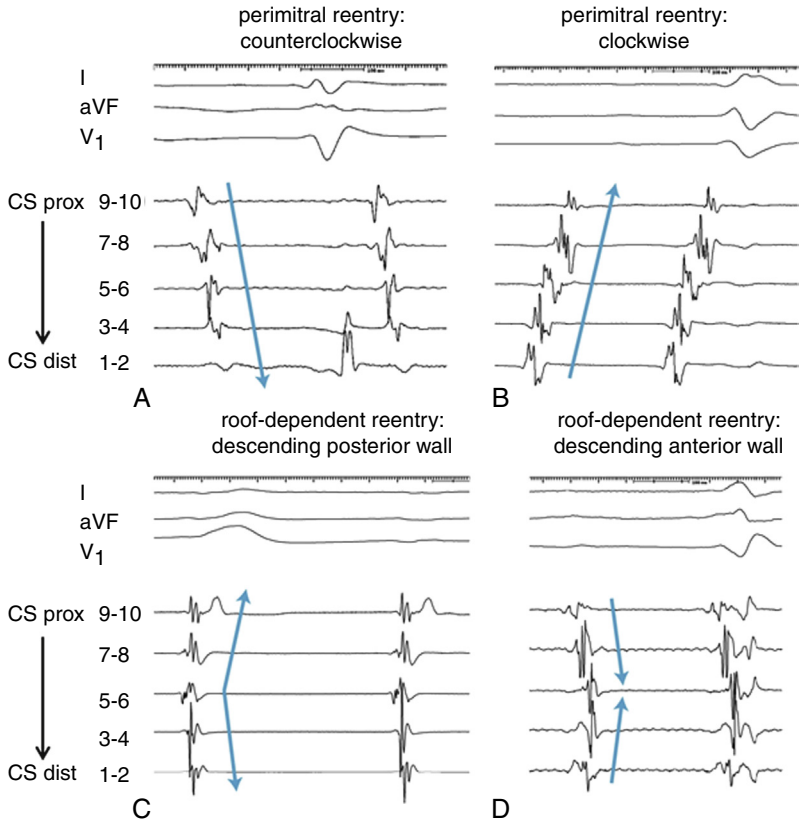


Fig. 12.5 Activation sequences of the coronary sinus (CS) in various forms of left atrial tachycardia. A, Counterclockwise perimitral reentry gives rise to proximal-to-distal CS activation. B, Clockwise perimitral reentry produces distal-to-proximal CS activation. C, Roof-dependent atrial tachycardia with a descending wave front in the posterior wall causes early activation in the mid CS, after which the impulse propagates both proximally and distally along the mitral annulus (chevron pattern). D, Roof-dependent atrial tachycardia with a descending wave front in the anterior wall causes fusion of wave fronts in the posterior mitral annulus (by the coronary sinus) because the impulse first activates the anterior mitral annulus and then bifurcates to activate the lateral and septal walls before fusing in the posterior annulus (reverse chevron pattern).

automated mapping technology uses algorithms that integrate bipolar and unipolar electrograms with neighboring activation times to annotate complex electrograms.[28] By displaying an atrial voltage map, areas of scar can be identified that facilitate the localization of channels that form potential reentrant circuits. Areas with no detectable voltage (defined as the noise limit of recording systems) represent a dense scar and thus a region of fixed conduction block.

Entrainment mapping may be combined with electroanatomic mapping to define critical components of a reentrant circuit. This combined technique is especially useful in situations where the electroanatomic map is ambiguous and it is difficult to distinguish critical components from bystander regions.

Misinterpretation of electroanatomic maps may occur unless care is taken to avoid the following pitfalls (Table 12.2):

1. Incomplete mapping and low resolution: It is important to acquire activation points during tachycardia with sufficient density and to sample different regions in the atrium of interest. Interpolation of activation times in the map may lead to misinterpretation of the rhythm and failure to identify critical components of the circuit. Advanced mapping technology that constructs high-density maps with shorter acquisition times is useful in addressing this pitfall.
2. Fractionated electrograms: If highly fractionated and wide potentials are present, it might be difficult to assign an activation time. In this case, the critical isthmus might not be identified, and the reentrant arrhythmia might be confused with a focal rhythm.
3. Central obstacles or conduction block: Failure to identify areas of scar or central obstacles to conduction may confuse interpretation of an electroanatomic map because interpolation of activation times through areas of conduction block may give the appearance of wave front propagation and obscure the reentrant circuit. If this occurs, it is impossible to identify a critical isthmus to target for ablation. Adjusting the voltage threshold for identifying areas of scar may ameliorate this. It should be recognized that very low-voltage areas (<0.05 mV) can serve as critical components of tachycardia circuit. Mapping with a relatively large-tip ablation catheter with widely spaced bipolar electrograms may not identify low-amplitude signals, which are more easily recorded with smaller, closely spaced electrodes and mapping systems that generate low noise levels. A line of block can be inferred if there are adjacent regions with wave front propagation in opposite directions, separated either by a line of double potentials or dense isochrones.[29,30]
4. Conduction delay in either atrium: Slow conduction or local block may prolong activation in either the RA or LA, and conduction time in the passively activated chamber may approach the tachycardia cycle length. For example, if conduction block is present in the CTI, an LA tachycardia can give rise to a craniocaudal activation pattern of the lateral RA (Fig. 12.9). Even if conduction is present in the CTI, RA activation in the lateral wall may be craniocaudal if conduction occurs over Bachmann bundle. In these situations,

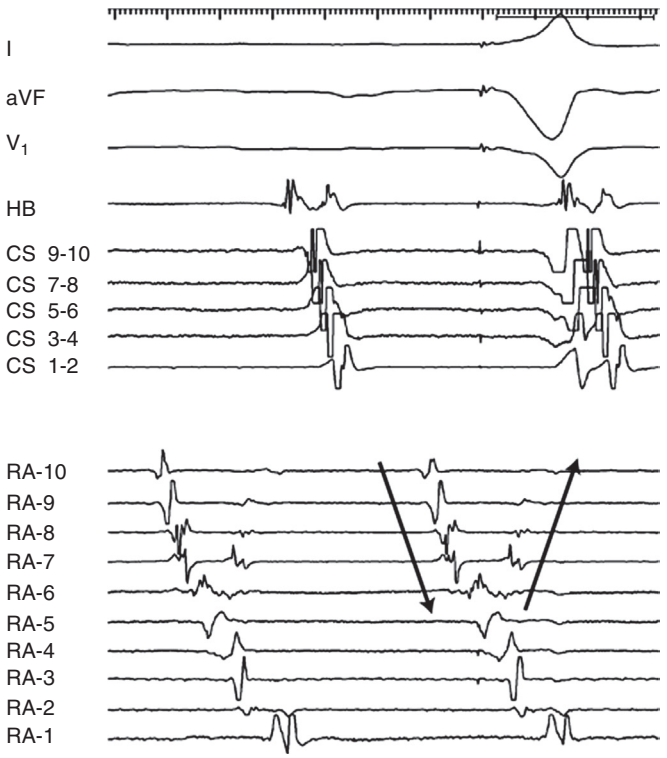


Fig. 12.6 Right atrial (RA) free-wall reentry. Double potentials in the lateral wall reflect descending and ascending wave fronts, with narrowing of the intervals between double potentials down the lateral wall and a fragmented electrogram reflecting a pivot point in pole RA-6 (RA-1, low lateral RA; RA-10, high lateral RA). *CS,* Coronary sinus.

entrainment is an important adjunctive tool, which would define the RA as a bystander and clarify the electroanatomic map.

5. Changing activation: The operator should be cognizant of changes in activation that occur during mapping, as the tachycardia may spontaneously transition to another rhythm or could be affected by ectopic beats induced by catheter manipulation. Also, a stable circuit can conduct variably to bystander regions, and these changes in activation can produce an uninterpretable map.
6. Misinterpretation of reentry: Ultrahigh-resolution mapping systems can identify small reentrant circuits, which are characterized by continuous rotation within a small area and activation encompassing the tachycardia cycle length. In some cases, the color-coded activation map appears to show localized reentry, but in fact this area of rotation is passively activated.[31] This phenomenon can be detected by tracing the wave front activation, which might show distant wave fronts that invade the area of slow conduction and produce incomplete rotation. Even larger circuits can appear to complete a single rotation but might actually be passively activated.[32] This situation can be demonstrated with entrainment to confirm if sites are constituents of a reentrant circuit.

Noncontact electroanatomic mapping uses a multielectrode array that records intracavitary potentials and software to construct virtual unipolar electrograms on a 3-dimensional representation of a given chamber. This technique is useful in delineating transitory arrhythmias and has been used to visualize upper loop and RA free-wall reentry.[33] Care must be taken to analyze atrial beats without ventricular depolarization or repolarization, which can obscure the atrial unipolar electrograms. The presence of lines of block can be inferred based on activation sequence rather than direct imaging of scar. Novel mapping systems use an ultrasound-electrode array to construct the anatomy of a chamber and compute activation through noncontact dipole density mapping.[34]

Body surface mapping has been employed using a 252-electrode vest combined with biatrial anatomy obtained from computed tomography. The feasibility of this technology has been demonstrated in identifying macroreentrant rhythms in both atria and distinguishing these from focal sources.[35]

ABLATION

The guiding principle in ablating atypical flutters is to target a critical isthmus or component that participates in the tachycardia circuit (Box 12.2). For successful ablation it is not always necessary to delineate the complete reentrant circuit, because interruption of the circuit at any one critical site will terminate the tachycardia and prevent its initiation. The critical isthmus may be a narrow channel or a relatively broad region. In the case of dual-loop tachycardias, it is helpful to identify the common isthmus or corridor. Ablation can be performed by targeting the common isthmus or each loop separately. The technique of ablation involves the creation of a linear lesion between two boundaries to transect the isthmus. Occasionally, a single radiofrequency (RF) application is enough to interrupt a narrow isthmus or localized reentry.

Standard RF with a 4-mm tip may be sufficient, but creation of long linear transmural lesions is usually performed with irrigated RF or larger tip (8 mm) catheters. Contact force sensing technology has proved useful, although it has been studied primarily in the context of AF ablation.[36] Effective RF lesions are characterized by at least a 10 Ω impedance drop during ablation and reduction of local electrogram amplitude. In cases of linear lesions, bidirectional conduction block should be verified after ablation by pacing from either side of the ablation line and recording from the contralateral side. The detour of wave fronts around completed ablation lines can be displayed graphically with a reconstructed electroanatomic map during pacing or sinus rhythm. Widely spaced double potentials along an ablation line, while pacing from one side, provide supporting evidence for conduction block after ablation. Noninducibility of AT is another end point; however, atypical flutters are frequently associated with other inducible atrial arrhythmias, and judgment is required to decide whether to target other inducible arrhythmias that may not have clinical significance. Strategies that involve ablation of all inducible atrial arrhythmias and channels for potential reentrant circuits have been successful in preventing recurrences, particularly in patients with prior surgery for congenital heart disease.[37] This strategy can be used for multiple interconverting ATs that are challenging to define with contact mapping (see Table 12.2).

Acute and long-term clinical outcomes depend on the specific arrhythmia targeted for ablation and the atrial substrate. For example, a series of patients with macroreentrant AT without a history of surgery or prior catheter ablation reported a freedom from atrial arrhythmias without antiarrhythmic drugs in 82% of patients with RA, and in 55% with LA tachycardias (average follow-up 37 months).[38] In another cohort of 91 patients with atypical flutters (comprising 171 ATs), the acute success rates for ablation were 97% for patients with nonseptal ATs and 77% for patients with septal ATs.[39] Long-term success rates were 82% for patients without septal ATs and 67% for those with at least one septal AT. Long-term success rates were higher for ablation of ATs that occurred after catheter ablation and cardiac surgery (75% and 88%, respectively) and lower for patients with idiopathic atrial scar (57%).[40–43] In a mixed cohort of 52 patients, many of whom had prior surgical or ablative procedures, macroreentrant ATs were ablated with an acute success rate of 90%, of whom 6% had early recurrences.[44] Successful outcome may require up to three staged procedures.[45,46]

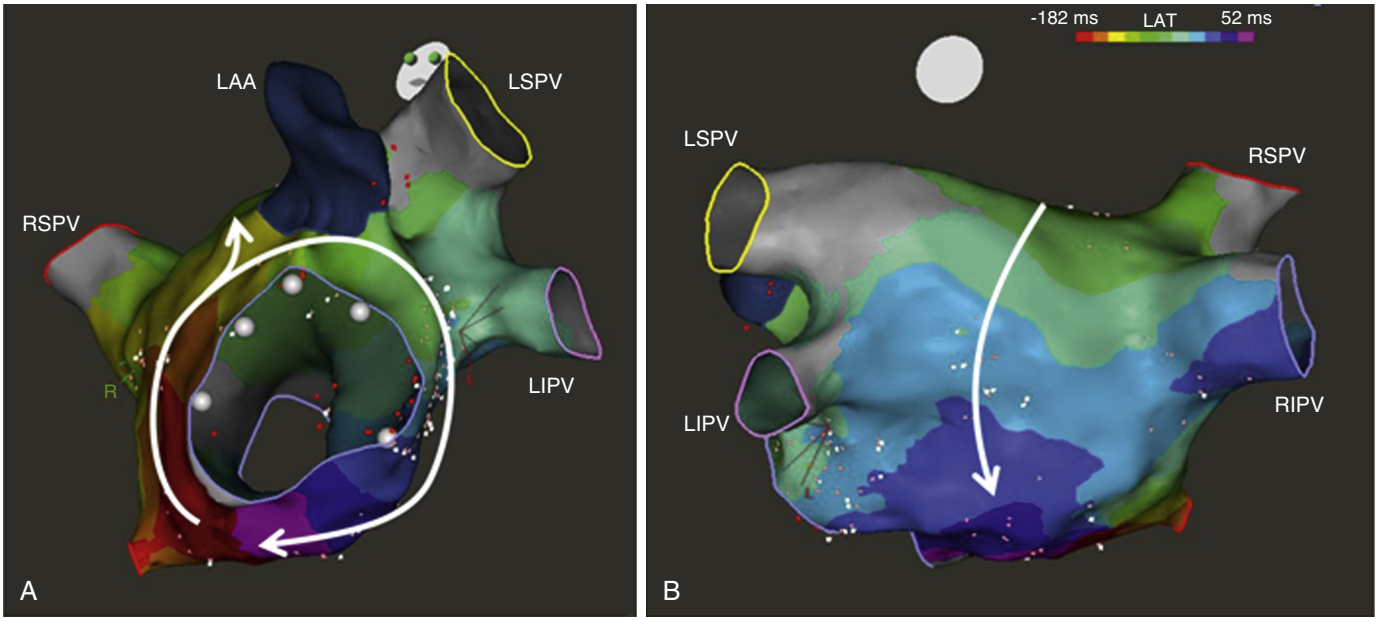


Fig. 12.7 Electroanatomic map of a left atrial tachycardia with isochronal steps of 20 ms. The map demonstrates a dual-loop tachycardia, with one clockwise loop around the mitral annulus (A) and a second roof-dependent circuit around the right pulmonary veins with descending activation of the posterior wall (B). *LAA,* Left atrial appendage; *LIPV,* left inferior pulmonary vein; *LSPV,* left superior pulmonary vein; *RIPV,* right inferior pulmonary vein; *RSPV,* right superior pulmonary vein.

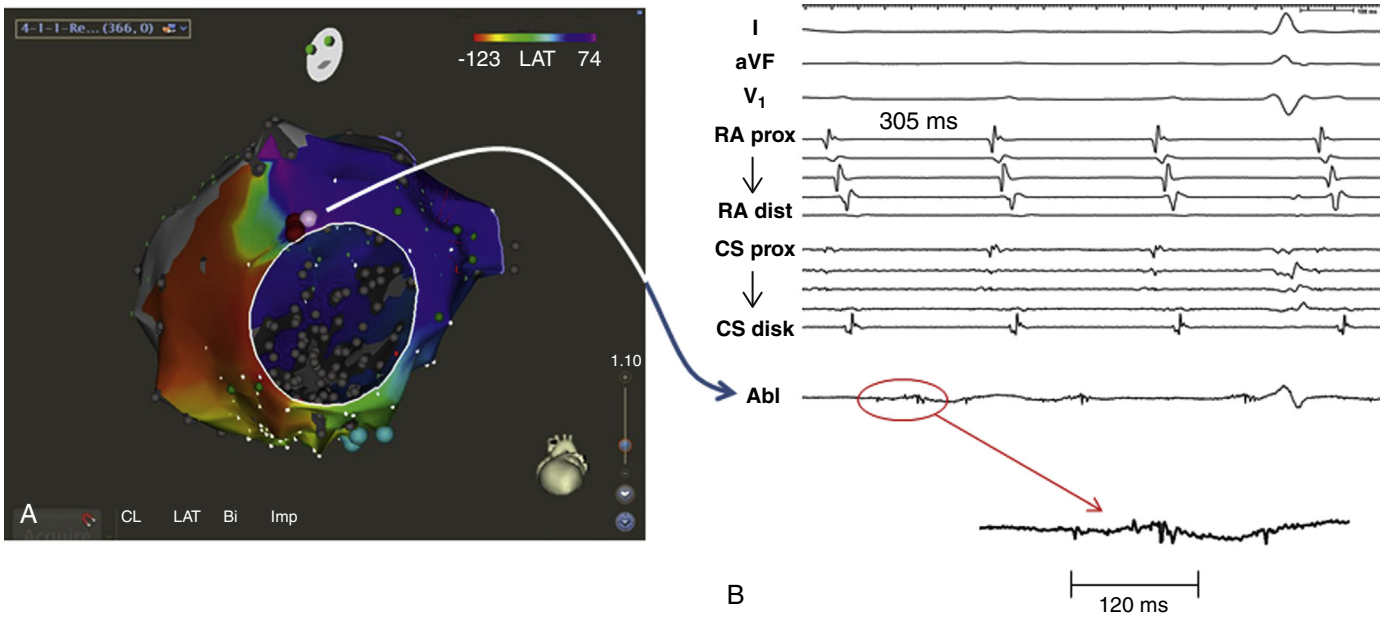


Fig. 12.8 Gap in electroanatomic map as a result of slowly conducting tissue. The patient had undergone mitral valve and maze surgery. A, An electroanatomic map of the left atrium shows counterclockwise activation of the mitral annulus (MA), but the activation times encompass only 197 ms (65% of the tachycardia cycle length: 305 ms). B, A careful search identifies a highly fractionated, low-amplitude electrogram (0.05 mV) along the anteroseptal mitral annulus, which accounts for 120 ms of the tachycardia cycle length *(pink tag).* This site is a gap along a cryomaze ablation (Abl) line, and focal radiofrequency ablation at this site terminated the atrial tachycardia. *CS,* Coronary sinus; *RA,* right atrium.

SPECIFIC FORMS OF ATYPICAL FLUTTER

Upper-Loop Reentry

This macroreentrant circuit involves reentry around the SVC and an area of fixed or functional conduction block adjacent to the SVC.[23,45] This arrhythmia may occur in patients who also have isthmus-dependent flutter, but it also occurs in isolation or in those with a prior right atriotomy (Fig. 12.10; Video 12.1). The ECG in upper-loop reentry mimics the morphology of clockwise CTI-dependent flutter, with an inferior flutter wave axis.[14] Electrograms may show a descending wave front in the lateral RA, but if scar or conduction block extends inferior to the SVC, the pattern of activation

TABLE 12.2 Troubleshooting Difficult Cases

Problem	Cause	Solution
Unstable rhythm interconverting with AF or other forms of atypical flutter	Instability of lines of block	Limit atrial pacing and entrainment mapping Consider noncontact electroanatomic mapping Consider long linear lesions and/or ablation of atrial fibrillation Identify and treat channels of slowly conducting tissue Low dose IC or III drug to stabilize reentry
Uninterpretable electroanatomic map	Incomplete map Failure to identify lines of block and areas of scar	Use high or ultrahigh density mapping Review voltage map to identify areas of scar and low voltage, fragmented electrograms Use entrainment to identify critical components of tachycardia Review previous surgery and ablation attempts
Failure to achieve conduction block in mitral isthmus	Persistent epicardial conduction	Use irrigated radiofrequency catheter Ablate within coronary sinus Alternative linear lesion
Failure to terminate tachycardia despite complete ablation line	Dual-loop reentry Inaccurate mapping of critical isthmus	Reevaluate cycle length, surface ECG, and activation sequence Reevaluate map or remap
Unstable catheter during linear ablation	Large left atrium, limited catheter mobility	Use adjustable or telescoping sheath system, change catheters, linear ablation array, robotic navigation

AF, Atrial fibrillation; *ECG,* electrocardiography.

may be more complex. Concealed entrainment may be demonstrated in the septum between the fossa ovalis and the SVC and in the high lateral right atrium.

Noncontact and contact electroanatomic mapping have been used to define the upper-loop reentry circuit.[33,46] In cases defined with noncontact mapping, gaps in the crista terminalis could be identified and targeted for ablation. Although the published literature on upper-loop reentry is limited and follow-up is relatively short (between 3 and 17 months), small clinical series reveal that ablation can be accomplished with a low recurrence rate of AF or other atrial flutters (23% in one clinical series).[33] When guided by noncontact electroanatomic mapping, an effective strategy is to make a line of RF applications through a gap in the crista terminalis. Another strategy is to connect the SVC to a scar elsewhere in the RA.

Right Atrial Free-Wall Macroreentry

Macroreentry may occur in the free wall of the RA in patients with an atriotomy, but also has been reported in patients without prior cardiac surgery.[24,29,30,33,47] The pathophysiology of this arrhythmia is reentry around a line of block in the lateral RA, defined by an electrically silent area or a line of double potentials. Lateral right atriotomies are used for repair of congenital heart disease (such as atrial septal defects and more complex congenital disease), mitral and tricuspid valve surgery, and removal of atrial myxomas. In patients without an atriotomy, free-wall reentry arises because of fixed scar in the lateral RA or rate-related functional block in the crista terminalis.

Mapping with conventional multipolar catheters reveals a line of double potentials in the lateral wall with single fractionated potentials at the inferior end of the line, reflecting the lower pivot point (see Fig. 12.6). Entrainment from both sides of the central line of block results in postpacing intervals within 30 ms of the tachycardia cycle length. The location of the upper pivot point may be in the upper free wall of the RA or involve the SVC.[24,33] Catheter ablation may be performed by making a line of RF lesions between the lateral RA (the area of double potentials) and the IVC. Alternatively, ablation may be performed in the corridor between the line of block in the lateral wall and the tricuspid annulus or the crista terminalis. It is important to confirm line of block after interrupting lateral wall reentry. When ablation is performed from the lateral wall to the IVC, line of block can be demonstrated by pacing and recording both anterior and posterior to the line (Fig. 12.11). Activation should proceed in a craniocaudal direction on the side opposite the pacing site.[30,48]

The acute success rate of ablating lateral wall reentry generally exceeds 85%, but late recurrences of atrial arrhythmias are common. In a report of 40 patients who had macroreentrant ATs as a late complication after lateral right atriotomies, ablation was acutely successful in 88%, but during follow-up (average 28 months) only 55% remained free of recurrent atrial arrhythmias.[49] Many of the recurrences were new arrhythmias, including AF. In another cohort of 20 patients who had catheter ablation of ATs resulting from surgical repair of atrial septal defects, 30% developed AF during long-term follow-up.[50] The issue of late AF is of particular importance because patients may require anticoagulation and additional medical or ablative therapy during follow-up.

Dual-Loop Right Atrial Macroreentry

Dual-loop reentry may occur in the RA in which two atypical circuits described earlier coexist or when one of the atypical circuits combine with rotation around the tricuspid annulus.[38,51] Entrainment and contact electroanatomic mapping have been used to identify various combinations of reentry around the IVC (lower-loop reentry), the tricuspid annulus, the SVC, and a conduction barrier in the lateral RA (see Fig. 12.3). Ablation of one component usually transforms the tachycardia to another circuit. Dual-loop reentry is common in patients who have prior right atriotomies. One example is reentry around the atriotomy scar that coexists with peri-tricuspid reentry. The flutter wave morphology may resemble typical flutter, and activation mapping may indicate reentry around the tricuspid annulus, which is confirmed with entrainment mapping in the CTI. If mapping in the lateral RA is not performed, the second component of this circuit might not be identified. A multipolar catheter is useful for demonstrating ascending and descending wave fronts on either side of a lateral atriotomy. Ablation in the CTI modifies but does not terminate the tachycardia, as evidenced by a change in activation pattern and/or cycle length (see Fig. 12.3). This form of dual-loop reentry can be treated by ablating the common isthmus (e.g., between the lateral tricuspid annulus and the lateral atriotomy) or by creating two linear lesions to interrupt each reentrant loop (one in the CTI and another from the lateral atriotomy to the IVC or SVC). As with single loop lateral wall reentry, it is important to verify line of block with pacing maneuvers, which is a more robust end point than noninducibility (see Fig. 12.11). Some dual-loop tachycardias

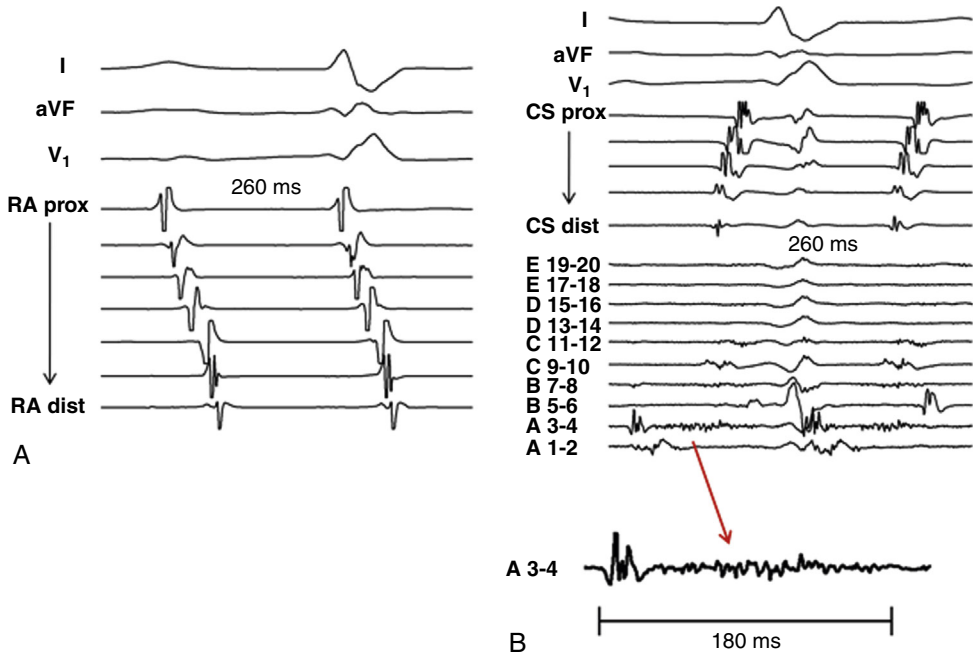


Fig. 12.9 Left atrial microreentrant tachycardia after catheter ablation of atrial fibrillation. A, The right atrial (RA) activation sequence demonstrates a descending wave front in the lateral wall because of prior ablation in the cavotricuspid isthmus. B, A PentaRay catheter positioned anterior to the left inferior pulmonary vein shows a highly fractionated, low-amplitude electrogram (0.12–0.24 mV) on spline electrode A 3–4, which spans most of the tachycardia cycle length. *CS,* Coronary sinus.

BOX 12.2 Target Sites for Ablation

General Principles

- Critical isthmus identified by electroanatomic mapping bounded by two conduction barriers
- Entrainment demonstrates orthodromic capture of most of atrium (*concealed entrainment* is present) and postpacing interval minus tachycardia cycle length ≤30 ms

Ablation Strategies for Specific Arrhythmias

Upper-Loop Reentry

- Gap in crista terminalis
- Linear ablation from SVC to scar or tricuspid annulus or intercaval line

RA Free-Wall Reentry

- Linear ablation from conduction barrier to IVC
- Linear ablation from conduction barrier to tricuspid annulus
- Linear ablation from conduction barrier to SVC

Perimitral Reentry

- Linear ablation from lateral mitral annulus to left inferior pulmonary vein
- Linear ablation from anterior mitral annulus to roof line or pulmonary vein
- Linear ablation from septal mitral annulus to right pulmonary veins

LA Roof-Dependent Reentry

- Linear ablation between left and right superior pulmonary veins (roof line)
- Linear ablation between left and right pulmonary veins on posterior wall

Left Septal Reentry

- Linear ablation from septum to mitral annulus
- Linear ablation from septum to right pulmonary

Lesional Tachycardia

- Linear ablation from scar boundary to anatomic barrier
- Gap within incomplete incisional lines

Localized Reentry

- Site with highly fragmented electrograms encompassing ≥35% of tachycardia cycle length

IVC, Inferior vena cava; *LA,* left atrium; *RA,* right atrium; *SVC,* superior vena cava.

share a narrow common isthmus in the lateral wall that can be targeted for focal ablation (Video 12.2).

Left Atrial Macroreentry

A variety of reentrant circuits have been described in the LA of patients with structural heart disease and after surgery for acquired heart disease.[21,22,29,38] Examples of LA macroreentrant circuits include circus movement around the mitral annulus, roof-dependent reentry, and reentry around electrically silent areas. Often times, transformation of a tachycardia during ablation, that is, subtle alterations in intracardiac activation sequence, cycle length, or P wave morphology, suggests the presence of a dual-loop tachycardia. Extensive ablation in one connecting line that eliminates voltage but fails to terminate a tachycardia should raise the possibility of a second limb (see Table 12.2).

Perimitral reentry is treated by making a line of RF lesions between the mitral annulus and another electrical barrier. This barrier can be an electrically isolated pulmonary vein or a line of block in the LA roof (Fig. 12.12). A common approach is to create a linear lesion from the lateral mitral annulus to the left inferior pulmonary vein (lateral mitral isthmus). Completion of the ablation line in the lateral mitral isthmus may be difficult to achieve, and incomplete ablation can result

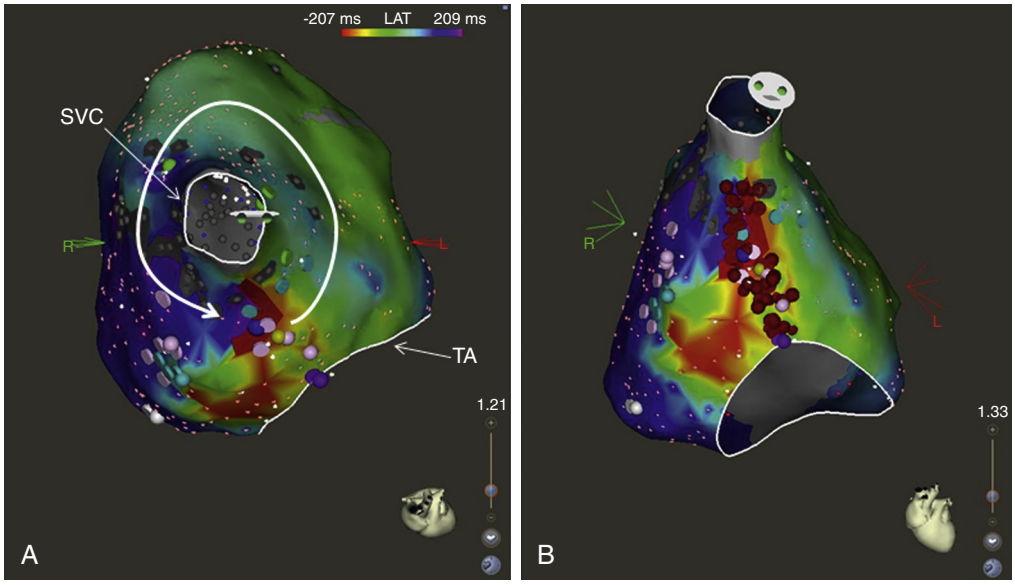


Fig. 12.10 Upper-loop reentry in patient with prior mitral valve and maze surgery. A, Superior projection of the electroanatomic map shows reentry around the superior vena cava (SVC). *Green tags* indicate sites where entrainment was consistent with participation in the tachycardia circuit. *Pink tags* indicate fractionated electrograms. B, An ablation line between the SVC and anterior tricuspid annulus (TA) terminated the tachycardia.

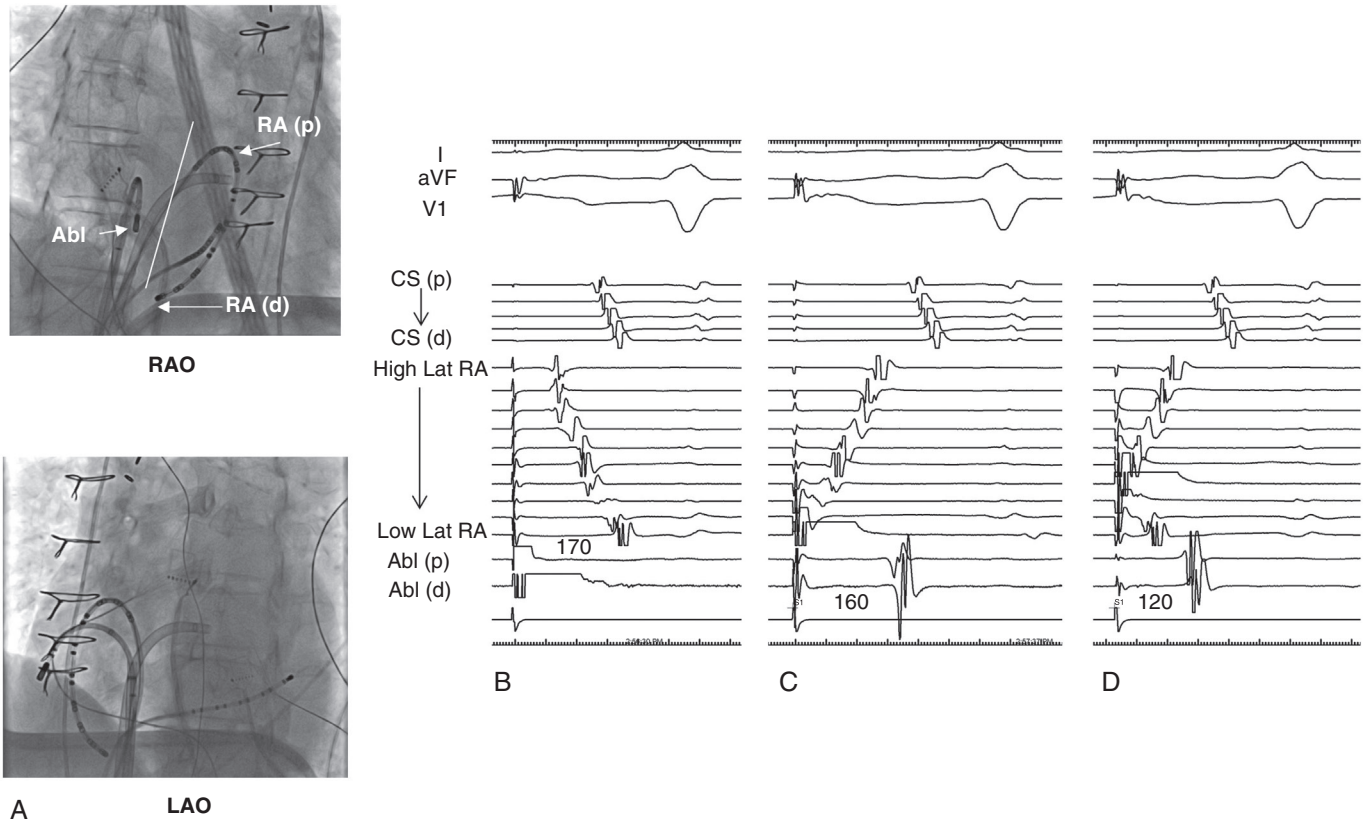


Fig. 12.11 Demonstration of bidirectional line of block in lateral wall of the right atrium (RA). The patient had incisional reentry around scar in the lateral RA. Ablation was performed between the scars in the lateral RA to the inferior vena cava. A, Fluoroscopic images in right and left anterior oblique show a multipolar catheter in the RA anterior to a line of block; an ablation catheter (Abl) in the lateral RA positioned posterior to the line of block, and a coronary sinus catheter (CS). B, During pacing from the ablation catheter, there is delay to the low right atrium (170 ms) with a descending wave front on the multipolar catheter, consistent with propagation around the superior end of the line and down the anterolateral wall. C, During pacing from the low lateral RA (RA 1-2) anterior to the line, there is a similar delay to the ablation catheter on the other side of the line (160 ms). D, Pacing higher on the anterolateral wall at RA 7–8 results in a shorter interval to the ablation catheter positioned posterior to the line (120 ms) because it is closer to the superior margin of the ablation line. *(d)* distal; *(p)* proximal; *lat,* lateral; *LAO*, left anterior oblique; *RAO*, right anterior oblique;

Fig. 12.12 Ablation lines for perimitral flutter. A, Lateral mitral isthmus: Ablation is performed from the lateral mitral annulus to an electrically isolated left inferior pulmonary vein. This lesion set is created lateral to the appendage. Ablation in the coronary sinus is often required to achieve line of block. In this case, *blue tags* represent endocardial ablation lesions, and the *orange tags* represent an ablation in the coronary sinus, which was required to achieve block in the lateral isthmus. B, Anterior mitral line: Ablation is performed from the anterior mitral annulus to a roof line or an isolated left superior pulmonary vein. This lesions set is created medial to the appendage *(shaded in blue).* C, Septal mitral line: Ablation is performed from the anterior or septal mitral annulus to an isolated right superior pulmonary vein.

in slow conduction and thus promote recurrent perimitral flutter. Achieving a line of block may require ablation within the CS with an irrigated-tip catheter.[52] Ablation within the CS is performed at power settings of 20 to 30 W with irrigation rates of 17 to 60 mL/minute. When an 8-mm-tip ablation catheter is used to create a line of block, initial power and temperature settings are 35 W and 50° C, respectively, and power and temperature can be gradually increased to a maximum of 70 W and 55° C, respectively. Verification of a line of block along the mitral isthmus can be demonstrated by pacing on either side of the ablation line; the CS catheter is useful for assessing activation lateral to the line (Fig. 12.13). In addition, the presence of double potentials along the line supports the existence of conduction block. A risk of ablation in the mitral isthmus, as with ablation in other locations in the LA, is cardiac tamponade, which can generally be avoided by limiting power to 42 W or less during endocardial ablation.[52]

Perimitral reentry can be also interrupted by creating a line of block in the anterior LA between the mitral annulus and the LA roof or the right pulmonary veins.[53] Some have reported that this line is more easily completed than ablation in the lateral mitral isthmus.[54] Conduction block can be verified by the demonstration of double potentials along

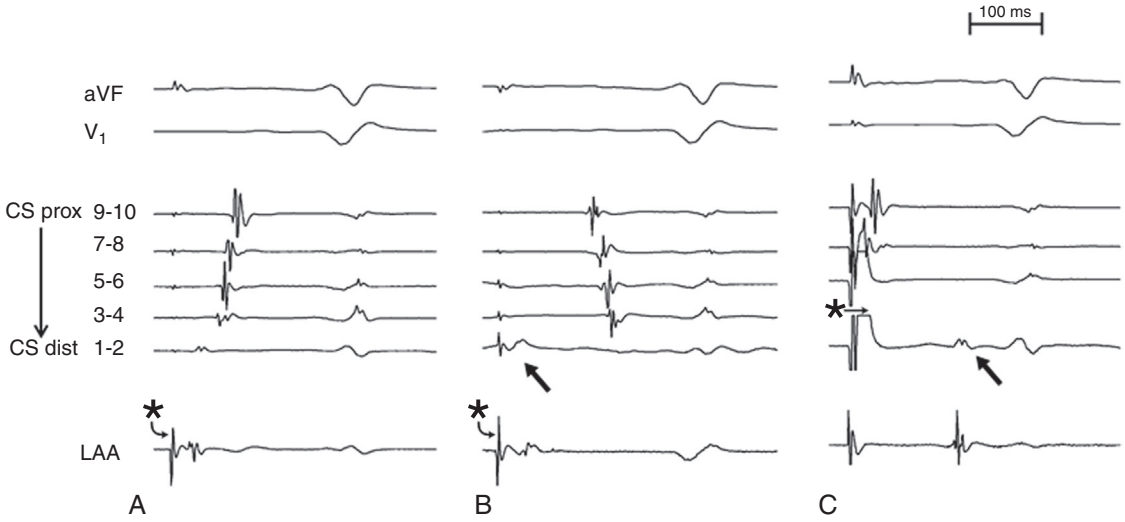


Fig. 12.13 Conduction block in the lateral mitral isthmus. A, Pacing *(asterisk)* is performed from the left atrial appendage (LAA) before the ablation line was made between coronary sinus (CS) 1–2 and CS 3–4. Pacing from the appendage activates the CS from distal to proximal. B, After the line is completed, conduction block is demonstrated by proximal to distal CS activation from CS 9–10 to 3–4. CS 1–2 is on the opposite (or anterior) side of the line *(arrow)*. C, To assess conduction in the opposite direction, pacing is performed from the distal CS, in this case poles 3–4. After the line is completed, there is late activation of the adjacent distal pole (CS 1–2, *arrow*) and the appendage.

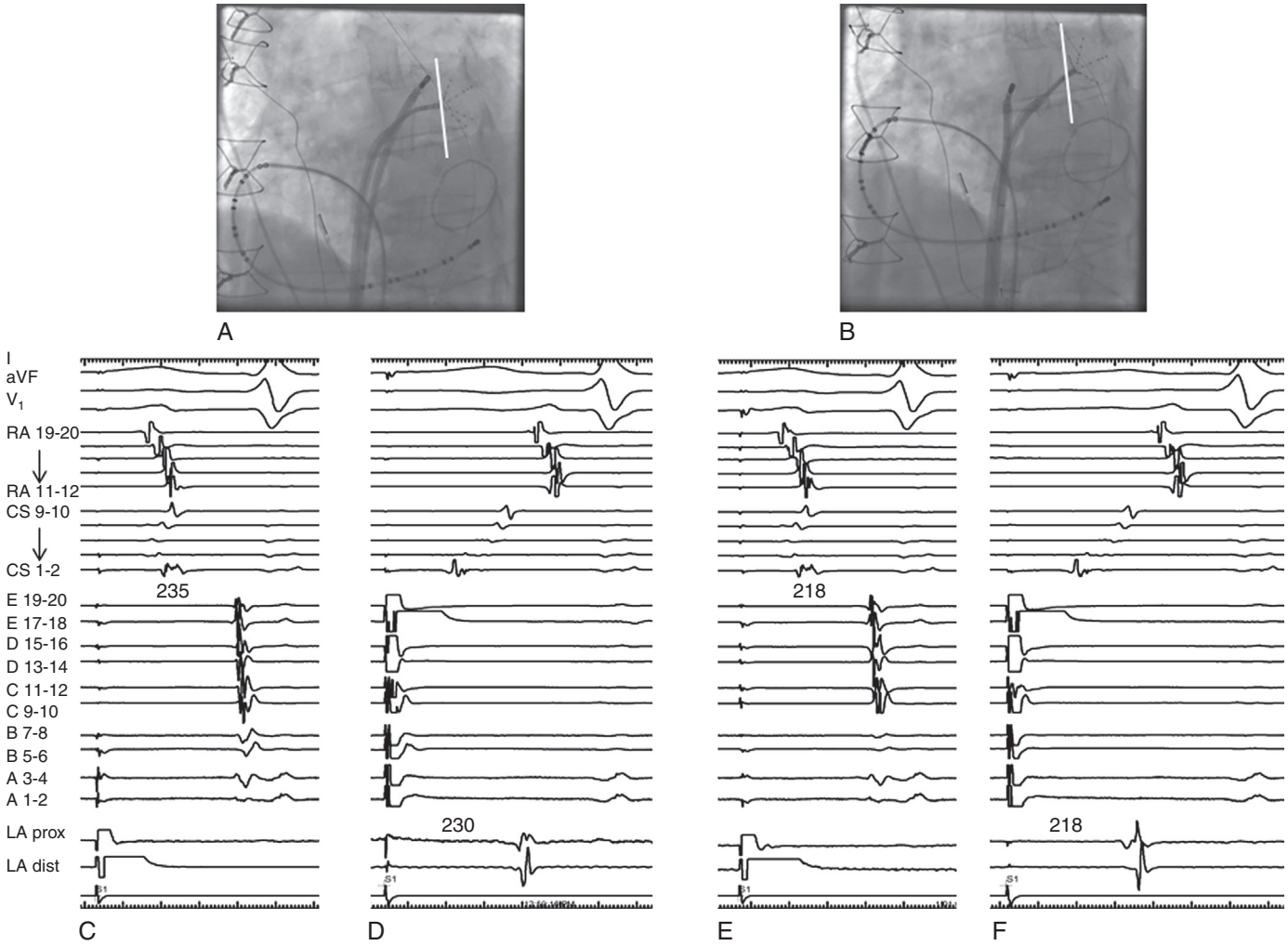


Fig. 12.14 Differential pacing to demonstrate line of block. An anterior mitral line was created to treat perimitral flutter. Fluoroscopic image (A) shows a Pentaray catheter in the left atrial appendage and an ablation catheter just medial to the ablation lesions, denoted by a *white line*. Pacing from the ablation catheter results in a delay of 235 ms to the appendage (C), and pacing from the appendage results in a similar delay of 230 ms of the ablation catheter on the medial side (D). When the ablation catheter is moved more medially and away from the line (B), there are shorter conduction times between the ablator and the appendage in both directions (E and F).

the line during pacing adjacent to the line. To distinguish slow conduction from complete block, the technique of differential pacing may be used, which involves measuring conduction times between two catheters positioned just across the ablation line and then moving a catheter further away from the line (Fig. 12.14).

Roof-dependent macroreentry is usually treated with linear ablation along the LA roof between the left and right superior pulmonary veins. Completeness of the line can be demonstrated by recording widely spaced double potentials along the ablation line during pacing from one side of the line.[55] The LA appendage may also be selected as a stable pacing site in the anterior segment of the LA. Activation of the posterior wall during appendage pacing should proceed from an inferior to superior direction as the impulse diverts around the pulmonary veins to engage the posterior wall (Fig. 12.15). Analogously, pacing from the posterior wall should result in late activation of the anterior segment, including the appendage.

Patchy areas of scar in the LA occur in a variety of disease conditions, including rheumatic valve disease, hypertrophic cardiomyopathy, and other atrial myopathies. Areas of low voltage consistent with scar have been identified in various locations, such as the anterior LA and posterior wall.[3,29,56] These areas of patchy scar create the substrate for single and dual-loop macroreentry. Approaches that involve ablating a slow conducting critical isthmus have been successful in interrupting these arrhythmias. Yet in patients with extensive scarring, such as those with rheumatic valve disease, AF commonly occurs during long-term follow-up.[56]

Left Septal Flutter

Reentry around the septum primum has been recognized as an uncommon mechanism of LA flutter in the absence of previous cardiac surgery and mostly occurs in patients with AF treated with antiarrhythmic drugs.[14,57] Left septal flutter also occurs in patients with prior open-heart surgery, typically mitral valve surgery that involves a

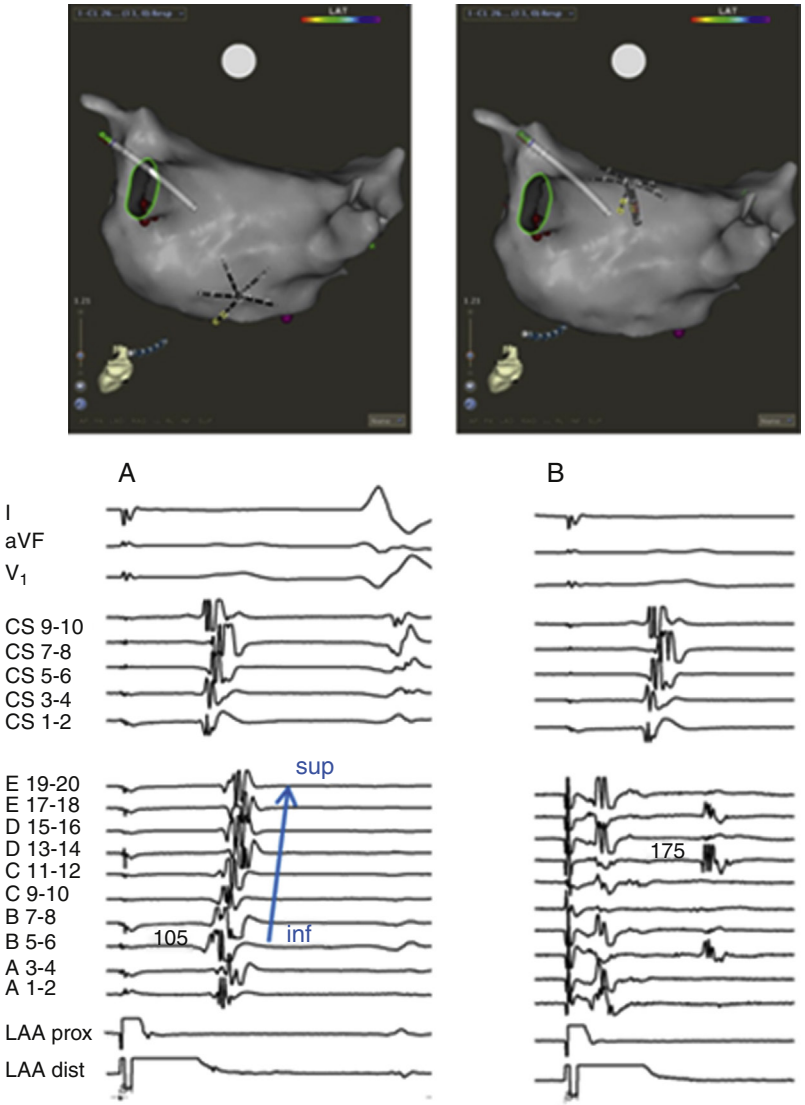


Fig. 12.15 Demonstrating line of block in the left atrial roof. A, Pacing is performed from the ablation catheter, which is positioned in the left atrial appendage (LAA). A PentaRay catheter positioned in the low posterior left atrium demonstrates earliest activation in the most inferior electrodes (inf) and propagation to the superior electrodes (sup). B, When the PentaRay is positioned in the anterior roof along the ablation line, straddling the roof ablation line, double potentials are demonstrated, with the second component activated later than any recording lower on the posterior wall as well as those recorded on the coronary sinus (CS) catheter. *dist,* Distal; *prox,* proximal.

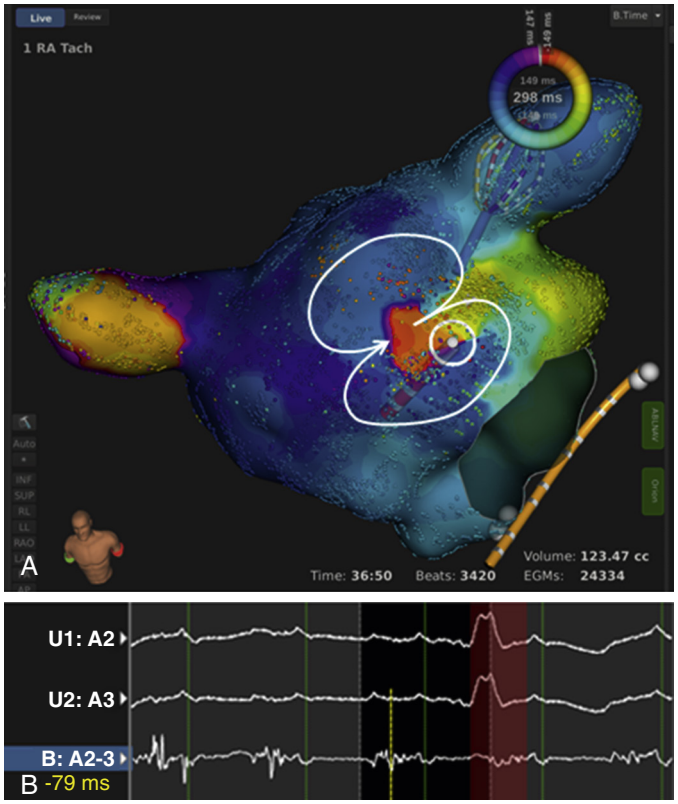


Fig. 12.16 Localized reentry in the left atrium: A, Ultrahigh-density map of the left atrium shows a dual-loop tachycardia confined to the anterior wall. B, An electrogram from the narrow isthmus is low amplitude, fragmented, and long duration (B: A2-3). Successful ablation was performed at the site of this abnormal electrogram. See associated Video 12.3.

left atriotomy.[58] The reentrant circuit is bounded by the right pulmonary veins posteriorly and the mitral annulus anteriorly, with double potentials defining a line of conduction block. The electrocardiogram shows positive or negative flutter waves in V_1 and low-amplitude flutter waves in the other leads. The ablation strategy involves creating a line between the septum primum and the right pulmonary veins, or between the septum primum and the mitral annulus. Thick septal tissue may prevent the creation of transmural lesions and account for a higher likelihood of recurrent flutter ablation.

Microreentrant Atrial Tachycardias

Small reentrant circuits can occur in myopathic right or left atria or after catheter or surgical ablation of AF. A characteristic feature of localized reentry or microreentry is the presence of low-amplitude, long-duration, fractionated electrograms that occupy a small region of the atrium (see Fig. 12.9).[10] Multielectrode catheters with closely spaced electrodes are useful for localizing the arrhythmia and identifying abnormal electrograms. (see Fig. 12.9). The catheter may record adjacent sites with complex electrograms that span most or all of the tachycardia cycle length.[59] Activation of the atria proceeds passively from this highly fractionated location. Electroanatomic maps will usually demonstrate a centrifugal activation pattern, but it may be difficult to assign activation times for highly fractionated points, which can make electroanatomic map difficult to interpret. Ultrahigh-resolution mapping with automated annotation has proved useful in delineating complex circuits with long-duration electrograms (Fig. 12.16).[27,28] Activation mapping can be supplemented with entrainment to localize these small circuits. Entrainment at or near the location of the small circuit will yield a postpacing interval within 30 ms compared with the tachycardia cycle length, whereas entrainment at increasing distance from the circuit results in a gradient of increasing postpacing intervals. Another characteristic of these arrhythmias is insensitivity to adenosine, which supports a reentrant mechanism rather than triggered activity or automaticity.[7] Focal ablation at sites with complex electrograms is often effective in terminating these arrhythmias (Video 12.3).

Atypical Flutter After Catheter Ablation for Atrial Fibrillation

LA macroreentrant AT may present as a late complication in less than 5% to 30% of patients who undergo catheter ablation for AF.[42,43,60–65] This complication is less common after segmental pulmonary vein isolation, cryoballoon ablation, and multielectrode phased-RF ablation compared with wide encircling antral ablation using unipolar RF catheters. Even higher rates of AT occur after procedures that add additional substrate modification techniques, such as linear ablation in the LA, ablation of complex fractionated electrograms, and targeting of rotational activity.[40,66–68]

A deductive stepwise approach using activation and entrainment mapping can be used to determine the mechanism of the AT and its location.[11] This strategy is based on the recognition that LA macroreentry after AF ablation is often as a result of either perimitral reentry or roof-dependent reentry, and basic activation and entrainment mapping can identify or exclude these circuits. First, cycle length variation is assessed, and variations of 15% or more should prompt a search for a focal tachycardia. Before transseptal catheterization, CTI-dependent flutter should be excluded. Pulmonary vein isolation is confirmed, and reisolation is performed if necessary. Activation mapping around the mitral annulus, supplemented by entrainment mapping from opposite segments (e.g., septal and lateral) is performed to assess for perimitral reentry. Similarly, activation and entrainment mapping of the posterior and anterior LA walls are performed to assess for a roof-dependent flutter. Gaps in prior ablation lines often demonstrate low-amplitude fragmented electrograms.

Between 20% and 40% of ATs that occur after catheter ablation of AF arise from focal sources.[10,42,43,62] The vast majority of these focal tachycardias are thought to be as a result of small reentrant circuits. These arrhythmias typically occur near previous ablation lines or may be related to gaps or inhomogeneities in the ablation lines. On occasion a reconnected pulmonary vein may be the source of an organized AT. If a macroreentrant mechanism is excluded through a combination of activation and entrainment mapping, a search should be conducted for a focal origin of the tachycardia. The CS activation can direct mapping to the region of interest. For example, if CS activation is distal to proximal, the focus may arise from the left pulmonary veins or lateral LA, whereas proximal to distal activation implicates a focus near the right pulmonary veins or the RA. Sites of successful ablation typically demonstrate long-duration, low-amplitude, fractionated electrograms.

For ATs that occur after catheter ablation of AF, freedom from recurrent AT or AF during follow-up is between 73% and 88% after one procedure,[40–43] and higher long-term success rates are attainable if patients have repeat procedures for recurrent arrhythmias.

Atypical Flutter After Maze Surgery

Macroreentrant AT may occur late after maze surgery for AF because of gaps in the operative lesions, similar to the situation described for arrhythmias after catheter ablation.[69–76] If surgical lesions are limited to the LA, typical RA flutter occurs in up to 10% of patients late after surgery. RA reentrant circuits can also occur related to cannula locations or gaps in the RA ablation lines. An example of a dual-loop tachycardia in the RA after maze surgery is shown in Fig. 12.17. In the LA, gaps in the posterior wall lesions and in connecting lesions to the mitral annulus have been described, resulting in roof-dependent and perimitral

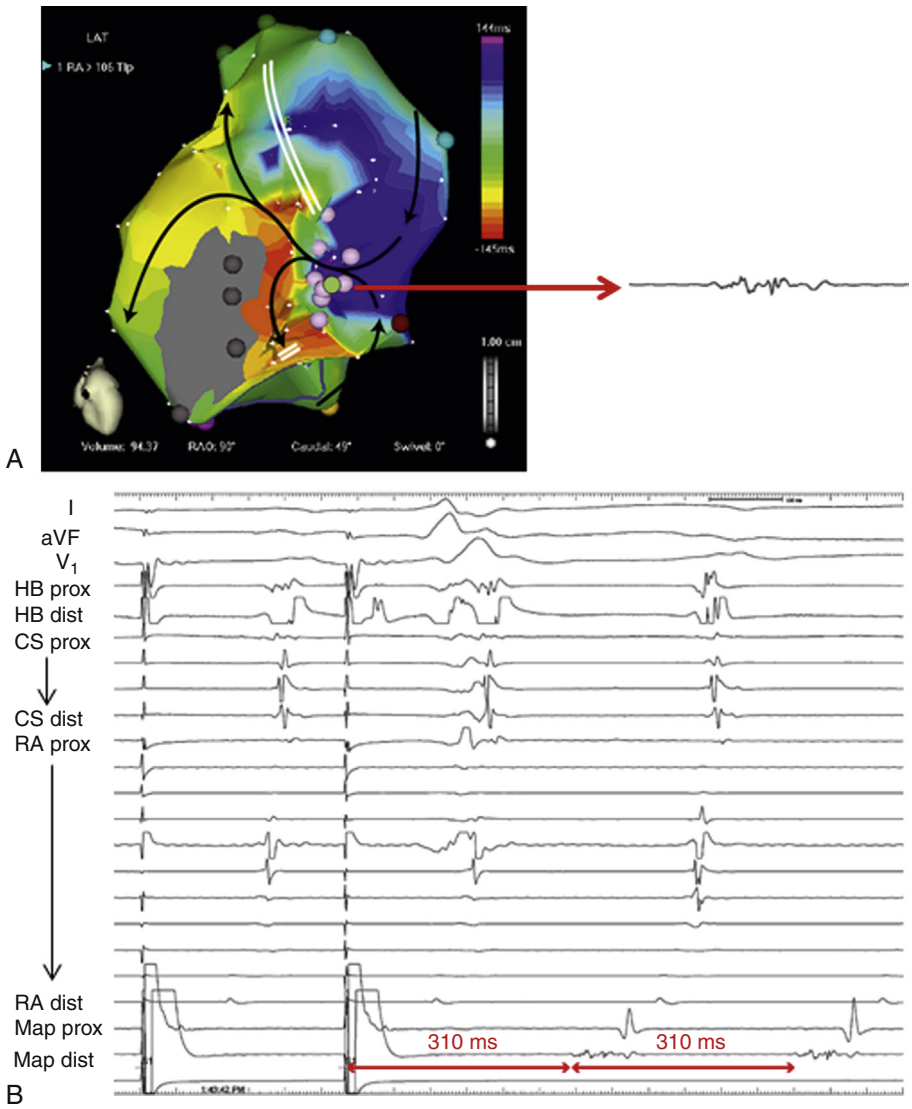


Fig. 12.17 Reentrant atrial tachycardia following maze surgery. This patient had surgical cryoablation in the cavotricuspid isthmus, an intercaval line, and a line between the right atrial (RA) appendage and lateral RA, in addition to left atrial (LA) lesions. A, RA electroanatomic activation map in right lateral caudal view, which demonstrates a line of block in the lateral wall with double potentials *(pink tags)*. A gap in this line served as a critical isthmus for the reentrant tachycardia. B, The electrogram at this site *(green tag)* was low amplitude and fractionated. Concealed entrainment from this site indicated participation in the tachycardia circuit (post-pacing interval 310 ms, identical to the tachycardia cycle length). Note the block in the cavotricuspid isthmus because of prior surgical ablation, thus preventing dual-loop tachycardia. *CS,* Coronary sinus; *HB,* His bundle; *IVC,* inferior vena cava; *Map,* mapping catheter; *TA,* tricuspid annulus

reentry.[69,72,75,76] Reconnection of the pulmonary veins also accounts for recurrences after surgical ablation of AF, particularly if surgery involved the use of unipolar RF energy.[73,76] It is not clear whether these gap-related flutters occur more commonly with a particular surgical modality (e.g., conventional incisions, bipolar RF, cryoablation, or microwave energy). Although some evidence suggests that gaps are less likely to occur across cut-and-sew lesions, reentrant arrhythmias caused by incomplete lines of block have been documented after this approach. Catheter ablation of gap-related atrial flutter after maze surgery is feasible, using the same mapping techniques for other complex atrial reentrant circuits.

Atypical Flutter After Mitral Valve Surgery

Atypical flutters occur in both the RA and LA after mitral valve surgery.[8,29,30,77] The substrate for these arrhythmias involves atriotomy incisions, as well as intrinsically diseased myocardium, giving rise to anatomic and functional regions of block as well as slowed conduction. Macroreentry is the predominant mechanism, and circuits often localize to the RA, including typical CTI-dependent flutter.[77] Reentry in the RA may be attributed to several factors, including surgical approaches to the mitral valve that involve RA incisions (such as the transseptal or superior septal approaches), cannula insertion in the RA, and underlying myocardial disease. Tachycardias in the RA are single- and dual-loop circuits that involve the RA free wall. The superior septal approach to the mitral valve, which involves an incision along the lateral RA extended to the interatrial septum and dome of the LA, is a particular situation that predisposes to reentry in the lateral RA. Both descending and ascending wave fronts can be identified in the lateral RA, representing reentry around the lateral atrial incision (see Fig. 12.6).

Participation of both limbs can be confirmed with entrainment. As with ablation of other forms of lateral RA reentry, an ablation line can be made between the lateral tricuspid annulus and the atriotomy. Alternatively, ablation can be performed between the atriotomy and the IVC or SVC. Often, a second loop exists around the tricuspid annulus, which may require ablation in the CTI. Reentry involving scar in the RA side of the septum has also been reported after transseptal access of the mitral valve.[78] In these cases, ablation can be accomplished by creating lesions between the septal scar and the tricuspid annulus.

In the LA, areas of low voltage are often identified anterior to the right pulmonary veins, which correspond to LA incisions. Roof-dependent AT may be present with reentry around the right pulmonary veins (involving this zone of low voltage), but other circuits may involve a posterior scar or the mitral annulus. Left atrial flutters are more common if mitral valve surgery is combined with a maze procedure.[77]

Atypical Flutters After Orthotopic Cardiac Transplantation

The late development of AT after cardiac transplantation may be a sign of acute rejection, but such arrhythmias also occur in less than 5% of stable patients.[79] The most common atrial arrhythmias presenting late after transplant are macroreentrant AT, but focal arrhythmias are also documented.[79–82] CTI-dependent flutter accounts for 30% to 60% of these arrhythmias. The flutter morphology of CTI-dependent tachycardias posttransplantation is usually atypical. A variety of other atypical focal and macroreentrant circuits can occur in these patients, and many involve scar or suture lines for the atrial anastomoses. A unique form of arrhythmia in transplant patients is related to conduction from recipient to donor atria across the atrial anastomoses. In cases of biatrial anastomoses, recipient to donor conduction can potentially occur in either the RA or LA. In cases of bicaval anastomoses, no recipient atrial myocardium exists in the RA, but the recipient LA can develop a connection to the donor LA. In some cases, tachycardia originating from a recipient atrium conducts to the donor atria. These tachycardias can be targeted by identifying the breakthrough site of recipient to donor conduction as the site of earliest activation in the donor atrium. Ablation at this site can dissociate the recipient and donor atria and prevent tachycardia in the donor heart. Macroreentrant circuits can also occur that involve isthmuses created by the anastomotic suture lines and other conduction barriers, such as the mitral annulus. Overall, organized ATs appear to arise more commonly from the RA than the LA of transplanted hearts. Catheter ablation of atrial tachyarrhythmias in this population is acutely successful in more than 90% of cases, although repeat ablation may be required in up to 20%.[82]

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Videos

Video 12.1 Upper-loop reentry. Ultrahigh-density maps of upper-loop reentry in the right atrium obtained with the Rhythmia system in a patient with atypical flutter after repair of an atrial septal defect. Left panel shows a right posterior oblique projection, and a wave front is seen to propagate slowly across the posterolateral wall and around the superior vena cava. Right panel (left anterior oblique projection). As the wave front emerges from the posteroseptal wall it descends the right atrial free wall lateral to the tricuspid annulus. The circuit therefore uses the superior vena cava and scar in the posterolateral wall as a central barrier.

Video 12.2 Dual loop right atrial reentry. Left panel is a right posterior oblique projection of an ultrahigh-density map showing dual loop reentry with a narrow isthmus in the posterolateral right atrium. One loop propagates around the superior vena cava, and a second loop propagates around the lower patch of scar. Right panel is a left anterior oblique projection that shows passive activation around the tricuspid annulus.

Video 12.3 Ablation of localized reentry in the left atrium. The movie shows ablation of tachycardia depicted in Fig. 12.16, a small dual loop tachycardia confined to the anterior wall of the left atrium. During focal ablation in the narrow isthmus, the tachycardia terminates.