Proarrhythmic Aspects of Atrial Fibrillation Surgery
Mechanisms of Postoperative Macroreentrant Tachycardias

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Background—Surgery for the treatment of atrial fibrillation may be associated with early or late postoperative atrial arrhythmias. In many cases, the arrhythmias that occur early in the postoperative course may be related to pericardial inflammation or increased catecholamine levels and tend to resolve without further therapy. In contrast, late postoperative arrhythmias often are persistent, highly symptomatic, and refractory to medical therapy. The incidence and mechanisms of these late atrial arrhythmias have not been thoroughly described.

Methods and Results—In this study, a total of 143 consecutive patients undergoing atrial fibrillation surgery from 1996 to 2005 were prospectively followed up. Sustained atrial tachycardia developed in 22 of 143 patients (15%) >8 weeks after the surgical procedure. These patients underwent electrophysiological study. A total of 25 separate tachycardias were mapped in these 22 patients, including 15 located in right atrium and 10 in the left atrium. Right atrial tachycardias included cavotricuspid isthmus–dependent atrial flutter (n = 7), non–isthmus-dependent right atrial reentry (n = 7), and 1 focal atrial tachycardia. Left atrial tachycardias included reentry around the mitral valve (n = 3) and in the roof of the left atrium (n = 7). All tachycardias were successfully treated with radiofrequency catheter ablation with no recurrences in long-term follow-up.

Conclusions—Postoperative arrhythmias are not uncommon late after surgery for atrial fibrillation and typically are reentrant and related to surgically created boundaries. Radiofrequency catheter ablation is highly effective in the management of these late arrhythmias. (Circulation. 2008;117:155-162.)

Key Words: ablation ■ arrhythmia ■ electrophysiology ■ fibrillation ■ surgery

Surgical operations for atrial fibrillation, although highly effective, are not uniformly so. Postoperative arrhythmias may include transient and self-limited or sustained atrial fibrillation or regular tachycardias related to macroreentry or focal mechanisms. Other than typical atrial flutter, the most commonly reported reentrant tachycardia after surgery for atrial fibrillation involves the coronary sinus musculature to support reentry around the mitral valve annulus. Recent reports of tachycardias after atrial fibrillation operations have included several arrhythmias that were incompletely mapped, often leaving the reentrant circuit undefined. Surgical boundaries, if incomplete, may promote iatrogenic reentrant circuits. Complete lesions, however, also could create an environment for macroreentrant circuits that depend on tissue with anisotropic conduction properties if a functional central obstacle is protected by surgical boundaries. In this case, the central functional barrier may provide a zone of slow conduction when anatomic boundaries have been surgically created.

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In this report, we describe the spectrum of tachycardias other than atrial fibrillation that are present late after surgery for atrial fibrillation, all of which were completely mapped and successfully ablated. We describe a reentrant tachycardia in the roof of the left atrium that may use Bachmann’s bundle or a region of sparse myocardial fibers as a central, slowly conducting functional barrier.

Methods

Study Population

The records of all patients who were treated with atrial fibrillation surgery at the University of Alabama at Birmingham from 1996 to 2005 were reviewed. All patients were seen in the clinic by the authors to evaluate cardiac symptoms and 12-lead ECG at 2 and 8 weeks postoperatively. Transient atrial arrhythmias occurring in the first 8 weeks were treated with atroventricular nodal blocking medications and were excluded from analysis. All patients experiencing palpitations after the 8-week blanking period were monitored intensively with 24-hour Holter monitors or transtelephonic event

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Either cryoablation using a 1-cm probe at the site near the left superior pulmonary vein. Thus, the remaining atrial appendage incision was made from the superior vena cava to the SVC posterior left atrium around the pulmonary veins using the left atrial groove (dashed lines). Radiofrequency lesions were created to extend from the left atrial surgical incision to encircle the pulmonary veins (PV) and join the pulmonary vein–encircling line to the mitral valve (MV) annulus and base of the left atrial appendage (LAA; dotted lines). The left atrial appendage was oversewn endocardially (hatched line). Cryoablation was used to ablate the cavotricuspid isthmus in the right atrium and the coronary sinus (CS) musculature along the mitral valve annulus and the base of the left atrial appendage (hatched circles). TV indicates tricuspid valve; RAA, right atrial appendage.

 recordings. If arrhythmias other than atrial fibrillation persisted for >8 weeks postoperatively, invasive electrophysiological testing was recommended.

Surgical Operation
All patients had preoperative atrial fibrillation and underwent a modified Cox Maze III operation performed by a single surgeon (D.C.M.) as described by Cox and colleagues.1–3,12 Surgical boundaries to electrical conduction were created by the combined use of surgical incisions, radiofrequency ablation, and/or cryoablation (Figure 1). The initial surgical dissection consisted of extensive mobilization of both caval veins and the roof of the left atrium. After bicaval cannulation and total cardioplegia, the heart was arrested, and the patient was supported by cardiopulmonary bypass. First, an intercalvar incision was made from the superior vena cava to the inferior vena cava posteriorly through the smooth portion of the right atrium. The left atrium was then opened by a vertical incision in Waterston’s groove. An encircling incision was then made in the posterior left atrium around the pulmonary veins using the left atrial antrum as its medial boundary. In the second half of the series, endocardial radiofrequency current was used to encircle the pulmonary veins and to create the right atrial intercaval barrier (Medtronic CardioAblate, Minneapolis, Minn). An incision or radiofrequency lesion was then made to join the pulmonary vein–encircling barrier to the posterior mitral valve annulus. The left atrial appendage was obliterated rather than amputated by endocardial oversewing at its base with cryoablation applied to electrically isolate this structure. The cavotricuspid isthmus, if treated, was ablated with either cryoablation using a 1-cm probe at −80°C or radiofrequency ablation. The surgical barrier extending from the pulmonary vein–encircling lesion to the mitral annulus was completed by applying cryoablation to the mitral annulus.

Electrophysiological Studies
All patients were evaluated in the Arrhythmia Clinic at the University of Alabama at Birmingham 8 weeks postoperatively. Patients who developed regular atrial tachycardias that persisted for >8 weeks underwent invasive electrophysiological study and catheter ablation in the fasting state with intravenous sedation. Multielectrode catheters were inserted percutaneously into the coronary sinus and across the tricuspid annulus to record His bundle activation. A deflectable, 7F, 20-electrode catheter (Irvine Biomedical, Irvine, Calif) was positioned around the tricuspid annulus. When the reentrant circuit involved the left atrium, transseptal catheterization was performed using standard techniques with biplane fluoroscopy. Rapid atrial pacing was performed at multiple sites in both atria at a cycle length that was 20 ms less than the tachycardia cycle length to transiently entrain the tachycardia with analysis of the postspacing interval at the pacing site. Sites where the postspacing interval minus the tachycardia cycle length was <20 ms were deemed to be within the tachycardia circuit. Rapid atrial pacing was performed at multiple sites in both atria to attempt to identify regions of the atria within and distant from the tachycardia circuit.

Mapping and Localization of Critical Isthmus
Electroanatomic maps also were created in most cases to further define the reentrant circuit (Carto, Biosense-Webster, Diamond Bar, Calif, or RPM, Boston Scientific, Natick, Mass). Entrainment mapping was relied on to localize the critical isthmus of tissue to be targeted for catheter ablation. Optimal ablation sites were those with a postspacing interval within 20 ms of the tachycardia cycle length at which fractioned electrograms were recorded. Areas with fractionated electrograms at which the postspacing interval exceeded the tachycardia cycle length by >20 ms were not ablated. Once a suitable ablation site was recorded, radiofrequency current was applied using a 4- or 5-mm-tip catheter (nonirrigated) in the temperature-controlled mode using a maximum power of 50 W and a target temperature of 60°C.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results
All patients had a preoperative history of atrial fibrillation, which was permanent in 15 patients, persistent in 5 patients, and paroxysmal in 2 patients. This proportion is similar to the entire surgical population in which atrial fibrillation was permanent in 105 of 143 patients.

Atrial Fibrillation Recurrence
Atrial fibrillation occurred or persisted after the 8-week blanking period in 10 of 143 patients (7%). Atrial fibrillation was targeted for catheter ablation in only one of these patients. In this patient, 3-dimensional electroanatomic mapping demonstrated persistent conduction from the posterior left atrium within the encircled pulmonary vein region at a site near the left superior pulmonary vein. Thus, the remainder of the left atrium was activated from this gap in the radiofrequency ablation line. This conduction gap was ablated with endocardial application of radiofrequency current, resulting in isolation of the pulmonary vein region and termination of atrial fibrillation. This patient has been free of recurrent atrial fibrillation for >18 months with no antiarrhythmic medications. The remaining 9 patients with late atrial fibrillation were treated with antiarrhythmic drug therapy (8 patients) or atrioventricular nodal ablation and permanent pacemaker implantation (1 patient).

Atrial Tachycardia Incidence
All other late postoperative arrhythmias were regular tachycardias and were based on atrial macrorreentrant (n=24) or focal (n=1) mechanisms. The technique for detecting
arrhythmias was a routine 12-lead ECG alone in 19 patients, a Holter monitor and an ECG in 1 patient, and transtelephonic event recording and an ECG in 2 patients.

The demographics of the patients who developed sustained regular tachycardia, including their age, sex, ejection fraction, structural heart disease, and concomitant mitral valve surgery, are detailed in Table 1. In the entire series of patients, 22 of 143 (15%) developed a sustained, regular postoperative atrial tachycardia (total of 25 distinct tachycardias) at least 8 weeks postoperatively. Fifteen of these circuits were localized to the right atrium, and 10 were localized to the left atrium. The summary of these arrhythmias, including the successful ablation site, tachycardia cycle length, and fluoroscopic exposure, is given in Table 2.

Mechanisms of Right Atrial Tachycardias

Right atrial arrhythmias included counterclockwise cavotricuspid isthmus–dependent atrial flutter in 7 patients. Macroreentrant right atrial arrhythmias that did not use the cavotricuspid isthmus were identified in 7 patients. For 3 right atrial tachycardias, the reentrant circuit involved a conduction gap at the junction of the superior vena cava and the right atrium along the superior margin of the posterior intercaval incision. Two tachycardias were based on reentrant circuits within the free wall of the right atrium in which the crista terminalis served as a central, functional obstacle. Ablation was targeted to the region of the crista terminalis demonstrating transcristal conduction. The number of entrained sites varied greatly between patients. The patients with reentry within the free wall of the right atrium all had highly fractionated potentials (spanning >30% of the tachycardia cycle length) with a stimulus-to-P-wave interval >30% of the tachycardia cycle length and a postspacing interval within 20 ms of the tachycardia cycle length. Two tachycardias were ablated in the interatrial septum. The ablation site was between the posterior limbus of the fossa ovalis and the right atrial intercaval line. A focal mechanism was mapped to the lateral right atrium for 1 tachycardia that could not be entrained.

Mechanisms of Left Atrial Tachycardias

Left atrial reentrant tachycardias were recorded in 10 patients. In 9 of 10 patients, the posterior left atrium inside the pulmonary vein–encircling surgical boundary was electrically isolated from the remainder of the atria. The 1 exception was a patient who had postoperative atrial fibrillation and left atrial macroreentry. In this patient, the gap in the posterior pulmonary vein–encircling barrier was ablated near the left superior pulmonary vein. Reentry around the mitral annulus accounted for 3 of the left atrial tachycardias. The number of entrained sites required to identify this mechanism was relatively few (4 to 6), and there was typically no discrete

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### Table 1. Patient Characteristics

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LVEF indicates left ventricular ejection fraction; LA, left atrium; and MV, mitral valve.

### Table 2. Arrhythmia Characteristics

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LA indicates left atrial; RA FW, right atrial free wall; CTI, cavotricuspid isthmus; IAS, intraatrial septum; and SVC-RA, superior vena cava–right atrial junction.
region of slow conduction identified. The site of ablation for these left atrial tachycardias was between the pulmonary vein–encircling surgical boundary and the mitral valve annulus. In addition, ablative energy was applied within the coronary sinus in these patients. Bidirectional block across the line was demonstrated in all patients by pacing of the coronary sinus and left atrium proximal and distal to the ablation site and by electroanatomic mapping of the left atrium. Interestingly, the surgical operation had targeted this region, with a surgical boundary line created in this same zone with a cryoablative lesion given at the mitral valve annulus that was designed to interrupt the musculature of the coronary sinus. Thus, in these patients, the postoperative macroreentrant tachycardia represented a failure of the surgical strategy.

The remaining 7 left atrial tachycardias were mapped to the roof of the left atrium. The surface ECG of these tachycardias was characterized by high-amplitude, upright flutter waves in leads V1 and V2 and the inferior limb leads (II, III, and aVF), with negative flutter waves in aVL (Figure 2). In all of these cases, a region of slow conduction was identified in the roof of the left atrium (Figure 3). These tachycardias were successfully ablated in the roof of the left atrium at the site of highly fractionated electrograms (Figures 4 and 5). In each case, the tachycardia was successfully ablated in the superior left atrium in a critical isthmus of tissue that recorded middiastolic (relative to the surface flutter waves) fractionated potentials and where the postpacing interval was within 10 ms of the tachycardia cycle length. The tachycardia slowed and terminated in all patients by a limited number of radiofrequency applications (1 to 2) at sites with these electrogram characteristics and response to pacing, suggesting that the isthmus was a relatively discrete and anatomically limited region. The stimulus-to-surface-P-wave interval was greater when entraining posterior to the expected location of Bachmann’s bundle than when the catheter tip was moved more anteriorly. Linear ablation to the posterior pulmonary vein–encircling line or the mitral valve annulus was not required.

Follow-Up and Efficacy of Strategy

After catheter ablation, all patients have remained free of recurrent arrhythmias over a period of 38±17 months. No patient undergoing catheter ablation has required antiarrhythmic medications or has developed atrial fibrillation in late follow-up.

Discussion

Surgical operations provide an effective, nonpharmacological treatment for atrial fibrillation that can be combined with the surgical repair of a variety of cardiac conditions. Although transient and self-limiting atrial arrhythmias are common in the early postoperative period, atrial fibrillation surgery may be followed by late reentrant arrhythmias that often are persistent and refractory to medical therapy. These arrhythmias may occur more commonly than previously appreciated and typically are based on a macroreentrant mechanism. The reentrant circuit may be located within either atria and often is related to persistence of electrical conduction through regions that were targeted for surgical ablation, a gap at the margin of a surgical incision as it joins an anatomic boundary, or the creation of a central anatomic obstacle to conduction. In each of these mechanisms, a stable macroreentrant circuit was created that was maintained by protected anatomic boundaries. In some cases, the reentrant circuit involved a region of incompletely ablated tissue such as the musculature of the coronary sinus or the junction of the intercaval right atrial incision and the superior vena cava. However, in other cases, the creation of surgical barriers may have allowed reentry to occur around a central obstacle that was functional (either the crista terminalis in the right atrium or Bachmann’s bundle in the left atrium).

Surgery for atrial fibrillation seems to produce a mechanism of reentrant tachycardia in the roof of the left atrium. This mechanism may use Bachmann’s bundle as a central, functional obstacle. The region of slow conduction in this circuit was, as expected, Bachmann’s bundle. This structure could serve to create a zone of slow conduction transverse to the long axis of this structure. This structure is organized as...
a thickened area of ordinary atrial myocardium with myofi-
bers oriented to rapidly conduct from the right atrium to the
left atrium. It has been shown to have rapid conduction along
its long axis and much slower transverse conduction.\textsuperscript{20–24} As
demonstrated by Spach et al,\textsuperscript{25} premature depolarizations may
block in the longitudinal axis while conducting slowly in the
transverse direction in canine cardiac structures such as the
crista terminalis or pectinate muscles. These authors demon-
strated that the safety factor for conduction is lowest when the
conduction velocity is highest.\textsuperscript{26} Such conditions would be
expected to be present in both the crista terminalis and
Bachmann’s bundle. Reentry based on nonuniform anisot-
ropy may result.\textsuperscript{26} The region of slow conduction in each of
our patients with reentry in the roof of the left atrium
appeared to be oriented in the posterior-anterior direction
across the expected location of Bachmann’s bundle, trans-
verse to its long axis. In these patients, the reentrant circuit
was protected by the mitral valve anteriorly, by the pulmo-
nary vein–encircling incision posteriorly, and by the left
atrial appendage lesion laterally. In each case, the tachycardia
could be interrupted and rendered noninducible by a rela-
tively discrete application of radiofrequency current to a site
where Bachmann’s bundle was anticipated to be located and
where long duration and fractionated potentials were
recorded.

It should be emphasized that there is at least 1 other
potential mechanism for reentry in the roof of the left atrium.
The roof of the left atrium immediately anterior to Bach-
mann’s bundle has relatively sparse myocardial fibers.\textsuperscript{27} This
paucity of fibers could provide the substrate for slow con-
duction across the left atrial roof and serve as a critical
isthmus for reentry.

In the right atrium, reentry often used the crista terminalis
as a functional central obstacle. When the crista terminalis was
the central functional obstacle, the tricuspid annulus served as a
boundary anteriorly, and the intercaval incision served as a
boundary posteriorly. In these cases, the region of slow con-
duction appeared to be transverse conduction across highly aniso-
 trope atrial myocardium.\textsuperscript{25,28–31} Thus, tachycardias in either
atrium may be dependent on the anisotropic conduction
properties of a longitudinally oriented bundle of atrial myo-
cardium that normally serves to enhance intraatrial or inter-
atrial conduction.\textsuperscript{20–25,28–31}
Arrhythmia Mechanisms and Their Surgical Implications

The 3 major mechanisms of macroreentrant tachycardias identified in this series have led to strategies designed to prevent these arrhythmias. First, because incomplete conduction block may result in cavotricuspid isthmus-dependent atrial flutter or reentry around the mitral valve annulus, great care must be taken to ensure that all conducting tissue within the surgical lines is completely eliminated. In each of our patients with these reentrant mechanisms, the gap occurred in a cryoablation lesion. Cryoablation has the advantage of protecting the cellular interstitial matrix of cardiac tissue but may be associated with reversible effects on myocardial conduction that are difficult to recognize immediately after ablation. Because only cryoablation was used at the cavotricuspid isthmus and at the mitral annulus targeting the coronary sinus musculature, no firm conclusions can be made regarding the relative efficacy of this ablative technique. Nevertheless, the macroreentrant arrhythmias around the mitral valve annulus and tricuspid valve annulus represent a failure of this surgical lesion to create lasting conduction block.

The second mechanism involved a linear lesion that did not extend to an anatomic boundary, leaving a small zone of atrial myocardium that remained capable of conduction. This was seen with right atrial tachycardias using the intercaval incision in which the cranial aspect of the incision did not extend far enough into the superior vena cava. Atrial myocardium can extend several centimeters into the superior vena cava. In these cases, the gap at the right atrial–superior vena caval junction provided enough slowing of conduction to sustain an atrial tachycardia over a relatively small anatomic circuit. It seems likely that a similar gap may occur in many patients in whom an atrial incision is constructed in close proximity to the superior vena cava, especially if the junction of these structures also was not ablated with a cryoablative lesion. We cannot exclude the possibility that caval vein cannulation for cardiopulmonary bypass may have contributed to this tachycardia. Incomplete anchoring of surgical barriers also may be responsible for some atrial tachycardias that involve reentry around the mitral valve. Conduction across the coronary sinus musculature has been shown to be responsible for many of these perimtrial tachycardias.14

The third mechanism of reentrant tachycardia after atrial fibrillation surgery appears to involve the anisotropic conduction properties of a functional central obstacle with slow conduction transverse to its long axis. These tachycardias were located in the roof of the left atrium where they appeared to use Bachmann’s bundle and in the free wall of the right atrium where they used the crista terminalis as a functional central obstacle with slow transverse conduction. Such tachycardias would seem unlikely to occur without the creation of artificial surgical barriers to conduction because unimpeded atrial conduction parallel to these structures would be expected to prevent slower transverse conduction because the tissue would be refractory once activated longitudinally. However, once boundaries have been created, it may be possible for this anisotropic obstacle to assume a role in transverse conduction. Such a mechanism has been observed for atypical atrial flutters in the right atrium that are dependent on the crista terminalis as a functional central obstacle with slow transverse conduction. The recognition that this anisotropic reentrant mechanism may be created by surgery has implications for surgical technique. In the right atrium, there is little or no evidence that an intercaval line improves the outcome of the Maze operation. Because this boundary may potentially promote reentry within the free wall of the right atrium, there may be a rationale for abandoning this ablation line. Although it remains unproven that it is Bachmann’s bundle that provides a central functional obstacle for reentry, techniques

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**Figure 5.** Demonstration of the postpacing interval equal to the tachycardia cycle length of 280 ms during pacing from the distal ablation catheter sinus electrode pair (Abl D). Surface ECG leads I, aVF, and V1, are recorded simultaneously with bipolar intracardiac electrograms recorded from the quadrapolar ablation catheter as shown in Figure 4, His bundle proximal (RPM His-P) and distal (RPM His-D) electrode pairs, and coronary sinus proximal (RPM CS 9/10) through distal (RPM CS 1/2) electrode pairs. Note the fractionated intracardiac electrograms from both the proximal and distal electrode pairs in the ablation catheter.
to prevent reentry using this structure as a central obstacle need to preserve the longitudinal conduction properties of interatrial conduction. Bachmann’s bundle is important for coordinated left atrial contraction and should not be intentionally interrupted. This may be especially important if the left atrial appendage is left intact. A surgical incision parallel to Bachmann’s bundle could potentially prevent reentry within the roof of the left atrium by preventing transverse conduction while maintaining longitudinal conduction through this important structure. Indeed, since recognition of this possible mechanism of late macroreentry, we have routinely incorporated a radiofrequency line anterior and parallel to Bachmann’s bundle into surgery for atrial fibrillation. There have been no episodes of this arrhythmia in the past 65 cases since this lesion was included in our standard surgical technique. However, the small number of cases in which this surgical boundary has been added limits our conclusions regarding the efficacy of this strategy.

Study Limitations
There are several important limitations to this study. First, the incidence of early and self-limited arrhythmias was not systematically collected in this study. Therefore, no correlation between early and late postoperative arrhythmias could be performed. Second, the incidence of sustained postoperative tachycardias reported is very likely to be an underestimate of their true frequency because only those patients with tachycardias that were recognized and underwent electrophysiologic study were included. No attempt was made to routinely monitor the cardiac rhythm of asymptomatic patients who were in sinus rhythm at the time of routine clinic follow-up visits. Thus, it is highly likely that less sustained or symptomatic atrial tachycardias were not identified. Third, this series occurred during an evolution from a cut-and-sew Maze III operation to an operation modified to include radiofrequency ablation for the pulmonary vein and intercaval barriers. The change in technique may have influenced the frequency and type of arrhythmias observed. Fourth, direct visualization of the anatomic structures was not performed during follow-up electrophysiologic procedures, which may have limited the precision of our identification of the structures responsible for these arrhythmias. Therefore, the identification of Bachmann’s bundle and the crista terminalis was made fluoroscopically and may have been less precise than direct visualization during surgery. In addition, intracardiac echocardiography was not used to define the crista terminalis. Finally, these operations were all performed by a single surgeon, a fact that may limit our ability to generalize conclusions to other surgeons and other techniques. To balance these limitations, all 22 patients in our series who developed late regular atrial tachycardias underwent electrophysiological study, thereby allowing a uniquely complete description of all such arrhythmias to enable us to fully describe their mechanisms and incidence.

Conclusions
Late atrial tachycardias are not uncommon after surgery for atrial fibrillation. The mechanism for these tachycardias is primarily reentry. Recognition that reentry can occur in the roof of the left atrium where it may use Bachmann’s bundle or a relative paucity of myocardial fibers as a central functional obstacle may lead to improved surgical strategies that prevent this arrhythmia. Elimination of the right atrial intercaval incision will likely prevent many of these arrhythmias. When late postoperative atrial tachycardias are recognized, catheter ablation techniques are highly effective for providing long-term control of these arrhythmias.

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References
CLINICAL PERSPECTIVE

Surgical procedures to prevent atrial fibrillation are being performed much more commonly with advances in surgical technologies for lesion creation. As a result, more patients are likely to experience proarrhythmic effects of these operations. Late macroreentrant atrial tachycardias may occur in either atria and may be the result of incomplete surgical lines (such as within the cavotricuspid isthmus or adjacent to the mitral valve annulus). Macroreentry also may occur at the site where surgical boundaries have not been extended through all conducting atrial myocardium (such as at the superior vena caval–right atrial junction). Additional mechanisms may involve the creation of slowly conducting zones of atrium based on the anisotropic conduction properties of structures such as the crista terminalis or Bachmann’s bundle. Although the precise mechanism of these functional central obstacles has not been completely defined, such structures appear to be the site of successful ablation for some arrhythmias in the right atrial free wall and in the roof of the left atrium. A critical aspect of any successful surgical program is close collaboration between the cardiovascular surgeon who is performing these operations and an experienced cardiac electrophysiologist capable of mapping and ablating these arrhythmias. Patients undergoing surgical ablation of atrial fibrillation require close follow-up postoperatively.
Proarrhythmic Aspects of Atrial Fibrillation Surgery: Mechanisms of Postoperative Macroreentrant Tachycardias

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