ELECTROPHYSIOLOGY
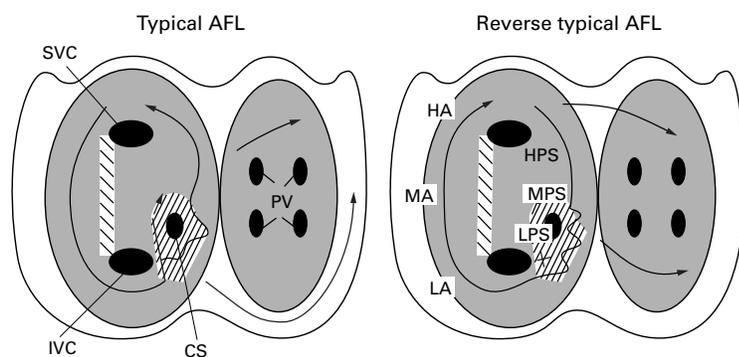
# Treatment of atrial flutter

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After atrial fibrillation, atrial flutter is the most important and most common atrial tachyarrhythmia. Although it was first described 80 years ago, techniques for its diagnosis and management have changed little for decades. The diagnosis rested almost entirely with the 12 lead ECG, and treatment options included only the use of a digitalis compound to slow and control the ventricular response rate, and/or the use of either quinidine or procainamide in an attempt to convert the rhythm to sinus rhythm or to prevent recurrence of atrial flutter once sinus rhythm was established.

The past 25 years have produced major changes. A series of studies has advanced our understanding of the mechanism(s) of atrial flutter. Old techniques to diagnose atrial flutter have been significantly refined, and new diagnostic techniques have been developed. Beginning with the advent of DC cardioversion in the 1960s, major advances in the treatment of atrial flutter have occurred. β Blockers and calcium channel blockers are now available for use as an adjunct to or in lieu of digitalis treatment to control the ventricular response rate. New antiarrhythmic agents are available for use to suppress atrial flutter or convert it to sinus rhythm. Atrial pacing techniques to interrupt or suppress atrial flutter have evolved. Catheter ablation techniques either to cure atrial flutter or to control the ventricular response rate have been developed, and related surgical treatments are available. Even automatic low energy cardioversion of atrial flutter to sinus rhythm has been developed.

Mechanisms and classification of atrial flutter

Most of the advances in our understanding of atrial flutter have come from our understanding its mechanism. There is a long history, summarised recently,[1] of studies in animal models which have contributed to our understanding of atrial flutter. While those studies have been and continue to be most helpful, a series of studies in patients—principally using catheter electrode mapping and pacing techniques—has established that classical atrial flutter is caused by a re-entrant circuit confined to the right atrium in which the impulse travels up the atrial septum, with epicardial breakthrough superiorly in the right atrium where the impulse then travels inferiorly down the right atrial free wall to re-enter the atrial septum (fig 1).[2–7] When the circulating wave front re-enters the atrial septum, it travels through an isthmus bounded by the inferior vena cava, Eustachian ridge, the coronary sinus os on one side and the tricuspid valve annulus on the other side (the "atrial flutter isthmus"). Atrial flutter caused by this mechanism is called *typical atrial flutter*,[8] although it also has been called common atrial flutter and counterclockwise atrial flutter. A 12 lead ECG during typical atrial flutter with characteristic negative "sawtooth" atrial flutter waves in leads II, III, and aVF is shown in fig 2. It is also recognised that impulses can travel in this re-entrant circuit in the opposite direction, so that the impulse travels down the atrial septum and breaks through to the epicardium via the same atrial flutter isthmus to travel up the right atrial free wall and then re-enter the septum superiorly (fig 1).[3] This form of atrial flutter is called *reverse typical atrial flutter*,[8] although it has in the past been called atypical atrial flutter, clockwise atrial flutter, uncommon atrial flutter, and rare atrial flutter. A 12 lead ECG during reverse typical atrial flutter with characteristic positive flutter waves in leads II, III, and aVF is shown in fig 3.

Figure 1. Left: atrial activation in typical atrial flutter (AFL). Right: activation in reverse typical AFL. The atria are represented schematically in a left anterior oblique view, from the tricuspid (left) and mitral rings. The endocardium is shaded and the openings of the superior (SVC) and inferior vena cava (IVC), coronary sinus (CS), and pulmonary veins (PV) are shown. The direction of activation is shown by arrows. Dashed areas mark approximate location of zones of slow conduction and block. Lettering on the right hand panel marks the low (LPS), mid (MPS), and high (HPS) posteroseptal wall, respectively. Modified after Cosío FG et al. J Cardiovasc Electrophysiol 1996;7:60–70.
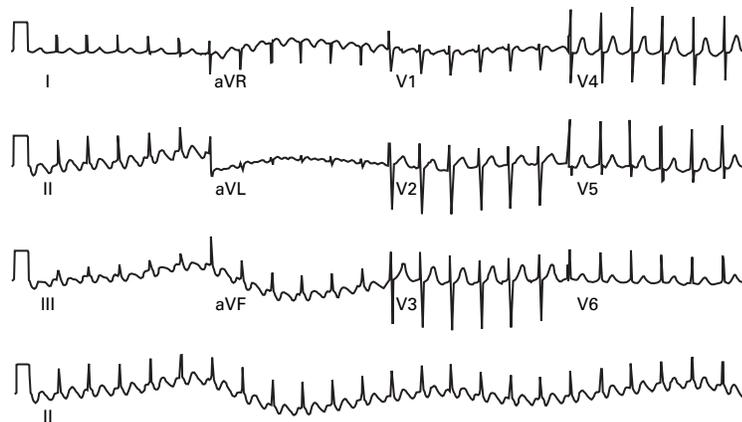
Figure 2. A 12 lead ECG in a case of typical type I atrial flutter. The atrial rate is 300 bpm and the ventricular rate is 150 bpm; 2:1 AV block is present. Note that the atrial activity is best seen in leads II, III, and aVF and is barely perceptible in lead I. Reproduced with permission from Waldo AL, Kastor JA: Atrial flutter. In: Kastor JA, ed. Arrhythmias. Philadelphia: WB Saunders Co, 1994:105–15.

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Types of atrial flutter

- Typical atrial flutter
- Reverse typical atrial flutter
- Incisional atrial re-entry
- Left atrial flutter
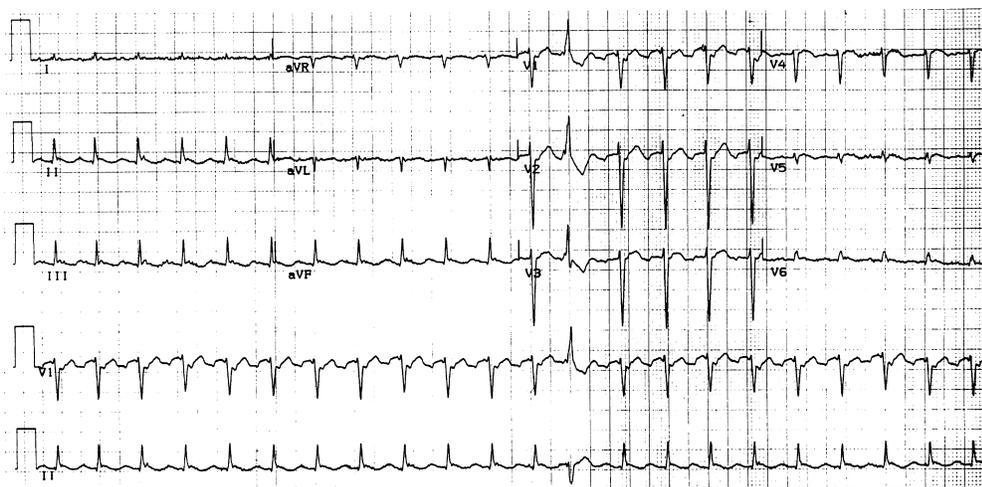- Atypical atrial flutter

Two other mechanisms of atrial flutter are now well recognised. One, *incisional atrial re-entry*,[8] is seen in patients after repair of congenital heart defects that involve one or more right atrial free wall incisions in which the re-entrant circuit travels around the line of block caused by the incision.[9] Interestingly, it has recently been shown[10] that when atrial flutter does occur chronically in patients following repair of congenital heart defects, it is usually caused by a re-entrant circuit that includes the atrial flutter isthmus. Additionally, *a left atrial flutter* is now recognised that is thought generally to circulate around one or more of the pulmonary veins or the mitral valve annulus, but this re-entrant mechanism has not been well characterised. And finally, there are some forms of atrial flutter which are quite unique, and have now been called truly atypical atrial flutter.[8]

All these types of atrial flutter fall under the category of type I atrial flutter as described by Wells and colleagues.[11] They are distinguished by the fact that they can always be interrupted by rapid atrial pacing, and have a rate range between 240–340 beats/min (bpm).[11] Type II atrial flutter[11] is a more rapid atrial flutter (rates > 340 bpm) which is still being characterised. It is presently thought to be caused by a re-entrant circuit with a very rapid rate which causes fibrillatory conduction to much or most of the atria, resulting in an atrial fibrillation pattern in the ECG.[12] [13]

Epidemiology and clinical significance

Atrial flutter typically is paroxysmal, usually lasting seconds to hours, but on occasion lasting longer. Occasionally, it is a persistent rhythm. Atrial flutter as a stable, chronic rhythm is unusual, as it usually reverts either to sinus rhythm or to atrial fibrillation, either spontaneously or as a result of treatment. However, atrial flutter has been reported to be present for up to 20 years or more. It can occur in patients with ostensibly normal atria or with abnormal atria. Atrial flutter occurs commonly in patients in the first week after open heart surgery. Patients with atrial flutter not uncommonly demonstrate sinus bradycardia or other manifestations of sinus node dysfunction. Atrial flutter is also associated with chronic obstructive pulmonary disease, mitral or tricuspid valve disease, thyrotoxicosis, and surgical repair of certain congenital cardiac lesions which involve large incisions or suture lines in the atria.[10] It is also associated with enlargement of the atria for any reason, especially the right atrium.

Atrial flutter is most often a nuisance arrhythmia. Its clinical significance lies largely in its frequent association with atrial fibrillation, its previously little appreciated association with thromboembolism, especially stroke,[14] [15] and its frequent association with a rapid ventricular response rate (fig 2). The association of atrial flutter with a rapid ventricular rate is important because the rapid ventricular rate is principally responsible for many of the associated symptoms. And, in the presence of the Wolff-Parkinson-White syndrome or a very short P-R interval ($\leqslant 0.115$ s) in the absence of a delta wave, it may be associated with 1:1 atrioventricular (AV) conduction, sometimes with dire consequences. Furthermore, if the duration of the rapid ventricular response rate is prolonged, it may result in ventricular dilatation and congestive heart failure.

Figure 3. 12 lead ECG from a patient with reverse typical atrial flutter confirmed at electrophysiological study. The atrial rate is 266 bpm with 2:1 AV conduction. Note the positive flutter waves in leads II, III, and aVF, and the negative flutter waves in lead V_1. Reproduced courtesy of N Varma, MD.

Management of atrial flutter

Acute treatment

When atrial flutter is diagnosed, three options are available to restore sinus rhythm: (1) administer an antiarrhythmic drug; (2) initiate DC cardioversion; or (3) initiate rapid atrial pacing to terminate the atrial flutter (fig 4). Selection of acute treatment for atrial flutter with either DC cardioversion, atrial pacing or antiarrhythmic drug therapy will depend on the clinical presentation of the patient and both the clinical availability and ease of using these techniques. Since DC cardioversion requires administration of an anaesthetic agent, this may be undesirable in the patient who presents with atrial flutter having recently eaten or the patient who has severe chronic obstructive lung disease. Such patients are best treated with either antiarrhythmic drug therapy or rapid atrial pacing to terminate the atrial flutter, or with an AV nodal blocking drug to slow the ventricular response rate. When atrial flutter is associated with a situation requiring urgent restoration of sinus rhythm—for example, 1:1 AV conduction or hypotension—prompt DC cardioversion is the treatment of choice. For the patient who develops atrial flutter following open heart surgery, use of the temporary atrial epicardial wire electrodes to perform rapid atrial pacing to restore sinus rhythm is the treatment of choice (fig 4).

Whenever rapid control of the ventricular response rate to atrial flutter is desirable, use of either an intravenous calcium channel blocking agent (verapamil or diltiazem) or an intra-venous β blocking agent (usually esmolol, although propranolol or metoprolol can also be used) is usually effective. Aggressive administration of a digitalis preparation, usually intravenously, to control ventricular rate (it might also convert the atrial flutter either to atrial fibrillation with a controlled ventricular response rate or to sinus rhythm) is also acceptable, but generally is not the treatment of choice except in the presence of pronounced ventricular dysfunction. DC cardioversion of atrial flutter to sinus rhythm has a very high likelihood of success. When this mode of treatment is selected, it may require as little as 25 joules, although at least 50 joules is generally recommended because it is more often successful. Because 100 joules is virtually always successful and virtually never harmful, it should be considered as the initial shock strength.

Antiarrhythmic drug treatment can be used to convert atrial flutter to sinus rhythm. Three drugs—ibutilide, flecainide, and propafenone—have a reasonable expectation of accomplishing this. Ibutilide, which can only be used intravenously, is associated with a 60% likelihood of converting atrial flutter to sinus rhythm.[16] Because ibutilide dramatically prolongs ventricular repolarisation, and consequently the Q-T interval, there is a small incidence of torsades de pointes associated with its use.[17] However, these episodes, should they occur, are usually self limited, and because of the short half life of this drug, the period of such risk is quite brief, usually less than one hour. Nevertheless, one should be prepared to administer intravenous magnesium and even perform DC cardioversion to treat a prolonged episode of torsades de pointes should it occur when using ibutilide. Flecainide and propafenone, when used intravenously[18] or when used orally but in a single high dose (300 mg for flecainide or 600 mg for propafenone) also may be effective in cardioverting this rhythm to sinus. When using either of these drugs, the atrial rate may slow dramatically—for example, to 200 bpm. Therefore, it is best given with a calcium channel blocker or β blocker to prevent the possibility of 1:1 AV conduction of the significantly slowed atrial flutter rate. Antiarrhythmic drug treatment also may be used before performing either DC cardioversion or rapid atrial pacing: (1) to slow the ventricular response rate (with a β blocker, a calcium channel blocker, digoxin or some combination of these drugs); (2) to enhance the efficacy of rapid atrial pacing in restoring sinus rhythm (use of procainamide, disopyramide or ibutilide); or (3) to enhance the likelihood that sinus rhythm will be sustained following effective DC cardioversion (use of a class IA, class IC or class III antiarrhythmic agent).

Long term treatment of atrial flutter

Recent improvements in the efficacy of catheter ablation techniques and the long recognised difficulty in achieving adequate chronic suppression of atrial flutter with drug treatment have significantly affected the approach to long term treatment of atrial flutter. In short, if atrial flutter is an important clinical problem in any patient, characterisation of the mech-
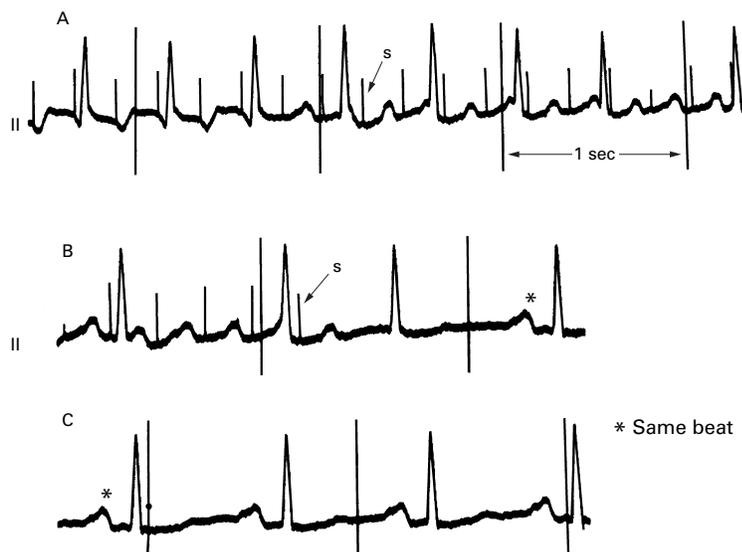
Figure 4. ECG lead II recorded from a patient with typical atrial flutter (spontaneous atrial cycle length of 264 ms). Rapid atrial pacing from a high right atrial site at a cycle length of 254 ms (not shown), at a cycle length of 242 ms (not shown), and at a cycle length of 232 ms (not shown) failed to terminate the atrial flutter. Panel A shows ECG lead II recorded during high right atrial pacing at a cycle length of 224 ms. Note that with the seventh atrial beat in this tracing, and after 22 seconds of atrial pacing at a constant rate, the atrial complexes suddenly became positive. Panel B shows ECG lead II recorded at the termination of atrial pacing in the same patient. Note that with abrupt termination of pacing, sinus rhythm occurs. In panel C, the first beat (asterisk) is identical to the last beat in panel B (asterisk). S, stimulus artifact. Time lines are at 1 second intervals. Modified from Waldo AL, et al. Circulation 1997;56:737–45.


Acute treatment of atrial flutter

- Depends on clinical presentation
  - need for prompt restoration of sinus rhythm: DC cardioversion
  - elective restoration of sinus rhythm: antiarrhythmic drug treatment (ibutilide or class IC agent), DC cardioversion or rapid atrial pacing
  - ventricular rate control: often required (β blocker or calcium channel blocker), especially with use of class IC antiarrhythmic agent

anism of atrial flutter followed by catheter ablation as treatment of choice (cure) is now recommended.

Catheter ablation treatment
Two types of catheter ablation are available for the treatment of chronic or recurrent atrial flutter, one curative and one palliative. Appropriate application of radiofrequency energy via an electrode catheter can be used to cure atrial flutter. Advances in both electrophysiologic mapping and radiofrequency catheter ablation techniques have improved the efficacy of this therapeutic approach to about a 95% cure rate for patients with typical or reverse typical atrial flutter,[7 19] making it the treatment of choice in most patients in whom the arrhythmia is clinically important. The technique involves electrophysiologic study of the atria during atrial flutter to identify the location of the re-entrant circuit and then to confirm that the re-entrant circuit includes a critical isthmus between the inferior vena cava–Eustachian ridge–coronary sinus ostium and the tricuspid valve (fig 5). When this latter area is identified, radiofre-
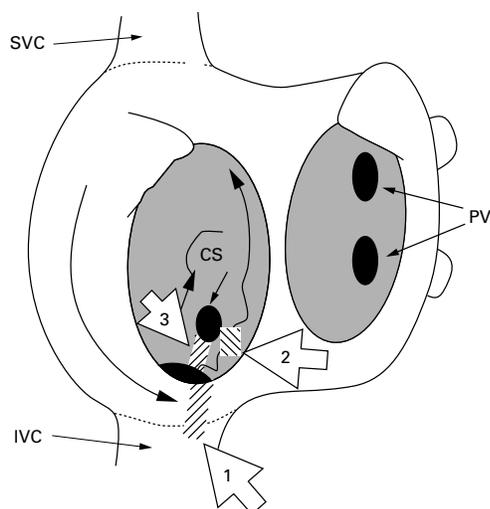
Figure 5. Targets for typical or reverse typical atrial flutter ablation. The schematic drawing shows the atria in an anterior view. The endocardium, inside the tricuspid (left) and mitral (right) rings, is shaded. The openings of the inferior vena cava (IVC), coronary sinus (CS), and left pulmonary veins (PV) are shown in black. Long arrows show activation sequence in common atrial flutter. The striped areas (large open arrows) mark ablation targets: 1, IVC–tricuspid valve isthmus; 2, CS–tricuspid valve isthmus; 3, CS–IVC isthmus. SVC, superior vena cava. Reproduced with permission from Cosío et al.[19]

quency energy is delivered through the electrode catheter to create a bidirectional line of block across it. This isthmus may be difficult to ablate completely,[7 19] but combined entrainment pacing and mapping techniques have now evolved which permit both the reliable demonstration that this isthmus is a part of the re-entrant circuit, and that application of radiofrequency energy has produced complete bidirectional conduction block in this isthmus. When the latter is demonstrated, successful ablation of atrial flutter has been accomplished.

Similarly, when incisional re-entrant atrial flutter is identified by electrophysiological mapping techniques, a vulnerable isthmus usually can be identified and successfully ablated using radiofrequency catheter ablation techniques.[9] There is insufficient information available to discuss the likely efficacy of successful radiofrequency ablation techniques to cure left atrial flutter or atypical atrial flutter, although contemporary electrophysiological mapping techniques are capable of identifying the location of the re-entrant circuits associated with these types of atrial flutter, making effective ablation treatment a possibility.

AV nodal–His bundle ablation to create high degree AV block (generally third degree AV block) can be used palliatively to eliminate the rapid ventricular response rate to atrial flutter. It does not prevent the atrial flutter, and requires placement of a pacemaker system. For patients in whom catheter ablation of atrial flutter is unsuccessful and in whom antiarrhythmic drug treatment is either ineffective or is not tolerated, or in whom atrial flutter with a clinically unacceptable rapid ventricular response rate recurs despite drug treatment, producing third degree AV block or a high degree of AV block provides a successful form of therapy. Selection of a pacemaker in such circumstances should be tailored to the needs of the patient, and may include a single chamber, rate responsive, ventricular pacemaker or a dual chamber pacemaker with mode switching capability.

Antiarrhythmic drug treatment
Atrial flutter is quite difficult to suppress completely with drug treatment. In fact, based on available long term data, drug treatment offers a limited ability to maintain sinus rhythm without occasional to frequent recurrences of atrial flutter, even when multiple agents are used. This is among the reasons why this form of therapy is no longer the long term treatment of choice in most patients with atrial flutter. For patients in whom drug treatment is selected, an important measure of efficacy should be the frequency of recurrence of atrial flutter rather than a single recurrent episode. For instance, recurrence only at long intervals—for example, once or twice per year—probably should be classified as a treatment success rather than a failure.

In the past, standard antiarrhythmic drug treatment consisted of administration of a class IA agent (quinidine, procainamide, or disopyramide) in an effort to prevent recurrence. However, recent studies indicate that the type

IC antiarrhythmic agents flecainide and propafenone are as effective, if not more effective, are generally better tolerated, and have less organ toxicity than class IA agents. Principally because of their serious adverse effects demonstrated in the cardiac arrhythmia suppression trial (CAST I), it is widely accepted that class IC agents should not be used in the presence of underlying ischaemic heart disease. In fact, this approach has generally been extrapolated to include the presence of underlying structural heart disease. Nevertheless, class IC agents are recommended for long term suppression of atrial flutter in the absence of structural heart disease.

Moricizine, a class I drug with A, B, and C properties, also may be effective in the treatment of atrial flutter. The long term data from CAST II, in which moricizine and placebo were no different in terms of mortality, suggests that moricizine may be a good choice for patients with atrial flutter and coronary artery disease late (> 3 months) after a myocardial infarction. However, more data are required to establish moricizine's efficacy and safety in this clinical setting.

In addition, the class III antiarrhythmic agents amiodarone, sotalol, and dofetilide also may be quite effective. When using sotalol or dofetilide, care must be taken to avoid Q-T$_c$ interval prolongation much beyond 500 ms in order to avoid precipitation of torsades de pointes. Amiodarone appears to be quite effective, but its potential toxicity is a well recognised concern, making widespread use of this drug to treat atrial flutter problematic.[20] Thus, the use of amiodarone as the drug of first choice to treat atrial flutter probably should be limited to patients with notably depressed left ventricular function. Since atrial flutter tends to recur despite antiarrhythmic drug treatment, it is important to remember that on a class IA (quinidine, procainamide, disopyramide) or especially a class IC or IC-like (flecainide, propafenone, moricizine) agent, the atrial flutter rate may be much slower (for example, 180–220 bpm) than in the absence of one of these drugs. Therefore, it is very important that adequate block of AV nodal conduction be present, usually with concurrent use of a β blocker or a calcium channel blocker, alone or in combination with digoxin.

Anticoagulant treatment
Although one study found neither atrial clot formation nor stroke associated with atrial flutter in a relatively small cohort of patients after open heart surgery, the association of the potential risk of stroke with atrial flutter has now been established.[14 15] Other data support this association. Thus, atrial flutter and atrial fibrillation often co-exist in patients. Additionally, using transoesophageal echocardiography, a high incidence of spontaneous echo contrast and atrial thrombi have been documented, as were striking abnormalities in the left atrial appendage in patients with atrial flutter. In short, in patients with atrial flutter, daily warfarin treatment to achieve an international normalised ratio (INR) of 2 to 3 is recom-

> **Long term treatment of clinically important atrial flutter**
>
> - Treatment of choice: radiofrequency catheter ablation to achieve cure
>
> - Alternative treatment (warfarin therapy usually required)
>   - drug treatment (class IC, III or IA antiarrhythmics plus an AV nodal blocking drug)
>   - device implantation (antitachycardia pacemaker or low energy atrial defibrillator)
>   - His bundle ablation plus pacemaker implantation

mended using the same criteria as for atrial fibrillation. Also, the same criteria apply for cardioversion. Thus, if the patient has had atrial flutter for greater than 48 hours and the INR is not therapeutic (INR ≥ 2), warfarin treatment should be either initiated or adjusted, and after achieving a therapeutic INR for three consecutive weeks, cardioversion may be attempted. Following cardioversion, the patient should remain on warfarin with a therapeutic INR for four weeks.

Permanent antitachycardia pacemaker treatment
Although rarely used as treatment, in selected patients consideration should be given to implantation of a permanent antitachycardia pacemaker to interrupt recurrent atrial flutter and restore sinus rhythm. While there is only a small published series of patients treated with such devices, it nevertheless has been shown to be safe and effective. Since precipitation of atrial fibrillation is always a potential problem when using any form of pacing to treat atrial flutter, if any pacing induced episodes of atrial fibrillation are clinically unacceptable, placement of a permanent antitachycardia pacemaker to treat atrial flutter should be avoided. To decrease or eliminate an incidence of inadvertent precipitation of atrial fibrillation as well as to decrease the frequency of atrial flutter episodes, chronic use of an antiarrhythmic drug may be desirable.

Surgical treatment
Presently, there is little if any role for surgical ablation of the atrial flutter. Nevertheless, there is a limited experience. Klein, Guiraudon and colleagues have reported on three operated patients in whom cryoablation of the region between the coronary sinus orifice and the tricuspid annulus successfully prevented recurrent atrial flutter in two.[21 22] However, the third patient had subsequent symptomatic atrial fibrillation. Similarities between these surgical data and the catheter ablation data are apparent. Also, Canavan and colleagues reported the successful surgical interruption of the atrial flutter re-entrant circuit after intraoperative mapping in an adolescent who had an atrial septal defect repair as a child.[23] The atrial flutter re-entrant circuit was around the atriotomy.

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Summary

Most atrial flutter is caused by re-entrant excitation in the right atrium. The 12 lead ECG remains the cornerstone for the clinical diagnosis. Acute treatment entails control of the ventricular response rate and restoration of sinus rhythm. Currently, radiofrequency catheter ablation treatment provides the expectation of cure, although atrial fibrillation may subsequently occur. Alternatively, antiarrhythmic drug treatment to suppress recurrent atrial flutter episodes may be useful, recognising that recurrences are common despite therapy. Use of an antitachycardia pacemaker may be useful in selected patients to terminate atrial flutter, as may His bundle ablation with placement of an appropriate pacemaker system to control the ventricular response rate. Anticoagulation with warfarin in patients with recurrent or chronic atrial flutter is recommended using criteria applied to patients with atrial fibrillation.

1. **Waldo AL.** Pathogenesis of atrial flutter. *J Cardiovasc Electrophysiol* 1998;**9**:518–25.
   • *Short review of the pathogenesis of atrial flutter.*

2. **Olshansky B, Okumura K, Hess PG,** *et al.* Demonstration of an area of slow conduction in human atrial flutter. *J Am Coll Cardiol* 1990;**16**:1639–48.
   • *Mapping studies of typical atrial flutter.*

3. **Cosío FG, Goicolea A, Lopez-Gil M,** *et al.* Atrial endocardial mapping in the rare form of atrial flutter. *Am J Cardiol* 1990;**66**:715–20.
   • *Mapping studies of reverse typical atrial flutter.*

4. **Olgin JE, Kalman JM, Fitzpatrick AP,** *et al.* Role of right atrial endocardial structures as barriers to conduction during human type I atrial flutter. Activation and entrainment mapping guided by intracardiac echocardiography. *Circulation* 1995;**92**:1839–48.
   • *Studies defining the boundaries of the typical atrial flutter re-entrant circuit.*

5. **Kalman JM, Olgin JE, Saxon LA,** *et al.* Activation and entrainment mapping defines the tricuspid annulus as the anterior boundary in atrial flutter. *Circulation* 1996;**94**:398–406.
   • *Studies defining the boundaries of the atrial flutter re-entrant circuit.*

6. **Nakagawa H, Lazzara R, Khastgir T,** *et al.* Role of the tricuspid annulus and the Eustachian valve/ridge on atrial flutter. Relevance to catheter ablation of the septal isthmus and a new technique for rapid identification of ablation success. *Circulation* 1996;**94**:407–24.
   • *Studies defining the boundaries of the atrial flutter re-entrant circuit.*

7. **Cosio FG, Arribas F, Lopez-Gil M,** *et al.* Atrial flutter mapping and ablation. I. Studying atrial flutter mechanisms by mapping and entrainment. *PACE* 1996;**19**:841–53.
   • *Electrode catheter mapping studies to identify the vulnerable part of the atrial flutter re-entrant circuit.*

8. **Saoudi N, Cosío F, Chen SA,** *et al.* A new classification of atrial tachycardias based on electrophysiologic mechanisms. *Eur J Cardiol* In press.
   • *Explanation and examples of the new classification of atrial flutter.*

9. **Van Hare GF, Lesh MD, Ross BA,** *et al.* Mapping and radiofrequency ablation of intraatrial reentrant tachycardia after the Senning or Mustard procedure for transposition of the great arteries. *Am J Cardiol* 1996;**77**:985–91.
   • *Studies of patients with chronic atrial flutter caused by incisional re-entry following surgical repair of a congenital heart lesion.*

10. **Chan DP, Van Hare GF, Mackall JA,** *et al.* Importance of the atrial flutter isthmus in post-operative intra-atrial reentrant tachycardia. *Circulation* In press.
    • *Studies of patients with chronic atrial flutter following surgical repair of a congenital heart lesion demonstrating that in 75% of these patients, the atrial flutter re-entrant circuit utilises the atrial flutter isthmus.*

11. **Wells JL Jr, MacLean WAH, James TN,** *et al.* Characterization of atrial flutter. Studies in man after open heart surgery using fixed atrial electrodes. *Circulation* 1979;**60**:665–73.
    • *Studies characterising type I and type II atrial flutter in patients.*

12. **Waldo AL, Cooper TB.** Spontaneous onset of type I atrial flutter in patients. *J Am Coll Cardiol* 1996;**28**:707–12.
    • *Studies demonstrating that atrial fibrillation generally precedes the onset of atrial flutter.*

13. **Matsuo K, Tomita Y, Khrestian CM,** *et al.* A new mechanism of sustained atrial fibrillation: studies in the sterile pericarditis model [abstract]. *Circulation* 1998;**98**:I–209.
    • *Demonstration of the nature of atrial fibrillation generated by a re-entrant circuit of very short cycle length (very rapid rate) which produces fibrillatory conduction.*

14. **Wood KA, Eisenberg SJ, Kalman JM,** *et al.* Risk of thromboembolism in chronic atrial flutter. *Am J Cardiol* 1997;**79**:1043–7.
    • *Study demonstrating important risk of stroke or systemic embolism in the presence of atrial flutter but in the absence of anticoagulation treatment.*

15. **Seidl K, Haver B, Schwick NG,** *et al.* Risk of thromboembolic events in patients with atrial flutter. *Am J Cardiol* 1998;**82**:580–4.
    • *Study demonstrating thromboembolic risk associated with atrial flutter.*

16. **Ellenbogen KA, Clemo HF, Stambler BS,** *et al.* Efficacy of ibutilide for termination of atrial fibrillation and flutter. *Am J Cardiol* 1996;**78**(suppl 8A):42–5.
    • *Study showing efficacy of ibutilide in conversion of atrial flutter to sinus rhythm.*

17. **Stambler BS, Wood MA, Ellenbogen KA,** *et al.* Efficacy and safety of repeated intravenous doses of ibutilide for rapid conversion of atrial flutter or fibrillation. *Circulation* 1996;**94**:1613–21.
    • *Study highlighting risks as well as efficacy of ibutilide therapy of atrial flutter.*

18. **Suttorp MJ, Kingma JH, Jessuren ER,** *et al.* The value of class IC antiarrhythmic drugs for acute conversion of paroxysmal atrial fibrillation or flutter to sinus rhythm. *J Am Coll Cardiol* 1990;**16**:1722–7.
    • *Study showing efficacy of class IC agents in conversion of atrial flutter to sinus rhythm.*

19. **Cosío FG, Arribas F, Lopez-Gil M,** *et al.* Atrial flutter mapping and ablation. II. Radiofrequency ablation of atrial flutter circuits. *PACE* 1996;**19**:965–75.
    • *Review of ablation techniques to cure atrial flutter.*

20. **Podrid PJ.** Amiodarone: reevaluation of an old drug. *Ann Int Med* 1995;**122**:689–700.
    • *Good review of use of amiodarone for atrial flutter, including data on adverse effects of this drug.*

21. **Klein GJ, Guiraudon GM, Sharma AD,** *et al.* Demonstration of macroreentry and feasibility of operative therapy in the common type of atrial flutter. *Am J Cardiol* 1986;**57**:587–91.

22. **Guiraudon GM, Klein GJ, Sharma AD,** *et al.* Surgical alternatives for supraventricular tachycardias. *Am J Cardiol* 1989;**64**:92J–6J.

23. **Canavan TE, Schuessler RB, Cain ME,** *et al.* Computerized global electrophysiological mapping of the atrium in a patient with multiple supraventricular tachyarrhythmias. *Ann Thorac Surg* 1988;**46**:232–5.