Overview of atrial flutter

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INTRODUCTION

Atrial flutter is an abnormal cardiac rhythm characterized by rapid, regular atrial depolarizations at a characteristic rate of approximately 300 beats/min and a regular ventricular rate of about 150 beats/min in patients not taking atrioventricular (AV) nodal blockers. It can lead to symptoms of palpitations, shortness of breath, fatigue, or lightheadedness, as well as an increased risk of atrial thrombus formation that may cause cerebral and/or systemic embolization.

Atrial flutter occurs in many of the same situations as atrial fibrillation, which is much more common. Atrial flutter may be a stable rhythm or a bridge arrhythmia between sinus rhythm and atrial fibrillation, or an organized rhythm in atrial fibrillation patients treated with antiarrhythmic drugs. It may also be associated with a variety of other supraventricular arrhythmias. (See "Epidemiology, risk factors, and prevention of atrial fibrillation".)

This topic will summarize key points regarding the causes, clinical presentation, diagnosis, and management approach to patients with atrial flutter. Other topics discuss management issues in detail. (See "Restoration of sinus rhythm in atrial flutter" and "Control of ventricular rate in atrial flutter" and "Atrial flutter: Maintenance of sinus rhythm" and "Embolic risk and the role of anticoagulation in atrial flutter".)

ELECTROPHYSIOLOGIC CLASSIFICATION

Atrial flutter was previously classified as either type I or type II. That terminology is no longer used.

Typical atrial flutter — The designation of "typical" atrial flutter involves a macroreentrant circuit traversing the cavo-tricuspid isthmus (CTI) (figure 1). This isthmus is the region of right atrial tissue between the orifice of the inferior vena cava and the tricuspid valve annulus (figure 2). If this isthmus is involved, it is called "typical" atrial flutter or CTI-dependent atrial flutter. The circuit is usually a counterclockwise rotation around the tricuspid valve (figure 2), exhibiting a classic sawtooth appearance in the inferior electrocardiogram (ECG) leads (II, III, aVF) (image 1B). If the circuit is clockwise, it is called "reverse" or "clockwise" typical flutter, exhibiting positive flutter waves in the inferior ECG leads (image 1C). The clockwise circuit occurs far less frequently than the counterclockwise circuit; rare patients exhibit both circuits at different times. The ECG hallmark of typical atrial flutter is discordance in flutter wave "direction" between the inferior leads and lead V1. In counterclockwise circuits, flutter waves are directly negative in the inferior leads but are positive in lead V1. In clockwise circuits, the opposite is true. These ECG rules are generally less reliable after atrial ablation or surgery.

Atypical atrial flutter — If the CTI is not involved in the underlying mechanism, then it is called "atypical" atrial flutter. This type of flutter can involve any region of the right or left atria, around areas of scar tissue due to intrinsic heart disease or surgical/ablated scar tissue (see "Electrocardiographic and electrophysiologic features of atrial flutter"). Surgical repair of congenital heart disease may lead to macroreentrant atrial flutter circuits, both typical (cavotricuspid isthmus dependent) and atypical. These circuits are usually right atrial, related to anatomic obstacles and surgical scars (cavotricuspid isthmus, right atriotomy scar, atrial septal defect repair, etc). Left atrial flutters that arise after AF ablation procedures constitute a large fraction of atypical flutters. Incomplete ablation lines created in attempts to cure atrial fibrillation with ablation can promote atypical atrial flutter circuits in the left atrium (mitral isthmus flutter, etc). Focal atrial tachycardias with atrioventricular block may also mimic atypical atrial flutter by ECG appearance, but by electrophysiologic study the focal mechanism can be differentiated from the macroreentry seen in atrial flutter. In atypical atrial flutters, the flutter waves in the inferior leads and lead V1 are often concordant. (See "Focal atrial tachycardia".)

ETIOLOGY AND RISK FACTORS

Atrial flutter is uncommon in the structurally normal heart [1-3].

A variety of underlying conditions can predispose to the development of atrial flutter. These include:

- **After antiarrhythmic drug initiation** – Atrial flutter may occur after initiation of an antiarrhythmic drug for the suppression of atrial fibrillation. It may occur in up to 15 percent of patients treated with flecainide or propafenone, and is also seen in patients treated with dronedarone or amiodarone.

- **After acute myocardial infarction** – Atrial flutter is a relatively uncommon complication of an acute myocardial infarction [4,5] and is rarely, if ever, a manifestation of digitalis toxicity [6,7]. (See "Supraventricular arrhythmias after myocardial infarction" and "Cardiac arrhythmias due to digoxin toxicity".)

- **Post-cardiac surgery** – Atrial flutter can occur after cardiac surgery, both as a postoperative complication and as a late arrhythmia. The atrial flutter in these patients is re-entrant and may be typical or involve atypical isthmuses between natural barriers, atrial incisions, and scar. (See "Atrial fibrillation and flutter after cardiac surgery", section on 'Pathogenesis'.)

- **Post-atrial fibrillation ablation** – Some patients develop atypical left atrial flutter after atrial fibrillation ablation. These arrhythmias may be due to circuits created by scar from left atrial (LA) ablations, but are often amenable to ablation themselves. This issue is discussed in detail separately. (See "Atrial fibrillation: Catheter ablation", section on 'Arrhythmic complications'.)

- **Other specific triggers** – Any of the disorders that can cause atrial fibrillation, including thyrotoxicosis, obesity, obstructive sleep apnea, sinus node dysfunction, pericarditis, pulmonary disease, and pulmonary embolism. (See "Epidemiology, risk factors, and prevention of atrial fibrillation".)

INCIDENCE

In the general population, the development of new onset atrial flutter is uncommon and occurs with significantly less frequency than atrial fibrillation.

In a population-based study, lone atrial flutter with neither identifiable recent predisposing events nor chronic preexisting comorbidities occurred in only 3 of 181 patients (1.7 percent) [1]. Sixteen percent of cases were attributable to heart failure and 12 percent to chronic obstructive pulmonary disease [1]. The incidence increased markedly with age, ranging from 5 per 100,000

person years under age 50 to 587 per 100,000 person years over age 80. The rate of lone atrial flutter was 8 percent in another series of 380 children and young adults [3].

Much of the information about atrial flutter has been derived from patients referred to tertiary care centers; as a result, the incidence of atrial flutter in the general population has been uncertain. This issue was addressed in a large database of 58,820 residents who obtained their care from one major medical center [1]. The overall incidence of new cases of atrial flutter during a four-year period was 88 per 100,000 person-years, ranging from 5 per 100,000 in those less than 50 and 587 per 100,000 in those more than 80 years of age. Atrial flutter was 2.5 times more common in men. Based upon these data, it was estimated that the incidence of atrial flutter in the United States is 200,000 new cases per year.

The incidence of this arrhythmia, as with atrial fibrillation, is greatest when underlying heart disease is associated with left atrial enlargement, or left ventricular or biventricular failure [1,8,9].

CLINICAL MANIFESTATIONS

History and physical examination — Typical complaints include palpitations, fatigue, lightheadedness, and/or mild shortness of breath. Less common problems include significant dyspnea, angina, hypotension, anxiety, presyncope, or infrequently, syncope. These symptoms are in large part attributable the rapid heart rate. (See 'Hemodynamics' below.)

The purpose of the remainder of the history is to define the onset of the arrhythmia as well as its frequency and duration, the precipitating causes and modes of termination, the previous response to drug therapy, and the presence of heart disease or potentially reversible causes. (See 'Etiology and risk factors' above.)

The physical examination may reveal tachycardia, hypotension, diaphoresis, and evidence of congestive heart failure. (See "Heart failure: Clinical manifestations and diagnosis in adults", section on 'Physical examination'.) Occasionally, cardiac auscultation may reveal an irregular rhythm, abnormal valve sounds, or a gallop.

Flutter waves may be seen in the jugular veins at a rate consistent with the atrial rate.

Electrocardiogram — For patients in atrial flutter at the time of the electrocardiogram (ECG), it generally shows an atrial rate of about 300 beats per minute (range 240 to 340) (image 1A-C). Typical P waves are absent, and the atrial activity is seen as a sawtooth pattern (also called F waves) in leads II, III, and aVF.

The ECG hallmark of typical atrial flutter is discordance in flutter wave "direction" between the inferior leads and lead V1. In counterclockwise circuits, flutter waves are directly negative in the inferior leads but are positive in lead V1. In clockwise circuits, the opposite is true. These ECG rules are generally less reliable after atrial ablation or surgery.

There is typically 2:1 conduction across the atrioventricular (AV) node, particularly in counterclockwise typical atrial flutter; as a result, the ventricular rate is usually one-half the flutter rate in the absence of AV node dysfunction. Even atrial to ventricular rate ratios (eg, 2:1 or 4:1 conduction) are much more common than odd ratios (eg, 3:1 or 5:1) (see "Electrocardiographic and electrophysiologic features of atrial flutter"). Odd ratios probably reflect bilevel block in the AV node. On the other hand, a 1:1 response suggests catecholamine excess, parasympathetic withdrawal, the presence of antiarrhythmic drug therapy with Class IA or IC agents (table 1), or the existence of an accessory bypass tract.

The ECG may also identify left ventricular hypertrophy, pre-excitation, bundle branch block, or prior myocardial infarction (MI). Overlapping flutter waves may complicate assessment of the QT interval, repolarization pattern, and even the presence of T waves.

Echocardiogram — A transthoracic echocardiogram should be obtained in all patients with atrial flutter to evaluate the size of the right and left atria, the size and function of the right and left ventricles, and to detect possible pericardial or valvular heart disease or left ventricular hypertrophy.

Transthoracic echocardiography has a low sensitivity for detecting thrombus, and transesophageal echocardiography (TEE) is preferred for this purpose. TEE may play an important role in the selection of patients for cardioversion as it does in atrial fibrillation. (See "Role of echocardiography in atrial fibrillation".)

Hemodynamics — Several hemodynamic changes occur with atrial flutter; many of these are consequent to the rapid atrial and ventricular rates. These changes include an increase in the mean right and left atrial pressures, a reduction in right and left ventricular end-diastolic pressures, a decrease in systolic blood pressure, and an increase in diastolic pressure. The cardiac index is generally unaltered [10]. The reduction in left ventricular pressure is a result of the rapid heart rate, while the increase in atrial pressure is due, in part, to contraction against closed atrioventricular valves. The hemodynamic changes lead to the symptoms presented below. (See 'History and physical examination' above.)

Additional testing — Exercise testing is sometimes useful to reproduce exercise-induced atrial flutter, to evaluate for associated ischemic heart disease, or to determine the maximum heart rate with exercise, which can help guide medical therapy. Holter monitoring or event recorders

are used to identify the arrhythmia if symptoms are nonspecific, to identify triggering events, to detect associated atrial arrhythmias, and to determine average and peak heart rates. (See "Ambulatory ECG monitoring".)

Serum electrolytes, renal and hepatic function, and pulmonary and thyroid function tests can be ordered when searching for predisposing causes.

DIAGNOSIS

The diagnosis of atrial flutter is almost always secured by the observation of a characteristic pattern on the electrocardiogram, which includes the presence of continuous, regular atrial electrical activity. If there are sawtooth negative flutter waves in the leads II, III, and aVF, it is typical atrial flutter (image 1B), especially at a characteristic atrial rate of approximately 300 beats/min with a regular ventricular rate of about 150 beats/min in patients not taking atrioventricular nodal blockers. (See 'Introduction' above and 'Electrophysiologic classification' above.)

DIFFERENTIAL DIAGNOSIS

Occasionally, the pattern of atrial activity on the electrocardiogram is not convincing for atrial flutter and raises the possibility of atrial fibrillation, other supraventricular arrhythmia, or even electrical artifact. If the latter has been excluded, an electrophysiology study is necessary to determine if the arrhythmia is atrial flutter. (See "Invasive diagnostic cardiac electrophysiology studies" and "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation", section on 'Types of narrow QRS complex tachycardia'.)

COMPLICATIONS

Serious complications of atrial flutter include myocardial ischemia, dizziness or syncope, heart failure (with either preserved or reduced left ventricular systolic function), stroke, or systemic embolism. Control of the ventricular rate or reversion to normal sinus rhythm will improve or prevent the first three; anticoagulation is frequently used to decrease the risk of embolization. (See "Atrial fibrillation in adults: Use of oral anticoagulants".)

Atrial flutter with a rapid ventricular response is also an important cause of tachycardia induced cardiomyopathy. Control of the ventricular rate or reversion to normal sinus rhythm will improve many symptoms in these patients. Rate-control is critical to treat heart failure (HF)

symptoms, but in patients with tachycardia-mediated cardiomyopathy, rate plus rhythm control may be more effective in improving symptoms. Effective treatment of atrial flutter, most commonly with ablation, frequently leads to improvement and sometimes normalization of left ventricular function. (See "Arrhythmia-induced cardiomyopathy".)

GENERAL TREATMENT ISSUES

As is true for atrial fibrillation, there are four major issues that must be considered in the management of atrial flutter:

- Control of the ventricular rate
- Reversion to normal sinus rhythm (NSR)
- Maintenance of NSR
- Prevention of systemic embolization

The following discussion will provide a brief summary of these four treatment issues, each of which is discussed in detail separately.

Rate control in atrial flutter — Rate control in atrial flutter, as in atrial fibrillation, usually involves the administration of a non-dihydropyridine calcium channel blocker or a beta blocker. Digoxin is used less often because its major action is an enhancement of vagal tone, which is offset during exertion. The main indication is concurrent heart failure in which it is often given in combination with a beta-blocker. In general, it is more difficult to affect rate control in atrial flutter, as compared with atrial fibrillation. While up-titration of atrioventricular (AV) nodal blocking agents typically lowers the mean rate in atrial fibrillation, patients with atrial flutter are frequently "stuck" at 2:1 AV conduction.

Rarely, amiodarone may also be also as a rate control agent, particularly in acutely ill patients, but is not generally used long term due to the risk of potential side effects. Ablation therapy of the AV node and pacemaker implantation ("ablate and pace" strategy) is also rarely indicated, but is a treatment option in drug-refractory cases. (See "Control of ventricular rate in atrial flutter".)

Reversion to normal sinus rhythm — Due to the high rate of recurrence of atrial flutter in patients without a correctable cause, and because of its high success rate with low rate of complications, definitive treatment with radiofrequency catheter ablation is the preferred treatment for most patients. (See "Atrial flutter: Maintenance of sinus rhythm", section on 'RF catheter ablation'.)

It is less preferable for most patients to consider antiarrhythmic drugs because of potential for side effects. Class IA and IC drugs (table 1) risk causing rapidly conducted atrial flutter.

These drugs can slow the atrial flutter rate, and in the absence of AV nodal blocking agents, lead to 1:1 A:V conduction and paradoxically faster rates than baseline flutter (generally with 2:1 A:V conduction). Amiodarone and dronedarone can also slow the atrial flutter rate, but rarely lead to 1:1 A:V conduction because they also slow AV nodal conduction. All of these agents can "organize" atrial fibrillation and lead to "slow" atrial flutter, with the risk of 1:1 A:V conduction as described.

For hemodynamically stable patients who will not require urgent catheter ablation, watchful waiting under anticoagulation and rate control medicines may be reasonable, as atrial flutter may convert to sinus rhythm spontaneously.

Cardioversion is also reasonable if the patient has had no prior episodes of atrial flutter, or if they choose to decline ablation as an invasive first-line strategy. Synchronized internal or external direct current (DC) is preferred to antiarrhythmic drug cardioversion (using quinidine, procainamide, disopyramide, flecainide, propafenone, amiodarone, ibutilide, or dofetilide).

If pharmacologic reversion is deemed necessary, ibutilide, which is approved by the United States Food and Drug Administration only for intravenous use, is the drug of choice [11] (see "Restoration of sinus rhythm in atrial flutter"). It can revert atrial flutter to a sinus mechanism in approximately 60 percent of patients and is more effective than procainamide, sotalol, or amiodarone [11-13]. Ibutilide therapy carries a risk of QT prolongation and torsades de pointes. One report noted an 8.3 percent incidence of torsades de pointes [14]. Although torsades de points is usually not sustained, electrical cardioversion was required for sustained arrhythmia in 1.7 percent. As a result, the use of ibutilide requires continuous monitoring, resuscitative equipment including a defibrillator, and personnel trained in the use of electrical cardioversion and resuscitation. (See "Therapeutic use of ibutilide".)

In the setting of ventricular preexcitation, patients with atrial flutter and rapid ventricular rates should be treated with intravenous ibutilide or procainamide, as is recommended in the treatment of AF with ventricular preexcitation [15] (see "Restoration of sinus rhythm in atrial flutter"). Administration of intravenous adenosine, beta blockers, digoxin (oral or intravenous), nondihydropyridine calcium channel antagonists (oral or intravenous), or amiodarone in patients with Wolff-Parkinson-White syndrome and pre-excited AF and/or atrial flutter is potentially harmful because these drugs accelerate the ventricular rate [15]. (See "Treatment of arrhythmias associated with the Wolff-Parkinson-White syndrome".)

Atrial overdrive pacing is another method to convert atrial flutter to sinus rhythm. The most frequent settings for this approach are patients with pre-existing permanent pacemakers where pacing can be performed via the programmer or following cardiac surgery when pacing via a temporary epicardial (atrial) wire can be performed. Of course, the usual precautions should be taken with regard to anticoagulation or transesophageal echocardiography (TEE) if the duration of atrial flutter is beyond 24 to 48 hours or of unknown duration.

Maintenance of normal sinus rhythm — The rate of recurrence of atrial flutter is difficult to determine because most published data combine atrial flutter with atrial fibrillation. However, the recurrence rate is substantial. In one report, for example, 50 patients were followed for a mean of 3.5 years after cardioversion for chronic atrial flutter; prophylactic antiarrhythmic drugs were not given [16]. Sinus rhythm was maintained at six months and five years in 53 and 42 percent of patients, respectively. In a second study of 59 patients with lone atrial flutter, 75 percent developed recurrent or chronic atrial flutter [17]. (See "Atrial flutter: Maintenance of sinus rhythm".)

Antiarrhythmic drug mechanisms, as with atrial fibrillation, may suppress triggering premature atrial complex (PAC; also referred to a premature atrial beat, premature supraventricular complex, or premature supraventricular beat), which may require the use of class IA and IC drugs, beta blockers, and amiodarone, and/or to prolong the atrial refractory period with class III drugs.

However, because of the high rate of recurrence in patients without a correctable cause, and because of its high success rate, radiofrequency catheter ablation is generally preferable to long-term pharmacologic therapy in patients with typical atrial flutter. The isthmus between the inferior vena cava and the tricuspid annulus (cavotricuspid isthmus) is an obligatory route for typical flutter, and, as such, is the preferred anatomic target for ablation (figure 1). This applies to the common counterclockwise circuit, as well as the less common clockwise circuit. A meta-analysis of 21 studies demonstrated an ablation success rate for a single procedure of 91.7 percent and for multiple procedures of 97.0 percent [18]. (See "Atrial flutter: Maintenance of sinus rhythm" and "Atrial fibrillation and flutter after cardiac surgery", section on 'Pathogenesis' and "Atrial fibrillation: Catheter ablation", section on 'Arrhythmic complications'.)

In a meta-analysis of 48 studies (between 1996 and 2015) of patients undergoing catheter ablation for typical atrial flutter, who were followed for an average of 2.5 years, those without prior atrial fibrillation had a 23 percent incidence of new atrial fibrillation diagnosis. Not surprisingly, those with prior history of paroxysmal atrial fibrillation undergoing atrial flutter ablation had a much higher recurrence rate of 52 percent, highlighting the need for individualized risk assessment for long-term anticoagulation [19].

Prevention of systemic embolization — Sustained atrial flutter, while much less common than atrial fibrillation, carries an elevated thromboembolic risk. The frequency of thromboembolism and the importance of anticoagulation were illustrated in a report of 100 patients who were referred to an electrophysiology laboratory for cardioversion of atrial flutter that was present for at least six months: Six patients had a thromboembolic event that was attributable to atrial flutter [20]. The event occurred during atrial flutter or after cardioversion and none of the patients were receiving adequate anticoagulation. There were no embolic events in patients on adequate anticoagulation. (See "Embolic risk and the role of anticoagulation in atrial flutter", section on 'Long-term flutter'.) One problem with interpreting these data is that many patients with chronic atrial flutter also have periods of atrial fibrillation.

Our approach to anticoagulation in patients with atrial flutter is identical to that for atrial fibrillation [21]. Although the risk of systemic embolism is probably a little lower compared with atrial fibrillation, it is appropriate to use anticoagulation in atrial flutter as similar to atrial fibrillation. Risk stratification using the CHA_2DS_2-VASc scoring system should be completed prior to deciding on the use of oral anticoagulation. (See "Atrial fibrillation in adults: Use of oral anticoagulants".)

Commonly, four weeks after successful ablation of isolated typical atrial flutter (ie, no prior atrial fibrillation history), anticoagulation is discontinued. However, in patients with prior atrial fibrillation history, anticoagulation should be continued long term based on the CHA_2DS_2-VASc scoring system.

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Atrial fibrillation" and "Society guideline links: Arrhythmias in adults".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5^{th} to 6^{th} grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10^{th} to 12^{th} grade reading

level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topics (see "Patient education: Atrial flutter (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Definition** – Atrial flutter is an abnormal cardiac rhythm characterized by rapid, regular atrial depolarizations at a characteristic rate of approximately 300 beats/min and a regular ventricular rate of about 150 beats/min (image 1B). (See 'Introduction' above and 'Electrophysiologic classification' above.)

- **Comorbid conditions** – Atrial flutter is unusual in patients without heart disease. It frequently coexists with atrial fibrillation and may be associated with valvular heart disease, cardiomyopathy, post-cardiac surgery, pericardial disease including pericardiotomy, prior heart surgery, and acute or chronic pulmonary diseases. (See 'Etiology and risk factors' above.)

- **Electrophysiologic classification** – Distinguishing typical from atypical atrial flutter has useful treatment implications, particularly the high success rate of catheter ablation in typical atrial flutter. (See 'Electrophysiologic classification' above.)

- **Clinical manifestations** – The clinical manifestations are similar to those of atrial fibrillation. (See 'Clinical manifestations' above.)

  - **Symptoms** – Typical complaints include palpitations, fatigue, lightheadedness, and/or mild shortness of breath.

  - **Physical examination** – This may reveal tachycardia, hypotension, diaphoresis, and evidence of congestive heart failure. Occasionally, cardiac auscultation may reveal an irregular rhythm, abnormal valve sounds, or a gallop. Flutter waves may be seen in the jugular veins at a rate consistent with the atrial rate.

- **Diagnosis** – The diagnosis can usually be made from a 12 lead electrocardiogram. (See 'Electrocardiogram' above and 'Diagnosis' above.)

- **Treatment strategies** – The management of rate-control and anticoagulation strategies for prevention of systemic thromboembolism are similar to those used for atrial fibrillation. However, long-term antiarrhythmic medications are infrequently used given the limitations of pharmacologic therapy and high rate of success of ablation for typical atrial flutter. (See 'General treatment issues' above.)

- **Anticoagulation** – Although the risk of systemic embolism is probably somewhat lower compared with atrial fibrillation, it is still appropriate to use anticoagulation in atrial flutter. Risk stratification using the CHA_2DS_2-VASc scoring system should be completed prior to deciding on the use of oral anticoagulation. (See 'Prevention of systemic embolization' above and "Atrial fibrillation in adults: Use of oral anticoagulants".)

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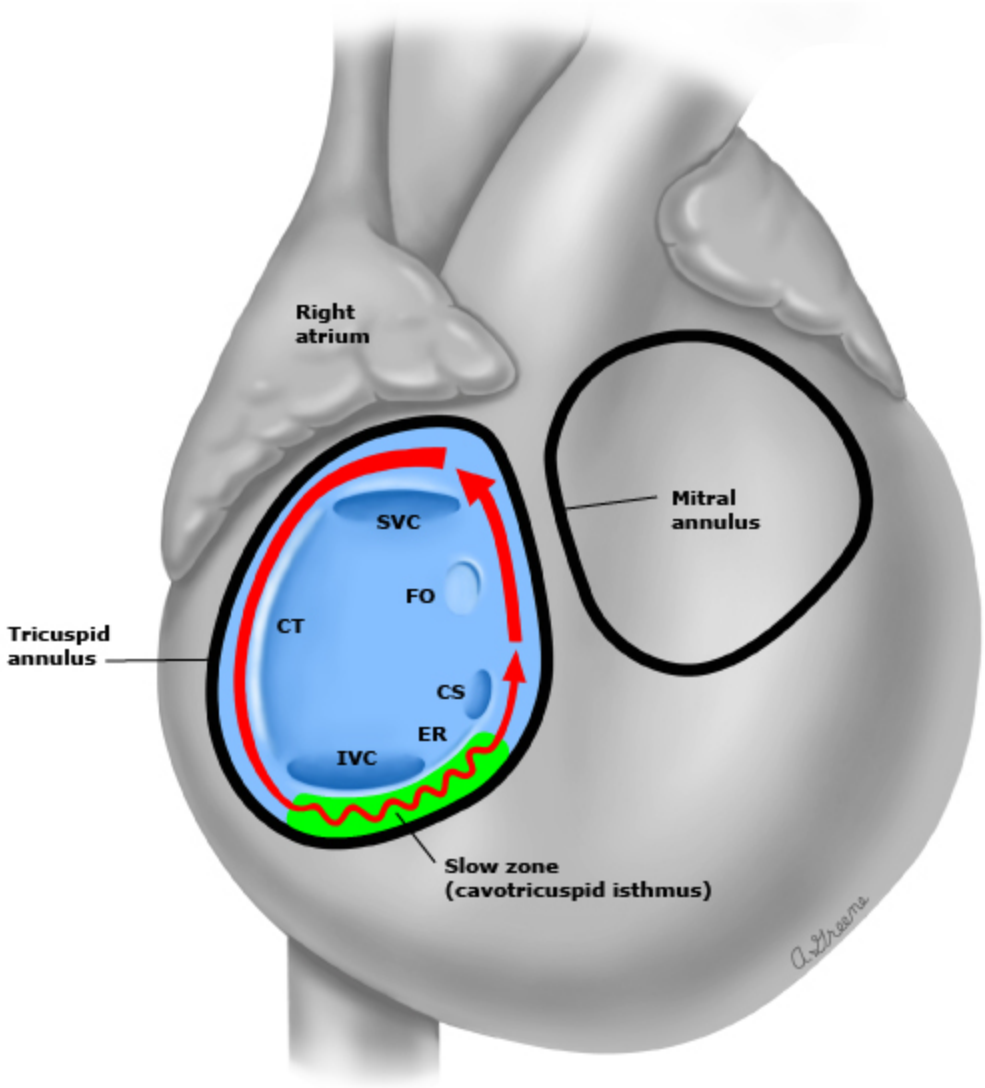
Topic 1048 Version 45.0

GRAPHICS

Reentrant circuit of typical atrial flutter within the right atrium



Schematic representation of reentrant circuit (red arrow) of typical (type 1) atrial flutter. The reentrant impulse rotates in a counterclockwise direction around the tricuspid annulus. The crista terminalis (CT) and eustacian ridge (ER) serve as lines of block, preventing the impulse from short-circuiting the annulus. Ablation is performed in the isthmus between the IVC and TA, which is an obligatory part of the circuit.

SVC: superior vena cava; CS: coronary sinus; IVC: inferior vena cava; FO: foramen ovale

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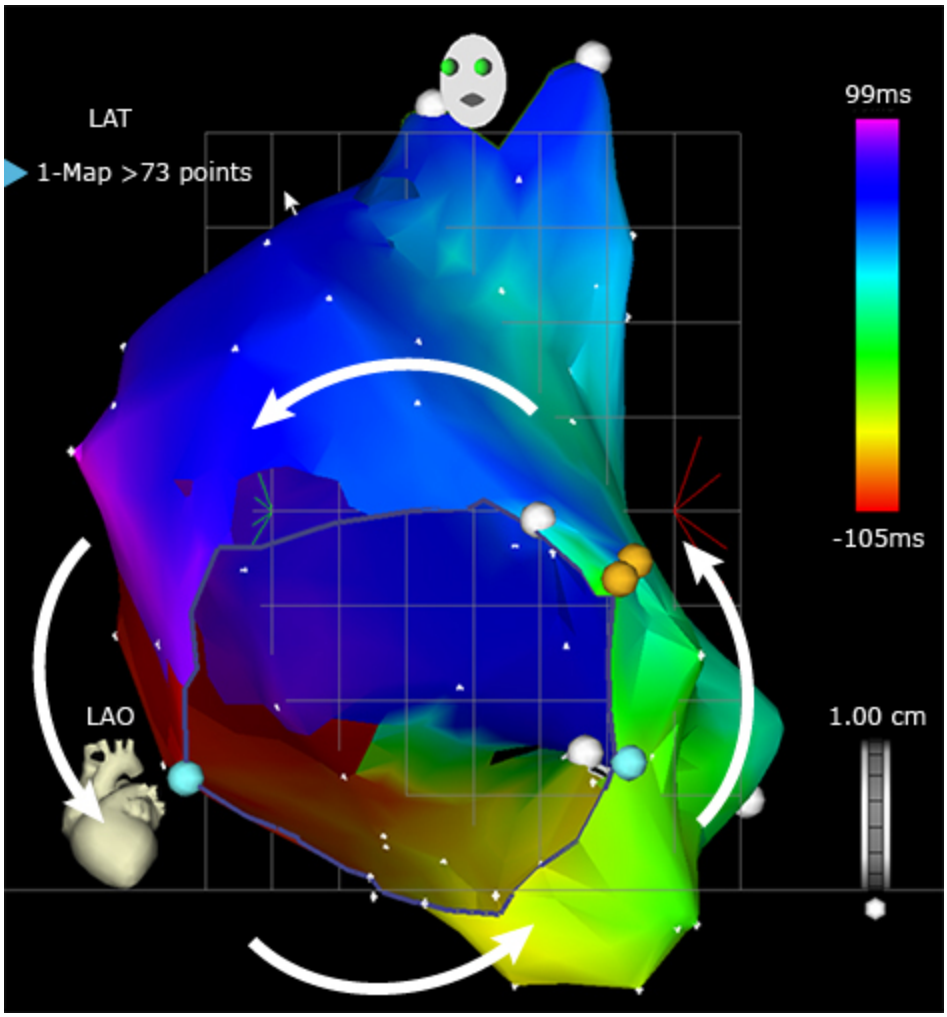
Graphic 52904 Version 5.0

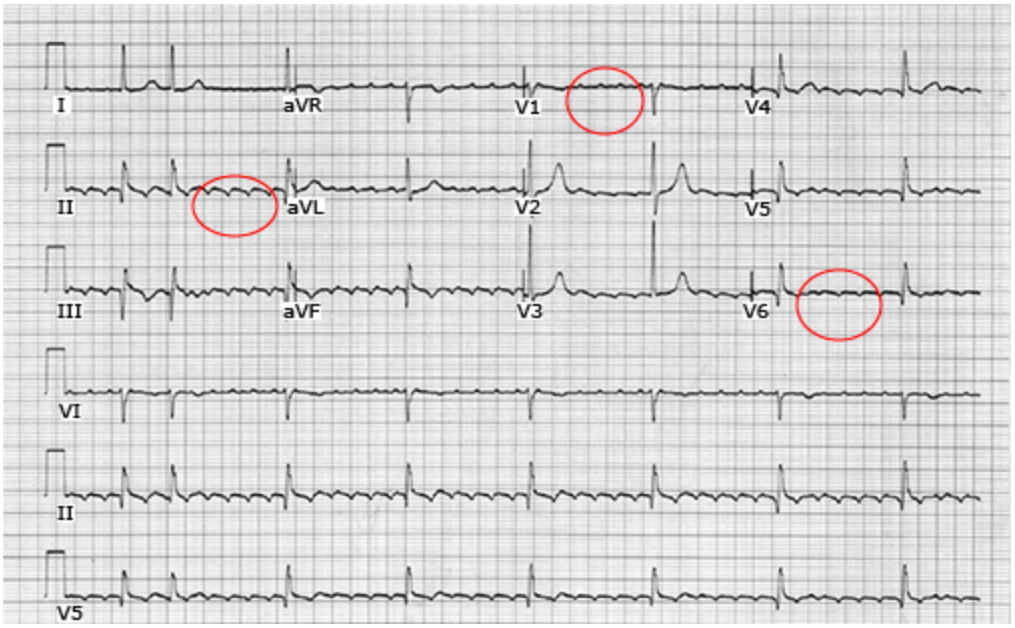
Electrical activation of the right atrium in atrial flutter



Electroanatomical (3D) mapping using the CARTO system. The right atrium (RA) is depicted in the LAO projection. The circular "cutout" area in the forefront of the picture represents the tricuspid valve annulus. The red area is the inferolateral RA, the yellow-green area at the bottom of the picture is near the inferior vena cava, the yellow dots are at the region of the His bundle (AV node), and the blue area is the superior RA. The top of the picture is the superior vena cava region. The color (red-orange-yellow-green-blue-purple) represents the direction of electrical activation in the RA during typical (counterclockwise) atrial flutter. Each flutter wave seen on ECG represents a single electrical activation of this entire circuit.

Graphic 89570 Version 1.0

ECG of counterclockwise typical atrial flutter



Sawtooth-like oscillations of baseline between flutter waves, best seen in the inferior leads.

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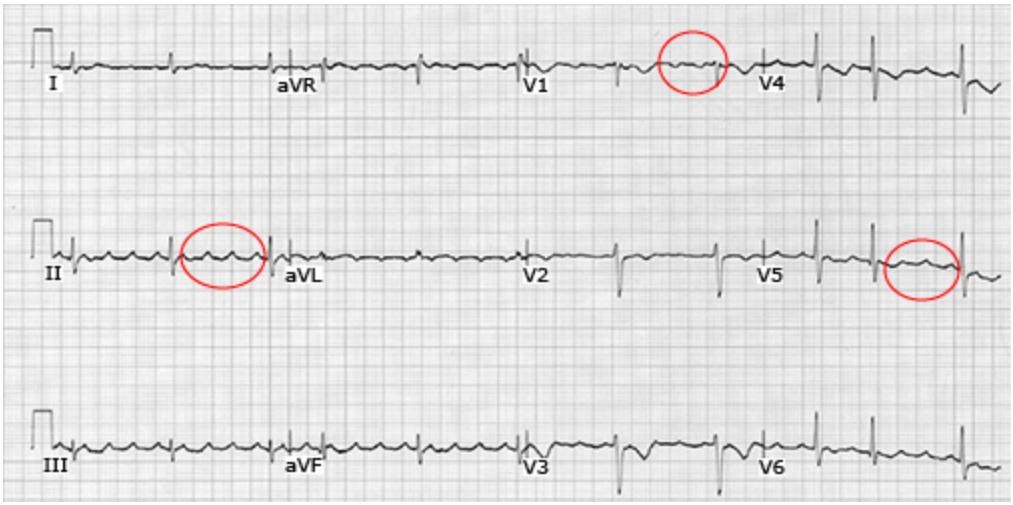
* Negative flutter waves in II, III, aVF, V6. Positive flutter waves in V1.

ECG: electrocardiogram.

Graphic 89571 Version 2.0

ECG of clockwise typical atrial flutter



Sawtooth-like oscillations of baseline between flutter waves, best seen in inferior leads.

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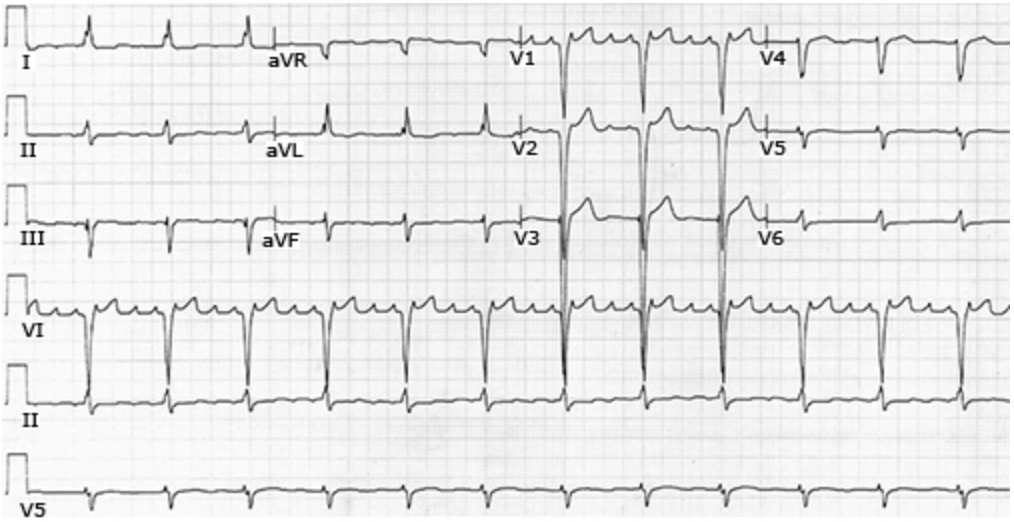
* Positive flutter waves in II, III, aVF, V6. Negative flutter waves in V1.

ECG: electrocardiogram.

Graphic 89576 Version 2.0

ECG of atypical atrial flutter



Note the lack of classic sawtooth flutter waves in the inferior leads II, III, and aVF.

ECG: electrocardiogram.

Graphic 89577 Version 4.0

Revised (2018) Vaughan Williams classification of antiarrhythmic drugs abridged table

Class 0 (HCN channel blockers)
Ivabradine
Class I (voltage-gated Na+ channel blockers)
Class Ia (intermediate dissociation):
▪ Quinidine, ajmaline, disopyramide, procainamide
Class Ib (rapid dissociation):
▪ Lidocaine, mexilitine
Class Ic (slow dissociation):
▪ Propafenone, flecainide
Class Id (late current):
▪ Ranolazine
Class II (autonomic inhibitors and activators)
Class IIa (beta blockers):
▪ Nonselective: carvedilol, propranolol, nadolol
▪ Selective: atenolol, bisoprolol, betaxolol, celiprolol, esmolol, metoprolol
Class IIb (nonselective beta agonists):
▪ Isoproterenol
Class IIc (muscarinic M2 receptor inhibitors):
▪ Atropine, anisodamine, hyoscine, scopolamine
Class IId (muscarinic M2 receptor activators):
▪ Carbachol, pilocarpine, methacholine, digoxin
Class IIe (adenosine A1 receptor activators):
▪ Adenosine
Class III (K+ channel blockers and openers)
Class IIIa (voltage dependent K+ channel blockers):
▪ Ambasilide, amiodarone, dronedarone, dofetilide, ibutilide, sotalol, vernakalant

Class IIIb (metabolically dependent K+ channel openers):
▪ Nicorandil, pinacidil
Class IV (Ca++ handling modulators)
Class IVa (surface membrane Ca++ channel blockers):
▪ Bepridil, diltiazem, verapamil
Class IVb (intracellular Ca++ channel blockers):
▪ Flecainide, propafenone
Class V (mechanosensitive channel blockers):
No approved medications
Class VI (gap junction channel blockers)
No approved medications
Class VII (upstream target modulators)
Angiotensin converting enzyme inhibitors
Angiotensin receptor blockers
Omega-3 fatty acids
Statins

HCN: hyperpolarization-activated cyclic nucleotide-gated; Na: sodium; K: potassium; Ca: calcium.

Graphic 120433 Version 3.0