Atrial flutter

The organised cousin of atrial fibrillation

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Atrial flutter shares many characteristics with atrial fibrillation, but it is important to distinguish between these arrhythmias because of the varying underlying electrophysiology and response to therapy.

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Key points

- **Although electrophysiologically different from atrial fibrillation (AF), atrial flutter (AFL) shares many characteristics, especially in terms of its pharmacological medical management.**
- **AFL can be classified as 'typical' or 'atypical' based on its anatomical and electrophysiological characteristics.**
- **Treatment of AFL focuses on identifying and treating reversible causes, controlling the ventricular rate and/or achieving sinus rhythm as well as reducing the risk of systemic embolisation.**
- **Radiofrequency ablation is generally regarded as a first-line therapy for typical flutter.**

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Atrial flutter (AFL) is one of the most common atrial arrhythmias seen in clinical practice, with an incidence of 88 per 100,000 person-years over four years.[1] Although electrophysiologically different from atrial fibrillation (AF), it shares many characteristics, especially in terms of its pharmacological medical management.

Classification and electrophysiology

Most typically AFL is characterised by regular atrial depolarisations at an atrial rate of approximately 300 beats/minute and a ventricular rate of half the atrial rate (150 beats/minute) in the absence of rate control pharmacotherapy or atrioventricular (AV) nodal disease. Various classifications can be applied to AFL, but the most common and clinically practical is to consider flutter as either 'typical' or 'atypical' based on its anatomical and electrophysiological characteristics (Table).[2]

Typical AFL, the most prevalent form of flutter, involves a macro re-entrant electrical circuit in the right atrium that traverses (and is dependent on) the cavotricuspid isthmus (right atrial tissue between the inferior vena cava orifice and the tricuspid valve annulus).[3-4] This AFL represents over 90% of all cases of flutter. In typical AFL, the circuit occurs in a counterclockwise rotation around the tricuspid valve. Much less often, the AFL circuit rotates clockwise around the tricuspid valve – referred to as 'reverse typical flutter'.

On initiation of AFL (e.g. with an atrial ectopic or rapid atrial activation), the atrial electrical activation travels across the cavotricuspid isthmus, with subsequent activation through the low septum and anterosuperiorly up the septal and posterior walls of the right atrium. It then descends over the lateral free wall to the crista terminalis. As the impulse travels along the re-entrant circuit, the initially excited (depolarised) area recovers its excitability and can

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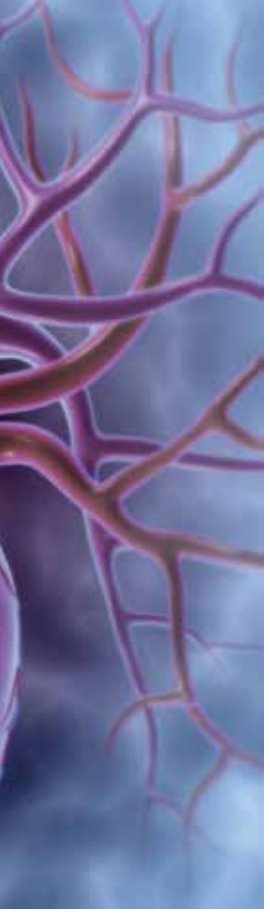
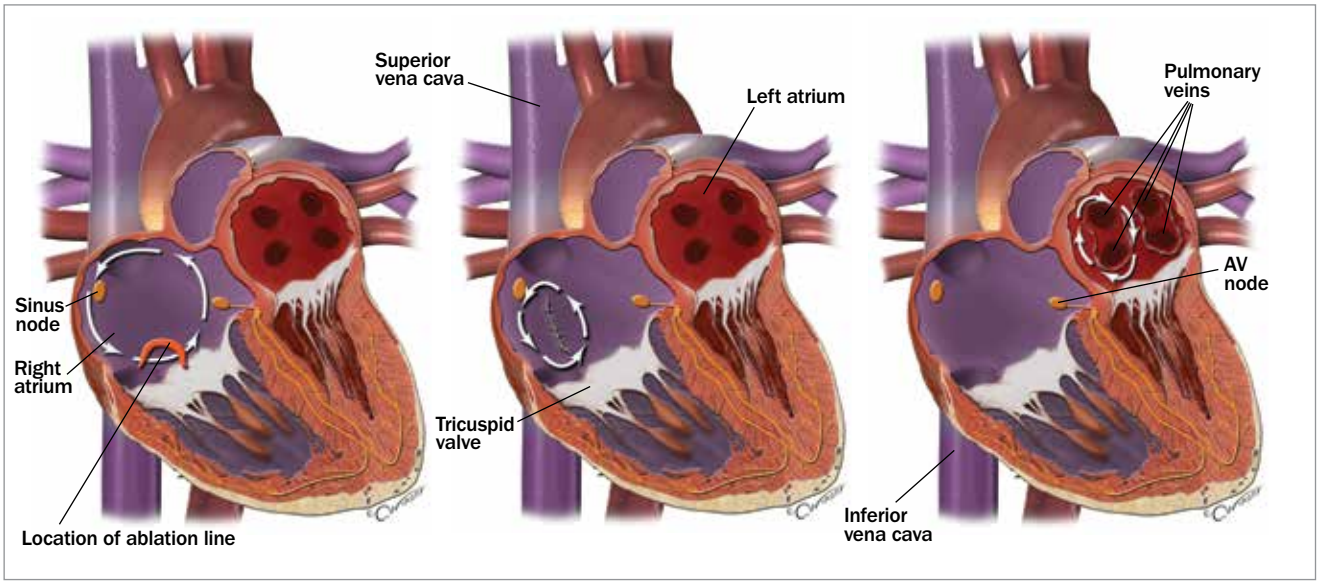


Table. Classification and characteristics of atrial flutter (AFL)

Characteristic	Typical AFL	Atypical AFL
Epidemiology	• Common	• Infrequent
Anatomy	• Macro re-entrant atrial tachycardia involving the cavotricuspid isthmus	• Intra-atrial re-entrant tachycardia that does not involve the cavotricuspid isthmus • It may revolve around a prior scar or other anatomical substrates
Rate • Atrial rate • AV nodal conduction	 • 300 beats/min • Usually 2:1	 • Variable (often slower than 300 beats/min) • Usually 2:1
ECG • P waves • Flutter waves on ECG	 • Absent • Sawtooth pattern • F waves usually do not have an isoelectric interval between them • F waves may have a negative axis in the inferior leads	 • Variable • May not appear as a sawtooth pattern • F waves may have an isoelectric interval between them • F waves are more likely to have a positive axis in the inferior leads

depolarise again creating a continuous circuit (Figure 1a). This counterclockwise circuit is characterised on an ECG by negatively deflected flutter waves (F waves) in the inferior leads, which appear as a typical sawtooth pattern, absent P waves and lack of isoelectric interval between F waves (Figure 2a). The ventricular rate is regulated by the rate at which the impulses exit the AV node and the variability of AV conduction (e.g. 2:1, 3:1, 4:1, etc). A 1:1 conduction response is rare, but can be seen in the context of catecholamine excess, parasympathetic withdrawal, and in the presence of class 1a or 1c antiarrhythmic agents, hyperthyroidism or an accessory pathway with pre-excitation.

All other types of AFL can be classified as 'atypical'. These flutters involve a re-entrant circuit that may involve atypical isthmuses between atrial incisions, scarring (e.g. from previous cardiac surgery or ablation procedures; Figures 1b and 1c) and natural (electrically silent) barriers. They can originate in the right atrium (e.g. lower loop re-entry, upper loop re-entry, fossa ovalis and superior vena cava) or the left atrium (e.g. perimitral, peripulmonary veins, posterior wall



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Figures 1a to c. Schematic representation of various flutter circuits. a (left). Typical counterclockwise atrial flutter (AFL) and location of the ablation line used to treat typical AFL. b (middle). Atypical right AFL located around an atriotomy scar. c (right). Left AFL following a previous vein isolation procedure.

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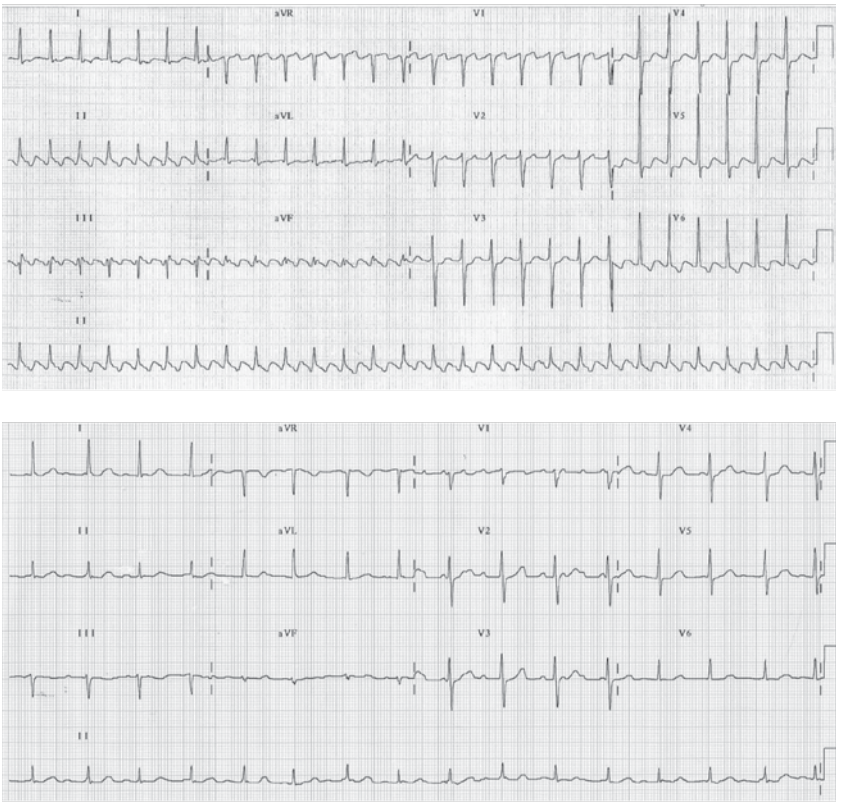

Figures 2a and b. a (top). ECG from a patient with counterclockwise typical atrial flutter. The flutter (F) waves are prominently negative (lead II) in a counterclockwise flutter. b (bottom). ECG from a patient with atypical flutter. Note the multiple P waves (seen in leads V1-V3) and the lack of a typical sawtooth pattern.

and septum). Previous pulmonary vein isolation (to treat AF) in particular, is a key risk factor for left-sided atypical flutters and 'small-circuit' flutters with a circuit diameter of less than 2 cm. Figure 2b shows an ECG of one type of atypical flutter, with multiple P waves and lack of the sawtooth pattern as seen in atypical flutter, but other patterns with different looking P waves can also be seen.

Risk factors

Lone AFL in the absence of identifiable risk factors is relatively uncommon; however, the incidence does increase with age. Factors that predispose to AFL include the following:

- cardiac disease – cardiac failure, left atrial enlargement, mitral or tricuspid valve disease, pericarditis, ischaemia, hypertension, AF, prior cardiac surgery and prior ablation of AF
- pulmonary disease – chronic obstructive pulmonary disease (COPD), bronchiectasis, obstructive sleep apnoea and pulmonary embolism
- other factors such as obesity, thyrotoxicosis, drugs (e.g. flecainide) and postoperative states.

Of these, heart failure and COPD are arguably the two principal risk factors, causing 16% and 12% of cases, respectively.[1]

It is also common for AFL to coexist with AF in the same patient. In this regard, risk factors that predispose to AF, also predispose to AFL. Antiarrhythmic medications (such as flecainide, amiodarone and sotalol) used to treat AF (which is inherently a disorganised atrial rhythm) can 'organise' the atrial rhythm into AFL, which may then be seen on an intermittent or a more persistent basis.

Haemodynamic effects

AFL can result in various haemodynamic changes, mostly as a result of rapid atrial and ventricular rates. These include:

- increased mean atrial pressure partly due to contraction against a closed AV valve
- reduction in ventricular end-diastolic pressures due to the rapid rate
- decreased blood pressure.

Clinical manifestations and complications

AFL often causes palpitations, dyspnoea, dizziness, chest pain, syncope and fatigue, although, as is the case with AF, patients can be asymptomatic. Other complications include an increased risk of thromboembolism (most commonly strokes), myocardial ischaemia and tachycardia-induced cardiomyopathy. The thromboembolism rate appears to be similar to that of AF, and many patients have alternating periods of AF. Atrial thrombi occur almost exclusively within the left atrial appendage. Studies have shown that 1.6 to 11% of patients develop left atrial thrombi as seen on transoesophageal echocardiography (TOE) after experiencing AFL for 28 to 36 days' duration in the absence of AF or valvular heart disease.[5,6]

Investigations

A transthoracic echocardiogram (TTE) should be performed routinely to evaluate the left ventricular size and function, as well as size of atria, and to assess for valvular heart disease. Serum electrolytes, full blood count and thyroid function tests should also be ordered to exclude predisposing causes. Assessment of myocardial ischaemia (e.g. by stress echocardiography or myocardial perfusion imaging) should be considered in patients at risk of coronary artery disease. Other investigations to consider include sleep studies and spirometry, depending on the clinical situation.

Management of atrial flutter

Treatment of patients with AFL focuses on identifying and treating reversible causes, controlling the ventricular rate and/or achieving sinus rhythm as well as reducing the risk of systemic embolisation.

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Rate control

Ventricular rate control in AFL (in the absence of AV node disease) can be notoriously challenging, as the slower atrial rate produces less refractoriness within the AV node than that occurring in AF. Medications to slow AV node conduction and increase AV nodal refractoriness include non-dihydropyridine calcium channel blockers such as verapamil or diltiazem and beta blockers. Digoxin is generally only used as adjunctive therapy rather than sole therapy, as its major action is enhancement of vagal tone and it often cannot achieve adequate rate control during exercise, especially in younger patients.

Higher doses of a single drug and in many cases multiple medications may be needed to achieve adequate rate control. Combination therapy at conventional doses is often needed to minimise toxicity of any individual drug.

Achieving sinus rhythm

Reversion to sinus rhythm is generally the cornerstone of AFL management. Urgent cardioversion may be needed in the event of haemodynamic compromise or identification of an accessory pathway with ventricular pre-excitation (Wolff-Parkinson-White syndrome). Elective cardioversion can also be considered to achieve sinus rhythm in new-onset or symptomatic AFL once patients are adequately anticoagulated or have had TOE to exclude an atrial thrombus.

Electrical cardioversion is preferred to pharmacological cardioversion as AFL is very sensitive to electrical cardioversion (96% success rate), with generally only low energy direct current shocks required for reversion.[6] Antiarrhythmic therapy has a much lower cardioversion success rate and can be associated with significant toxicity. The cardioversion success rate is approximately 19% for sotalol and 29% for amiodarone.[7,8] Class 1 antiarrhythmic agents should be used with extreme caution as they can slow the AFL rate, and in patients who are not taking AV nodal blocking agents can lead to 1:1 AV conduction, with paradoxically faster ventricular rates than those seen within the original flutter. Although AFL can be considered a 'supraventricular tachycardia', there is no role for adenosine in terminating this arrhythmia.

After cardioversion, antiarrhythmic medications are generally continued to maintain sinus rhythm; however, the long-term recurrence rate of AFL is high, at 58 to 75% at five years.[9-11] Predictors of recurrence include large left atrial size, prolonged duration of flutter and previous cardioversion.

Typical flutter

Definitive treatment with radiofrequency ablation is generally regarded as a first-line therapy for typical flutter. The cavotricuspid isthmus is the anatomical target for ablation in this arrhythmia. An ablation catheter is positioned at the isthmus via the femoral approach, generally under fluoroscopic guidance. With ablation, bidirectional block (clockwise and counterclockwise) is created across the isthmus, preventing further AFL in this region. Ablation is highly effective in typical flutter, with a meta-analysis demonstrating a success rate of 91.7% for a single procedure and 97.0% for multiple procedures.[12] Restoration of sinus rhythm can result in improvement in left ventricular ejection fraction in those with tachycardia-induced cardiomyopathy and reduced need for antiarrhythmic drugs.[13,14]

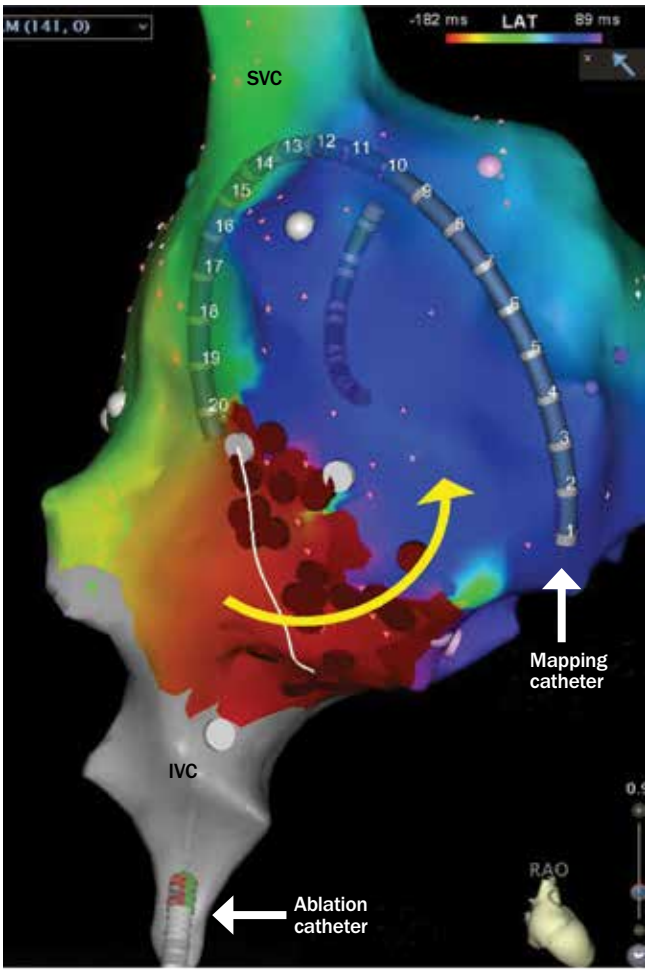

Figure 3. 3D activation map demonstrating ablation of atypical right atrial flutter due to a previous atriotomy scar. Propagation of the electrical signal within the right atrium (in the direction of the yellow arrow) is shown, created using a CARTO mapping system. Red circles represent areas of radiofrequency ablation to treat this flutter.

Abbreviations: IVC = inferior vena cava; SVC = superior vena cava.

First-line therapy with catheter ablation results in fewer hospitalisations (11 *vs* 56%), greater maintenance of sinus rhythm and improved quality of life compared with antiarrhythmic pharmacotherapy at 12 months.[14] The complication rates following radiofrequency ablation for typical AFL are generally low.[12] Patients with AFL do remain at risk of other atrial arrhythmias and approximately 7 to 44% of patients develop AF or atypical AFL,[15,16] which is likely to be due to underlying abnormalities in the atria. As AFL often coexists with AF, a pulmonary vein isolation (to treat AF) may be required in a number of patients, and in some cases this ablation also abolishes AFL.

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Atypical flutter

Treatment of atypical flutter involves the use of the same medications as those used for typical AFL. Ablation of atypical AFL is technically a more complex procedure. It is generally considered only for patients with recurrent, poorly tolerated flutter, or in whom pharmacotherapy has failed. The ablation requires exact identification of the flutter circuit using three-dimensional cardiac mapping equipment and various electrophysiological techniques. These include activation mapping (identification of the direction of wavefront propagation through a series of catheters; Figure 3) and entrainment mapping (pacing from various atrial sites to identify which are within the re-entrant circuit). The success rates of ablation for atypical AFL vary depending on the aetiology and location of the flutter circuit.

Coexisting atrial flutter and atrial fibrillation

The management of patients with coexistent AFL and AF involves a similar pharmacological approach. Ablation can be more extensive in these patients because both arrhythmias may need to be targeted. Patients randomised to pulmonary vein isolation with or without cavotricuspid isthmus ablation were shown to be more likely to be arrhythmia free (64%) at 21 ±9 months than those randomised to cavotricuspid isthmus ablation alone (19%) in one single-blind randomised trial.[17]

Anticoagulation

Anticoagulation should be prescribed for patients with AFL as it would be for those with AF and guided by the CHA2DS2-VASc score, given the similar thromboembolic risk and often alternating periods of AF. Anticoagulation with warfarin (target INR 2 to 3) has been recommended to prevent thromboembolisation. There are limited data about the use of novel oral anticoagulants (dabigatran, rivaroxaban and apixaban) in AFL, but they are likely to have similar efficacy to that in AF. Despite the ease of administration, it is more difficult to monitor compliance with these novel agents and there is a risk of thrombus formation with interrupted therapy. For this reason, some centres still recommend TOE before cardioversion in patients taking these novel agents to exclude a left atrial thrombus. For patients scheduled for electrical cardioversion or radiofrequency ablation, it is generally recommended that they have at least four weeks of adequate anticoagulation before the procedure.

Following cardioversion or catheter ablation, anticoagulation should be continued for at least one month and often indefinitely in those with recurrent flutter or other atrial arrhythmias associated with increased thromboembolic risk.

Conclusion

AFL is a common atrial arrhythmia, often seen in conjunction with AF. Although it shares many characteristics with AF, it is important to distinguish between these arrhythmias because of the varying underlying electrophysiology and response to therapy. Ablation should be considered as first-line therapy for typical AFL, and anticoagulation remains a cornerstone of management to reduce thrombotic complications. **CT**

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