BMJ Best Practice

Atrial flutter

The right clinical information, right where it's needed

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Summary

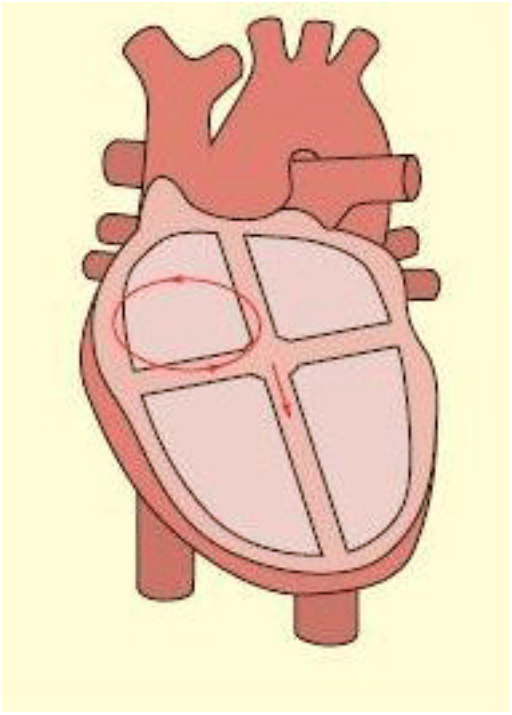
- In the typical form, this entity is characterised electrocardiographically by flutter waves, which are a saw-tooth pattern of atrial activation, most prominent in leads II, III, aVF, and V1.
- Atrial rates are typically above 250 bpm and up to 320 bpm.
- Ventricular rates range from 120 to 160 bpm, and most characteristically 150 bpm, because an associated 2:1 AV block is common.
- This rhythm is commonly associated with atrial fibrillation, into which it may degenerate. Atrial fibrillation may also convert to atrial flutter.
- If the rhythm persists despite treatment of the underlying cause or in the absence of a reversible cause, electrical cardioversion is used to terminate the arrhythmia.
- If electrical cardioversion is unavailable or not acceptable to the patient, pharmacological cardioversion may be attempted.
- Because of alterations in atrial activation, the ECG often fluctuates between both rhythms in the same patient.

BASICS

Definition

Typical atrial flutter (anti-clockwise cavotricuspid isthmus-dependent atrial flutter) is a macro-reentrant atrial tachycardia with atrial rates usually above 250 bpm up to 320 bpm. It results from organised electrical activity in which large areas of the atrium take part in the reentrant circuit. The typical form depends on the so-called cavotricuspid isthmus for part of the circuit: tricuspid annulus as the anterior boundary and the crista terminalis/eustachian ridge as the posterior boundary, as well as the endocardial cavity of the right atrium. The term anti-clockwise refers to the direction of activation when the tricuspid annulus is viewed en face, whereby activation occurs up the septum, down the right atrial free wall in an anti-clockwise fashion. Characteristic features on ECG are negatively directed saw-tooth atrial deflections (f waves) seen in leads II, III, and aVF, with positively directed deflections in lead V1.[1]
[Fig-1]

This rhythm is closely related to atrial fibrillation.[2] [3] [4]
[Fig-2]



Atrial flutter typically involves a circuit in the right atrium

From: Cox D, Dougall H. Student BMJ. 2001;9:399-442; used with permission

Epidemiology

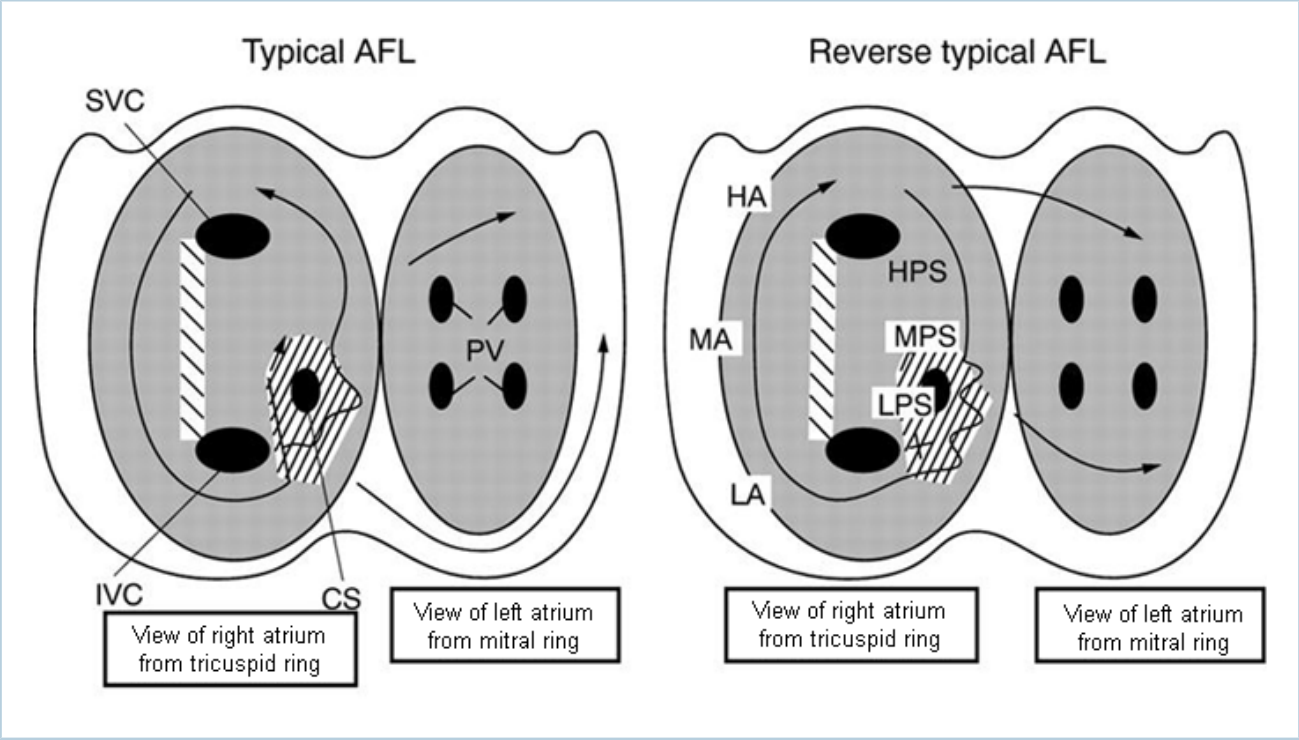
The overall incidence has been reported as 88/100,000 person-years, with increasing rates with older age. The incidence is 5/100,000 in people <50 years of age and 587/100,000 in those >80 years of age.[5] It occurs 2.5 times more frequently in men than in women. It is seen in 25% to 35% of patients with atrial fibrillation. Typical atrial flutter tends to be more common (diagnosed in 90% of patients in a population-based epidemiological study). Most cases (>98%) are associated with an identifiable predisposing event or a pre-existing comorbidity.[5]

Aetiology

Atrial flutter generally results from structural or functional conduction abnormalities of the atria. Structural abnormalities include atrial dilation due to a number of processes (see Risk Factors); incisional scars from prior atrial surgery, particularly for congenital heart disease; prior atrial ablation sites; and idiopathic fibrosis within the atrium. In addition, it can be precipitated by toxic and metabolic conditions such as thyrotoxicosis, alcoholism, or pericarditis. Patients taking anti-arrhythmics for chronic suppression of atrial fibrillation may convert to atrial flutter, noted most commonly with Vaughan Williams class Ic drugs (flecainide and propafenone) and with class Ia (disopyramide, procainamide, quinidine) and amiodarone.[6] [7] Occasionally, it can be congenital.[8]

Pathophysiology

The typical form of atrial flutter is a prototypic macro-reentrant arrhythmia, in which the reentrant wavefront travels up the interatrial septum and down the right atrial free wall ('typical' form) or vice versa ('reverse typical' form). The lateral anatomical boundaries are critical to the development and maintenance of the circuit. One of these boundaries is the tricuspid valve annulus and is fixed or anatomical. The other is generally a functional line of block between the venae cavae.[9] Atypical flutters are seen when the cavotricuspid isthmus is not part of the circuit, and are characterised by a continuously undulating pattern by ECG that does not fit strict criteria for the typical and reverse typical forms of atrial flutter. This is a limitation in the mechanistic/anatomical classification scheme, as the exact mechanism can be determined only by electrophysiological mapping studies and not from the ECG alone. Clinical presentations and electrophysiological features of atypical atrial flutter and other types of atrial tachycardia can overlap.[2]



Left panel: atrial activation in typical atrial flutter (AFL). Right panel: 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

From: Waldo AL. Heart. 2000;84:227-227; used with permission

Classification

American College of Cardiology/American Heart Association/Heart Rhythm Society classification of atrial flutter/atrial tachycardias[4]

Atrial flutter is a macro-reentrant atrial tachycardia with constant P wave/flutter morphology with a rate usually >250 bpm. It is distinguishable from focal atrial tachycardia, which has discrete P waves with an intervening isoelectric segment. Focal atrial tachycardia is caused mechanistically by micro-reentry or increased automaticity and generally has atrial rates in the range of 100-250 bpm.

Cavotricuspid isthmus dependent (typical atrial flutter):

- Anti-clockwise atrial flutter with ECG flutter waves characterised by:
    - Negative deflection in leads II, III, aVF
    - Positive deflection in lead V1.
- Clockwise atrial flutter (reverse typical atrial flutter) with ECG flutter waves characterised by:
    - Positive deflection in leads II, III, aVF
    - Negative deflection in lead V1.

Non cavotricuspid isthmus dependent (atypical atrial flutter):

- Re-entry that does not depend upon conduction through the cavotricuspid isthmus
- Circuit is typically defined by atrial scars due to prior heart surgery, ablations, or idiopathic causes
- Location determines ablation approach and risks
- Multiple sites of re-entry may be present
- Can occur in both the left and right atria.

Secondary prevention

Anticoagulation is recommended for persistent or paroxysmal atrial flutter, as it is for atrial fibrillation, for the prevention of thromboembolic events including acute stroke.

Case history

Case history #1

A 77-year-old man presents with complaints of palpitations and new shortness of breath, especially with exertion. He has a history of rheumatic fever in childhood. He has been told he has a murmur but does not recall having had an echocardiogram. He is otherwise healthy.

Case history #2

A 76-year-old man presents with progressive symptoms of dyspnoea and increasing peripheral oedema. He denies palpitations. He has a history of CHF from hypertensive heart disease. He reports that he is taking his medications as directed and has had no recent medication or dietary changes.

Step-by-step diagnostic approach

Patients with recent-onset atrial flutter generally complain of palpitations, fatigue, light-headedness, chest pain, and/or dyspnoea. In addition, exercise intolerance, worsening heart failure symptoms, and pulmonary complaints can also occur. Less commonly, hypotension, syncope, or embolic events can be the presenting symptoms.[4] The history should be directed at identifying the onset, frequency, and duration, and the presence of structural heart disease or precipitating cause.

History

In about 60% of patients, atrial flutter occurs as part of an acute disease process, and resolves with that process.[5] Identifying that underlying cause or trigger is therefore central to management. The history should establish whether there are any concurrent symptoms of an acute disease process, such as fever and cough in the case of pneumonia. A thorough past medical history should identify whether there is a history of any risk factors:

- Hypertensive heart disease
- Heart failure
- Asthma
- COPD
- Hyperthyroidism
- Mitral or tricuspid valve disease
- Atrial fibrillation
- Structural heart disease, such as hypertrophic cardiomyopathy or congenital heart disease
- Recent cardiac or thoracic procedures, including ablation
- Prior MI or pulmonary embolism or other pathology that can cause atrial dilation
- Diabetes.

A drug history will reveal whether the patient is taking anti-arrhythmics for chronic suppression of atrial fibrillation such as class Ic drugs (flecainide and propafenone) or class Ia (disopyramide, procainamide, quinidine) and amiodarone. Digitalis drugs (e.g., digoxin) are also known to be a risk factor for atrial flutter.

Physical examination

The pulse in a patients with atrial flutter may be regular or irregularly irregular. On examining the JVP, jugular venous pulsations with rapid flutter waves may be seen. Manifestations of the underlying cause should be sought through a thorough physical exam. Wheezing, rales, and hyperinflation suggest lung disease. Murmurs or rubs on auscultation suggest valvular disease, pericarditis, or congenital heart disease. Signs of hyperthyroidism may be present, such as tachycardia, fine tremor, goitre with or without nodules, palmar erythema, and hair thinning. Blood pressure should be taken and scars from recent cardiac or thoracic surgery looked for. These may suggest underlying conditions that may complicate therapy.

Investigations

The ECG pattern may fluctuate between atrial flutter and atrial fibrillation.
[Fig-1]

[Fig-2]

The ECG is diagnostic:

- In the typical form (anti-clockwise atrial flutter): negatively directed saw-tooth atrial deflections (f waves) in leads II, III, and aVF, and positive deflections in V1 with atrial rates of 240 to 320 bpm.
  [Fig-1]

  [Fig-5]
- Typically there is 2:1 AV block and the characteristic ventricular rate is 150 bpm.
- Variable block may occur leading to an irregular rate.
- In the reverse typical form (clockwise isthmus-dependent flutter): positive flutter waves in leads II, III, and aVF, and negative flutter waves in lead V1.
  [Fig-6]

  [Fig-7]
- Atypical flutter has an ECG pattern of continuous undulation of the atrial complex, not meeting criteria for typical or reverse typical flutter, with atrial rates >240 bpm.[3] [4] [6]
  [Fig-8]

  [Fig-9]

Electrolyte abnormalities are generally not the sole cause of atrial flutter, but imbalances should be checked and corrected. TFTs should be checked to rule out thyroid disease as an underlying cause. Cardiac enzymes should be checked if acute MI is suspected, and digitalis levels taken if a patient is taking digitalis drugs (e.g., digoxin).

A CXR should be performed and pulmonary function tests should be considered, as atrial flutter is often associated with a known history of, or suggestive presentation for, lung disease. Spiral CT with pulmonary embolism protocol may be considered if pulmonary embolism is suspected.

The trans-thoracic echocardiogram should be used to detect structural heart disease. Specifically, atrial sizes can be measured and valvular disease, ventricular function, and pericardial disease assessed. Right ventricular systolic pressures can also be measured and indicate the presence or absence of pulmonary HTN, which can be seen in pulmonary processes.

Electrophysiological studies are usually performed on patients with recurrent atrial flutter, particularly if anti-arrhythmic drug therapy has been ineffective or not tolerated because of adverse effects, if the ventricular rate is difficult to control with drugs, or if atrial flutter persists despite resolution of acute underlying illness. Electrophysiology studies require the input of electrophysiologists and may assist in diagnosis, mapping for the critical isthmus (i.e., the cavotricuspid isthmus), and therapeutic ablation.

An atrial electrogram recording can help visualise flutter waves when diagnosis is not clear. They are recorded by post-surgical epicardial leads, dual chamber pacemaker, or oesophageal lead.

Risk factors

Strong

increasing age

- The incidence is 5/100,000 in people <50 years of age and 587/100,000 in those over 80 years of age. It is especially uncommon in children or young adults unless associated with structural heart disease.[5] [10]

valvular dysfunction

- Mitral or tricuspid valve stenosis or regurgitation can lead to atrial dilation. Dilation promotes the development and maintenance of reentrant circuits.

atrial septal defects

- Can lead to atrial dilation. Dilation promotes the development and maintenance of reentrant circuits.

atrial dilation

- Some pathological conditions (e.g., MI or pulmonary embolism) can lead to atrial dilation; or, rarely, this occurs in the absence of structural heart disease. Dilation promotes the development and maintenance of reentrant circuits.

recent cardiac or thoracic procedures

- Although atrial fibrillation is the most common post-cardiac surgery arrhythmia, atrial flutter can occur and is due to pericarditis, alterations in autonomic tone, or atrial ischaemia.[4] [11]
- Transient atrial flutter in the first 2 months after pulmonary vein isolation procedures is common and may not necessitate long-term treatment. Can occur in up to 55% of such patients.[12]

surgical or post-ablation scarring of atria

- Any surgical incision involving the atria can result in atrial flutter with the flutter circuit involving atypical isthmuses between anatomical barriers, prior atrial incision sites, and scarred regions, as well as the cavotricuspid isthmus.[4] [10] [12] [13] [14] [15] [16]
- Scarring from prior atrial ablation lesions can lead to development of a reentrant circuit.[12]
- Approximately 10% to 30% incidence at 5- to 10-year follow-up after congenital heart disease operative corrections.[17] May also be associated with procedures such as valve surgery, in which atrial incisions or maze procedures were performed.

heart failure

- The risk of developing atrial flutter is increased 3.5 times in the presence of heart failure. In 16% of patients with atrial flutter, the arrhythmia was attributable to heart failure in a population-based epidemiological study.[5]

hyperthyroidism

- May precipitate atrial fibrillation or atrial flutter.

COPD

- In 12% of patients with atrial flutter, the arrhythmia was attributable to COPD in a population-based epidemiological study.[5] May precipitate atrial fibrillation or atrial flutter.

asthma

- May precipitate atrial fibrillation or atrial flutter.

pneumonia

- May precipitate atrial fibrillation or atrial flutter.

Weak

anti-arrhythmic drugs for atrial fibrillation

- Conversion of paroxysmal atrial fibrillation to chronic, incessant atrial flutter has been noted most commonly with Vaughan Williams class Ic drugs (flecainide and propafenone) and with class Ia (disopyramide, procainamide, quinidine) and amiodarone.[6] [7] Can occur in up to 15% to 20% of patients treated with propafenone, flecainide, and amiodarone.[4]

diabetes

- Atrial flutter is more common in those with a history of diabetes.[5]

digitalis use

- Rarely, atrial flutter occurs as a result of digitalis (e.g., digoxin) toxicity.

male sex

- Incidence is 2.5 times higher in men than in women.[5]

congenital or lone atrial flutter

- This is rare.[8]

History & examination factors

Key diagnostic factors

presence of risk factors (common)

- Risk factors include surgical or post-ablation scarring of atria, increasing age, valvular dysfunction, chronic ventricular failure, atrial septal defects, atrial dilation, recent cardiac or thoracic procedures, heart failure, hyperthyroidism, COPD, asthma, or pneumonia.

worsening heart failure or pulmonary symptoms (common)

- These are common underlying conditions, and worsening of symptoms may indicate other decompensation or new-onset atrial flutter. May present as exercise intolerance.

Other diagnostic factors

palpitations (common)

- Classic symptom, but because AV block usually results in normal ventricular rate, patients may not have this symptom.

fatigue or lightheadedness (common)

- Nonspecific but can be the main presenting symptom.

jugular venous pulsations with rapid flutter waves (uncommon)

- Rapid flutter waves can be noted in the jugular venous wave form.

chest pain (uncommon)

- Can be symptom of atrial flutter, underlying myocardial ischaemia, or a pulmonary embolic event.

dyspnoea (uncommon)

- Can be a symptom of atrial flutter, underlying myocardial ischaemia, or a pulmonary embolic event.

syncope (uncommon)

- Rare presentation of atrial flutter.

hypotension (uncommon)

- Rare presentation of atrial flutter. AV block typically results in normal ventricular rate and preservation of blood pressure

embolic events (uncommon)

- Such as stroke. Rare presentation of atrial flutter.

Diagnostic tests

1st test to order

Test	Result
ECG • Essential for the clinical diagnosis of atrial flutter. • In the typical form (anti-clockwise atrial flutter), negatively directed saw-tooth atrial deflections (f waves) in leads II, III, and aVF, and positive deflections in V1 with atrial rates of 240 to 320 bpm are seen. [Fig-1] [Fig-5] • 2:1 AV block is usually present in the typical form, resulting in the characteristic ventricular rate of 150 bpm. However, variable block may occur, leading to an irregular rate. • In the reverse typical form (clockwise isthmus-dependent flutter), positive flutter waves in leads II, III, and aVF, and negative flutter waves in lead V1 are seen. [Fig-6] [Fig-7] • Atypical flutter has an ECG pattern of continuous undulation of the atrial complex, not meeting criteria for typical or reverse typical flutter, with atrial rates >240 bpm. [Fig-8] [Fig-9] • Carotid massage or giving adenosine during ECG recording generally slows the ventricular rate to allow better visualisation of the flutter waves if the diagnosis is not clear.	**changes diagnostic of typical, reverse typical, or atypical atrial flutter**
TFTs • To rule out underlying thyroid disease.	**normal; abnormal if underlying thyroid disease is present**
serum electrolytes • Electrolyte abnormalities are generally not the sole cause of atrial flutter, but imbalances should be checked and corrected.	**normal; abnormal if underlying electrolyte disturbances are present**
CXR • Should be ordered if there is clinical suspicion of lung disease as a cause.	**normal; may be abnormal if underlying lung disease is present**

Other tests to consider

Test	Result
PFTs • Should be ordered if there is clinical suspicion of lung disease as a cause.	**normal; abnormal if underlying lung disease is present**

Test	Result
digitalis level • Rarely a cause of atrial flutter, but digitalis toxicity may be considered in patients taking digitalis drugs (e.g., digoxin).	**normal; elevated in digitalis toxicity**
cardiac enzymes • May be considered if acute MI is suspected.	**normal; elevated in MI**
spiral CT with pulmonary embolism protocol • May be considered if pulmonary emboli are suspected.	**normal; direct visualisation of thrombus in a pulmonary artery in pulmonary embolism**
trans-thoracic echocardiogram • Atrial sizes can be measured. • Can also assess for valvular disease, ventricular function, and pericardial disease. • Right ventricular systolic pressures (RVSP) can also be measured. • Elevated RVSP indicates the presence of pulmonary HTN, which can be seen in pulmonary processes.	**possible structural heart disease**
atrial electrogram recording • Recorded by post-surgical epicardial leads, dual chamber pacemaker, or oesophageal lead. • Can help visualise flutter waves when the diagnosis is not clear.	**flutter waves**
electrophysiological studies • Requires the input of electrophysiologists. • May be required for diagnosis, mapping of the critical isthmus (i.e., the cavotricuspid isthmus), and therapeutic ablation.	**map of reentrant circuit**

DIAGNOSIS

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Atrial fibrillation	• No differentiating signs/ symptoms	• ECG shows uncoordinated atrial activation with rapidly oscillating, fibrillatory waves that vary in amplitude, shape, and timing.[6]
Atrial tachycardia	• No differentiating signs/ symptoms	• Generally, by ECG, atrial tachycardia has isoelectric intervals between the P waves in all leads. At very high atrial rates, it may be extremely difficult to distinguish the two.[6]

Diagnostic criteria

American College of Cardiology/American Heart Association/Heart Rhythm Society classification of atrial flutter/atrial tachycardias[4]

Atrial flutter is a macro-reentrant atrial tachycardia with constant P wave/flutter morphology with a rate usually >250 bpm. It is distinguishable from focal atrial tachycardia, which has discrete P waves with an intervening isoelectric segment. Focal atrial tachycardia is caused mechanistically by micro-reentry or increased automaticity and generally has atrial rates in the range of 100-250 bpm.

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Non cavotricuspid isthmus dependent (atypical atrial flutter):

- Re-entry that does not depend upon conduction through the cavotricuspid isthmus
- Circuit is typically defined by atrial scars due to prior heart surgery, ablations, or idiopathic causes
- Location determines ablation approach and risks
- Multiple sites of re-entry may be present
- Can occur in both the left and right atria.

Step-by-step treatment approach

Haemodynamically unstable patients require urgent synchronised cardioversion. Haemodynamically stable patients can be treated with pharmacological therapy; however, cardioversion (either electrical or pharmacological) is an option in patients who do not respond to rate-control drugs. Patients with recurrent atrial flutter, or those who do not respond to elective cardioversion, may require catheter ablation of the cavotricuspid isthmus (CTI). Anticoagulation and treatment of any co-existing disease processes are important adjunctive therapies in all patients.

Haemodynamically unstable

If atrial flutter is associated with acute haemodynamic collapse involving symptomatic hypotension, congestive heart failure evidenced by pulmonary oedema and/or elevated serum brain natriuretic peptide (BNP), or myocardial ischaemia (acute ischaemic ECG changes, angina), emergency direct current (DC)-synchronised cardioversion is indicated.[18] This rhythm is generally successfully cardioverted with monophasic shocks using <50 J of energy, although the higher-energy initial shocks are indicated for emergent therapy and may be needed for elective cardioversions.[4]

Haemodynamically stable: rate control

More commonly, patients in atrial flutter present with 2:1 or higher grades of AV conduction block and are thus stable haemodynamically. In about 60% of patients, it occurs as part of an acute disease process.[5] When the underlying process resolves, sinus rhythm is generally restored and chronic therapy is not needed.

AV nodal blocking agents (e.g., beta-blockers, calcium-channel blockers, and amiodarone) are considered a first-line therapy for rate control in the immediate acute setting. Adequate rate control is more difficult with atrial flutter than with atrial fibrillation. However, most randomised controlled trials of AV nodal blocking agents generally do not report data for atrial flutter alone, but rather combined groups of patients with atrial fibrillation and/or flutter.[4] In addition, with slowing of the atrial rate can come rapid one-to-one AV conduction, particularly with class Ic anti-arrhythmic drugs in the absence of AV nodal-blocking agents.[4] [7] For example, atrial flutter with a rate of 300 bpm that conducts 2:1 will result in a ventricular rate of 150 bpm. An anti-arrhythmic drug that slows the flutter rate to 200 bpm might allow 1:1 AV nodal conduction in the absence of AV nodal blockers, thus resulting in a potentially clinically dangerous ventricular rate of 200 bpm.

Haemodynamically stable: cardioversion or pacing

If the rhythm persists despite pharmacological therapy and treatment of the underlying cause (or in the absence of a reversible cause), elective synchronised cardioversion is generally preferred, both because atrial flutter is extremely responsive to electrical cardioversion and because it is relatively difficult to rate control chronically.[4] [6] The success rate for external DC cardioversion, using 5 to 50 J of energy, is 95% to 100%.[19] [20] [21] Lower amounts are most successful with biphasic rather than monophasic waveforms.

Rate-control agents are continued before cardioversion and discontinued when sinus rhythm is restored. However, they can be continued afterwards to prevent rapid ventricular rate in case of recurrence. Dosage may need to be decreased after cardioversion if there is bradycardia or hypotension.

Rapid atrial pacing is useful for acute conversion of atrial flutter in patients who have pacing wires in place as part of a permanent pacemaker or an implantable cardioverter-defibrillator, or for temporary atrial pacing after cardiac surgery.[4]

The decision to perform a trans-oesophageal echocardiogram prior to cardioversion (both electrical and chemical) to assess for left atrial or appendage thrombus should follow the recommendations for atrial fibrillation.[6]

Haemodynamically stable: pharmacological cardioversion

If atrial flutter persists despite resolution of acute provocation, and electrical cardioversion is unavailable or not acceptable to the patient, pharmacological cardioversion may be attempted if the patient has a normal QT interval and no structural heart disease. It is also an option when sedation is not tolerated or available.

Intravenous ibutilide is the preferred agent for pharmacological cardioversion; however, oral dofetilide may also be used.[4] Dofetilide is contra-indicated in patients with long QT syndrome, QT prolongation, renal failure, and torsade de pointes. It requires specialist inpatient monitoring and should only be initiated by a physician experienced with its use.

Pharmacological cardioversion is less effective than synchronised cardioversion, with potential for being pro-arrhythmic. The success rate is 38% to 76% for conversion of atrial flutter to sinus rhythm, with the mean time to conversion reported to be 30 minutes in those who respond. Ventricular pro-arrhythmia, specifically sustained polymorphic VT, occurs at a rate of 1.2% to 1.7%.[22] [23] [24] [25] For this reason, these drugs should not be given to those with severe structural heart disease and prolonged QT interval.

The major risk associated with pharmacological conversion is torsade de pointes, for which patients with reduced left ventricular ejection fraction are at the highest risk. Pre-treatment with magnesium may reduce the risk of torsade de pointes. Continuous ECG monitoring is required during administration of these agents and for at least 4 hours after completion of therapy (ibutilide), or at least 3 days (or 12 hours after conversion to normal sinus rhythm, whichever is greater) after completion of therapy (dofetilide).

Intravenous class Ic agents and oral sotalol have relatively poor efficacy in acute conversion, are associated with significant adverse effects, and are not recommended.[4]

The decision to perform a trans-oesophageal echocardiogram prior to cardioversion (both electrical and chemical) to assess for left atrial or appendage thrombus should follow the recommendations for atrial fibrillation.[6]

Recurrent or refractory atrial flutter: catheter ablation

Catheter ablation of the cavotricuspid isthmus (CTI) is useful in patients with atrial flutter that is symptomatic or refractory to pharmacological rate control, patients in whom at least one anti-arrhythmic drug has failed, patients who develop atrial flutter as a result of anti-arrhythmic therapy for atrial fibrillation, and patients with recurrent atrial flutter.

Catheter ablation has a class I indication in the following clinical scenarios:[4] [26]

- Symptomatic or refractory to pharmacological rate control
- Recurrent symptomatic CTI-dependent flutter after failure of at least one anti-arrhythmic agent

Reasonable (class II) indications include:

- CTI-dependent atrial flutter that occurs as the result of flecainide, propafenone, or amiodarone used for the treatment of atrial fibrillation
- Patients undergoing catheter ablation of atrial fibrillation who also have a history of documented clinical or induced CTI-dependent atrial flutter
- Primary therapy of recurrent symptomatic non-CTI-dependent flutter before therapeutic trials of anti-arrhythmic drugs after carefully weighing the potential risks and benefits of treatment options
- Asymptomatic patients with recurrent atrial flutter.

Catheter ablation is effective at maintaining sinus rhythm in typical atrial flutter in which the CTI is a necessary part of the arrhythmic circuit. This invasive technique involves a femoral venous approach. An ablation catheter is placed at the isthmus between the inferior vena cava and the tricuspid annulus using either fluoroscopic guidance or a 3-dimensional electroanatomic mapping system. Radiofrequency energy is then applied to create a line of ablation from the tricuspid annulus to the inferior vena cava.

The success rate for treatment of typical atrial flutter is high and has been reported at 92% for the first procedure and 97% for multiple procedures.[26] [27] [28] Atypical flutter is more difficult to ablate, particularly when associated with congenital heart disease. In such situations, referral to an experienced centre ought to be considered.

Recurrent or refractory atrial flutter: long-term anti-arrhythmic therapy

Chronic pharmacological therapy is generally not required. In 60% of cases, atrial flutter arises in the setting of a precipitating cause and, once that acute process resolves, sinus rhythm is restored.[5]

Most studies evaluating long-term anti-arrhythmic therapy have grouped atrial flutter patients with atrial fibrillation patients. Therefore, exact efficacy rates are difficult to determine, but are probably around 50% for class I anti-arrhythmics.[4]

Anti-arrhythmic drug choice depends on the presence or absence of underlying heart disease and any comorbidities. Options include amiodarone, dofetilide, sotalol, flecainide, and propafenone.

AV nodal agents such as beta-blockers or calcium-channel blockers should be used in conjunction with class Ic drugs (e.g., flecainide, propafenone) because of the concern for slowing of the atrial flutter rate with resultant 1:1 AV conduction at high rates. However, class Ic drugs are contra-indicated in patients with structural heart disease.

Class III agents such as oral dofetilide, sotalol, and amiodarone resulted in maintenance of sinus rhythm in 73% of atrial flutter patients.[4] Amiodarone is generally less effective than dofetilide; however, it is less pro-arrhythmic than other anti-arrhythmics and is relatively safe in patients with structural heart disease.[29] Dofetilide is contra-indicated in patients with long QT syndrome, QT prolongation, renal failure, and torsade de pointes. Continuous ECG monitoring is required during administration and for at least 3 days (or 12 hours after conversion to normal sinus rhythm, whichever is greater) after completion of therapy. It should only be initiated by a physician experienced in its use. Sotalol has both class II beta-blocking and class III properties, and thus provides rate control.

Anticoagulant therapy

The guidelines for thromboembolic prophylaxis in atrial flutter are the same as those for atrial fibrillation.[6] [30] [31] [32] [33] [34] [35] [36]

Observational studies have demonstrated a 1.7% to 7% risk of embolisation during cardioversion from atrial flutter.[37] Anticoagulation management prior to ablation should be handled similarly to that before cardioversion for atrial fibrillation. Anticoagulation after catheter ablation for atrial flutter should follow the same approach for that after atrial fibrillation ablation. The incidence of thrombus or echo-dense material in the atria in patients with atrial flutter who are not anticoagulated ranges from 0% to 34% and increases with atrial flutter duration more than 48 hours.[38] Atrial mechanical stunning has also been documented to persist for several weeks after cardioversion.[39]

Initial anticoagulation is with intravenous heparin or subcutaneous low-molecular weight heparin, which is continued until an INR of 2-3 is achieved with warfarin therapy. Warfarin is continued for at least 4 weeks after cardioversion. Novel oral anticoagulants (NOACs) such as dabigatran (a direct thrombin inhibitor), and apixaban, edoxaban, or rivaroxaban (direct factor Xa inhibitors), can be used as alternatives to heparin or enoxaparin plus warfarin. Bridging with a parenteral anticoagulant is not necessary. NOACs do not require monitoring of anticoagulant activity; however, they must be used with caution in patients with renal impairment and a dose adjustment may be necessary. Dabigatran is contra-indicated in patients with mechanical heart valves. Its effects can be reversed in the event of major bleeding with idarucizumab. The other NOACS are also not recommended in patients with mechanical heart valves at the current time. Edoxaban should not be used in patients with a CrCL >95 mL/minute because of an increased risk of ischaemic stroke.

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Presumptive (summary)

Patient group	Tx line	Treatment
haemodynamically unstable	**1st**	**synchronised cardioversion**

Acute (summary)

Patient group	Tx line	Treatment
haemodynamically stable	**1st**	**beta-blocker or calcium-channel blocker or amiodarone**
	plus	**anticoagulation**
	plus	**treat co-existing acute disease process**
	2nd	**synchronised cardioversion**
	plus	**anticoagulation**
	plus	**treat co-existing acute disease process**
	3rd	**pharmacological conversion**

Acute (summary)

	plus	anticoagulation
	plus	treat co-existing acute disease process
with a permanent pacemaker or an implantable cardioverter-defibrillator or after cardiac surgery	plus	rapid atrial pacing

Ongoing (summary)

Patient group	Tx line	Treatment
recurrent atrial flutter or failure of elective cardioversion	1st	catheter ablation of the cavotricuspid isthmus (CTI)
	plus	anticoagulation
recurrent atrial flutter or failure of elective cardioversion	1st	beta-blocker or calcium channel blocker
	plus	long-term anticoagulation
	adjunct	anti-arrhythmic therapy

Treatment options

Presumptive

Patient group	Tx line	Treatment
haemodynamically unstable	**1st**	**synchronised cardioversion** » If atrial flutter is associated with acute haemodynamic collapse involving symptomatic hypotension, congestive heart failure evidenced by pulmonary oedema and/or elevated serum brain natriuretic peptide (BNP), or myocardial ischaemia (acute ischaemic ECG changes, angina), emergent DC-synchronised cardioversion is indicated.[18] » This rhythm is generally successfully cardioverted with monophasic shocks using <50 J of energy, although the higher-energy initial shocks are indicated for emergency therapy.[4]

Acute

Patient group	Tx line	Treatment
haemodynamically stable	**1st**	**beta-blocker or calcium-channel blocker or amiodarone** » Used for rate control in acute setting. » Beta-blockers are particularly useful if acute MI, angina, or association with exercise is present. They should be used with caution in COPD. » Calcium-channel blockers (CCB) are preferred if chronic lung disease is also present, where beta-blockers might provoke bronchospasm. CCBs are generally contra-indicated or used with extreme caution in heart failure. » Intravenous amiodarone is useful for acute control of the ventricular rate (in the absence of pre-excitation) in patients with atrial flutter and systolic heart failure when beta-blockers are contra-indicated or ineffective. **Primary options** » metoprolol: 2.5 to 5 mg intravenous bolus over 2 minutes initially, may repeat every 5 minutes to a total of 3 doses, followed by

Acute

Patient group	Tx line	Treatment
		25-100 mg orally (immediate-release) twice daily
		OR
		Primary options
		» esmolol: 500 micrograms/kg intravenously over 1 minute as a loading dose, followed by 50 micrograms/kg/min infusion for 4 minutes, if no response after 5 minutes, repeat loading dose and increase infusion; consult specialist for further guidance on dose
		OR
		Primary options
		» diltiazem: 0.25 mg/kg/dose intravenous bolus over 2 minutes initially, may give second dose of 0.35 mg/kg/dose bolus over 2 minutes if necessary, followed by 5-15 mg/hour infusion
		OR
		Primary options
		» verapamil: 2.5 to 10 mg intravenous bolus over 2 minutes initially, may give second dose of 5-10 mg bolus after 30 minutes if necessary followed by 0.005 mg/kg/min infusion
		OR
		Secondary options
		» amiodarone: 150 mg intravenously over 10 minutes initially, followed by 0.5 to 1 mg/minute infusion
	plus	**anticoagulation**
		» The guidelines for thromboembolic prophylaxis in atrial flutter are the same as those for atrial fibrillation.[6] [30] [35]
		» Heparin or enoxaparin plus warfarin should be initiated in all patients and the parenteral anticoagulant continued until the warfarin levels are therapeutic (INR 2-3).
		» Novel oral anticoagulants (NOACs) such as dabigatran, apixaban, edoxaban, and rivaroxaban can be used as alternatives to heparin or enoxaparin plus warfarin. Bridging with a parenteral anticoagulant is not necessary. NOACs do not require monitoring

Acute

Patient group	Tx line	Treatment
		of anticoagulant activity; however, they must be used with caution in patients with renal impairment and a dose adjustment may be necessary. Dabigatran is contra-indicated in patients with mechanical heart valves. Its effects can be reversed in the event of major bleeding with idarucizumab. The other NOACS are also not recommended in patients with mechanical heart valves at the current time. Edoxaban should not be used in patients with a CrCL >95 mL/minute because of an increased risk of ischaemic stroke. **Primary options** » heparin: see local protocol for dosing guidelines, maintain aPTT at 45-60 seconds Continue treatment until warfarin levels are therapeutic (INR 2-3). **-or-** » enoxaparin: 1 mg/kg subcutaneously every 12 hours Continue treatment until warfarin levels are therapeutic (INR 2-3). **--AND--** » warfarin: 2-5 mg orally once daily initially, adjust dose to target INR of 2-3 **OR** **Primary options** » dabigatran: 150 mg orally twice daily **OR** **Primary options** » apixaban: 5 mg orally twice daily **OR** **Primary options** » edoxaban: 60 mg orally once daily **OR** **Primary options** » rivaroxaban: 20 mg orally once daily
	plus	**treat co-existing acute disease process** » In 60% of patients, atrial flutter accompanies an acute illness and resolves with that disease process.[5]

Acute

Patient group	Tx line	Treatment
	2nd	**synchronised cardioversion** » Recommended for acute treatment of patients who do not respond to pharmacological therapy. » The decision to perform a trans-oesophageal echocardiogram prior to cardioversion to assess for left atrial or appendage thrombus should follow the recommendations for atrial fibrillation.[6] » The success rate for external direct current cardioversion is 95% to 100% and can be generally achieved using 5 to 50 J of energy, the lower amounts being most successful with biphasic versus monophasic waveforms. However, higher energies may be needed.[19] [20] [21] » Rate-control agents are continued before cardioversion and discontinued when sinus rhythm is restored. However, they can be continued afterwards to prevent rapid ventricular rate in case of recurrence. Dosage may need to be decreased after cardioversion if there is bradycardia or hypotension.
	plus	**anticoagulation** » The guidelines for thromboembolic prophylaxis in atrial flutter are the same as those for atrial fibrillation.[6] [30] [35] » Heparin or enoxaparin plus warfarin should be initiated in all patients and the parenteral anticoagulant continued until the warfarin levels are therapeutic (INR 2-3). » Novel oral anticoagulants (NOACs) such as dabigatran, apixaban, edoxaban, and rivaroxaban can be used as alternatives to heparin or enoxaparin plus warfarin. Bridging with a parenteral anticoagulant is not necessary. NOACs do not require monitoring of anticoagulant activity; however, they must be used with caution in patients with renal impairment and a dose adjustment may be necessary. Dabigatran is contra-indicated in patients with mechanical heart valves. Its effects can be reversed in the event of major bleeding with idarucizumab. The other NOACS are also not recommended in patients with mechanical heart valves at the current time. Edoxaban should not be used in patients with a CrCL >95 mL/minute because of an increased risk of ischaemic stroke.

Acute

Patient group	Tx line	Treatment
		Primary options » heparin: see local protocol for dosing guidelines, maintain aPTT at 45-60 seconds Continue treatment until warfarin levels are therapeutic (INR 2-3). **-or-** » enoxaparin: 1 mg/kg subcutaneously every 12 hours Continue treatment until warfarin levels are therapeutic (INR 2-3). **--AND--** » warfarin: 2-5 mg orally once daily initially, adjust dose to target INR of 2-3 **OR** **Primary options** » dabigatran: 150 mg orally twice daily **OR** **Primary options** » apixaban: 5 mg orally twice daily **OR** **Primary options** » edoxaban: 60 mg orally once daily **OR** **Primary options** » rivaroxaban: 20 mg orally once daily
	plus	**treat co-existing acute disease process** » In 60% of patients, atrial flutter accompanies an acute illness and resolves with that disease process.[5]
	3rd	**pharmacological conversion** » May be considered if electrical cardioversion is unavailable, or if the patient does not consent to electrical cardioversion. It is also an option when sedation is not tolerated or available. To be a candidate, a patient must have a normal QT interval and no structural heart disease. » Intravenous ibutilide is the preferred agent for pharmacological cardioversion; however, oral dofetilide may also be used.[4] Dofetilide is contra-indicated in patients with long QT

Acute

Patient group	Tx line	Treatment
		syndrome, QT prolongation, renal failure, and torsade de pointes. It requires specialist inpatient monitoring and should only be initiated by a physician experienced with its use. » Less effective than synchronised cardioversion, with potential for being pro-arrhythmic. Mean time to conversion is reported to be 30 minutes in those who respond (38% to 76%). Ventricular pro-arrhythmia, specifically sustained polymorphic VT, occurs at a rate of 1.2% to 1.7%.[22] [23] [24] [25] » The major risk associated with pharmacological conversion is torsade de pointes, for which patients with reduced left ventricular ejection fraction are at the highest risk. Pre-treatment with magnesium may reduce the risk of torsade de pointes. » Continuous ECG monitoring is required during administration of these agents and for at least 4 hours after completion of therapy (ibutilide), or at least 3 days (or 12 hours after conversion to normal sinus rhythm, whichever is greater) after completion of therapy (dofetilide). **Primary options** » ibutilide: 0.01 mg/kg/dose intravenously (maximum 1 mg/dose) over 10 minutes initially, may repeat 10 minutes after initial dose if no response **OR** **Secondary options** » dofetilide: dose depends on QTc and renal function; consult specialist for guidance on dose
	plus	**anticoagulation** » The guidelines for thromboembolic prophylaxis in atrial flutter are the same as those for atrial fibrillation.[6] [30] [35] » Heparin or enoxaparin plus warfarin should be initiated in all patients and the parenteral anticoagulant continued until the warfarin levels are therapeutic (INR 2-3). » Novel oral anticoagulants (NOACs) such as dabigatran, apixaban, edoxaban, and rivaroxaban can be used as alternatives to heparin or enoxaparin plus warfarin.

Acute

Patient group	Tx line	Treatment
		Bridging with a parenteral anticoagulant is not necessary. NOACs do not require monitoring of anticoagulant activity; however, they must be used with caution in patients with renal impairment and a dose adjustment may be necessary. Dabigatran is contra-indicated in patients with mechanical heart valves. Its effects can be reversed in the event of major bleeding with idarucizumab. The other NOACS are also not recommended in patients with mechanical heart valves at the current time. Edoxaban should not be used in patients with a CrCL >95 mL/minute because of an increased risk of ischaemic stroke. **Primary options** » heparin: see local protocol for dosing guidelines, maintain aPTT at 45-60 seconds Continue treatment until warfarin levels are therapeutic (INR 2-3). **-or-** » enoxaparin: 1 mg/kg subcutaneously every 12 hours Continue treatment until warfarin levels are therapeutic (INR 2-3). **--AND--** » warfarin: 2-5 mg orally once daily initially, adjust dose to target INR of 2-3 **OR** **Primary options** » dabigatran: 150 mg orally twice daily **OR** **Primary options** » apixaban: 5 mg orally twice daily **OR** **Primary options** » edoxaban: 60 mg orally once daily **OR** **Primary options** » rivaroxaban: 20 mg orally once daily
	plus	**treat co-existing acute disease process**

Acute

Patient group	Tx line	Treatment
		» In 60% of patients, atrial flutter accompanies an acute illness and resolves with that disease process.[5]
with a permanent pacemaker or an implantable cardioverter-defibrillator or after cardiac surgery	**plus**	**rapid atrial pacing** » Useful for acute conversion of atrial flutter in patients who have pacing wires in place as part of a permanent pacemaker or an implantable cardioverter-defibrillator, or for temporary atrial pacing after cardiac surgery.

Ongoing

Patient group	Tx line	Treatment
recurrent atrial flutter or failure of elective cardioversion	**1st**	**catheter ablation of the cavotricuspid isthmus (CTI)** » Useful in patients with atrial flutter that is symptomatic or refractory to pharmacological rate control, patients in whom at least one anti-arrhythmic drug has failed, patients who develop atrial flutter as a result of anti-arrhythmic therapy for atrial fibrillation, and patients with recurrent atrial flutter. » Catheter ablation is effective at maintaining sinus rhythm in typical atrial flutter in which the CTI is a necessary part of the arrhythmic circuit. This invasive technique involves a femoral venous approach. An ablation catheter is placed at the isthmus between the inferior vena cava and the tricuspid annulus using either fluoroscopic guidance or a 3-dimensional electroanatomic mapping system. Radiofrequency energy is then applied to create a line of ablation from the tricuspid annulus to the inferior vena cava. » The success rate for treatment of typical atrial flutter is high and has been reported at 92% for the first procedure and 97% for multiple procedures.[26] [27] [28] » Atypical flutter is more difficult to ablate, particularly when associated with congenital heart disease. In such situations, consider referral to an experienced centre.
	plus	**anticoagulation**

Ongoing

Patient group

Tx line

Treatment

» The guidelines for thromboembolic prophylaxis in atrial flutter are the same as those for atrial fibrillation.[6] [30] [35]

» Heparin or enoxaparin plus warfarin should be initiated in all patients and the parenteral anticoagulant continued until the warfarin levels are therapeutic (INR 2-3).

» Novel oral anticoagulants (NOACs) such as dabigatran, apixaban, edoxaban, and rivaroxaban can be used as alternatives to heparin or enoxaparin plus warfarin. Bridging with a parenteral anticoagulant is not necessary. NOACs do not require monitoring of anticoagulant activity; however, they must be used with caution in patients with renal impairment and a dose adjustment may be necessary. Dabigatran is contra-indicated in patients with mechanical heart valves. Its effects can be reversed in the event of major bleeding with idarucizumab. The other NOACS are also not recommended in patients with mechanical heart valves at the current time. Edoxaban should not be used in patients with a CrCL >95 mL/minute because of an increased risk of ischaemic stroke.

Primary options

» heparin: see local protocol for dosing guidelines, maintain aPTT at 45-60 seconds
Continue treatment until warfarin levels are therapeutic (INR 2-3).
-or-
» enoxaparin: 1 mg/kg subcutaneously every 12 hours
Continue treatment until warfarin levels are therapeutic (INR 2-3).

--AND--

» warfarin: 2-5 mg orally once daily initially, adjust dose to target INR of 2-3

OR

Primary options

» dabigatran: 150 mg orally twice daily

OR

Primary options

» apixaban: 5 mg orally twice daily

OR

Ongoing

Patient group	Tx line	Treatment
		Primary options » edoxaban: 60 mg orally once daily **OR** **Primary options** » rivaroxaban: 20 mg orally once daily
recurrent atrial flutter or failure of elective cardioversion	**1st**	**beta-blocker or calcium channel blocker** » Can be useful to control the ventricular rate in patients with haemodynamically tolerated atrial flutter. **Primary options** » metoprolol: 25-100 mg orally (immediate-release) twice daily **OR** **Primary options** » diltiazem: 120-360 mg/day orally (regular-release) given in 3-4 divided doses **OR** **Primary options** » verapamil: 240-320 mg/day orally (regular-release) given in 3-4 divided doses
	plus	**long-term anticoagulation** » The guidelines for thromboembolic prophylaxis in atrial flutter are the same as those for atrial fibrillation.[6] [30] [35] » Heparin or enoxaparin plus warfarin should be initiated in all patients and the parenteral anticoagulant continued until the warfarin levels are therapeutic (INR 2-3). » Novel oral anticoagulants (NOACs) such as dabigatran, apixaban, edoxaban, and rivaroxaban can be used as alternatives to heparin or enoxaparin plus warfarin. Bridging with a parenteral anticoagulant is not necessary. NOACs do not require monitoring of anticoagulant activity; however, they must be used with caution in patients with renal impairment and a dose adjustment may be necessary. Dabigatran is contra-indicated in patients with mechanical heart valves. Its effects

Ongoing

Patient group	Tx line	Treatment
		can be reversed in the event of major bleeding with idarucizumab. The other NOACS are also not recommended in patients with mechanical heart valves at the current time. Edoxaban should not be used in patients with a CrCL >95 mL/minute because of an increased risk of ischaemic stroke. **Primary options** » heparin: see local protocol for dosing guidelines, maintain aPTT at 45-60 seconds Continue treatment until warfarin levels are therapeutic (INR 2-3). **-or-** » enoxaparin: 1 mg/kg subcutaneously every 12 hours Continue treatment until warfarin levels are therapeutic (INR 2-3). **--AND--** » warfarin: 2-5 mg once daily orally initially, adjust dose to target INR of 2-3 **OR** **Primary options** » dabigatran: 150 mg orally twice daily **OR** **Primary options** » apixaban: 5 mg orally twice daily **OR** **Primary options** » edoxaban: 60 mg orally once daily **OR** **Primary options** » rivaroxaban: 20 mg orally once daily
	adjunct	**anti-arrhythmic therapy** » Can be useful to maintain sinus rhythm in patients with symptomatic recurrent atrial flutter. » Drug choice depends on the presence or absence of underlying heart disease and any comorbidities. Options include amiodarone, dofetilide, and sotalol. Flecainide or propafenone must be given with a rate-control drug and

Ongoing

Patient group | Tx line | Treatment

should only be considered in patients who do not have structural or ischaemic heart disease.

» Amiodarone is less pro-arrhythmogenic than other anti-arrhythmics and relatively safe in patients with structural heart disease.[29]

» Dofetilide is contra-indicated in patients with long QT syndrome, QT prolongation, renal failure, and torsade de pointes. Continuous ECG monitoring is required during administration and for at least 3 days (or 12 hours after conversion to normal sinus rhythm, whichever is greater) after completion of therapy. It should only be initiated by a physician experienced in its use.

» Sotalol has both class II beta-blocking and class III properties, and thus provides rate control. It is contra-indicated in patients with renal failure or creatinine clearance <40 mL/minute.

» Anti-arrhythmic dosing follows atrial fibrillation guidelines, as not many of the studies looked at atrial flutter separately.[4] [30]

Primary options

» amiodarone: 100-400 mg orally once daily

OR

Primary options

» dofetilide: dose depends on QTc and renal function; consult specialist for guidance on dose

OR

Primary options

» sotalol: 80-160 mg orally twice daily

OR

Primary options

» flecainide: 50-150 mg orally twice daily

OR

Primary options

» propafenone: 150-300 mg orally (immediate-release) three times daily

Recommendations

Monitoring

Patients presenting with acute atrial flutter who have paroxysmal, persistent, or permanent atrial flutter need long-term follow-up.

- Depending on the nature of the underlying cause (i.e., CAD, valvular heart disease, or heart failure), patients should have regular follow-up at a minimum of every 3 to 6 months.
- Patients who are taking anti-arrhythmic agents need follow-up ECG monitoring. Exercise stress testing is recommended to assess for drug-related ventricular tachycardia due to pro-arrhythmic effects of flecainide and propafenone.
- A regular follow-up to check and monitor INR is mandatory for patients who are taking warfarin for anticoagulation.

For patients with persistent atrial flutter that is felt to be secondary to a reversible cause, long-term follow-up may not be necessary after the initial treatment and evaluation of the atrial flutter, if the cause has been removed. For patients requiring continued therapy due to high risk of recurrence, follow-up should generally include the following general categories:

- Adequate treatment of the associated medical/cardiac diagnosis
- Periodic assessments of efficacy of therapy
- Periodic evaluation for the adverse effects or complications of the therapy
- Maintenance and monitoring of adequate anticoagulation
- Re-evaluation of the current therapy if the underlying cardiovascular problem changes or progresses or the atrial flutter becomes permanent.

In the few patients with atrial flutter who have asymptomatic recurrences irrespective of the therapy (anti-arrhythmic drug or ablation), chronic anticoagulation should be considered long term. If the patient is symptomatic, evaluation with Holter monitor, event monitor, or pacemaker/implantable cardioverter-defibrillator interrogations can be considered, largely to assure that ventricular rates are controlled, and to measure the number and duration of atrial flutter episodes. Clearly any change in the clinical status of a patient with a history of atrial flutter should prompt the physician to look for a recurrence as the cause of the clinical change.

Patient instructions

Recurrent palpitations should be reported to the cardiologist. Associated symptoms of chest pain, SOB, or hypotension warrant a call to activate emergency medical services.

Complications

Complications	Timeframe	Likelihood
beta-blocker-associated exacerbation of reactive airway disease	**short term**	**medium**

FOLLOW UP

Complications	Timeframe	Likelihood
Beta-blockers may cause bronchospasm. They should be avoided in patients with known asthma or history of lung disease with prominent history of wheezing.		
myocardial ischaemia	**short term**	**low**
Demand ischaemia can result from poor rate control.		
acute stroke	**short term**	**low**
Patients usually have paroxysmal to persistent atrial flutter with risk factors to thromboembolic events, such as left atrial dilation, CHF, HTN, diabetes, hyperlipidaemia, and advanced age. Controversy exists as regards treatment of established embolic acute stroke in the presence of acute atrial flutter. Anticoagulation in this setting may cause haemorrhagic stroke. Consultation with specialist (neurologist) is highly recommended. Novel approaches to interrupt thrombus propagation by intravascular devices are being studied.		
medication-related bradycardia	**variable**	**high**
Secondary to AV nodal blocking effects of beta-blockers and calcium-channel blockers. Can occur due to profound effects on the AV node while in atrial flutter or to the effects on the sinus node when in sinus rhythm, particularly in patients with underlying sinus node dysfunction. Can occur with anti-arrhythmic drugs (e.g., flecainide or amiodarone) as well. May require changing drug, or adding permanent pacing.		
medication-related hypotension	**variable**	**medium**
Secondary to the vasodilatory effects of beta-blockers and calcium-channel blockers, as well as the AV nodal effects slowing the heart rate. If this occurs, may resolve with lowering the dosage. Other causes of hypotension should also be explored. If the strategy was rate control only, consider AV nodal ablation for rate control; can also reconsider rhythm control in some patients.		
medication-related heart failure	**variable**	**medium**

Complications	Timeframe	Likelihood
Due to the negative inotropic effects of beta-blockers and calcium-channel blockers, as well as some anti-arrhythmic drugs. These drugs should be used cautiously in patients with poor left ventricular function and not at all in patients with overt clinical heart failure until the heart failure is treated and compensated. If heart rate control or rhythm control is the goal, amiodarone can be used to afford rate control or rhythm control with little depression of contractility. Optimisation of heart failure regimen is also helpful.		
medication-related pro-arrhythmia	**variable**	**medium**
May take many forms depending on the type and drug used. The most dangerous is ventricular arrhythmias such as ventricular tachycardia or torsades de pointes. Occurs with anti-arrhythmic drugs that prolong the QT or with drugs that prolong conduction in patients with underlying coronary disease.		
medication-related thyroid dysfunction	**variable**	**medium**
Amiodarone contains iodine. Elevated TSH and hypothyroidism more common than hyperthyroidism unless the patient has a predilection to hyperthyroidism or a previous goiter. Can replace thyroid hormone orally for hypothyroidism but usually need to discontinue the drug if hyperthyroid and treat the symptoms of hyperthyroidism.		
complications of catheter ablation	**variable**	**medium**
Complications vary with the technique and experience, and some have decreased with modification of ablation strategies. However, ablation for atrial flutter is generally quite safe.[26] [27] May include pneumothorax (0.2%), AV fistulae, haematomas (<0.3%), CHF exacerbation (<0.1%), cerebrovascular events (0% to 4%), pericardial effusion usually uncomplicated (25%), pericardial tamponade (1%), organized atrial tachyarrhythmias (13%), AV block (<1%), need for a pacemaker (<0.2%), and pulmonary embolus (<1%). Procedure-related death is extremely rare (estimated at 0.03%).[26] Most complications are handled acutely.		
tachycardia-mediated cardiomyopathy	**variable**	**low**
From poor rate control and loss of active atrial contraction, a cardiomyopathy can result and support the need for rhythm conversion.		
medication-related pulmonary toxicity	**variable**	**low**

FOLLOW UP

Complications	Timeframe	Likelihood
Acute toxicity presents as ARDS within hours of giving amiodarone. The long-term toxicity causes inflammation and/or fibrosis. Symptoms of an unexplained cough or dyspnoea should trigger investigation with a CXR and PFTs with a diffusion capacity. PFTs should be considered as routine follow-up every 6 to 12 months while using the drug. Will require discontinuation of the drug in most cases.		

Prognosis

In approximately 60% of cases, atrial flutter occurs in the setting of an acute process.[5] Once that process has been treated, sinus rhythm is usually restored and chronic therapy is not required. The thromboembolic risk is similar to that of atrial fibrillation and guidelines for prophylaxis should follow the atrial fibrillation guidelines. If all acute treatments and attempts at cardioversion and ablation fail, chronic atrial flutter is very difficult to rate control and is not as responsive to anti-arrhythmic agents as atrial fibrillation.

Diagnostic guidelines

Europe

2016 ESC guidelines for the management of atrial fibrillation

Published by: European Society of Cardiology **Last published:** 2016

Summary: Evidence-based recommendations for the evaluation of atrial flutter.

Atrial fibrillation: management

Published by: National Institute for Health and Care Excellence **Last published:** 2014

Summary: Provides recommendations on the diagnosis and assessment of adults with suspected or diagnosed atrial fibrillation.

Cardiac arrhythmias in coronary heart disease: a national clinical guideline

Published by: Scottish Intercollegiate Guidelines Network **Last published:** 2007

International

ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia

Published by: American College of Cardiology; American Heart Association; Heart Rhythm Society **Last published:** 2016

Summary: Provides a contemporary guideline for the management of adults with all types of supraventricular tachycardia other than atrial fibrillation.

North America

Guideline for the management of patients with atrial fibrillation

Published by: American College of Cardiology; American Heart Association; Heart Rhythm Society **Last published:** 2014

Summary: Provides recommendations on the management of atrial fibrillation, including information on diagnosis.

Treatment guidelines

Europe

2016 ESC guidelines for the management of atrial fibrillation

Published by: European Society of Cardiology **Last published:** 2016

Summary: Evidence-based recommendations for the treatment of atrial flutter.

Europe

Resuscitation guidelines 2015

Published by: Resuscitation Council **Last published:** 2015

Summary: Provides guidelines on treatment of atrial flutter using cardioversion.

Atrial fibrillation: management

Published by: National Institute for Health and Care Excellence **Last published:** 2014

Summary: Provides recommendations on the management of adults with atrial fibrillation including paroxysmal (recurrent), persistent, and permanent atrial fibrillation, and atrial flutter.

Consensus document on antithrombotic therapy in the setting of electrophysiological procedures

Published by: European Heart Rhythm Association **Last published:** 2008

Summary: Recommends catheter ablation of isthmus for isthmus-dependent atrial flutter in combination with anti-thrombotic therapy.

Cardiac arrhythmias in coronary heart disease: a national clinical guideline

Published by: Scottish Intercollegiate Guidelines Network **Last published:** 2007

International

ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia

Published by: American College of Cardiology; American Heart Association; Heart Rhythm Society **Last published:** 2016

Summary: Comprehensive treatment strategy for atrial flutter.

North America

Prevention and management of perioperative atrial fibrillation and flutter for thoracic surgical procedures

Published by: American Association for Thoracic Surgery **Last published:** 2014

Summary: Evidence-based guidelines for the prevention and treatment of perioperative/postoperative atrial fibrillation and flutter for thoracic surgical procedures.

North America

2014 focused update of the CCS Guidelines for the management of atrial fibrillation

Published by: Canadian Cardiovascular Society **Last published:** 2014

Summary: The 2014 focused update deals with advances in oral anticoagulant (OAC) therapy and presents a new CCS algorithm that will allow clinicians to easily determine which patients with atrial fibrillation will benefit from OAC therapy. The update also outlines the optimal approach to perioperative OAC management and updates rate and rhythm management in atrial fibrillation including catheter ablation.

Guideline for the management of patients with atrial fibrillation

Published by: American College of Cardiology; American Heart Association; Heart Rhythm Society **Last published:** 2014

Summary: Provides recommendations on the management of atrial fibrillation.

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Images

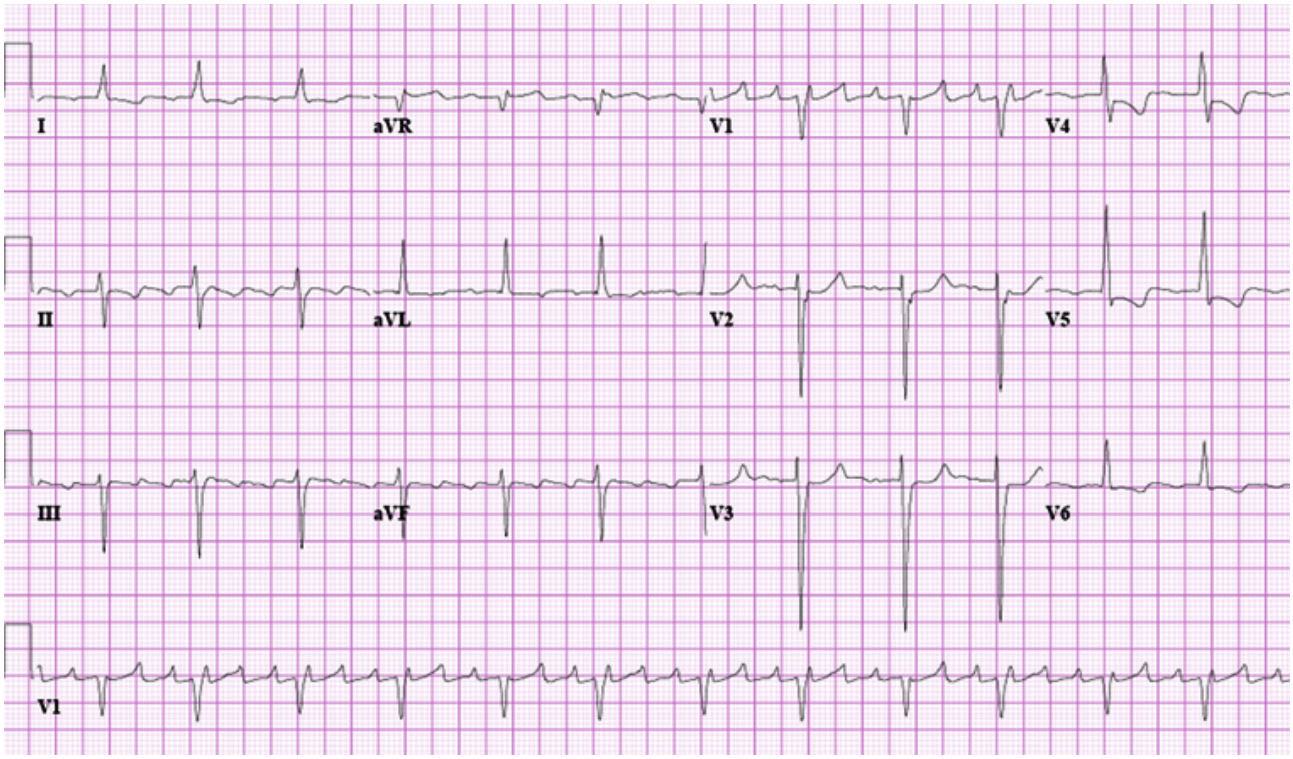

Figure 1: Typical atrial flutter with variable (3 to 4:1) block

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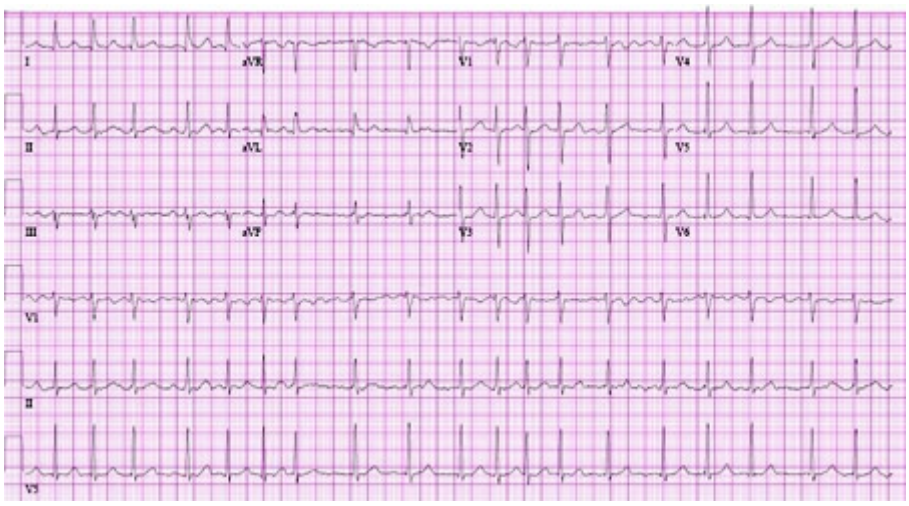

Figure 2: Atrial fibrillation

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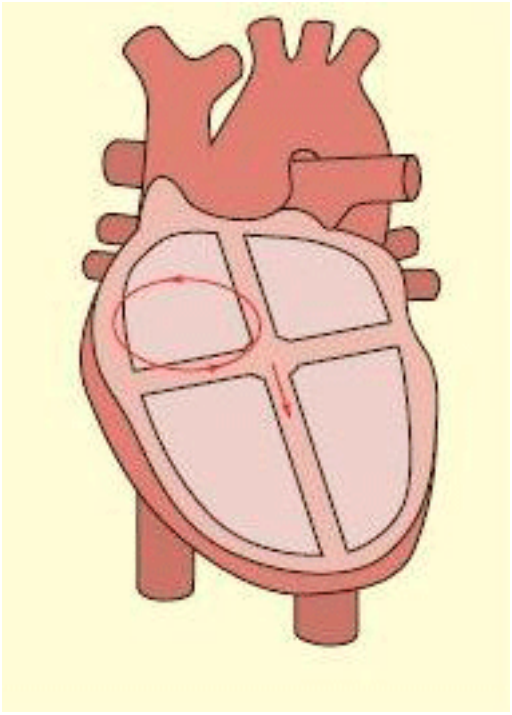

Figure 3: Atrial flutter typically involves a circuit in the right atrium

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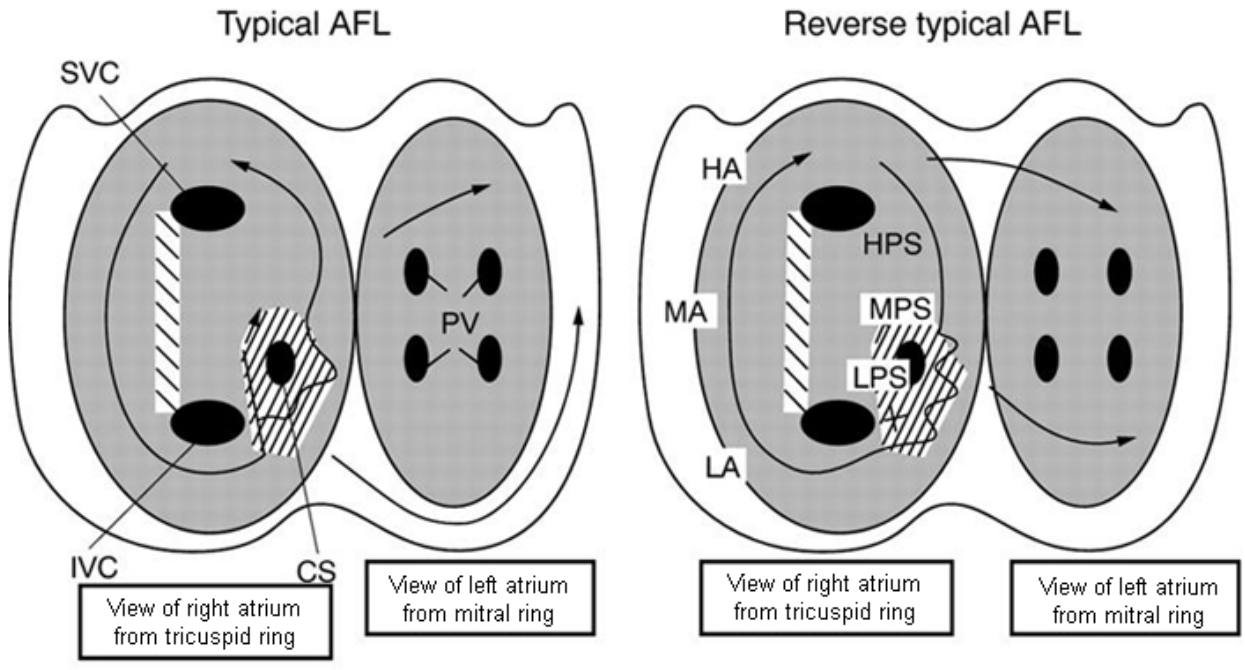

Figure 4: Left panel: atrial activation in typical atrial flutter (AFL). Right panel: activation in reverse typical AFL. The atria are represented schematically in a left anterior oblique view, from the tricuspid (left) and mitral rings. The endocardium is shaded and the openings of the superior (SVC) 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

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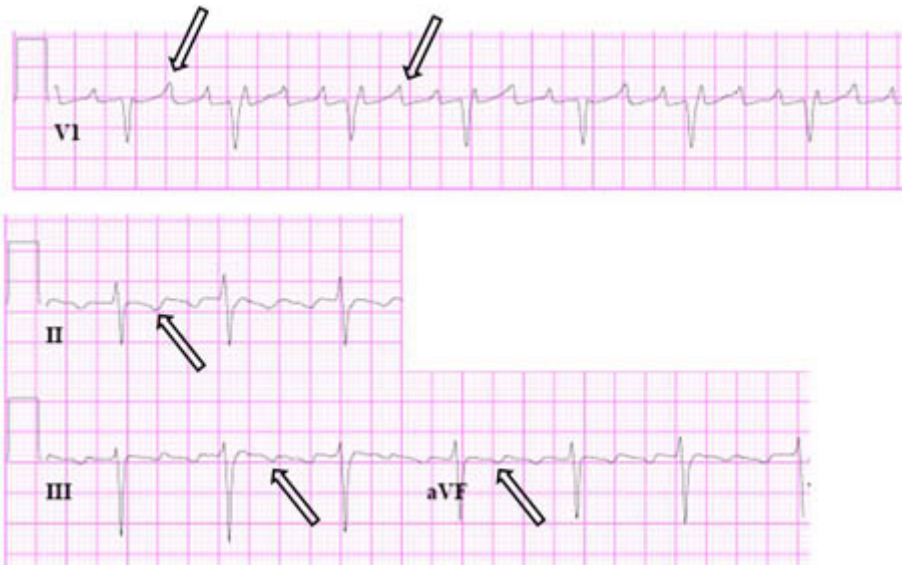

Figure 5: Close-up images of leads V1, II, III, aVF demonstrating the features of typical atrial flutter: positive saw-tooth deflections in lead V1 and negative deflections in leads II, III, aVF (arrows)

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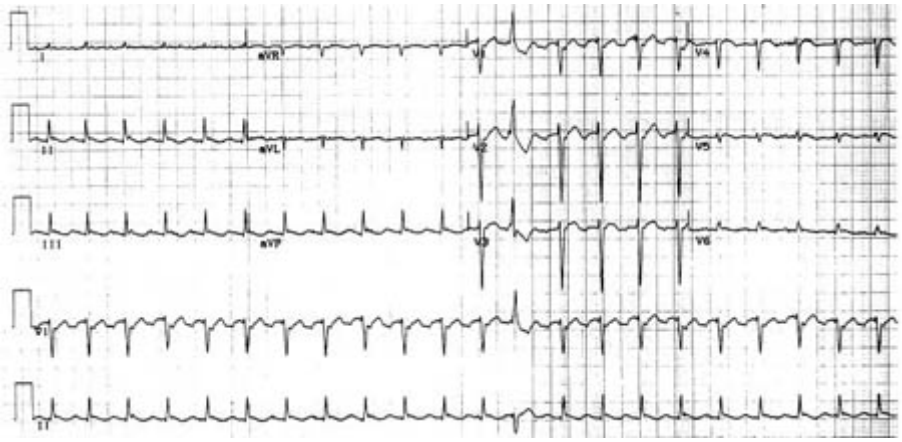

Figure 6: Reverse typical atrial flutter

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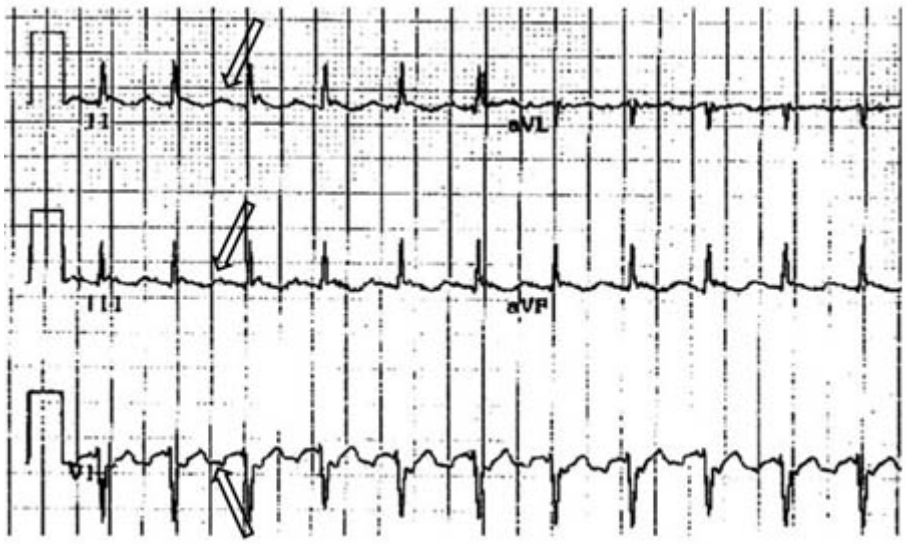

Figure 7: Selected leads from a patient with reverse typical atrial flutter confirmed at electrophysiological study. The atrial deflections are negative in lead V1 and positive in leads II, III, aVF (arrows)

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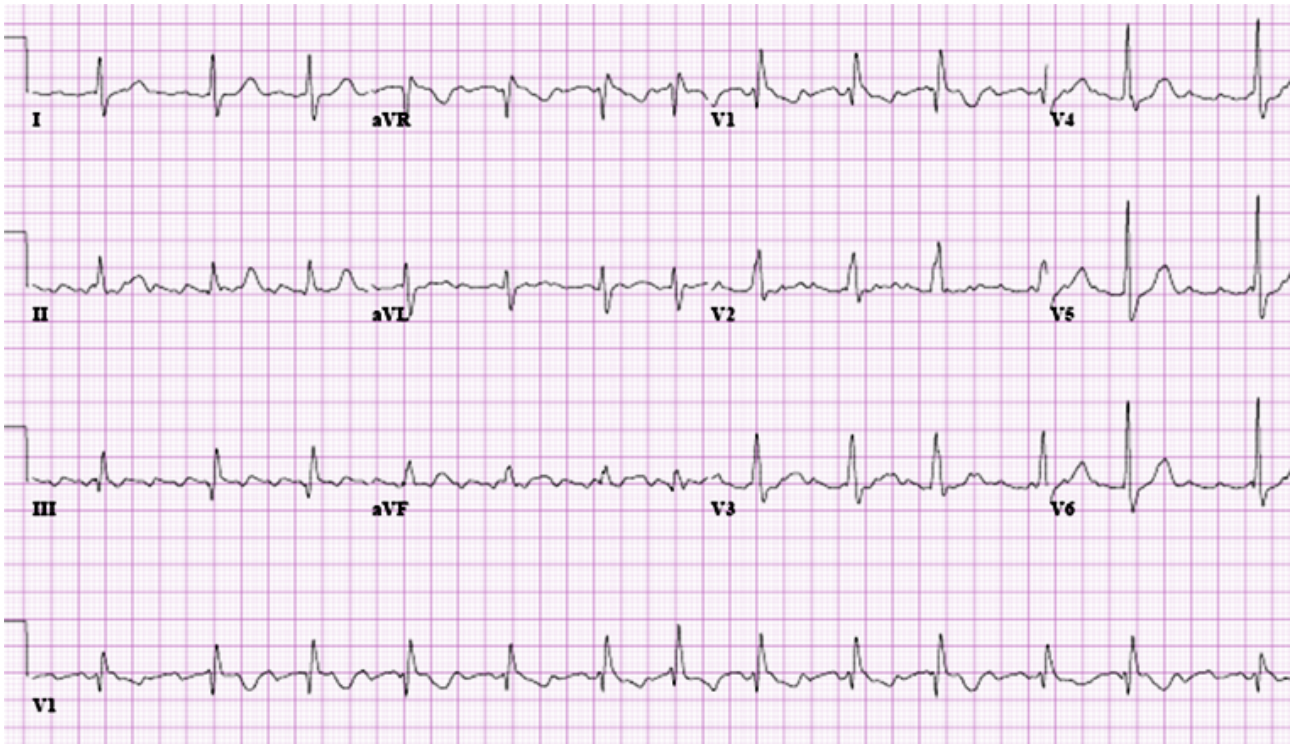

Figure 8: Atypical flutter with right bundle branch block

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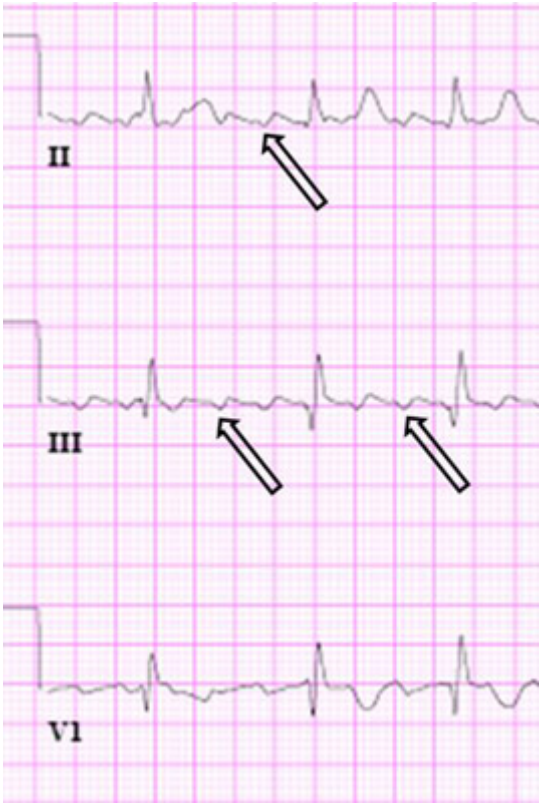

Figure 9: Close up of leads II, III, V1 showing the continuously undulating pattern of atrial deflections not fitting the criteria for typical or reverse typical atrial flutter

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