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Atrial Flutter — Diagnosis, Management and Treatment

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Abstract

Atrial flutter and atrial fibrillation are the two most common arrhythmias which originate in the atrium and cause a narrow complex tachycardia which has thromboembolic risk and coexist clinically. Atrial flutter has been traditionally defined as a supraventricular arrhythmia with an atrial rate of 240–360 beats per minute (bpm). It is due to a macro-reentrant atrial activation around an anatomical barrier. Atrial flutter can be described as typical and atypical. Due to recent innovations in technology, catheter ablation has emerged as the most viable option with a success rate of more than 90 %. Three-dimensional electroanatomical mapping is useful in the treatment of atypical atrial flutter.

Keywords: Typical atrial flutter, Atypical atrial flutter, Cavo-tricuspid isthmus (CTI), Radio-frequency ablation (RFA), Differential pacing, Bidirectional block, Mapping, Entrainment

1. Introduction

Atrial arrhythmias are significant contributors for cardiac co-morbidity especially for stroke, heart failure and recurrent hospitalisations. The more frequent clinically encountered atrial tachyarrhythmias include atrial tachycardia, atrial flutter and atrial fibrillation. Although they are supraventricular in origin, apart from atrial tachycardia, they are not generally included in the nomenclature of supraventricular tachycardia. Atrial flutter has been traditionally defined as a macro-reentrant arrhythmia around a macroscopic (more than 2 cm in area)

anatomical barrier that is confined within the atria. The atrial rate in atrial flutter is approximately 240–360 beats per minute (bpm) with no distinct isoelectric period between the flutter 'F' waves. It is generally paroxysmal in nature in a structurally healthy heart. If the tachycardia persists for a prolonged period, it frequently can degenerate into atrial fibrillation, particularly if the patient already has structural heart disease. As such, atrial flutter and atrial fibrillation often coexist.

Atrial tachycardia is typically characterised by atrial rates >100 bpm but less than 240 bpm with discrete activation sequences and non-sinus P waves including a baseline isoelectric period between these waves on ECG. Its mechanism can be due to triggered activity or increased automaticity of atrial cells. These mechanisms are distinct from that of atrial flutter which is macro-reentrant; however, atrial tachycardia can also be re-entrant in mechanism similar to atrial flutter but on a microscopic level (re-entry around barriers of less than 2 cm).

Atrial fibrillation is due to fibrillatory waves in the atria with rates that are typically greater than 300 bpm in the atria. Currently these waves are considered chaotic and do not behave like the macro-reentry wavefront of atrial flutter. Re-entry however is still thought to play a role in atrial fibrillation, but its exact involvement is unknown.

In this chapter, we will discuss the classification, pathophysiology, clinical presentation, electrocardiographic characteristics, electrophysiological testing and both the pharmacological and ablative management of atrial flutter.

2. Epidemiology

Evidence based on epidemiological studies in the USA suggests that the overall incidence of atrial flutter is about 88/100,000 person-years. When adjusted for age, the incidence of atrial flutter in men is more than 2.5 times that of women. The age-specific incidence of atrial flutter increases exponentially with age from 5/100,000 person-years in those less than 50 years old to 587/100,000 person-years among individuals more than 80 years [1].

The risk factors that are identified as the highest risk for developing atrial flutter include male gender, increasing age, heart failure, chronic obstructive pulmonary disease (COPD) and diabetes mellitus.

3. Classification

Classification for atrial flutter can be based on electrocardiography (ECG) or anatomical and electrophysiological mechanisms [2].

Originally, atrial flutter was classified as types I and II [3]. Type I atrial flutter is the designated classical sawtooth-appearing atrial tachycardia with rate >240 to 360 bpm, lacking an isoelectric baseline between deflections (i.e. continuous flutter wave). Type II

atrial flutter was defined on the basis of a rapid rate (>350 bpm) and the inability to be entrained. However, there are no further systematic electrophysiological studies of type II atrial flutter, and the mechanism is unknown. Now, atrial flutter is referred to as being either typical or atypical.

For clinical and practical purposes, atrial flutter can be broadly classified as per Table 1.

Cavo-tricuspid isthmus (CTI)-dependent atrial flutter or typical flutter	Non-CTI-dependent atrial flutters or atypical atrial flutters
Typical atrial flutter (counterclockwise right atrial flutter)	Right atrial free wall
Clockwise or reverse typical right atrial flutter	Upper-loop re-entry
	Lower-loop re-entry
	Left atrial flutter, including mitral annular atrial flutter, scar and pulmonary vein-dependent atrial flutter and coronary sinus atrial flutter, left septal atrial flutter

Table 1. Classification of atrial flutter

In 2001, the European Society of Cardiology and the North American Society of Pacing and Electrophysiology proposed a classification [4] that takes into consideration both anatomic features and electrophysiological mechanisms.

3.1. Typical atrial flutter (Counterclockwise CTI-Dependant right atrial macro-reentry)

Counterclockwise re-entry is the most common type of macro-reentrant atrial tachycardia. The anatomical boundaries for this re-entrant tachycardia are anteriorly the tricuspid orifice and posteriorly the orifices of vena cavae and the eustachian ridge and the region of the crista terminalis [5, 6]. The conduction of macro-reentrant circuit is up the inter-atrial septum and around the roof towards the crista terminalis and then down the anterolateral wall (RA free wall anterior to the crista terminalis) to the lateral aspect of the tricuspid annulus (Figure 2).

3.2. Reverse typical atrial flutter (Clockwise right atrial macro-reentry)

A reverse direction of rotation of the above circuit in the right atrium (i.e. ascending the lateral wall and descending the posterior and septal walls; see Figure 2) can occur clinically in the typical atrial flutter circuit in 10 % of cases [7]. This is still called typical atrial flutter because the re-entry path is the same, even though the direction of activation is reversed. Reverse typical atrial flutter has also been called clockwise atrial flutter, referring to the direction of endocardial activation from a left anterior oblique fluoroscopic perspective. It is proposed that there is a 9:1 clinical predominance of typical (counterclockwise) atrial flutter compared to clockwise re-entry. This may be related to the localisation of an area with a low safety factor for conduction in the atrial flutter isthmus, close to the atrial septum.
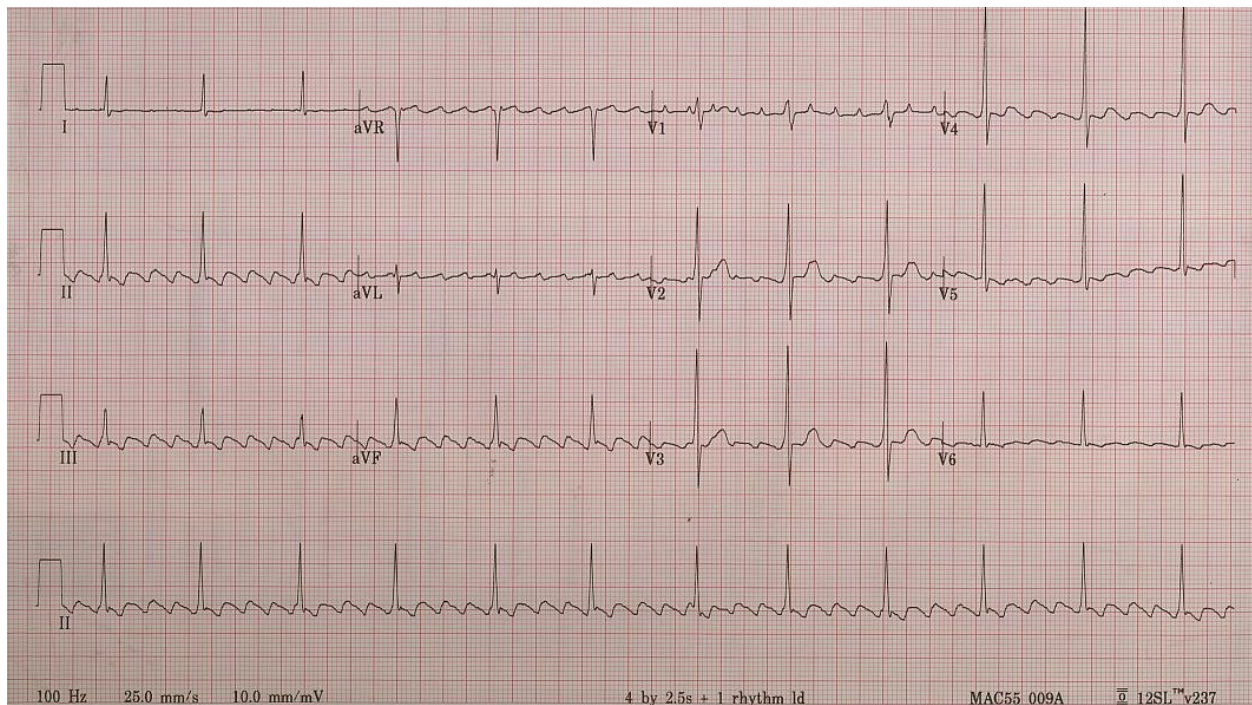
Figure 1. ECG of counterclockwise CTI-dependant atrial flutter: Flutter waves are continuous without an isoelectric baseline, best seen in the inferior leads. The 'F waves' (Flutter waves) are most commonly conducted in the ventricle in a 2:1 manner, giving a regular ventricular response during the arrhythmia typically 150 beats per minute (bpm); however, other multiples of conduction can occur such as 3:1 or 4:1 (Figure 1), giving slow ventricular rates during the arrhythmia. Less commonly, irregular rhythms can be encountered with a variable pattern in conduction to the ventricle

3.3. Lower-Loop Re-entry

Counterclockwise re-entry around the inferior vena cava (see Figures 2 and 12) where the anterior arm of the circuit is the inferior vena cava. The posterior arm is the low posterior right atrial wall with conduction across the crista terminalis [8]. Electroanatomical or conventional mapping shows activation rotating around areas of low-voltage electrograms in the right atrial free wall, not due to surgical scars.

3.4. Atypical atrial flutter

3.4.1. Lesion macro-reentrant atrial tachycardia

In this macro-reentrant atrial tachycardia, the central obstacle of the circuit is an atriotomy scar, a septal prosthetic patch, a suture line or a line of fixed block secondary to radio-frequency ablation or other causes of scar [9]. This can also lead to complicated tracts for the re-entry circuit.

3.4.2. Right atrial free (Lateral) wall atriotomy tachycardia

The best characterisation of atriotomy macro-reentrant atrial tachycardia is due to activation around an area of low voltage or scar in the lateral right atrial wall, with a main superoinferior axis.
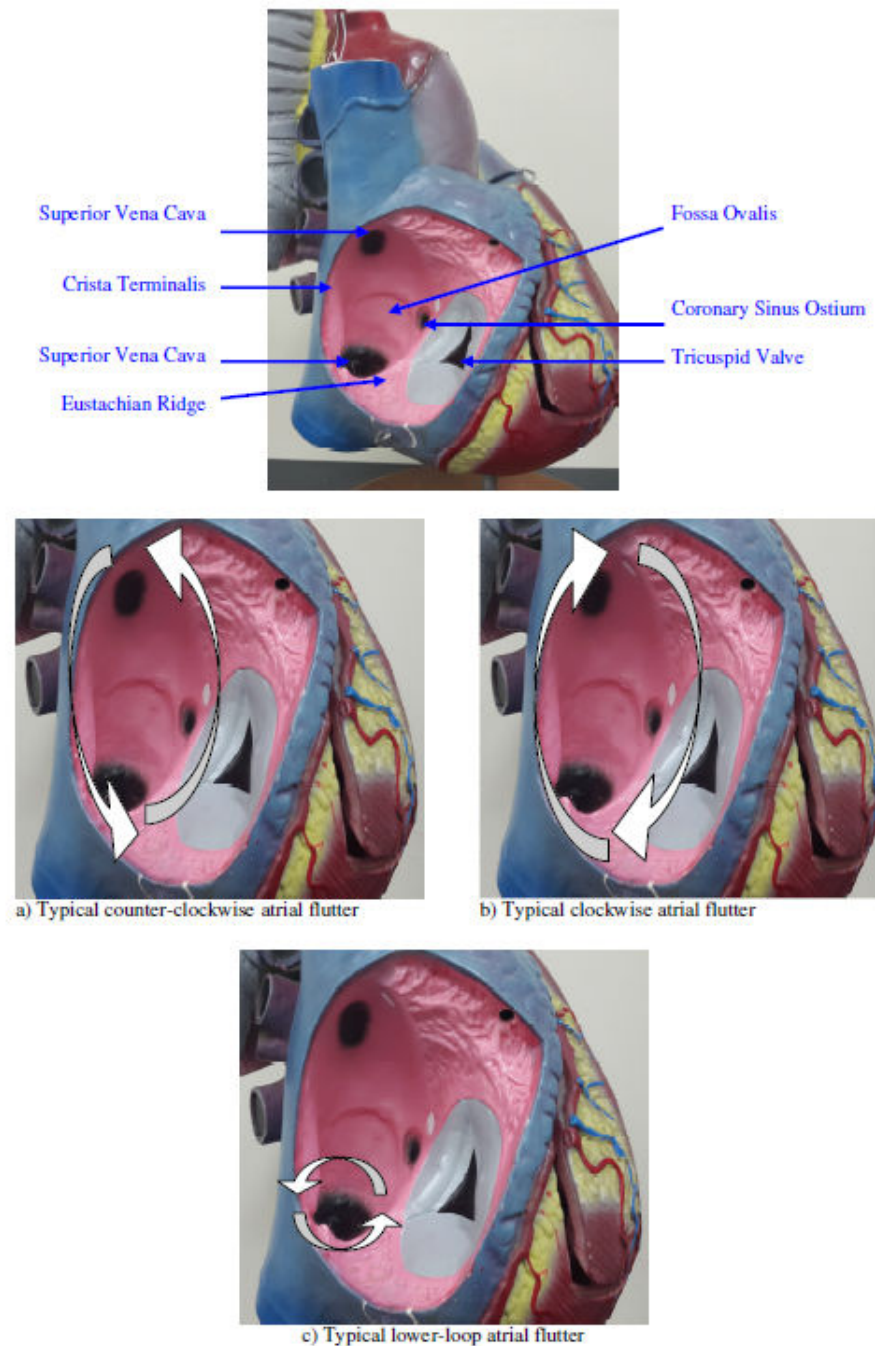
Figure 2. Typical Atrial flutter

3.5. Double-wave re-entry

In this macro-reentrant tachycardia, two wavefronts circulate simultaneously in the same re-entrant circuit. A stable macro-reentrant atrial tachycardia can originate in the left atrium. The clinical incidence is not well known but may be 1/10th that of typical atrial flutter. There is still little information on the anatomical bases of left atrial macro-reentry tachycardia, although

recent reports have characterised the substrate as showing wide scarred areas with low voltage or absent electrograms [10].

4. Clinical presentation

Atrial flutter can be paroxysmal or persistent. When atrial flutter is associated with an increased ventricular response, it can result in palpitations, shortness of breath, chest pain, fatigue or pre-syncope. If a patient presents with atrial flutter and a rapid ventricular rate, stroke, tachycardia-induced cardiomyopathy and rarely myocardial infarction are complications that can be encountered. Syncope in the setting of atrial flutter is rare if there is no significant cardiac history [11]. When presenting because of a more prolonged episode, increased symptoms of heart failure may be evident. Occasionally, atrial flutter is an incidental finding on ECG with patients who are completely asymptomatic.

5. Management

Therapy for atrial flutter has two goals: management of the arrhythmia itself with either rate control or rhythm control and management of the complications of the arrhythmia with stroke prophylaxis [12].

5.1. Non-invasive management

5.1.1. Rate control

Rate control is generally reserved for patients who are in permanent atrial flutter and have no or minimal symptoms and cannot achieve rhythm control due to co-morbidities or are not willing to undergo procedures or take medications. There is debate in the literature about what exactly is adequate rate control; this however pertains to atrial fibrillation as it has not been specifically studied in atrial flutter. The same parameters however could generally be applied as the goal is to avoid tachycardia-induced cardiomyopathy whilst preserving exercise capacity. Typically, the aim is an average 24-hour heart rate over 24 hours of 80 bpm and a maximum of less than 130 bpm [13]. An emerging data however shows that less strict control such as heart rates less than an average 24-hour heart rate of less than 110 bpm is adequate [13, 14]. Various pharmacological agents which are used in non-invasive management are presented in Table 2.

Rate control of atrial flutter can be very difficult to achieve pharmacologically. It is important to understand that atrial flutter ablation has a high success rate unlike atrial fibrillation, and extreme methods of rate control such as pacemaker implantation and AV nodal ablation are rarely used as a management strategy.

Rate control	Rhythm control	Stroke prophylaxis
Beta-blocker	Class Ia: procainamide	Coumadin
Calcium channel blockers	Class Ic: flecainide, propafenone	Newer anticoagulation agents: dabigatran
Digoxin	Class III: sotalol, amiodarone, ibutilide,	Rivaroxaban
Amiodarone	dofetilide	Apixaban

Table 2. Pharmacological management therapies

5.1.2. Rhythm control

In an acute setting, atrial flutter with hemodynamic compromise or rarely significant cardiac symptoms (i.e. severe chest pain), synchronised direct current cardioversion is indicated to revert patients to sinus rhythm. Previously, lower defibrillator outputs such as 50 J were employed to prevent pain, but the current recommendation is 200 J biphasic with either anteroposterior or midline and lateral defibrillation pad positioning. There is no difference in pain and potential of ventricular fibrillation induction with lower energy levels.

In clinically stable atrial flutter, the various non-invasive management therapies include cardioversion (electrical or pharmacological) and rate control with pharmacotherapy to slow down AV nodal conduction (see Table 2) or if an atrial pacing lead is in situ rapid atrial pacing for overdrive termination. This is commonly burst pacing to depolarise tissue in the macro-reentrant circuit into which the activation front of the arrhythmia is depolarising to terminate the tachycardia. The pacing is typically for three to five seconds at rate approximately 20 ms less than flutter wave cycle length; though this also has the potential to cause atrial fibrillation.

For a list of antiarrhythmic drugs for cardioversion of atrial flutter, refer to Table 2. Classes Ia and Ic can result in slowing the rate of atrial flutter which can facilitate 1:1 conduction of atrial flutter via AV node and cause a rapid ventricular rate. Thus, it is recommended to have concurrent AV blocking agents to control the ventricular response. These antiarrhythmic agents are not very well studied in atrial flutter patient population. The data about their efficacy is derived from clinical trials where atrial flutter was grouped with atrial fibrillation. In terms of prevention of atrial flutter recurrence, flecainide and dofetilide have a long-term efficacy of 50 % and 70 %, respectively [15, 16].

5.2. Stroke prevention

Anticoagulation is key to preventing ischemic cerebrovascular events [17]. It is extremely important when either above-mentioned management option is considered. Even though the evidence for anticoagulation in the atrial flutter patient population is not as robust as for atrial fibrillation, it is felt that the risks are similar especially for typical cavo-tricuspid isthmus-dependent atrial flutter. Thromboembolic prophylaxis is indicated in the management of chronic atrial flutter similar to atrial fibrillation. In our clinical practice, we use CHA2DS-VASc score which has superseded the older CHADS2 score to calculate the annual risk of cerebral thromboembolic event [18]. For oral anticoagulants that are used to prevent stroke, refer to Table 2. Patients who have atrial flutter and undergo DC cardioversion should be on novel

anticoagulation or on warfarin with therapeutic international normalised ratio (INR) (>2) for a minimum period of three weeks prior to the DCCV and for a minimum of one month later. The new oral anticoagulants do not have as much data for direct-current cardioversion as warfarin therapy, and some clinicians perform trans-oesophageal echocardiograph prior to direct-current cardioversion [19]. There is no role for aspirin in stroke prophylaxis as the risks of bleeding are greater than the benefit of stroke reduction.

5.3. Invasive strategy

5.3.1. Typical cavo-tricuspid-dependent atrial flutter

A standard electrophysiological (EP) case for typical CTI-dependent atrial flutter most often requires the use of three catheters; these include the ablation catheter, a multipolar coronary sinus catheter and a right atrial (RA) mapping catheter. The RA mapping catheter can be either a decapolar or duodecapolar catheter (sometimes referred to as orbital catheter) and is placed along the lateral RA wall anterior to the crista terminalis with the tip down to the lateral inferior RA and tricuspid annulus. Some operators also prefer to additionally use a His and/or right ventricular catheter (Figure 3). Radio-frequency ablation (RFA) is the most commonly used ablation modality in treatment of atrial flutter.
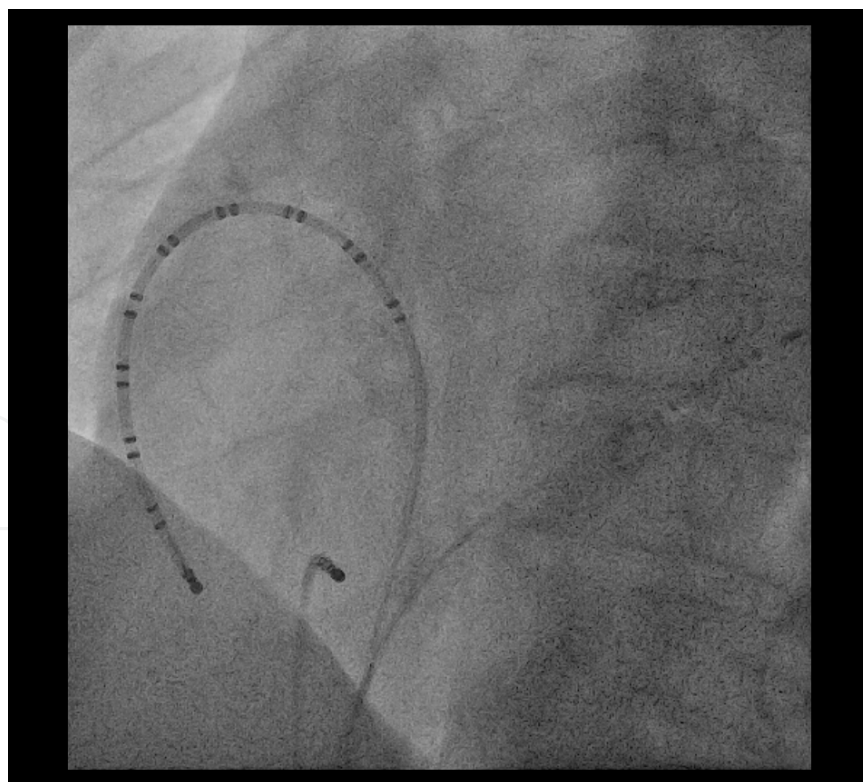

Figure 3. Fluoroscopic image of catheters used for a typical atrial flutter study with duodecapolar catheter in the right atrium, ablation catheter at the cavo-tricuspid isthmus and decapolar catheter in the coronary sinus (courtesy of PA hospital EP Lab)

A similar catheter configuration may be used for an atypical or non-CTI-dependent atrial flutter although a 3D mapping system for these arrhythmias is highly beneficial and can negate the need for other electrogram-based mapping catheters. The mapping system can be utilised to define the entire macro-reentrant circuit by identifying the anatomical boundaries and the ablation target which is generally the area of slow conduction. A line of block is achieved by making a linear ablation line between areas of conduction block (discussed under Radio-Frequency Ablation).

5.4. Electrophysiological study

Generally with cavo-tricuspid isthmus-dependent ablation, there is little electrophysiological study performed as the target of ablation and arrhythmia circuit are known. However, manoeuvres such as entrainment (discussed below) can still be used to confirm cavo-tricuspid isthmus dependence. In atypical atrial flutter ablation, entrainment manoeuvres can be used to localise the arrhythmia circuit. However, with the advent of non-fluoroscopic mapping, entrainment is being employed less frequently to confirm isthmus dependence.

Entrainment is used to determine if a specific anatomical site is a part of the re-entrant circuit [20]. To confirm that atrial flutter is typical and cavo-tricuspid isthmus dependant (not a bystander), entrainment from the CTI and/or another site such as the proximal coronary sinus is performed. By entraining in the atria at a cycle length approximately 10 to 20 ms faster than the tachycardia cycle length and then measuring the post-pacing interval (PPI) (return cycle length), one can determine if CTI (or pacing site) is a part of the re-entrant circuit (Figures 4). The post-pacing interval is the time between the last pacing stimulus that entrained the tachycardia and the next recorded electrogram at the pacing site. The pacing site is considered to be a part of the circuit if the post-pacing interval (PPI) is equal or within 30 ms of tachycardia cycle length.

If entrainment from the isthmus does not demonstrate isthmus involvement, further thought needs to be carried out regarding treatment options, as the tachycardia is likely to be atypical and 3D mapping may be more appropriate.

5.5. Radio-Frequency Ablation (RFA)

Ablation therapy for typical atrial flutter targets the cavo-tricuspid isthmus (CTI). It is a narrow point of the circuit and the slow conduction part of the circuit between two nonconductive boundaries; anteriorly this is bounded by the tricuspid annulus and posteriorly by IVC. It is easily accessible percutaneously and distant from the AV node. Conventionally, atrial flutter ablation involved delivering RF energy in a linear ablative line across the entire isthmus to create bidirectional block. Ablation is started on or near the tricuspid annulus and the catheter brought (dragged) back along the isthmus towards the IVC. Ablation can be performed in both atrial flutter and during atrial pacing (empirically) from the coronary sinus but can equally be performed with pacing from the lower right atrium.
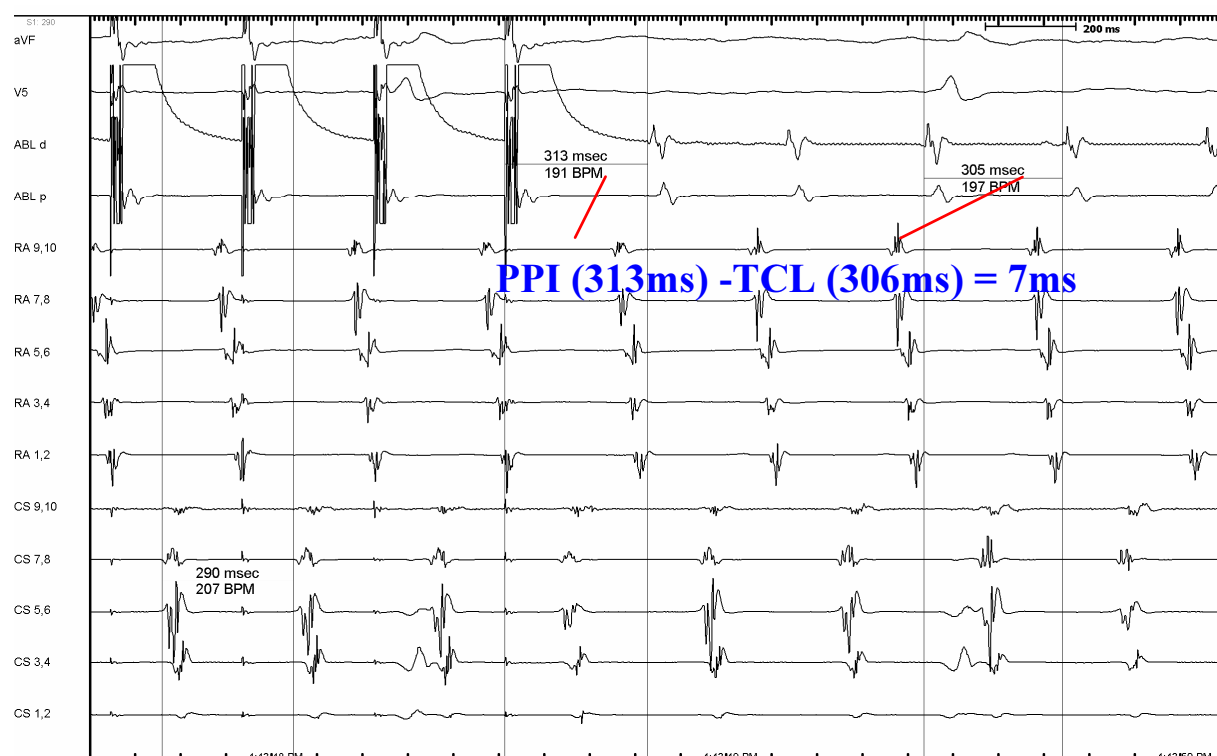
Figure 4. Entrainment of typical CTI ablation demonstrating that pacing site (ablation catheter) is within the circuit of the tachycardia. PPI − TCL = 7 ms. Note also the unidirectional activation of the lateral RA wall superiorly (RA 9,10) to inferiorly (RA 1,2) (courtesy of PAH EP Lab)

6. Post-ablation bidirectional electrophysiological study

6.1. Bidirectional block

This is the electrophysiological endpoint of ablation of the isthmus in CTI-dependent atrial flutter [21]. Activation mapping to measure the trans-isthmus conduction time, differential pacing and online double potentials are the three commonly used techniques that can be employed to determine if the ablation has been successful and bidirectional block has been achieved.

6.2. Activation mapping

Activation mapping involves pacing from either side of the ablation line (proximal CS pacing low lateral RA are commonly used). With incomplete CTI ablation, the wavefront along the RA mapping catheter will be fused as conducts in different directions as depicted in Figure 5 and 6.
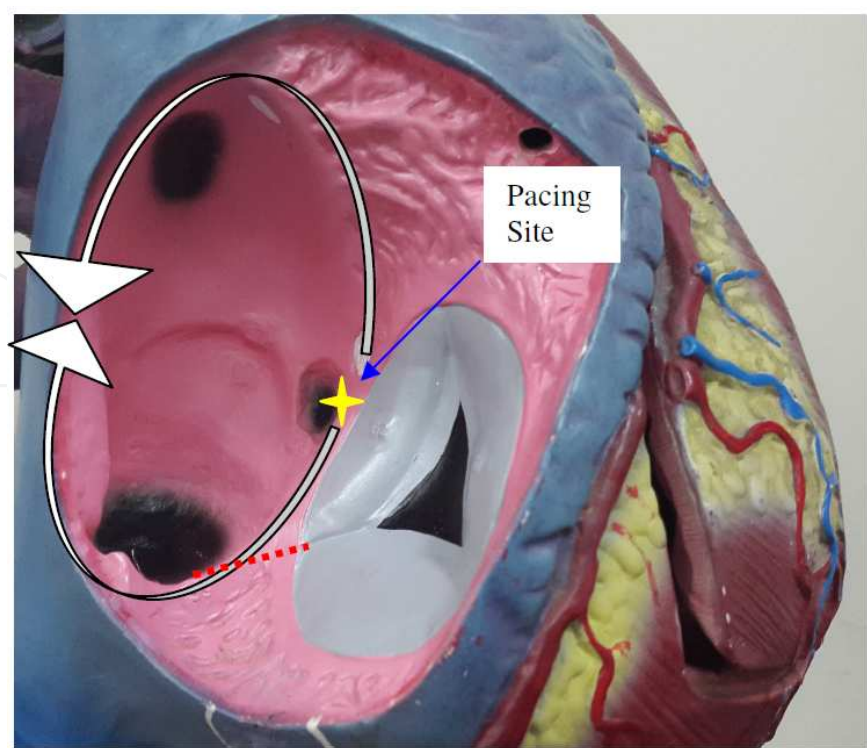
Figure 5. Pacing site from proximal coronary sinus.
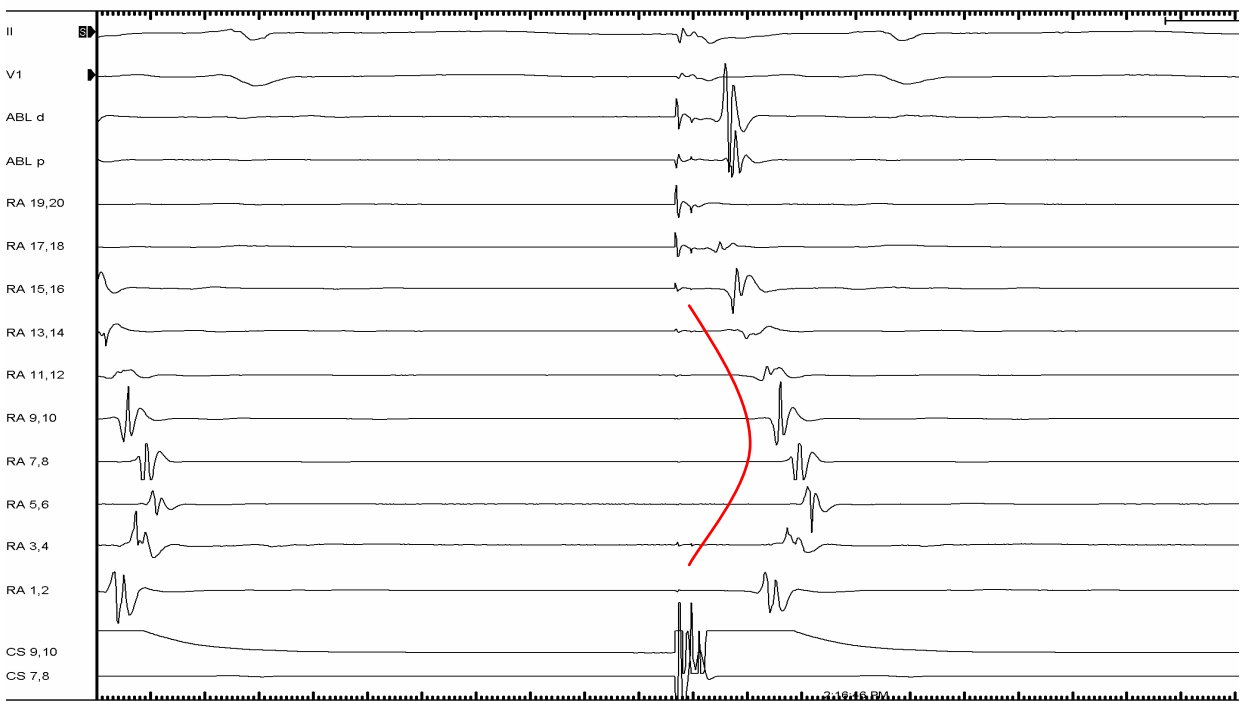

Figure 6. Prior to CTI ablation depicting activation pattern in the right atrium. It is chevron shaped due to persistent conduction through the isthmus and fusion of wavefront in the right atrium

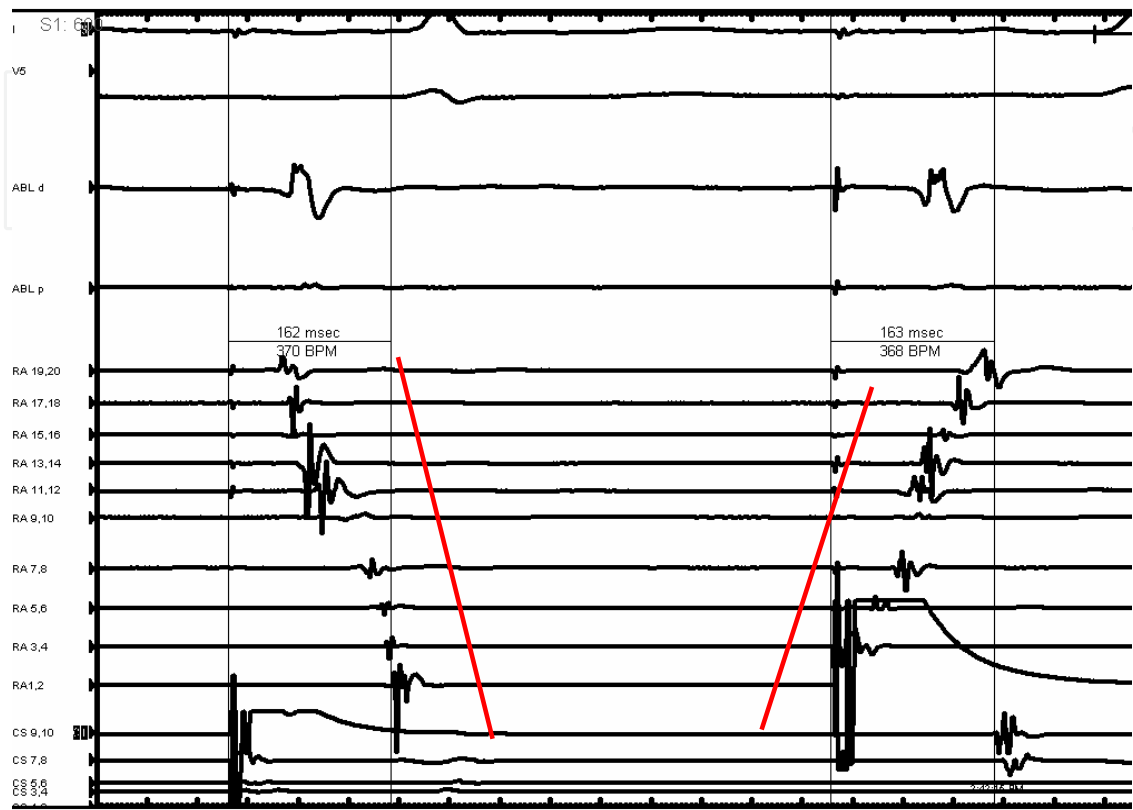With successful bidirectional block, the activation along the RA catheter is uniform (Figures 7 and 8).

Figure 7. Bidirectional block demonstrated by activation; A, indicates proximal CS pacing and sequential activation around the RA from proximal to distal RA catheter; B, pacing from distal RA with sequential activation from distal to proximal RA – both showing conduction block over the cavo-tricuspid isthmus region (Courtesy of PAH EP Lab)
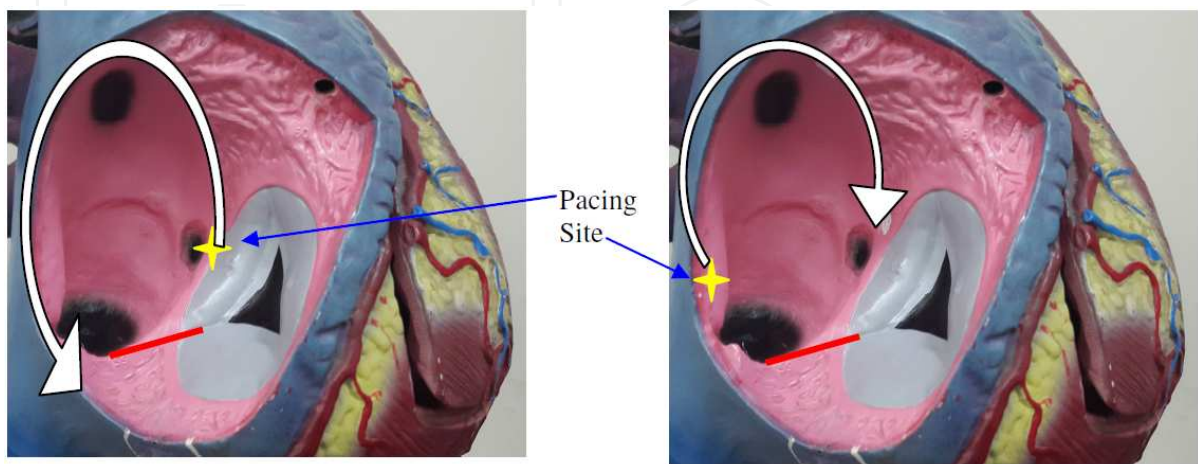

Figure 8. Pacing from proximal sinus and from RA lateral/anterior wall respectively

6.3. Trans-isthmus conduction time

During the flutter circuit, the isthmus conduction time accounts for ~33 % of the cycle time, and the RA cycle accounts for ~66 % of the cycle length [22]. The isthmus conduction time is measured from the pacing site to the signal arising on the other side of the cavo-tricuspid isthmus.

At baseline, if the patient is in sinus rhythm, pacing from proximal CS, the measurement would be to low in RA and vice versa. When there is no damage to the isthmus, conduction time should be <90 ms. Post-successful CTI ablation trans-isthmus conduction time increases by 50 % [23] or from <90 ms to >140 ms.

6.4. Differential pacing

One important manoeuvre to establish bidirectional block is to perform differential pacing [24]. Differential pacing is most useful when there is a very slow conduction across CTI with long conduction time, leaving it difficult to confirm bidirectional block with trans-isthmus time measurements and activation mapping. It involves pacing at two different sites on the lateral side of the CTI line (along the lateral wall of the RA).

During differential pacing, the wavefront conducts in two directions, counterclockwise towards the isthmus line and clockwise around the right atria towards the septal border of the CTI line (or the CS ostium). When isthmus block has been achieved, moving the catheter further away from the CTI line should result in a decreased clockwise activation time from lateral pacing site to the CS ostium. It can also be performed the other way by pacing from a standard site (CS ostium) and then positioning the mapping catheter in two sites on the lateral side of the CTI line.

6.5. Online double potentials

Online double potentials represent an area of local conduction block at the line of ablation. A 110 ms separation of signals has been identified as a 100 % positive predictor value for isthmus block [25]. The early first signal results from the initial wavefront from the pacing stimulus reaching the line, which then encounters block. The second delayed signal results from activation entering the opposite side of the line of ablation.

The online double potential technique is an assistance guide to determining bidirectional block and should be used in conjunction with other methods.

6.6. Atypical atrial flutter

An isthmus for atypical flutter can be formed by a number of anatomical barriers, including the tricuspid or mitral annuluses, superior or inferior vena cava, pulmonary veins or the CS ostium. It can also form a circuit secondary to scars which can occur after infarction, myocarditis or congenital or valvular surgery. Electrically, the isthmus can be represented by low amplitude or fractionated signals, areas of slow conduction and/or double potentials. Double
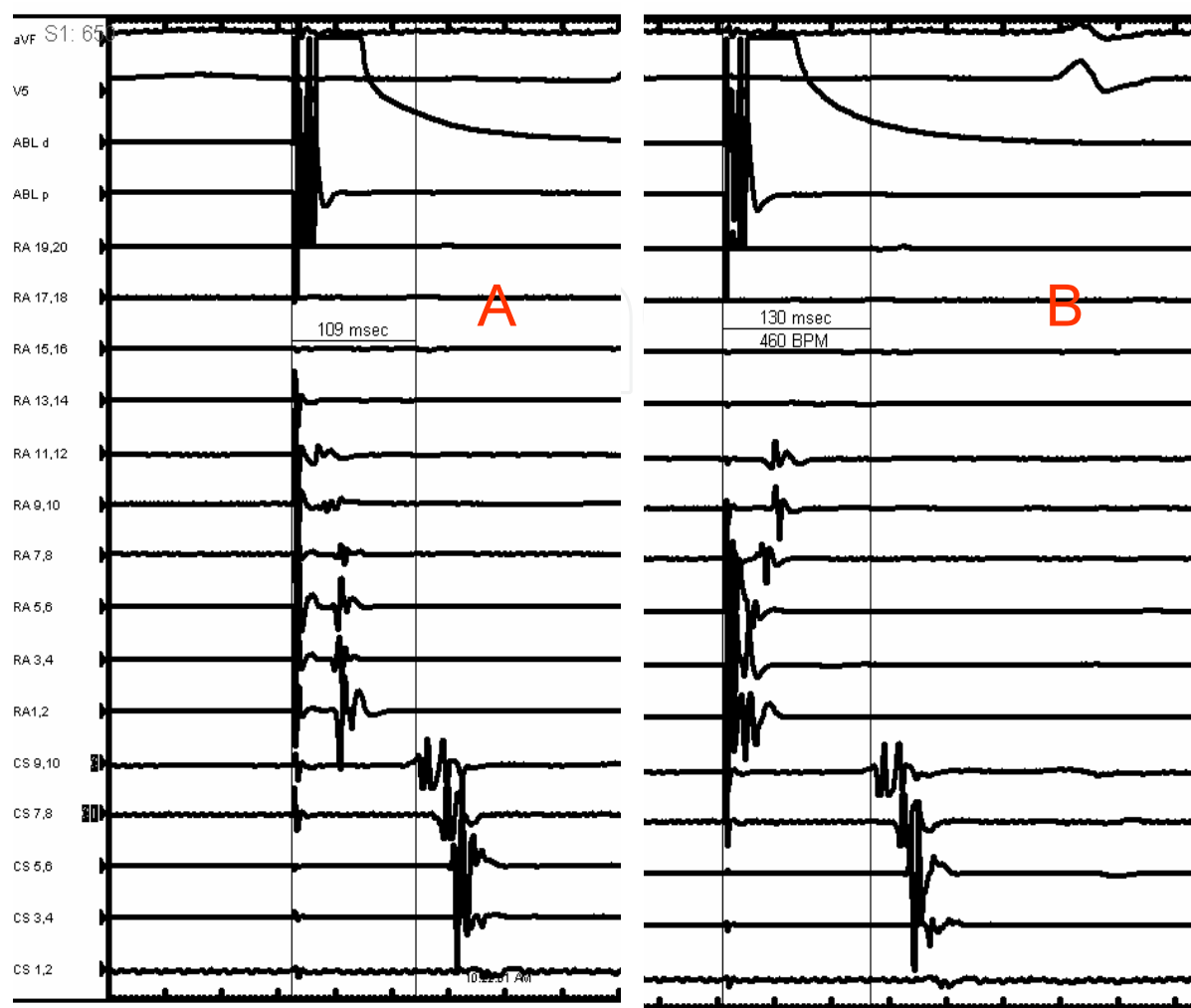
Figure 9. Differential pacing with pacing from RA 11, 12 of a duodecapolar catheter (lateral) and then more laterally from site RA 3,4 of the duodecapolar catheter (courtesy of PA EP Lab)

potential indicates line of block and fragmented potentials and mid-diastolic potentials indicate critical zones of slow conduction.

Atypical atrial flutter can be very difficult to assess and treat. The use of anatomical and 3D mapping systems has an important role in identification and successful ablation of atypical atrial flutter.

6.7. 2D mapping and electrophysiology

To assist with 2D and signal mapping, it is very important to know the chamber the flutter is originating from.

6.7.1. Localising atypical flutter to the right atrium

Activation of the coronary sinus from proximal to distal is the first clue that the flutter is most likely to be originating from the RA. Other clues include RA activation time accounting for

Figure 10. Online double potentials; an immediate signal post-ablation site (medial side of isthmus line) and a signal out later (lateral side of the isthmus line)

more than 50 % of the tachycardia cycle length, entrainment at multiple sites in the RA and post-pacing interval of equal or less than 30 ms including CTI and right free wall but not RA septum. Variability in LA cycle length with relatively fixed RA cycle length is also a clue that atypical flutter is localised to the right atrium.

6.7.2. Localising Atypical Flutter to the Left Atrium

Electrogram activation is usually, but not exclusively, distal to proximal on the CS catheter with passive RA conduction, or early septal RA activation, indicating breakthrough to the RA from the LA. Entrainment within the RA at multiple sites should elicit a PPI – TCL response of more than 30 ms.

6.8. 3D or non-fluoroscopic mapping

Mapping atypical flutter with a 3D mapping system (CARTO and NavX) allows direct visualisation of the re-entrant circuit [26]. When mapping the flutter, it is important to get high resolution and to cover as much as the cycle length as possible (Figure 11). Analysing up to 80–100 points increases accuracy, and covering >90 % of the circuit allows for the whole circuit to be covered. 3D mapping can be used to demonstrate the typical activation pattern. Scar can be determined by areas of signals less than 0.5 mV or inability to pace capture at 20 mA.
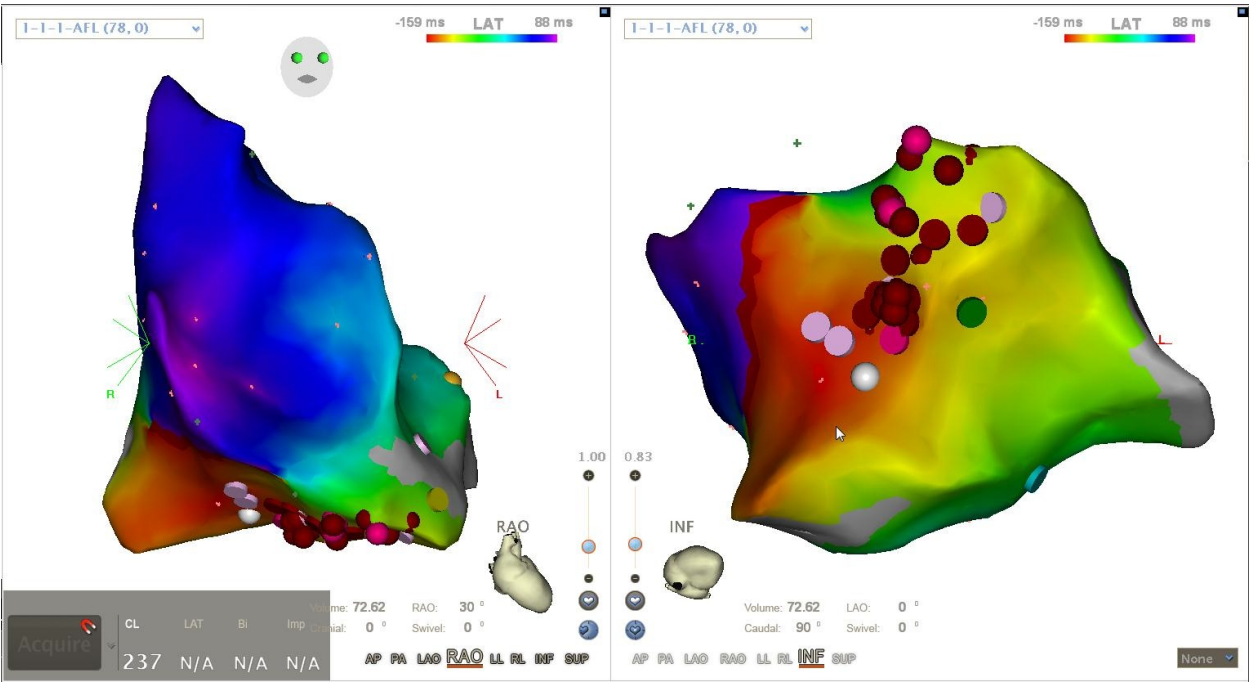
Figure 11. Figure 11: A 3-dimensional CARTO map of cavo-tricuspid isthmus-dependant atrial flutter in a patient with a history of congenital heart disease (tetralogy of Fallot, s/p repair) and symptomatic typical atrial flutter. Right anterior oblique (RAO) view and inferior view of RA demonstrating the macro-reentrant mechanism and the ablation sites
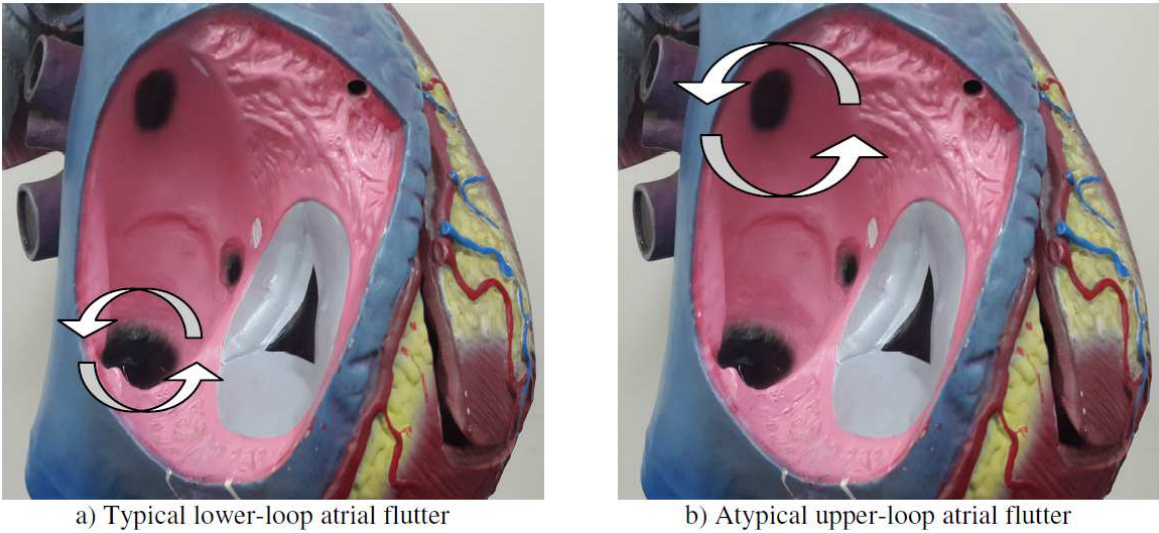

a) Typical lower-loop atrial flutter   b) Atypical upper-loop atrial flutter

Figure 12. Activation sequence in upper-loop re-entry compared to lower-loop re-entry. Lower-loop re-entry is classified in the same group as typical atrial flutter because the circuit is dependent on CTI

6.8.1. Upper-loop re-entry atrial flutter

This atypical flutter has clockwise activation sequence around the SVC and breaks out through the crista terminalis (Figure 12). This type of flutter is typically seen in patients with hyper-

tension or some structural heart disease. Entrainment should be performed between the fossa ovalis and the superior vena cava (SVC) in the atrial septum. The targeted ablation site is at the excitable gap in the crista terminalis.

6.8.2. RA free wall atrial flutter

This atrial flutter is seen in patients with no structural heart disease, who develop scar in the right atria and in postsurgical patients with a scar caused by an atriotomy procedure. When mapping for the scar relating to the flutter, online double potentials will be present, and fractionation at the end of the line denotes the pivot point for the flutter. The ablative strategy for this flutter is to create a linear line from the lateral RA (at the end of the line) to the IVC or to create a line of block between the scar and the crista terminalis. It is also recommended that the cavo-tricuspid isthmus line be performed. It is important to pace in the right atrium at the proposed ablation point to avoid phrenic nerve injury.

6.8.3. LA atrial flutter

Flutter arising from the left atria is almost certainly related to the patient having some sort of structural heart disease. It is most likely to arise from the mitral annulus, the pulmonary veins or a spontaneous scar. The target ablation site for left atrial flutters depends on the circuit of the flutter and the position of the anatomical block. A new class of left atrial flutter has arisen in the recent past which is idiopathic in origin, post-pulmonary vein isolation and left atrial ablation for atrial fibrillation. This form of atrial flutter occurs between ablation lines performed to isolate and compartmentalise the left atrium to modify the burden of atrial fibrillation in patients. It has rapidly become a very common form of left atrial flutter, perhaps the most common.

7. Ablation

7.1. Radio-frequency ablation

Radio-frequency ablation uses radio-frequency energy in the form of electrical current delivered from typically 4 to 8 mm catheter tip and collects it on an indifferent electrode patch which is commonly placed on the mid-spine in the lumbar region. The density of the current at the catheter tip causes an ablation lesion 4–6 mm deep in the cardiac tissue which interrupts electrical conduction. An 8 mm catheter tip is increasingly being used as the first choice in CTI ablation due to the length and depth of ablation line needed. Irrigated catheters with a shorter tip length of 3.5–4 mm are employed in redo cases where deeper ablation lesions are needed.

In the management of especially typical atrial flutter, ablation has evolved as the first-line treatment, even after a single episode of documented symptomatic atrial flutter above antiarrhythmic pharmacotherapy [27]. In this multicentric prospective randomised study, amiodarone and radio-frequency ablation were compared in 104 patients (aged 78 +/-5 years; 20 women) with atrial flutter. The recurrence of atrial flutter was 3.8 % versus 29.5 %, $P<0.0001$.

Ablation therapy for atrial flutter has a success rate depending on the studies of ~90–95 % [28]. The other forms of atrial flutter generally also respond best to ablation if the circuit can be mapped and a line of conduction is performed.

The conventional method of CTI ablation was to make a linear ablation line along CTI as guided by electrograms; however recently, the 'maximum voltage-guided' technique using radio-frequency application has been studied as an alternative method and has demonstrated to be equivocal in achieving bidirectional cavo-tricuspid isthmus block. Ablation for atrial flutter using the "Maximum Voltage Guided" technique results in significantly less ablation applications than the traditional approach, potentially by concentrating ablation lesions on the muscle bundles responsible for Trans-isthmus conduction [29, 30].

7.2. Complications

During ablation, signs that can indicate that the catheter is ablating in a possibly unsafe position can include ventricular ectopy or a sharp increase in measured catheter tip impedance. Ventricular ectopy could indicate that the ablation catheter is in close proximity to the tricuspid annulus or right ventricle and potentially result in valve damage. A sharp impedance rise could indicate that the catheter has been pulled back into the IVC or wedged into a pouch on the isthmus which can create a sharp temperate increase and a possibility of a steam pop.

In patients who have had persistent atrial flutter, ablation can result in post-reversion asystole. This is commonly due to underlying sinus node disease. So one should always be ready to pace in either the atria (assuming normal AV node conduction) or ventricle (assuming a ventricular catheter is in place). Rarely, cavo-tricuspid isthmus ablation can lead to atrioventricular block or myocardial infarction if the right coronary artery or the artery to the atrioventricular node is occluded by an ablation lesion.

Other complications which can potentially occur during atrial flutter ablation include venous access/puncture (one in 100) such as hematoma, bruising and deep vein thrombosis, with or without pulmonary embolism. A small risk of stroke (1 in 500), pericardial effusion or death (1 in 1,000) remains with radio-frequency ablation method.

7.3. Emerging trends and novel technologies

Recently, the focus of atrial flutter ablation has been on emerging technology. There has been some promising data about the use of contact force catheter and its effectiveness at predicting the lesion size using the lesion size index (LSI). It is based on an algorithm which integrates contact force, radio-frequency power and ablation duration to make a key parameter, force-time integral [31]. Contact force catheters are used for pulmonary vein isolation. Contact force parameters however have not yet been employed for atrial flutter ablation. We are currently involved in a research study of a contact force catheter, and the initial results have shown that the guidance of CTI ablation using novel CF parameters can be performed successfully. It is independent of traditional parameters and demonstrates excellent short- to medium-term results.

8. Other ablation modalities

Cryoablation is another ablation modality which also has a good success rate with a 10 % recurrence rate [32]. The short- and long-term results are comparable to RFA, and it is relatively pain-free.

Microwave ablation has been used successfully to cure atrial fibrillation during open-heart surgery, and it has been demonstrated to be safe and effective in a preliminary feasibility study [33].

High-frequency ultrasound and diode laser balloon ablation catheter [34] have shown some promising results in the treatment of atrial fibrillation [35]. The use of these technologies in treatment of atrial flutter needs further research.

9. Conclusions

Atrial flutter is a common atrial arrhythmia with a characteristic mechanism. Its morbidity and mortality are similar to atrial fibrillation; but unlike atrial fibrillation, it can be cured. However, a thorough understanding of electrophysiological properties and anatomical landmarks is essential in achieving a successful ablation outcome and in reducing complication rates. The advent of newer technology potentially has a favourable outlook in reducing the time required to perform the procedure.

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