ATRIAL FLUTTER ABLATION OUTCOMES AND PREDICTORS OF RECURRENCE

PROJECT REPORT
Submitted during the course of
DM Cardiology

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DECLARATION

I, Dr Suraj Narasimhan A, hereby declare that the project in this book, titled “Atrial flutter ablation outcomes and predictors of recurrence” was undertaken by me under the supervision of the Faculty, Department of Cardiology, Sree Chitra Tirunal Institute for Medical Sciences and Technology.

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“ATRIAL FLUTTER ABLATION OUTCOMES AND PREDICTORS OF RECURRENCE”

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I express my sincere thankfulness to the patients of this hospital with whose cooperation this study has been possible. I hope this thesis would help in planning better patient care.

Dr. Suraj Narasimhan A
### ABBREVIATIONS

<table>
<thead>
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<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>AFL</td>
<td>Atrial Flutter</td>
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<tr>
<td>CL</td>
<td>cycle length</td>
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<tr>
<td>RA</td>
<td>right atrium</td>
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<tr>
<td>LA</td>
<td>to left atrial</td>
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<tr>
<td>CTI</td>
<td>Cavotricuspid Isthmus</td>
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<tr>
<td>MRAT</td>
<td>macroreentrant atrial tachycardias</td>
</tr>
<tr>
<td>AF</td>
<td>Atrial Fibrillation</td>
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<tr>
<td>CCW</td>
<td>Counterclockwise</td>
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<td>CW</td>
<td>Clockwise</td>
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<tr>
<td>AES</td>
<td>Atrial Extra Stimulus</td>
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<td>CS</td>
<td>Coronary Sinus</td>
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<tr>
<td>PPI</td>
<td>Post Pacing Interval</td>
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<tr>
<td>LAO</td>
<td>Left Anterior Oblique</td>
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<tr>
<td>RAO</td>
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INTRODUCTION

Atrial flutter (AFL) or Macro reentrant atrial tachycardia is a supra ventricular tachycardia whose reentry circuit is entirely located either in the right atrium or in the left atrium(1). Macroteventrant atrial tachycardia is typically described as “circular activation (reentrant) that revolves around a large obstacle, arbitrarily defined as being several centimeters in diameter” (2). Flutter describes a specific electrocardiographic pattern of atrial tachycardia ≥240/min, with a regular and uniform continuous waveform without an isoelectric baseline. Contrasting to AFL, atrial tachycardia has regular P waves separated by isoelectric lines and is usually less than 240/min.

The incidence of AFL is reported in previous studies as 88/100 000 person years. 58% of patients with flutter also have a documented episode of atrial fibrillation in the past (1). It’s 2.5 times more common in male as compared to females. Like that of atrial fibrillation the incidence of atrial flutter increases with age. The mean age of presentation is 67 years. Case fatality is 1%, mostly related to underlying disease (3).

Atrial flutter is a result of either structural or functional conduction abnormalities of the atria. The etiology and risk factors for atrial flutter are similar to that of atrial fibrillation. Identifying the underlying etiology is very important as the treatment of the primary cause is frequently warranted to prevent the recurrences of arrhythmia.

Typical atrial flutter is facilitated by the anatomical and functional properties of the right atrium (RA) and it is the most frequent atrial macroreentrant tachycardia constituting around 80% of cases. The circuit is bounded in front by the tricuspid ring and behind by a mixed functional and anatomical obstacle formed by the crista terminalis and inferior
venacava. Cavotricuspid, or subeustachian isthmus is the critical link of the circuit and is the target for radiofrequency ablation (10). Cycle length is usually around 240-200 ms, with great stability, but under the influence of pharmacological treatment or due to prior ablation, the cycle length (CL) can reach 300 ms.

There is a growing incidence of macroreentrent tachycardia due to structural lesions like atrial scars due to heart surgery with atriotomy or due to left atrial (LA) ablation for the treatment of atrial fibrillation. Non CTI dependent atrial tachycardia were grouped as atypical flutter which may have origin right or left atrium(13). Regardless of the atrial cycle length (CL), atypical AFL is a term commonly used to describe all other macroreentrant atrial tachycardias (MRATs).

AFL is symptomatic in the majority of patients and is associated with morbid events such as inappropriate heart rate during effort and marked reduction of exercise tolerance especially in patients with advanced structural heart disease, tachycardic cardiomyopathy, thromboembolism and sometime associated with atrial stunning after therapeutic termination. Radiofrequency ablation of the Cavotricuspid isthmus for the treatment of typical atrial flutter which started out as an experimental technique in early 1990’s by Feld and coworkers, has evolved to become the therapy of choice for the treatment of AFL. Atrial flutter ablation for atypical flutter has evolved with the advent of electroanatomical mapping. Atypical flutter ablation has a high procedural success rate and acceptable long term recurrence free survival.

Single centre radiofrequency experiences reported in the past showed a high initial success rate but there was relatively high recurrence rate of around 11–25%. With the application of the anatomical approach
of radiofrequency ablation and incorporating bidirectional isthmus block as a hard endpoint of success has transformed the technique of ablation of AFL into a safe and effective means of therapy and improved substantially the long-term results. The 1998 NASPE Prospective Catheter Ablation Registry showed that out of the total 477 patients underwent ablation of the tricuspid valve annulus-inferior vena caval isthmus for attempted cure of atrial flutter, acute success was achieved in 85.8% of patients. On follow-up, recurrent flutter was documented in 64 (14.7%) (37).

E Bertaglia at all published a study in Heart 2004 which showed Atrial flutter ablation proved successful in 367 of 383 patients (95.8%). The mean follow up duration was 20.5 months (range 6–55 months) which showed recurrence of typical AFL was documented in 41 of 367 patients (11.2%)(30).Sebastian Schmieder et al achieved acute success rate of 90% and during the follow-up period, 310 patients (90%) were free of AFL recurrences. In 33 patients (10%) had recurrences of typical AFL (33).

Published data of atrial flutter ablation in Indian population are scarce. Long term follow up studies addressing the issues of clinical outcomes and recurrence free survival in patients who underwent ablation flutter ablation and the predictors of recurrence are not there.

This study is designed to find out the Short term and Long term outcomes of atrial flutter ablation. This study also aims to find out the procedural complications, predictors of atrial flutter recurrence, the incidence of atrial fibrillation in post ablated patients and incidence of thromboembolic manifestations/stroke in this patient group.
AIM AND OBJECTIVES

• Aim of the study is to assess acute and long term outcomes of patients who underwent atrial flutter ablation.

• To determine the predictors of Atrial flutter recurrence.
REVIEW OF LITERATURE

Atrial flutter (AFL) or Macro reentrant atrial tachycardia is a supra ventricular tachycardia whose reentry circuit is entirely located either in the right atrium or in the left atrium (1). Macreoreentrant atrial tachycardia is typically described as “circular activation (reentrant) that revolves around a large obstacle, arbitrarily defined as being several centimeters in diameter” (2).

Epidemiology

Commission on Profession and Hospital Activity (CPHA) published the database of the nationwide patients admitted with arrhythmia as the principal diagnosis. AF accounted for 35% of admissions and atrial flutter being the second highest with 5%.

The incidence of AFL is reported in previous studies as 88/100 000 person years. 58% of patients with flutter also have a documented episode of atrial fibrillation in the past (1). It’s 2.5 times more common in male as compared to females. Like that of atrial fibrillation the incidence of atrial flutter increases with age and at 80 years of age 9% of males and 5% of females have atrial flutter (Figure 1). The mean age of presentation is 67 years. Case fatality is 1%, mostly related to underlying disease (3).
Etiology

Atrial flutter is a result of either structural or functional conduction abnormalities of the atria. The etiology and risk factors for atrial flutter are similar to that of atrial fibrillation. Identifying the underlying etiology is very important as the treatment of the primary cause is frequently warranted to prevent the recurrences of arrhythmia. Previous studies noted that around 30% have no underlying cardiac disease and 70% had underlying structural heart disease.

Structural abnormalities of atrium include atrial dilatation due to any etiology (valvular heart disease, hypertension, heart failure), prior scars from atrial surgery especially surgeries for congenital heart disease, prior atrial ablation sites and idiopathic scars within the atrium. Metabolic conditions and toxic conditions like thyrotoxicosis, pericarditis and alcoholism can also precipitate flutter. Anti arrhythmics like class Ic drugs (propafenone, flecainide), class Ia and amiodarone which are used in the

Figure 1. Incidence rates of atrial flutter by age and gender (100,000 person-years). Black squares = males; black circles = females.
Review of Literature

treatment of atrial fibrillation can convert atrial fibrillation into a more organized atrial flutter. Other causes include pulmonary embolism, pulmonary parenchymal disease, post operative period, anemia (4).

Hypertension is probably the most common cause of atrial flutter. Obstructive sleep apnea is seen in around 40% of patients but the proportion of flutter directly caused by obstructive sleep apnea is not certain.

Genetic etiology for atrial flutter was identified in Genome-wide association studies (GWAS) (5). The $PITX2$ (paired-like homeodomain 2) gene on chromosome locus 4q25 is said have an important role in left-right asymmetry of the heart and found to have a strong association with typical atrial flutter and atrial fibrillation.(6)

**Clinical Presentation**

AFL may present in paroxysms, lasting seconds to hours together or may exists as persistent rhythm. Symptomatic patients are those who have flutter which is paroxysmal and with rapid ventricular rate. The most common symptom being palpitations, other symptoms include dyspnea, presyncope, and weakness. In the absence of significant cardiac disease it is rare for AFL to cause syncope (7). AFL can also precipitant of CHF and, inappropriate heart rate during effort and which leads onto marked reduction of exercise tolerance in patients with structural heart disease. AFL is also an important cause of tachycardiomyopathy and also a cause for thromboembolism (8).

**Types of atrial flutter**

Working Group of Arrhythmias of the European Society of Cardiology and the North American Society of Pacing and
Electrophysiology in 2011 came out with a consensus document and developed terminology for AFL. Consensus describe “CTI-dependent, right atrial macro-reentry tachycardia in the counter clockwise direction as “typical” AFL, and clockwise direction as “reverse typical” AFL. Other right atrial isthmus-dependent flutters include lower loop reentry, double loop reentry and intra isthmus reentry (9). Non CTI dependent atrial tachycardia was grouped as atypical flutter which may have origin right or left atrium. Regardless of the atrial cycle length (CL), Atypical AFL is a term commonly used to describe all other macroreentrant atrial tachycardias (MRATs) (Table 1).

**Classification of Atrial Flutter**

<table>
<thead>
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<tr>
<td>Right atrial CTI-dependent flutter</td>
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<tr>
<td>Counterclockwise (CCW) flutter</td>
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<td>Clockwise (CW) flutter</td>
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<tr>
<td>Double-wave reentry</td>
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<tr>
<td>Lower loop reentry</td>
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<tr>
<td>Intra-isthmus reentry</td>
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<td>Right atrial non-CTI-dependent flutter</td>
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<tr>
<td>Scar-related flutter</td>
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<td>Upper loop flutter</td>
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<td>Left atrial flutter</td>
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<td>Mitral annular flutter</td>
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<tr>
<td>Scar- and pulmonary vein-related flutter</td>
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<tr>
<td>Coronary sinus flutter</td>
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<tr>
<td>Left septal flutter</td>
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*CTI = Cavo-tricuspid isthmus.*

Counterclockwise vs. clockwise direction of wavefront rotation, when visualized from the left anterior oblique fluoroscopic view.

Table 1 Classification of atrial flutter
Right Atrial Cavotricuspid-Isthmus-Dependent Flutter

Counterclockwise Atrial Flutter

Counterclockwise (CCW) Atrial flutter is the most common type of flutter. It accounts for up to 90% of clinical cases. Even in patients with right atriotomy for repair of congenital heart defects, CCW is the commonest form of atrial flutter. The characteristic ECG findings include negative saw tooth waves in the II, III, aVF with positive waves in V1 and transition to negative in V6. Viewing from the LAO view in fluroscopy the activation wave front proceeds superiorly over the right atrial posterior and septal walls and inferiorly over the right atrial anterior and lateral walls rotating in a counter clockwise fashion, anteriorly it is bounded by the tricuspid orifice and posteriorly by the anatomic barriers of the vena cavae(10). The superior margin is not very well defined but may include the right atrial roof. The inferior margin is marked anteriorly by the tricuspid orifice and posteriorly by the IVC orifice and its continuation in the eustachian ridge (Figure 2). Cavotricuspid, or subeustachian isthmus is the critical link of the circuit. CTI is the target for radiofrequency ablation.

Clockwise (CW) Atrial Flutter

The reversed version of CCW CTI dependent AFL is the pathway for CW flutter (Figure 2). It accounts for up to 10% of cases and has ECG findings that include positive deflections in the inferior leads and negative in V1. The circuit has the same boundaries as that of counterclockwise flutter, and complete interruption of the CTI is the treatment of choice.
Double-Wave Reentry

In 1998 Cheng et al reported that Double Wave Reentry was responsible for pacing-induced AFL acceleration. He postulated single extra stimulus critically precisely timed and delivered to the isthmus results in unidirectional antidromic block of the paced impulse and acceleration of CCW AFL. Surface ECG flutter wave morphology showed that tachycardia acceleration was due to two successive activation wave fronts traveling simultaneously in the same direction in the reenstrant circuit. They were all ill sustained. It has been speculated that Double Wave reentry may be considered as a trigger for AF (11).

Lower Loop Reentry

Lower loop reentry circuit that localizes to the lower right atrium. The tachycardia circuit rotates around the IVC, either CCW or CW, or around both the IVC and the tricuspid annulus resulting in a figure-of-eight (Figure 3). Surface ECG findings are similar to those of CCW or CW CTI-dependent AFL (12).
Intra-Isthmus Reentry

Yang et al described an Atrial Flutter circuit which is entirely confined within the CTI itself. The circuit does not involve lateral CTI and its bounded by the medial CTI and the coronary sinus ostium. RF ablation of the medial isthmus alone leads to a cure of this tachycardia.

Right Atrial Non-Cavotricuspid-Isthmus-Dependent Flutter

Scar-Related Atrial Flutter

Macro-reentrant AFL can also occur around an anatomic obstacle which is remote from the CTI. Right atrial scars due to prior cardiac surgery especially for congenital heart defects can serve as anatomic substrate for macro-reentry (13). Scars especially located in the posterolateral and inferolateral regions of right atrium have been found to
have increased incidence of atrial flutter. A linear RF lesion drawn from the region of the scar to the IVC can eliminate the tachycardia. Tachycardia can also be eliminated by focal ablation of the critical channels (14).

**Upper Loop Reentry**

While scar-related AFL circuits provide an anatomic obstacle other CTI, upper loop AFL circuit is because of functional obstacles away from CTI. Tai et al have shown that a tachycardia circuit localizes to the right atrial upper portion with the crista terminalis (CT) and its slowed conduction through it acts as the functional obstacle (15). The direction of rotation can be Counterclockwise (descending activation sequence in the free wall anterior to the CT) or CW (ascending activation sequence in the free wall anterior to the CT). The CT conduction gap is critical for maintenance of tachycardia and ablation at this site can eliminate the tachycardia.

**Left Atrial Flutter**

**Overview**

Left AFLs are much less common when compared with the right atrial CTI AFL. They are commonly seen in patients with left side structural heart disease, rarely few cases have been reported in structurally normal heart. Electro anatomical mapping have shown areas of slowed conduction or block and electrically silent regions which can serve as substrates for macro-reentry. The circuits are complex with more than one loop. Surface ECG findings are variable, commonly flutter wave are of low amplitude. They often coexist with AF.
Mitral Annular Atrial Flutter

In Mitral Annular Atrial Flutter wave fronts rotates around the mitral annulus, either counter clockwise or clockwise. The critical isthmus is bounded anteriorly by the mitral annulus and posteriorly by the low-voltage areas or scars in the posterior wall of the left atrium. The ECG patterns in left atrial CCW circuits are notable for low amplitude flutter waves in the inferior leads and positive waves in V1 and V2. In one case, the ECG pattern mimicked its right atrial CCW CTI-dependent circuit counterpart. A linear RF ablation extending from the mitral annulus to another obstacle anatomic obstacle like left superior pulmonary vein, right superior pulmonary vein, posterior scar, or left atrial roof can result in a cure (16).

Scar- and Pulmonary Vein-Related Atrial Flutter

Atrial flutter can also have a re entry circuit which rotates around one or more pulmonary veins or a scar in the posterior wall of the left atrium. They can have multiple loops. The circuits around the pulmonary veins can be cured by creating ablation lines from a pulmonary vein to the mitral annulus or to the other pulmonary vein (16).

Coronary Sinus Atrial Flutter

Olgin et al reported a patient with no structural heart disease who had an AFL circuit which included the ostium of the coronary sinus in it. The circuit traveled from the coronary sinus, to the lateral left atrium, down the interatrial septum, and back to the coronary sinus. Double potentials were noted in the coronary sinus with circumferential RF ablation within the coronary sinus terminated the arrhythmia (17).
Left Septal Atrial Flutter

AFL circuits rotating around the left septum primum have been reported in the past. The critical isthmus is located between the septum primum and the mitral annular ring or between the septum primum and the pulmonary veins (18). Marrouche et al reported that these AFL can be eliminated by delivering RF lesion from the septum primum to the right inferior pulmonary vein or and from the septum primum to the mitral annulus. There were no recurrences during 13 month follow up.

Principles of Management

Acute therapy

Acute therapy for patients presenting with AFL depends on the clinical presentation. Options include cardioversion and slowing the ventricular rate by using AVN blockers. When the patient is hemodynamically unstable then cardioversion is the treatment of choice. Cardioversion can be electrical or chemical. Electrical cardioversion with relatively low energies (less than 50 J) is almost always successful in terminating AFL. Chemical cardioversion can be attempted with intravenous anti arrhythmic drugs but the conversion rate is lower. Ibutilide is successful in 38% to 76% of cases, and is found to be more effective than IV amiodarone, sotalol, and other class IC agents. Overdrive atrial pacing can be attempted by placing a catheter in the esophagus or the RA but it also degenerate into AF (14). Achieving ventricular rate control with either oral or intravenous AVN blockers such as diltiazem, beta blockers, verapamil, and digoxin is an option but it is more difficult to achieve in AFL when compared with AF because of the slower and more regular atrial rates(4).
Chronic therapy

AFL which occur secondary to an acute disease process usually restores to sinus rhythm on treatment of underlying disease process. The long term success rate of antiarrhythmic drugs to maintain sinus rhythm appears to be limited, and complete prevention of AFL recurrence is difficult to achieve. Catheter ablation is highly successful with high procedural success and with low-risk. Hence catheter ablation of the CTI is the treatment of choice for typical AFL. Antiarrhythmic drugs which can be used in suppression of AFL, including class IA (quinidine, procainamide, and disopyramide), class IC (flecainide and propafenone), and class III (sotalol, amiodarone, dofetilide, and dronedarone) agents. Class IC agents are the drugs of choice in patients with no structural heart disease (19). AVN blockers is to be initiated in conjunction with antiarrhythmic agents in order to avoid the risk of rapid ventricular rates secondary to the vagolytic effects of class I drugs and slowing of the flutter rate. AV node ablation and pacemaker implantation may be considered as an option for patients in whom curative ablation of the AFL, antiarrhythmic therapy, and rate control strategies have failed. Prevention of stroke is recommended either with aspirin or an oral anticoagulant depending on the calculated patients risk for stroke like that of atrial fibrillation.
Electrophysiological Testing

Typical atrial flutter:

A typical EP study in AFL is done with a decapolar catheter positioned into the CS with the proximal electrodes bracketing the CS os and a multipolar Halo catheter is positioned around the tricuspid annulus are used to map AFL. The distal tip of the Halo catheter is positioned at 6 to 7 o’clock in the LAO view, so that RA septum is recorded by the proximal electrodes, anterolateral RA by the middle electrodes and the distal tip records the middle and lateral aspects of CTI(Figure 5).
Induction of tachycardia

Programmed electrical stimulation protocol by atrial burst pacing and AESs either single or double extra at various CLs (600 to 200 milliseconds) from the high RA and CS can induce atrial flutter. Isoproterenol infusion may be needed for tachycardia induction. In most patients with a clinical history of AFL, programmed electrical stimulation can easily induce tachycardia. Induction of flutter in patients with documented AFL is as high as 95% (20). The chance for tachycardia induction is more likely with rapid atrial pacing than with single AES, but as likely as introducing two AES. Clockwise AFL is more likely to be induced with low lateral RA pacing and Counterclockwise AFL by pacing from the CS os. The clinical significance of accidental occurrence of AF during AFL induction is uncertain.

Tachycardia Features

Typical AFL has a constant cycle length, polarity, morphology, and amplitude of the recorded bipolar electrograms and characteristic single constant macroreentrant circuit with a same atrial activation
sequence. It has a stable cycle length with cycle-to-cycle variation of < 2%. The atrial CL is mostly between 190 and 250 milliseconds, in patients who are on antiarrhythmic agents or had a previous unsuccessful ablation of the CTI can have a slower cycle length. Ventricular conduction is usually 2:1, but variable AV conduction and larger multiples are also seen. Variable AV block is because of block at multilevel. If there is associated anterogradely conducting bypass tracts with a short ERP can result in preexcited AFL with rapid AV conduction. When the atrial rate is low, infusion of isoproterenol can facilitate 1:1 AV conduction (21). Adenosine causes transient AV block, but it can also reduce atrial refractoriness and hence causes AFL to degenerate into AF.

Diagnostic Maneuvers during Tachycardia

- **Atrial extra stimulation during Atrial Flutter:** Atrial Extra stimuli results in resetting of the AFL circuit. Closer the site of atrial stimulation is, the easier the resetting of the AFL circuit at longer coupling intervals will be. If there is atrial capture without resetting the AFL circuit indicates that the pacing point is outside the AFL circuit.

- **Atrial pacing during atrial flutter:**
  - Entrainment: Overdrive atrial pacing can almost always entrain typical AFL. Achievement of entrainment establishes a reentrant mechanism of the tachycardia and excludes abnormal automaticity and triggered activity as potential mechanisms. Entrainment with manifest fusion shows that pacing site is outside the CTI, such as lateral RA and CS, whereas entrainment with concealed fusion occurs when pacing site is in CTI (22).
Termination: Rapid atrial burst pacing results in termination of AFL in most of the patients.

Overdrive suppression: Overdrive suppression analogous to that seen with automatic AT is not expected in AFL.

Transformation: Rapid atrial burst pacing may convert AFL into AF. This is less likely with a slower pacing CL or pacing from sites within the AFL circuit.

Mapping of the circuit

There are four main techniques used for mapping the flutter circuit

1. Activation mapping
2. Entrainment mapping
3. Electro anatomic mapping
4. Non contact mapping

Activation mapping

The activation sequence in right atrium during counterclockwise AFL occurs in a sequence CS OS, up the atrial septum, over the roof of LA, down the lateral RA wall and adjacent to the crista terminalis, across the CTI (with some delay because of slow conduction across the CTI) in a proximal to- distal direction along the Halo electrodes (Figure 6) . The activation sequence in clockwise AFL is opposite to that of CCW flutter, from distal to Proximal in Halo catheter. The atrial activation sequence in patients with sinus rhythm or focal Atrial Tachycardia originating from the upper RA or LA is from the middle or proximal Halo down both the
RA septum and lateral wall in a cranio caudal direction, toward the distal and proximal-most Halo electrodes.

**Figure 6** Atrial activation pattern in a patient with counterclockwise atrial flutter arrows indicates the atrial activation in Halo catheter

**Entrainment Mapping**

Entrainment Mapping shows three important facts about atrial flutter circuit

- Sites of the RA or LA that are part of the reentrant circuit
- Sites of atrium which are outside the circuit
- Critical isthmus of the AFL circuit

Entrainment also tells about how far the reentrant circuit is from the pacing site. Pacing is performed from the CTI, high RA, mid-lateral RA, and proximal and distal CS, to distinguish between RA and LA
flutters. Entrainment with concealed fusion indicates that the pacing site is in a protected isthmus of the reentrant circuit. The diagnosis of CTI dependent AFL is established when pacing from the CTI results in entrainment with concealed fusion and a PPI that is equal (within 20 milliseconds) to the flutter CL.

1. Pacing from sites outside the AFL:
   
   Manifest atrial fusion on the surface ECG
   
   PPI – tachycardia CL > 20 msec

2. Pacing from sites inside the AFL circuit
   
   Manifest atrial fusion on surface ECG or intracardiac recordings
   
   PPI – tachycardia CL < 20 msec

3. Pacing from a protected isthmus inside the circuit (cavotricuspid isthmus)
   
   Concealed atrial fusion
   
   PPI – tachycardia CL < 20 msec

**Electroanatomical Mapping**

3D mapping is help to find out the mechanism of tachycardia, to distinguish between a focal origin and macroreentrant tachycardia by providing sequence of atrial activation during the tachycardia and by visualization of the activation wavefront. Anatomical and EP landmarks are tagged. The catheter slowly advanced around the chamber walls to various points along the endocardium, and acquiring the location of its tip together with the local electrogram. Then activation mapping is
performed to find out the atrial activation sequence and mapping of endocardial sites around the tricuspid annulus and CTI. The local activation time at each site bipolar electrogram and is measured and compared to the electrogram obtained from the reference catheter. Activation maps display the local activation time by a color-coded overlay on the reconstructed 3-D geometry. The activation map demonstrates a continuous progression of colors around the tricuspid annulus with close proximity of earliest and latest local activation and an activation time in a similar range to tachycardia CL, consistent with macroreentry. The 3-D electroanatomical maps can also provide information about the voltage characteristics of the tissues involved in the CTI. Thus, 3-D mapping also helps in choosing the easiest path in the CTI to ablate.

**Noncontact Mapping**

The EnSite 3000 noncontact mapping system uses a balloon which is positioned in the center of the atrium and does not come in contact with the atrial walls being mapped. Detailed geometry of the chamber is then reconstructed by moving the mapping catheter around the atrium. Although typical AFL is usually readily treated using standard ablation techniques, noncontact mapping can be used to confirm the anatomical location of the flutter circuit, reduce fluoroscopy time, and confirm CTI block after ablation.

**Ablation**

**Target site for Ablation**

The CTI is the ideal target of AFL ablation because it is accessible, short, safe to ablate, and essential for the AFL circuit (24). The central
portion of the isthmus the 6 o’clock region appears to be the optimal target site because it is the thinner part of the isthmus and therefore it is easier RF ablation and less chance of recurrence. Other advantages include the increased distance from the paraseptal isthmus, and from the right coronary artery. Alternatively, the tricuspid annulus–CS or IVC-CS isthmuses may be targeted but those approaches were less successful in treating AFL.

Figure 7: 3D reconstruction and fluoroscopic image of the right atrium in LAO view and the various anatomical approach to AFL ablation. Red dots indicate Cavo Tricuspid Isthmus ablation points, Blue dots line from CS to TA, Yellow line between CS os to IVC

**Catheter positioning**

A steerable irrigated ablation catheter 4 or 8 mm is generally used. The ablation catheter is advanced into the RV under fluoroscopy and the tip is deflected to achieve good contact with the RV inferior wall and is withdrawn progressively until the electrogram shows small atrial and large ventricular electrograms with the ablation tip pointing toward 6 o’clock in a 45-degree LAO view. Position of the ablation catheter can be
inferred from the ratio of the atrial and ventricular electrogram amplitude(25).

**Radiofrequency ablation**

Radiofrequency energy is applied starting from near the tricuspid annulus and the catheter is gradually pulled back toward the IVC during continuous energy application (50 to 70 W, 60 to 120 seconds, targeting a temperature of 55°C to 60°C), RF energy (50 to 70 W, 30 to 60 seconds, targeting a temperature of 55°C to 60°C). RF lesion is initiated from the tricuspid annulus edge where there is large ventricular and small atrial electrograms, the last lesion is completed at the IVC edge. After ablation, the electrogram loses voltage and may become fragmented; the catheter is then withdrawn (2 to 4 mm at a time) toward the IVC and the next RF application is delivered. Ablations are repeated until the lack of atrial electrograms indicates that the catheter has reached the IVC. AFL can terminate or its CL can increase transiently or permanently during delivery of RF energy.

**Role of three-dimensional electroanatomical Mapping navigation systems**

Electroanatomical mapping systems provides precise spatial localization and tracking of the ablation catheter along the CTI that potentially help shorten fluoroscopy time. The main advantage is the visibility of an ablation line as the procedure is carried out so that no area is left out or repeatedly ablated. Therefore, it facilitates the creation of ablation lines devoid of gaps across the entire isthmus (26). The benefits of electroanatomical mapping are much more in patients with atypical flutter/Non CTI dependent atrial flutter.
Cryoablation

CTI block can also be achieved by cryothermal ablation. Cryothermal ablation has the advantage of being less painful. Acute success rates are comparable to those for RF ablation (27). A randomized control trial comparing cryothermal and RF for ablation of typical AFL has shown that lesion durability from cryoablation was significantly inferior to that of RF ablation. Persistence of bidirectional CTI block in patients treated with cryoablation studied 3 months after ablation was inferior to that in patients treated with RF ablation, as evidenced by the higher recurrence rate of symptomatic, ECG-documented AFL and higher asymptomatic conduction recurrence rates (27).

Endpoints of Ablation

Ablation can be performed during AFL or CS pacing (28). If ablation is done while patient is in AFL, the first endpoint is to terminate of the tachycardia during RF energy delivery. On termination of the tachycardia, programmed electrical stimulation and burst atrial pacing should be performed immediately to look for re inducible of tachycardia. If there is continuation of tachycardia or is re inducible then ablation should be repeated. If AFL is terminated and is not re inducible, maneuvers are to be performed for determine of bidirectional block in the CTI. Termination of AFL during RF delivery by itself should not be considered as an end point because it is often not associated with complete bidirectional CTI block. On achievement of complete bidirectional CTI block, reconfirmation should be repeated 30 minutes after the last RF application. If ablation is to be performed when the patient is in sinus rhythm, then it is carried out by pacing from CS to help monitor the activation sequence in the lateral RA wall. During ablation of
the CTI, gradual delay in activation of the low lateral RA can be observed prior to achieving complete Bidirectional CTI block (29).

**Confirmation of bidirectional cavotricuspid Isthmus block**

As the confirmation of Bidirectional block is one of the important parameters of AFL outcomes various parameters are taken as an indirect evidence of CTI block.

1. **Atrial activation sequence during atrial pacing**

   Bidirectional CTI block is inferred by pacing from the CS os and RA lateral wall and looking at the atrical activation sequence. Normally, during atrial pacing from the CS os, one wave front propagates from the CS pacing site in a clockwise direction across the CTI to activate the low lateral RA and the other wave front ascends upwards along the atrial septum to the high RA in a counterclockwise direction, with ultimately resulting collision of wavefronts at the upper part of the lateral RA and generating an atrial activation sequence with a chevron pattern. Clockwise CTI block is indicated by the looking at the purely descending wave front at the lateral wall to the CTI (proximal-to-distal activation sequence on the Halo) when paced from the CS os. This block is associated with marked prolongation of the CTI conduction duration. Incomplete clockwise CTI block is said to occur when the CTI still allows signals to reach lateral RA when paced from the CS os in a clockwise direction but at a slower conduction velocity, hence resulting in displacement of collision of the wave fronts to the lower part of lateral RA.
2. **Transisthmus conduction interval.**

   The transisthmus conduction interval between the stimulus artifact from one side of the isthmus to the atrial electrogram recorded on the contralateral side, while pacing from CS os or low lateral RA pacing. Prolongation of transisthmic conduction time when compared with the baseline value more than 50% suggests Bidirectional CTI block. Absolute value > 150ms can also be taken as a surrogate marker. This criterion has sensitivity and negative predictive values of 100% and the specificity and positive predictive values are less than 90%.

3. **Double potentials.**

   Double potential recorded at the site of ablation confers CTI block. Double potentials, separated by an isoelectric interval of 30 milliseconds or longer qualify for a conduction block. It is considered the gold standard for determining complete bidirectional block. When there is a gap in a line of block, the isoelectric period which lies between double potentials becomes shorter the electrograms are to the gap. At the gap, in the line of block, double potentials are no longer present, and the electrogram is typically long and fractionated but can also be discrete. Interval between the double potentials is > 110 milliseconds, indicates CTI block and values less than 90 milliseconds, indicates absence of complete bidirectional block (Figure 7).

4. **Unipolar electrogram configuration.**

   In a patient with CTI conduction while pacing from the proximal CS pacing, the unfiltered unipolar signals recorded demonstrate an RS configuration as the paced impulse propagates clockwise across the isthmus. When there is complete clockwise CTI block, depolarization of
the electrode just medial to the line of block retains its original polarity, but its morphology monophasic R wave because the recording site since it’s a dead end for impulse conduction.

5. Differential pacing

Double potentials with an isoelectric interval are recorded over the ablation line during pacing just lateral to the line. The stimulus to first electrogram component represents the time to activation of the ipsilateral side of the ablation line and the second component represents the contralateral side of ablation line activation. The pacing site is changed to about 15 mm further away from the ablation line and paced. If there is persistent CTI conduction, both components of the split electrogram will be delayed. If there is CTI block, the time to the first electrogram component is delayed but the terminal component is advanced or unchanged in timing. It has sensitivity is 100%, specificity 75%, negative-predictive value 94%, and positive-predictive value 100% for detection of CTI block.

6. Electroanatomical mapping.

Activation mapping can be used to confirm CTI block. When clockwise block in the CTI is achieved, pacing from the proximal CS results in an activation wave front propagating in a counterclockwise fashion, with the latest activation in the CTI immediately lateral to the ablation line. When CTI conduction is still present, CS pacing produces an activation wave front to propagate rapidly through the CTI, with the anterolateral RA wall being activated at last.
Review of Literature

**FIGURE 12.6** The use of coronary sinus (CS) pacing to verify the presence of clockwise counterclockwise (CTI) block. **Upper panels:** Intracardiac recordings from the right atrium (RA), CS, and CTI. **Lower panels:** Left anterior oblique fluoroscopic view illustrating the position of the ablation catheter at the CTI and the Halo catheter around the tricuspid annulus (TA), with the distal end at the lateral end of the CTI. When CTI conduction is intact (left panel), pacing from the coronary sinus results in the collision of activation wavefronts in the lateral RA wall (red and blue arrows). **Middle panel:** The collision point moves toward the low lateral RA wall when incomplete block is present. **Right panel:** Complete clockwise CTI block is indicated by the observation of a purely descending wavefront at the lateral wall of the RA to the CTI (proximal-to-distal Halo sequence). The bipolar electrogram recording from the CTI initially shows a single atrial potential (left panel, gold arrow). With partial CTI ablation, the atrial electrogram splits into two closely adjacent potentials (middle panel, gold arrows). Complete CTI block is indicated by the observation of double potentials separated by an isoelectric interval (right panel, gold arrows). Additionally, bipolar electrogram polarity reversal (left panel) is observed in the distal Halo and the distal ablation electrode recordings when complete block is achieved (as compared with before complete CTI block is achieved in the right and middle panels), thus indicating reversal of the direction of the activation wavefront lateral to the line of block (green and gold arrows). Positive (R wave) morphology of the bipolar recording lateral to the line of block also indicates complete CTI block. In contrast to bipolar SR electrogram morphology, when intact conduction or only incomplete block is present, ABL = ablation site; CS = coronary sinus; CTI = counterclockwise; CS = coronary sinus; TA = tricuspid annulus; TA = tricuspid annulus; PR = proximal.
Review of Literature

Table 2 Showing various parameters for achievement bidirectional block and their sensitivity and specificity

Outcomes of Atrial flutter ablation

Number of studies has been done in the past in relation to the outcomes of atrial flutter ablation both retrospectively and prospectively. Outcomes include acute procedural success, complication rates, and long term maintenance of sinus rhythm without recurrence.
Acute Procedural success

Acute procedural success has been taken in most of the studies as Termination of tachycardia with non inducibility of tachycardia with pacing maneuvers and in CTI dependent AFL documentation of bidirectional conduction block in CTI.

E Bertaglia et al published a prospective multicentre study, 383 patients (75.4% men) who underwent transisthmic ablation for typical AFL were analyzed. In 239 patients (62.4%) AF was present before ablation. Ablation proved successful in 367 patients (95.8%)(30). The 1998 NASPE Prospective Catheter Ablation Registry showed that out of the 477 patients, acute success was achieved in 85.8% of patients.

Hugh Calkins et al out of the 150 ablation fornpatients with typical atrial flutter, acute procedural success was 88%. In the largest meta-analysis so far, comprising 10,719 patients, the acute procedural success rate for ablation with irrigated or large-tip RF catheters was 94%. Large degree to these improved results could be attributed to the use of cooled irrigation but also the use of bidirectional conduction block as a procedural end point.

In patients with structural heart disease the results of ablation were not as good as CTI dependent flutter ablation. Short-term success rates are reasonably good (approximately 90%). Atrial septal defect repair is the most common cause of incisional reentry in adults. Reentrant circuits usually rotate around the atriotomy scar in the lower right atrium and can be disrupted by creating a lesion from the scar to the IVC or SVC. In 134 patients with incisional-related flutter who have undergone ablation, 50-88% have had no tachycardia recurrences at 2 years of follow-up(31).
Long term follow up

Documented atrial fibrillation either by holter or ECG post ablation is considered as the definition for Recurrence. The recurrence rates of AFL after ablation are greatly reduced by the use of irrigated or large-tip ablation catheters (6.7%) compared with standard RF ablation (14%). Most of the recurrences occur within 6 months of ablation. The recurrence rates of AFL are higher for cryoablation than for RF ablation. During invasive follow-up 3 months after ablation, 15% of patients undergoing RF CTI ablation had documented recovery of CTI conduction, compared with 34% of patients undergoing cryoablation. In this study, no patient undergoing RF ablation had clinical AFL recurrence, compared with 11% undergoing cryoablation. By meta-analysis, the recurrence rates of AFL were not statistically different for cooled ablation (6.7%) versus cryoablation (11%). Despite the excellent acute results and long-term outcome after RF catheter ablation for freedom from type 1 AFL, the development of atrial fibrillation or atypical AFL occurs at a high rate in this population of patients (up to 67% over 5 years), especially if there is a history of atrial fibrillation or underlying heart disease. Stringent endpoint of bidirectional block significantly reduces the AFL recurrence rate. For patients in whom typical AFL recurs after ablation, conduction through the CTI is usually responsible. Presumably, such recurrences reflect a failure to achieve bidirectional CTI block during the initial procedure, incorrect initial assessment of bidirectional block, or resumption of conduction across an initially blocked isthmus. The incidence of AFL recurrence does not increase beyond 1 to 6 months of follow-up, a finding suggesting that if recovery of isthmus conduction is going to occur, it will have done so by 6 months.
Hugh Calkins et al noted that during follow up for 6 month recurrent typical atrial flutter was observed in 13% of patients (32). E Bertaglia at al reported during the follow up of 20.5 (12.4) months (range 6–55 months), recurrence of typical AFL was documented in 41 of 367 patients (11.2%) (30). Sebastian Schmieder et al reported in his study the long term outcomes where in 343 patients (95%) during follow up for a mean of 496±335 days and 310 patients (90%) remained free of AFL recurrences(33). NAPSE registry stated that during their follow up, recurrent flutter was documented in 64 (14.7%) patients.

<table>
<thead>
<tr>
<th>Catheter Type</th>
<th>No. of Studies/No. of Patients</th>
<th>Acute Success (% [95%] CI)</th>
<th>AFL Recurrence (No. of Studies/No. of Patients)</th>
<th>AFL Recurrence (% [95%])</th>
<th>Atrial Fibrillation Incidence Postablation (% [95%])</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-6 mm RF</td>
<td>55/2449</td>
<td>80 (84, 91)</td>
<td>56/2516</td>
<td>14 (11, 17)</td>
<td>21 (13, 33)</td>
</tr>
<tr>
<td>8-10 mm or irrigated RF</td>
<td>54/3098</td>
<td>94 (90, 95)</td>
<td>49/3052</td>
<td>7 (5, 8)</td>
<td>25 (19, 31)</td>
</tr>
<tr>
<td>Cryoablation</td>
<td>11/489</td>
<td>89 (79, 94)</td>
<td>10/442</td>
<td>11 (8, 16)</td>
<td>21 (14, 34)</td>
</tr>
</tbody>
</table>

*Only studies using cryo/irrigated ablation as the end point were included.
†Follow-up duration of 14±6.3 months.
‡Data from: Perez FJ, Schobert CM, Pareve B, et al. Long-term outcomes after catheter ablation of cavotricuspid isthmus dependent atrial flutter: a meta-analysis. Circ Arrhythmia Electrophysiol. 2009;2:99–101. Follow-up duration 15±0.4 months and number of studies/patients are 11/233, 24/1936 and 6/325 for 4-6 mm RF, 8-10 mm or irrigated RF and cryoablation, respectively.
AFL, atrial flutter; CI, confidence interval; RF, radiofrequency.

Atrial fibrillation on follow up

It was noted that there is an increased incidence of atrial fibrillation post RF ablation AFL, even in patients without prior documented history of AF. AF can develop in approximately 20% to 30% with short-term follow-up, approximately 1 year of patients with or without a prior history of AF (34). It is postulated that AFL is often an early marker of atrial electrical disease that frequently progresses to AF even after curative treatment for AFL. This thought has led some investigators to ablate of AF at the time of AFL ablation (35). In a meta-analysis, during an average follow-up of around 16 months, 23.1% of patients with no pre ablation history of AF and in 52.7% of patients with
a history of AF, had AF despite successful AFL ablation. The occurrence of AF in the post procedural period was not influenced by the ablation technology or procedural endpoints. This finding has greater influence on patient selection, long-term arrhythmia-free success rates, continuation of anti arrhythmic drug use, and anticoagulation. AF prior to ablation of AFL is very common, thus reducing the ability to stop antiarrhythmic medications or anticoagulation in many patients, the potential benefits of ablation of only AFL should be seriously scrutinized in those patients (36).
MATERIALS AND METHODS

Setting: Cardiology OPD and Medical records, SreeChitra Institute for medical sciences and technology. All patients who underwent atrial flutter ablation during the study period and who gave informed written consent to be a part of the study were included.

Ethical committee approval was obtained prior to the initiation of the study

Study Design: Single centre Observational Retrospective - Prospective study.

Study period: Jan 1, 2005 to June 31, 2016.

Retrospective study of the patients who underwent atrial flutter ablation from Jan 2005 till June 2014 were analyzed and were followed up prospectively till the June 31 2016. Patient who underwent Atrial flutter ablation from June 2014 till June 31 2016 were also included and followed up prospectively in the study

Inclusion criteria:

All consecutive patients who have underwent atrial flutter ablation in SCTIMST from Jan 1, 2005 till June 31, 2016 and gave informed written consent to be a part of the study were included.

Exclusion criteria:

- Patients without adequate procedural data or in whom there were inadequate follow up data (at least 3 Months of follow up) were excluded from the study.
Endpoints of the study

Primary endpoint:

- Acute procedural success: defined as “Termination of tachycardia, non inducibility of tachycardia 30 mins post ablation and without recurrence within 48 hours post procedure”

- Recurrence on follow up: defined as “any documented atrial flutter either by surface ECG or in holter recorded 48 hrs after the procedure”. Just the presence of palpitations and no documented AFL was not considered as recurrence.

Secondary Endpoints:

- Procedural complications

- Occurrence of AF on follow up

- Stroke

Patient’s clinical data and procedural data were collected from hospital records and analyzed. Patients were interviewed by principal investigator in review outpatient department during their routine hospital visits after their written consent. No patient was asked to come to hospital for follow up when there is no cardiac indications.

For a few patients for whom logistical problems made clinic attendance impossible, after obtaining oral consent from the patient, their clinical status, complaints and follow-up data were obtained from the treating local cardiologist/physician or by telephonic questionnaires.
Since all the patient attending hospital are for cardiological indications and not for collection of data alone, no reimbursement for travel or daily wages lost were given to patients participating in the study.

The data was analyzed by the principal investigator. Data was expressed as mean of continuous variables with standard deviations. All data was handled with care to maintain patient confidentiality. Records were maintained in both computer and paper formats. The closing point for any 1 patient was the time of their last visit to the follow-up clinic during study period. Data was interpreted as the percentage of patients who had acute procedural success, procedural complications, percentage of recurrence on follow up and predictors of atrial flutter recurrence. Descriptive summaries were presented as frequencies and percentages for categorical data, and as means and standard deviations for continuous variables. Continuous variables were compared using Student’s t test or Mann-Whitney U test as appropriate, Group comparisons were made using χ² tests. All statistical analyses were performed using the SPSS statistical software package (release 23.0, SPSS Inc.; Chicago,Ill).
RESULTS AND OBSERVATIONS

Baseline Characteristics:

548 patients were admitted with the diagnosis of atrial flutter in the department of cardiology in our hospital. 107 (20%) patients underwent atrial flutter ablation during our study period. Out of them, 1 patient had incomplete procedural data and 2 of them had follow up data less than 3 months and hence they were excluded from the analysis. Study group consisted predominantly of males constituting 63 (61%) and females 41 (39%), with the male female ratio of 1.5:1 (Figure 9). Mean age was 50 years (range 9 – 81).

Figure 9 Showing the sex distribution of the study group

Only 30/104 (28.9%) had a structural heart disease, rest of the patients had structural abnormalities 74/104 (71.1%). Post surgical closure of ASD being the most common structural abnormality 37/104 (36%). 10 (10.4%) had coronary artery disease, 4 had rheumatic heart disease and 16 patients had reduced LV function (ejection fraction < 50 %) (Figure 10). Reduced ejection includes patients with underlying LV
dysfunction which could have probably precipitated Atrial flutter and also those patients who had developed LV dysfunction because of sustained tachycardia (tachycardiomyopathy). 10(9.6%) had documented history of atrial fibrillation prior to ablation (Table 3).

**Figure 10** Showing the distribution of structural heart disease
### Results and Observations

**Table 3** Showing baseline characteristics

<table>
<thead>
<tr>
<th>Clinical Characters</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no of patients</td>
<td>104</td>
</tr>
<tr>
<td>Mean age yrs (range)</td>
<td>50(9-82)</td>
</tr>
<tr>
<td>Females</td>
<td>41(39)</td>
</tr>
<tr>
<td>Male: Female</td>
<td>1.5 : 1</td>
</tr>
<tr>
<td>Structurally Normal heart</td>
<td>30(28.9)</td>
</tr>
<tr>
<td>Structural abnormality</td>
<td>74(71.1)</td>
</tr>
<tr>
<td>ASD Surgical closure</td>
<td>37</td>
</tr>
<tr>
<td>Other CHD Post OP</td>
<td>11</td>
</tr>
<tr>
<td>CAD</td>
<td>10</td>
</tr>
<tr>
<td>DCM</td>
<td>2</td>
</tr>
<tr>
<td>RHD</td>
<td>4</td>
</tr>
<tr>
<td>HCM</td>
<td>2</td>
</tr>
<tr>
<td>EMF</td>
<td>4</td>
</tr>
<tr>
<td>ASD</td>
<td>2</td>
</tr>
<tr>
<td>Others</td>
<td>2</td>
</tr>
<tr>
<td>Reduced LV function (&lt; 50%)</td>
<td>16(15)</td>
</tr>
<tr>
<td>Atrial fibrillation prior to ablation</td>
<td>10(9.6)</td>
</tr>
</tbody>
</table>
**Procedural/Electrophysiological characteristics**

Atrial flutter circuit is entirely located either in the left atrium or Right atrium. 101/104 had their circuit in right atrium and 2 in their left atrium, 1 patient had circuits located both in left and right atrium (Figure 11). CTI dependent atrial flutter was noted in 78% of patients (81/104) (Figure 12). Out of the patients with CTI dependent AFL, 55 had Counterclockwise circuit, 23 had Clockwise circuit and 3 of them had both types of circuit (Figure 13). CTI independent atrial flutter was noted in 12 (12%) and 11(10%) had both CTI dependent and CTI independent circuit. Of the patients with CTI independent flutter, 10 had the circuit from the RA free wall and 2 had circuit in LA (Table 4).

![Figure 11 Showing the location of the flutter circuit](image-url)

**Location of the circuit**
Results and Observations

Figure 12 Showing the types of atrial flutter circuit

CTI dependent Atrial flutter

Figure 13 Showing the type of CTI dependent Atrial flutter
<table>
<thead>
<tr>
<th>Characteristics</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial flutter types</td>
<td></td>
</tr>
<tr>
<td>CTI Dependent</td>
<td>81(78)</td>
</tr>
<tr>
<td>Counterclockwise</td>
<td>55(51)</td>
</tr>
<tr>
<td>Clockwise</td>
<td>23(22)</td>
</tr>
<tr>
<td>Both types</td>
<td>3(3)</td>
</tr>
<tr>
<td>CTI Independent</td>
<td>12(12)</td>
</tr>
<tr>
<td>RA Free wall</td>
<td>10</td>
</tr>
<tr>
<td>Roof of LA</td>
<td>2</td>
</tr>
<tr>
<td>CTI Dependent + Independent</td>
<td>11(11)</td>
</tr>
<tr>
<td>CTI Dependent + LA</td>
<td>1</td>
</tr>
<tr>
<td>CTI Dependent + RA Free wall</td>
<td>7</td>
</tr>
<tr>
<td>Lower loop re-entry</td>
<td>2</td>
</tr>
<tr>
<td>Multiple</td>
<td>1</td>
</tr>
<tr>
<td>Location of the circuit</td>
<td>RA 101 (97)  LA 2(1.9)  RA+LA 1(1)</td>
</tr>
</tbody>
</table>

*Table 4* showing Procedural characteristics
Results and Observations

Ablation Outcomes

Most of the patients had ablation done with 3D electro anatomical mapping (83/104) constituting around 80%. Acute procedural success which was the primary endpoint of the study was achieved in 94/104 (90.3%) (Table 5). In patients with CTI dependent atrial flutter, Bidirectional conduction block across CTI was achieved in 77/92 (82%), Bidirectional delay in 4(4%), Unidirectional block in 4(4%) and no conduction block/delay was achieved in 7(8%) (Figure 14).

Figure 14 showing outcomes based on Bidirectional CTI conduction
Results and Observations

<table>
<thead>
<tr>
<th>Procedural characteristics</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>3D vs 2D</td>
<td>3D 83(80) 2D 21(20)</td>
</tr>
</tbody>
</table>

Conduction

<table>
<thead>
<tr>
<th>Type</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1:1 Conduction</td>
<td>1</td>
</tr>
<tr>
<td>2:1 Conduction</td>
<td>48(47)</td>
</tr>
<tr>
<td>3:1 Conduction</td>
<td>9(9)</td>
</tr>
<tr>
<td>4:1 Conduction</td>
<td>3(3)</td>
</tr>
<tr>
<td>Variable</td>
<td>43(41)</td>
</tr>
</tbody>
</table>

Acute success

<table>
<thead>
<tr>
<th>Type</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>94(90.3)</td>
<td></td>
</tr>
</tbody>
</table>

BD block achieved

<table>
<thead>
<tr>
<th>Type</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>77(82)</td>
<td></td>
</tr>
</tbody>
</table>

BD delay

<table>
<thead>
<tr>
<th>Type</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4(4)</td>
<td></td>
</tr>
</tbody>
</table>

Unidirectional delay

<table>
<thead>
<tr>
<th>Type</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4(4)</td>
<td></td>
</tr>
</tbody>
</table>

No Bi/ Uni directional block

<table>
<thead>
<tr>
<th>Type</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7(8)</td>
<td></td>
</tr>
</tbody>
</table>

Table 5 showing ablation outcomes

Peri procedural events (prior to discharge)

Peri procedural events were minimal during the study period. 1 patient had access site related hematoma which resolved on its own. 1 patient had transient high degree AV block which reverted to sinus rhythm in the next day and did not warrant any pacemaker implantation. 2 patients had atrial fibrillation in the post procedural period and 3 had recurrence within 48 hours (Table 6).
Results and Observations

<table>
<thead>
<tr>
<th>Complications</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor Complications</td>
<td></td>
</tr>
<tr>
<td>Access site - 1 (0.9)</td>
<td></td>
</tr>
<tr>
<td>Pericardial effusion/ tamponade</td>
<td>0</td>
</tr>
<tr>
<td>AF &lt;48hrs</td>
<td>2(1.9)</td>
</tr>
<tr>
<td>Flutter recurrence &lt;48hrs</td>
<td>3 (2.8)</td>
</tr>
<tr>
<td>DC Version</td>
<td>6 (5.7)</td>
</tr>
<tr>
<td>CVA</td>
<td>0 (2.8)</td>
</tr>
<tr>
<td>Mortality</td>
<td>0</td>
</tr>
<tr>
<td>High degree AV block</td>
<td>1 (0.9)</td>
</tr>
</tbody>
</table>

Table 6 showing Periprocedural complications

Follow up data

All the patients who underwent atrial flutter ablation were followed up till the end of study period. Mean follow up period in our study was 1074 days (36 Months), range (111- 3406 days). Atrial flutter recurrence was documented in 18/92 (19%) with successful ablation (Figure 14,15). Reablation was attempted in 7 of those patients. Atrial fibrillation was documented in 11 of those patients. Other than atrial flutter and atrial fibrillation, 4 patients had atrial tachycardia and 1 had AVNRT. 4 had CVA on follow up, out of which 3 had recurrence of AFL (Table 7).
AFL Ablation Follow up

- Recurrence
- No Recurrence

Figure 14 showing percentage of recurrence on follow up

19% Recurrence
81% No Recurrence
### Results and Observations

<table>
<thead>
<tr>
<th>Follow up data</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean follow up duration (Range)</td>
<td>1074 days (111 – 3406)</td>
</tr>
<tr>
<td>AFL Recurrence on follow up</td>
<td>18(19)</td>
</tr>
<tr>
<td>Re ablation done</td>
<td>7</td>
</tr>
<tr>
<td>AF on f/u</td>
<td>11(11)</td>
</tr>
<tr>
<td>Other tachycardia on f/u</td>
<td>5 (4 AT, 1 AVNRT)</td>
</tr>
<tr>
<td>CVA on f/u</td>
<td>4</td>
</tr>
</tbody>
</table>

**Table 7** showing outcomes on follow up

![Graph showing recurrence free survival](image)

**Figure 15** Graph showing recurrence free survival
Characteristics of patients with no recurrence and with recurrence

Patients who had successful atrial flutter ablation were divided into two groups (with and without recurrence) and their baseline, procedural and ablation outcomes were compared to find out the predictors of recurrence and their statistical significance. The mean age in patients without recurrence was 50.3 years and in patients without recurrence was 48.3 years. Follow up period in patients with recurrence were higher when compared to patients without recurrence (1484 Vs 784 days). Presence or absence of structural heart disease did not predict recurrence. Specific type of structural heart disease which were categorized into various subgroup based upon etiology also did not have statistical significance. CTI dependent atrial flutter has a trend towards lesser incidence of recurrence (OR 0.79, CI 0.22 – 2.76, P 0.712) and CTI Independent atrial flutter has a trend towards increased incidence of recurrence (OR 2.33, CI 0.52 – 10.39, P 0.266) though both of them were not statistically insignificant(Table 8,9).

Achievement of bidirectional block in patients with CTI dependent atrial flutter leads to reduce chances of recurrence (OR 0.19, CI 0.06 – 0.59, P <0.005), presence of bidirectional delay(OR 9.5, CI 0.94 – 95.6, P 0.005) or non achievement conduction block in the CTI(OR 23.18, CI 1.06 – 505.75, P 0.045) leads to increased chance of recurrence. Other parameters like unidirectional conduction block in CTI or LV ejection fraction < 50% were not predictors of recurrence during follow up(Figure 16).
Results and Observations

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No recurrence (n, %)</th>
<th>Recurrence (n, %)</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of Patients</td>
<td>76</td>
<td>18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>50.3(9-76)</td>
<td>48.3(27-74)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follow up</td>
<td>874 days</td>
<td>1484 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females</td>
<td>28</td>
<td>8</td>
<td>1.37(0.48 - 3.88)</td>
<td>0.551</td>
</tr>
<tr>
<td>Structurally normal heart</td>
<td>23</td>
<td>5</td>
<td>0.89(0.28 - 2.77)</td>
<td>0.835</td>
</tr>
<tr>
<td>Structural heart disease</td>
<td>53</td>
<td>13</td>
<td>1.12(0.36 – 3.53)</td>
<td>0.835</td>
</tr>
<tr>
<td>Post OP ASD</td>
<td>26</td>
<td>7</td>
<td>1.22(0.42 – 3.53)</td>
<td>0.708</td>
</tr>
<tr>
<td>Other CHD Post OP</td>
<td>10</td>
<td>1</td>
<td>0.39(0.04 – 3.24)</td>
<td>0.382</td>
</tr>
<tr>
<td>CAD</td>
<td>8</td>
<td>2</td>
<td>1.06(0.20 – 5.5)</td>
<td>0.942</td>
</tr>
<tr>
<td>RHD</td>
<td>1</td>
<td>1</td>
<td>4.41(0.26-74.12)</td>
<td>0.302</td>
</tr>
<tr>
<td>ASD non operated</td>
<td>1</td>
<td>1</td>
<td>4.41(0.26-74.12)</td>
<td>0.302</td>
</tr>
<tr>
<td>DCM+EMF+HCM</td>
<td>5</td>
<td>1</td>
<td>0.83(0.9-7.62)</td>
<td>0.873</td>
</tr>
</tbody>
</table>

Table 8 showing comparison of characteristics between patients with recurrence and no recurrence
## Results and Observations

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No recurrence (n, %)</th>
<th>Recurrence (n, %)</th>
<th>OR (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Others (Myxoma, RV cardiomyopathy)</td>
<td>2</td>
<td>0</td>
<td>0.80 (0.03-17.5)</td>
<td>0.890</td>
</tr>
<tr>
<td>CTI Dependent</td>
<td>62</td>
<td>14</td>
<td>0.79 (0.22-2.76)</td>
<td>0.712</td>
</tr>
<tr>
<td>CTI Independent</td>
<td>6</td>
<td>3</td>
<td>2.33 (0.52–10.39)</td>
<td>0.266</td>
</tr>
<tr>
<td>Bidirectional block</td>
<td>66</td>
<td>10</td>
<td>0.19 (0.06 – 0.59)</td>
<td>&lt; 0.005</td>
</tr>
<tr>
<td>Bidirectional delay</td>
<td>1</td>
<td>2</td>
<td>9.5 (0.94 – 95.6)</td>
<td>0.005</td>
</tr>
<tr>
<td>Bi/Uni directional block not achieved</td>
<td>0</td>
<td>2</td>
<td>23.18 (1.06-505.75)</td>
<td>0.045</td>
</tr>
<tr>
<td>Unidirectional block</td>
<td>3</td>
<td>1</td>
<td>1.43 (0.14 – 14.62)</td>
<td>0.762</td>
</tr>
<tr>
<td>AF after ablation</td>
<td>9</td>
<td>2</td>
<td>0.93 (0.18 – 4.73)</td>
<td>0.930</td>
</tr>
</tbody>
</table>

Table 9 showing comparison of characteristics between patients with recurrence and no recurrence
**Results and Observations**

**Figure 16** Forrest plot showing predictors of recurrence

- Post-op ASD: 1.22 (0.42, 3.53)
- CII dependent: 0.79 (0.22, 2.76)
- Bidirectional block: 0.19 (0.06, 0.59)
- Bidirectional delay: 9.5 (0.94, 95.6)
- Bidirectional block not achieved: 23.18 (1.0, 505.7)
- Unidirectional block: 1.43 (0.14, 14.62)
- AF after ablation: 0.93 (0.18, 4.73)
- LVEF < 50%: 0.67 (0.13, 3.28)
DISCUSSION

This study was aimed to find out the acute and long term outcomes of atrial flutter ablation, find out the arrhythmia free survival during long term follow up and to find out the predictors of atrial flutter recurrence. Since most of the patients in this study underwent atrial flutter ablation with electroanatomical 3 D mapping and with standard anatomical approach, results of this study reflect the therapeutic power of a mature and standardized radiofrequency ablation approach for this arrhythmia.

Baseline characteristics

The mean age of ablation in the study was 50 years and it was noted that with the increasing age, the incidence of atrial flutter also increases. As previously stated the incidence of atrial flutter is 2.5 times more common in males when compared with females, in our study the sex ratio was 1.5: 1 (1). Structural heart disease was seen in 71% of patients which is higher when compared to previous ablation studies where in structural heart diseases accounted for just under half the patients (30). Sebastian Schmieder et al published a large series of atrial flutter ablation in which 72% of patients had structural heart disease which is similar to this study but then in western population the most common structural abnormality which is noted is hypertensive heart disease (52%), coronary artery disease (26%) and post cardiac surgery constituted just 8%. The most common structural heart disease in our study was post ASD surgical closure which constituted the majority of cases. This difference could be because of selection bias but it is worthy to note the difference in etiology in our population (33).

10% of patients had prior documented atrial fibrillation prior to ablation procedure. In the study by Sebastian Schmieder et al it was noted
that 55% of patients had documented Atrial fibrillation. The arrhythmic burden in such persons is higher. There is an overlap between risk factors and etiology for atrial flutter and atrial fibrillation and hence there is a frequent association between them. It is also postulated that atrial flutter could be a fore runner or early stage of atrial disease which in future can lead onto development of atrial fibrillation. These postulate has lead operators to combine AF ablation after completion of CTI ablation during the same procedure (35).

**Electrophysiological characteristics**

RA flutter was more common in our study constituting 97% of patients (101/104) and only 2 had LA flutter. Both the patients with LA flutter had structural heart disease. In our study more than 2/3 rd of the patients had right atrial typical atrial flutter (CTI dependent atrial flutter) which is comparable to previous studies stating 70 – 75 % and is the most common type of atrial flutter (9). In patients with CTI dependent atrial flutter the circuit is most commonly Counterclockwise. 55% had CCW AFL which is lesser when compared to prior studies which quoted up to 90% for CCW(2). In patients with atypical flutter (11%) most of them had the tachycardia circuit in RA free wall and few had it in LA roof. 10 % of patients had circuit which was CTI dependent and also had a non CTI dependent flutter. In those patients they underwent CTI ablation and also their non CTI circuit was also ablated. AV conduction depends upon the intrinsic conduction properties of the node, influence of antiarrhythmic drugs, atrial flutter cycle length and autonomic influence on the node. Most of the patients in our study had 2:1 or variable AV conduction in the tachycardia induced prior to ablation.
Ablation outcomes

Most of the patients underwent ablation under 3D electroanatomical mapping. Electroanatomical approach is of greater importance in Non CTI dependent and scar related flutters. Precise location of the circuit and scar area by voltage mapping and propagation mapping makes those complicated ablations easier. Acute procedural success which was the primary outcome of the study was defined as “termination of tachycardia, Non inducibility of tachycardia post ablation, No recurrence of atrial flutter < 48 hrs post procedure”. Acute procedural success was reached in 94/104 (90.3%). Achievement of bidirectional conduction block is considered as one of the important endpoints of CTI dependent atrial flutter ablation. It was achieved in 77/93 (82%) of patients, Bidirectional delay and unidirectional block was achieved in 4 patients each and 7 of them did not have any conduction block through CTI. In a study by Hugh Calkins et al of 150 patients with typical atrial flutter ablation that underwent ablation had an acute procedural success of 88 % (32). NAPSE published their ablation registry where In their AFL ablation acute success was achieved in 85.8% of patients (37). Sebastian Schmieder et al published the results of 363 with typical atrial flutter who underwent CTI ablation and BD block was achieved in 90% of patients (33). E Bertaglia et al in their study published in heart 2004, 367/383(95%) had acute procedural success. Increased incidence of structural heart disease and inclusion of patients with atypical atrial flutter could explain the lesser slightly lesser acute procedural success rate.

Reasons for failure to produce a Bidirectional conduction block across isthmus block are not very clear. Anatomic factors like thicker isthmus myocardium or an abnormal isthmus geometry which prevents a close catheter tissue contact throughout the isthmus entirety or long
Eustachian valve could be the cause (38)(39). Another possible explanation could be, with the use of conventional mapping technique the possibility of rapid conduction around the posterior aspect of the inferior vena cava cannot be excluded. In this situation the completeness of the isthmus block cannot be evaluated. (40)

Patient in whom Bidirectional conduction block was demonstrated during the initial procure when studied for recurrence, some of them showed CTI conduction. The exact factors for the resumption of the conduction are not clear, the possible role of acute ischaemia and oedema caused by radiofrequency ablation might be considered as factors that participate in acute interruption of the trans-isthmus conduction, masking the presence of still viable isthmus myocardium. Since these factors fade gradually after acute damage, viable myocardium at the isthmus might restore capacity for conduction.(33)

**Procedural complications**

Peri procedural events were minimal during the study period. 1 patient had access site related hematoma which resolved on its own. 1 patient had transient high degree AV block which reverted to sinus rhythm in the next day and did not warrant any pacemaker implantation. 2 patients had atrial fibrillation in the post procedural period and 3 had recurrence within 48 hours.

RF catheter ablation of the CTI for type 1 AFL is relatively safe, with complication rates of 2.5% to 3.5% in previous studies (41). Most complications are peripheral vascular injury (0.4%). Serious complications can rarely occur, including heart block (0.2%), pericardial effusion and tamponade (0.1%), myocardial infarction from right
coronary artery injury, and thromboembolic events, including pulmonary embolism and stroke.

**Follow up results**

Most of the patients who underwent atrial flutter ablation were followed up till the end of study period. Mean follow up period in our study was 1074 days (36 Months), range (111- 3406 days). Atrial flutter recurrence was documented in 18/92 (19%) with successful ablation. Reablation was attempted in 7 of those patients. Atrial fibrillation was documented in 11 of those patients. Other than atrial flutter and atrial fibrillation, 4 patients had atrial tachycardia and 1 had AVNRT. 4 had CVA on follow up, out of which 3 had recurrence of AFL.

Hugh Calkins et al on follow up for 6 month recurrent typical atrial flutter was observed in 13% of patients (32). E Bertaglia at al reported during the follow up of 20.5 (12.4) months (range 6–55 months), recurrence of typical AFL was documented in 41 of 367 patients (11.2%) (30). Sebastian Schmieder et al reported in his study the long term outcomes where in 343 patients (95%) were followed up for a mean of 496±335 days, during which 310 patients (90%) remained free of AFL recurrences(33). NAPSE registry stated that during their follow up, recurrent flutter was documented in 64 (14.7%) patients.

Compared to the previous studies, our patients had increased incidence of recurrence possible explanation could be in this study most patients had structural heart disease and study included atypical flutter also, which is well known for higher recurrence. Follow up duration in our study was longer than the previous studies and hence could have increased documentation of arrhythmia recurrence.
Characteristics of patients with no recurrence and with recurrence

Patients who had successful atrial flutter ablation were divided into two groups (with and without recurrence) and their baseline, procedural and ablation outcomes were compared to find out the predictors of recurrence and their statistical significance. The mean age in patients without recurrence was 50.3 years and in patients without recurrence was 48.3 years. Follow up period in patients with recurrence were higher when compared to patients without recurrence (1484 Vs 784 days). Presence or absence of structural heart disease did not predict recurrence. Specific type of structural heart disease which were categorized into various subgroup based upon etiology also did not have statistical significance. CTI dependent atrial flutter has a trend towards lesser incidence of recurrence (OR 0.79, CI 0.22 – 2.76, P 0.712) and CTI Independent atrial flutter has a trend towards increased incidence of recurrence (OR 2.33, CI 0.52 – 10.39, P 0.266) though both of them were not statistically insignificant.

Achievement of bidirectional block in patients with CTI dependent atrial flutter leads to reduce chances of recurrence (OR 0.19, CI 0.06 – 0.59, P <0.005). Presence of bidirectional delay (OR 9.5, CI 0.94 – 95.6, P 0.005) or non achievement conduction block in the CTI (OR 23.18, CI 1.06 – 505.75, P 0.045) leads to increased chance of recurrence. Other parameters like unidirectional conduction block in CTI or LV ejection fraction < 50% were not predictors of recurrence during follow up.

Previous studies have shown that five independent predictors of AFL recurrence: fluoroscopy time (p<0.001), atrial fibrillation after AFL ablation (p0.01), lack of bidirectional block (p0.02), reduced left ventricular function (p0.035) and right atrial dimensions (p0.046). Atrial
fibrillation occurrence was significantly reduced after AFL ablation (112 in 343 patients, 33%) as compared to occurrence of atrial fibrillation before radiofrequency ablation (198 in 363 patients, 55%, p<0.001).(33)

**Atrial fibrillation on follow up**

Atrial fibrillation post RF ablation for flutter on follow up in our study was 11/104(11%) patients. Prior to the procedure 10 patients had documented AF. This number is very less when compared to previous studies which has documented up to 20% to 30% (with short-term follow-up, approximately 1 year) and in up to 82% (with long-term follow-up, approximately 4 years) of patients with or without a prior history of AF(42).

In a meta-analysis, average follow-up of 16 months, AF occurred in 23.1% of patients with no preablation history of AF and in 52.7% of patients with a history of AF, despite successful AFL ablation. The occurrence of AF was not influenced by the ablation technology or procedural endpoints. This finding has implications for patient selection, long-term arrhythmia-free success rates, post procedure antiarrhythmic drug use, and post procedure anticoagulation. Because AF predating ablation of AFL is very likely to recur, thus limiting the ability to discontinue antiarrhythmic medications or anticoagulation in many patients, the potential benefits of ablation of only AFL should be seriously scrutinized (43).
LIMITATIONS

Limitation of this study is that it is a retrospective analysis of data. All patients in the study group did not undergo holter evaluation and hence there is a real chance that patients with asymptomatic recurrence could have been missed in our study. Even though the study includes procedure done from 2005, the mean follow up duration is only 3 years; this signifies that many patients are lost to follow up few years after the procedure.
CONCLUSION

Atrial flutter ablation has a high procedural success rate with low procedural complications. Achieving a bidirectional conduction block in CTI dependent Atrial flutter leads to lower recurrence during follow up.
REFERENCES


References


32. Hugh Calkins, MD, Robert Canby, MD, for the 100W Atakr II Investigator Group Results of Catheter Ablation of Typical Atrial Flutter (Am J Cardiol 2004;94:437–442)


37. Melvin M. Scheinman and Sue Huang The 1998 NASPE Prospective Catheter Ablation Registry (PACE 2000; 23:1020-1028)


## Appendix

### Originality Report

**10%**

### Similarity Index

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<td>1. <a href="http://www.herzrhythmusst%C3%B6rungen.org">www.herzrhythmusstörungen.org</a></td>
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<td>2. circep.ahajournals.org</td>
<td>55</td>
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<td>3. Ziad F. Issa. <em>Isthmus-Dependent Atrial Flutter</em>, <em>Clinical Arrhythmology and Electrophysiology, 2009</em></td>
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<td>4. dev.revespcardiol.elsevier.es</td>
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PROFORMA

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<tr>
<td>Patient ID:</td>
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<tr>
<td>Name:</td>
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<td>Age (in years):</td>
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**Symptoms (as per records of hospital visit)**

<table>
<thead>
<tr>
<th>Dyspnoea on exertion: Yes/ No</th>
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<tr>
<td>If yes, NYHA functional class-</td>
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<td>Orthopnoea: Yes/no</td>
</tr>
<tr>
<td>PND: Yes/no</td>
</tr>
</tbody>
</table>

| Angina on exertion: Yes/ No   |
| If yes, NYHA functional class-|

| Syncope: Yes/ no              |
| Presyncope: Yes/ no           |
| Palpitations: Yes/ no         |
| Edema: Yes/no                 |

| Past history of Acute coronary syndrome: Yes/ No |
| Past history of rheumatic fever: Yes/ no        |
| History of Hyperthyroidism: Yes/ no             |
| History of Atrial Fibrillation: Yes/ no         |
| History of Stroke: Yes/ no                      |

| History of Prior cardiac surgery: Yes/ no       |
| If yes then what Surgery........................ Date of procedure......... |

**Coronary artery disease risk factors:**

| Diabetes: Yes/ no |
| Systemic Hypertension: Yes/ no |
| Dyslipidemia: Yes/ no |
| Smoking: Yes/ no |
Appendix

ECG findings

Rate:

Type of flutter: Clockwise/ Counterclockwise/Right atrial flutter/ Right free wall /Left atrial flutter

Ventricular rate: QRS axis: PR interval:
QRs duration:

Description (if required)

Chest roentgenogram:

CT ratio: LAE:Yes/no RAE: yes/no
PVH grade: Absent/ I/ II/ III PAH: yes/no

Echocardiographic findings:

<table>
<thead>
<tr>
<th>LVIDd(mm)</th>
<th>MR GRADE (1+ to 4+)</th>
</tr>
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<tbody>
<tr>
<td>LVIDs(mm)</td>
<td>AR GRADE(1+ to 4+)</td>
</tr>
<tr>
<td>VEF(M mode,%)</td>
<td>TR GRADE(1+ to 4+)</td>
</tr>
<tr>
<td>LA (mm)</td>
<td>RVSP(mmhg)</td>
</tr>
<tr>
<td>Aorta (mm)</td>
<td>PUL.VELOCITY(m/s)</td>
</tr>
<tr>
<td>EDV (ml)</td>
<td>PR GRADE (1+ to 4+)</td>
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<td>ESV(ml)</td>
<td>PEAK PR (mmhg)</td>
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<tr>
<td>EF(AL METHOD,%)</td>
<td>AORTIC.VELOCITY(m/s)</td>
</tr>
</tbody>
</table>

EP study

Rate

AV Conduction

Type of Atrial flutter

3 D Mapping done Yes/No

Site of Ablation:
Radiofrequency delivered and duration

Ablation Endpoint: Unidirectional Block/ Bidirectional Block

Induction of tachycardia Post procedure

Procedural Complications

Pericardial effusion: Yes/No

Cardiac Tamponade: Yes/No

Vascular complications:

Heart block:

Per procedural Stroke :

Post Ablation Follow up

**Symptoms (as per records of hospital visit)**

**Dyspnoea on exertion:** Yes/ No Post ablation time period

If yes, NYHA functional class-

Orthopnoea : Yes/no PND: Yes/no

**Syncope:** Yes/ no Post ablation time period

**Presyncope:** Yes/ no Post ablation time period

**Palpitations:** Yes/ no Post ablation time period

Post procedural Echo

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<td>VEF(M mode,%)</td>
<td>TR GRADE(1+ to 4+)</td>
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<tr>
<td>LA (mm)</td>
<td>RVSP(mm/hg)</td>
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<tr>
<td>Aorta (mm)</td>
<td>PUL.VELOCITY(m/s)</td>
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<td>EDV (ml)</td>
<td>PR GRADE (1+ to 4+)</td>
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<td>PEAK PR (mm/hg)</td>
</tr>
<tr>
<td>EF(AL METHOD,%)</td>
<td>AORTIC.VELOCITY(m/s)</td>
</tr>
</tbody>
</table>
Appendix

Recurrence Of tachycardia

Documented tachycardia Yes/ no

If yes then Atrial flutter/Atrial fibrillation/Other tachycardia……………………

How was the tachycardia detected: ECG/Holter

Holter findings

Sustained tachycardia/ ill sustained tachycardia

Recurrent Atrial Flutter

Time interval between 1st recurrence and procedure…………………..

Tachycardia Morphology :Similar to the Previous tachycardia/ Not Rate………… AV conduction………..

Treatment modality opted : Rate control/Rhythm control/ Ablation

Recurrence of Atrial fibrillation :

Time interval between 1st recurrence and procedure…………………..

Type of atrial fibrillation: Paroxysmal/Persistent/ Permanent

Ventricular rate ……..

Treatment modality opted : Rate control/Rhythm control/ Ablation

Recurrence of other tachycardia:

Time interval between 1st recurrence and procedure…………………..

Type of tachycardia:

Description of tachycardia

Treatment modality opted: Rate control/Rhythm control/ Ablation
PATIENT INFORMATION FORM

Title of the study: Atrial flutter ablation outcomes and predictors of recurrence

Name of the Investigators: Dr. Suraj Narasimhan A,
Dr. Krishna Kumar M, Dr. K.K Narayanan Namboodiri, Dr Ajith Kumar VK

You are being requested to participate in this study to assess Atrial flutter ablation outcomes and predictors of recurrence.

This is a Retrospective-Prospective observational study to understand follow up of patients who underwent the procedure of Radiofrequency ablation in patients with atrial flutter. Study aims to assess efficacy of Radiofrequency ablation as a short term and long term treatment modality of patients with Atrial flutter. It also aims to study the predictors of recurrence during the follow up of patients.

We are going to find out the outcomes of atrial flutter ablation. We are also going to find out the predictors of atrial flutter recurrence and occurrence of atrial fibrillation in ablated patients.

Who will be included in this study?

- All consecutive patients who have underwent Atrial flutter ablation in SCTIMST from Jan 1, 2005 till date and those who undergo ablation from now till June 31, 2016 will be enrolled. Patients without adequate follow up data (at least 3 Months of follow up) will be excluded from the study.
If you take part what will you have to do?

After you have consented to be part of the study, you will be interviewed for your disease status and hospital records will be reviewed for clinical status, primary rhythm abnormality, ECG findings, electrophysiological study reports and your follow up data.

No patient will be asked to come to hospital for follow up when there is no cardiac indications, just for collection of data.

For a few patients for whom logistical problems made clinic attendance impossible, after obtaining oral consent from the patient clinical status, complaints and follow-up data will be obtained from your treating local cardiologist/physician or by telephonic questionnaires.

Since all the patient attending hospital are for cardiological indications and not for collection of data alone, no reimbursement for travel or daily wages lost will be given to patients participating in the study.

Who will undergo Atrial flutter radiofrequency ablation?

Patient with atrial flutter ablation will be selected based upon guidelines oriented approach for management of atrial flutter. Patient will be well explained about the procedure, complications and outcomes after detailed discussion with principal co investigators. Patient will be selected after consent and without known bias.

Can you withdraw from this study after it starts?

Your participation in this study is entirely voluntary and you are also free to decide to withdraw permission to participate in this study. If
you do so, this will not affect your usual treatment at this hospital in any way.

**Whether there is any risk related to this study?**

There is no individual risk or benefit to the patient himself as it is a observational study. Rarely if any harm happen to patients, they will be managed but no monetary benefits will be provided. This study will assess impact of radio frequency ablation for atrial flutter both in short term and long term. This study will provide insight to clinicians about the outcomes of atrial flutter ablation and also about the predictors of recurrence.

**Will your personal details be kept confidential?**

The results of this study will be published in a medical journal but you will not be identified by name in any publication or presentation of results. However, your medical notes may be reviewed by people associated with the study, without your additional permission, should you decide to participate in this study.

If at any time you experience any problems, or if you have any further questions, please ask, Dr. Suraj Narasimhan A (principle investigator), drasuraj@sctimst.ac.in or mobile :08594055605

For further information or anxiety about the ethical aspects of the study, please contact institute ethics committee member secretary Ph : 04712524234, email : iec.mem.sec@sctimst.ac.in
CONSENT FORM

Title of the study : Atrial fultter ablation outcomes and predictors of recurrence

Participant’s name: Age (in years):

I________________, son/daughter/husband/wife/-------------- of
________________________ declare that  (Please tick boxes)

- I have read the above information provide to me regarding the study:[ ]
- I have clarified any doubts that I had. [ ]
- I also understand that my participation in this study is entirely voluntary and that I am free to withdraw permission to continue to participate at any time without affecting my usual treatment or my legal rights [ ]
- I understand that the study staff and institutional ethics committee members will not need my permission to look at my health records even if I withdraw from the trial. I agree to this access [ ]
- I understand that my identity will not be revealed in any information released to third parties or published [ ]
- I voluntarily agree to take part in this study [ ]
• I have been provided with the contact numbers of the principle investigator, in case I want to know more about the study and participants rights [ ].

• I received a copy of this signed consent form [ ]

Name: 

Name of witness: 

Relation to participant: 

Signature: 

Signature: 

Signature:
Person Obtaining Consent

I attest that the requirements for informed consent for the medical research project described in this form have been satisfied. I have discussed the research project with the participant and explained to him or her in nontechnical terms all of the information contained in this informed consent form, including any risks and adverse reactions that may reasonably be expected to occur. I further certify that I encouraged the participant to ask questions and that all questions asked were answered.

Name :

Signature :

Date :

Place : SCTIMST, Thiruvananthapuram
Appendix

നായികാരി വിവിധ സ്ഥലങ്ങൾ.

നായികാരി നിർദ്ദേശത്തിന്റെ നാമത്തില്‍ സ്ഥാനം നല്‍കുന്നതു വഴി നിലവില്‍ നിന്ന് വെച്ച്

നായികാരി ഭാഷയില്‍ - മാനവ സ്വാഭാവികാണം, മാന്യ അതിന്റെ പരിപാലനം വേണം.

മാന്യത്തിന് മരണം. ആരോഗ്യം, മാനവ കഴിവുകള്‍ നടപ്പാക്കുക.

ജീവിതത്തെ വിധിയ്ക്കുന്നതു വഴി നിന്ന് പഠിക്കാം ഭാഷയില്‍ നിന്ന് വെച്ച്

നായികാരി നിർദ്ദേശത്തിന്റെ നാമത്തില്‍ സ്ഥാനം നല്‍കുന്നതു വഴി നിലവില്‍ നിന്ന് വെച്ച്

നായികാരി ഭാഷയില്‍ - മാനവ സ്വാഭാവികാണം, മാന്യ അതിന്റെ പരിപാലനം വേണം.

മാന്യത്തിന് മരണം. ആരോഗ്യം, മാനവ കഴിവുകള്‍ നടപ്പാക്കുക.

നായികാരി ഭാഷയില്‍ - മാനവ സ്വാഭാവികാണം, മാന്യ അതിന്റെ പരിപാലനം വേണം.

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നായികാരി ഭാഷയില്‍ - മാനവ സ്വാഭാവികാണം, മാന്യ അതിന്റെ പരിപാലനം വേണം.

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Appendix
Appendix

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Institutional Ethics Committee
(IEC Regn No. ECR/189/Inst/KL/2013)

SCT/IEC/83/APRIL-2016

Dr. Suraj Narasimhan A
Resident
Department of Cardiology
SCTIMST, Thiruvananthapuram

Dear Dr. Suraj Narasimhan,

The Institutional Ethics Committee reviewed and discussed your application to conduct the study entitled "ATRIAL FULTTER ABLATION OUTCOMES AND PREDICTORS OF RECURRENCE" (IEC/883) on 16th April, 2016.

The following documents were reviewed:

Original submission
1. Covering letter addressed to the Chairperson, IEC, SCTIMST, dated 20.03.2016 with check list
2. Letter addressed to the Chairperson, IEC, SCTIMST, dated 20.03.2016 received from Dr. Narayanan Nambodiri, Additional Professor, Department of Cardiology, SCTIMST
3. TAC Approval Letter
4. IEC Application Form
5. Project Proposal
6. Proforma
7. Consent Form in English and Malayalam
8. CV of Principal Investigator and Co- Investigators

Revised submission
1. Covering letter addressed to Chairperson, IEC, SCTIMST dated 13.05.2016 with check list
2. Copy of the IEC Recommendation letter dated 03.05.2016
3. TAC Approval Letter
4. IEC Application Form
5. Project Proposal
6. Proforma
7. Patient Information Sheet and Consent Form in English and Malayalam
8. CV of Principal Investigator and Co- Investigators
The following members of the Ethics Committee were present at the meeting held on 16th April, 2016 at G. Parthasarathi Board Room, AMCHSS, SCTIMST.

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<tr>
<th>SL. No.</th>
<th>Member Name</th>
<th>Highest Degree</th>
<th>Gender</th>
<th>Scientific/Non Scientific</th>
<th>Affiliation with Institution(s)</th>
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<td>Justice Gopinathan, P.S.</td>
<td>BSc, LLB</td>
<td>Male</td>
<td>Legal Expert (Chairperson)</td>
<td>No</td>
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<tr>
<td>2</td>
<td>Dr. Aoho Kirhora</td>
<td>MD, DM</td>
<td>Female</td>
<td>Clinician (Neurologist)</td>
<td>Yes</td>
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<td>3</td>
<td>Shri. O.S. Neelakantan Nair</td>
<td>BE</td>
<td>Male</td>
<td>Engineer</td>
<td>Yes</td>
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<tr>
<td>4</td>
<td>Dr. Meenu Hariharan</td>
<td>DM</td>
<td>Female</td>
<td>Clinician (Gastro-Enterologist)</td>
<td>No</td>
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<tr>
<td>5</td>
<td>Dr. Rema M. N</td>
<td>MD</td>
<td>Female</td>
<td>Pharmacologist</td>
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<tr>
<td>6</td>
<td>Dr. V. Raman Kutty</td>
<td>MPH (Harvard) MPhil, MD</td>
<td>Male</td>
<td>Public Health</td>
<td>Yes</td>
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<tr>
<td>7</td>
<td>Dr. K R S Krishnan</td>
<td>ME, PhD</td>
<td>Male</td>
<td>Biomedical Scientist/Engineer</td>
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<tr>
<td>8</td>
<td>Dr. Kala Kesavan, P.</td>
<td>MD</td>
<td>Female</td>
<td>Pharmacologist</td>
<td>No</td>
</tr>
<tr>
<td>9</td>
<td>Smt. Sathis Nair</td>
<td>MA</td>
<td>Female</td>
<td>I ay Person</td>
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</tr>
<tr>
<td>10</td>
<td>Dr. Christine George</td>
<td>MD</td>
<td>Female</td>
<td>Psychiatrist</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>Dr. Mala Ramanathan</td>
<td>MSc, PhD, MA</td>
<td>Female</td>
<td>Ethicist/Social Scientist (Member Secretary)</td>
<td>Yes</td>
</tr>
</tbody>
</table>

IEC Decision

The IEC approved the conduct of the study in the present form.

Remarks:
The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information/informed consent and asks to be provided a copy of the final report.

There was no member of the study team who participated in voting / decision making process. The ethics committee is organized and operated according to the requirements of Good Clinical Practice and the requirements of the Indian Council of Medical Research (ICMR).

Sincerely,

Mala Ramanathan
Member Secretary, IEC