The Role of Radiofrequency Catheter Ablation in the Management of Atrial Arrhythmias

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ATRIAL FIBRILLATION

Atrial Fibrillation is the most common cardiac arrhythmia, with an incidence of approximately 2% in the adult population that increases with each decade of life. It has been estimated that atrial fibrillation is present in up to 10% of the population over 60 years of age and up to 79% of the patients with mitral valve disease; this accounts for at least one million people in the United States with this arrhythmia. The sequellae of atrial fibrillation are 1) increased risk of stroke (up to 5% per year in nonrheumatic atrial fibrillation), 2) loss of the atrial "kick" and AV synchrony, and 3) a change in the ventricular rate resulting in an irregular rhythm and a rapid ventricular response.

In view of all of these complications, physicians aggressively try to convert the rhythm to sinus mechanism. Unfortunately, the efficacy of pharmacological therapy is at best 50%. Class IA agents such as quinidine, procainamide and disopyramide were commonly used in the past, but their safety was put into question recently following the Coplen analysis, which indicated that the use of quinidine may be associated with a possible increase in cardiac mortality. The use of class IC agents such as flecainide and encainide have also been shown to result in an increase mortality in patients with coronary artery disease (CAST study). Today, most patients with structural heart disease are being treated with class III agents such as amiodarone and sotalol. Unfortunately these drugs are associated with serious side effects (mainly proarrhythmia with sotalol and potential lung and liver toxicity with amiodarone therapy). In patients who fail pharmacological therapy, treatment is directed at controlling ventricular rate and anticoagulation alone. Unfortunately, rate controlling agents such as calcium channel blockers and beta blockers may not be well tolerated in patients with left ventricular dysfunction and could have an adverse effect on mortality. Therefore, drug therapy is suboptimal and subjects patients to potential serious side effects.

Radiofrequency (RF) catheter ablation in the management of atrial fibrillation was first used to control the rate in patients who failed drug therapy; this is usually achieved with atrioventricular (AV) junction ablation and subsequent pacemaker implantation, or with AV nodal modification to slow the ventricular response. More recently, research has concentrated on RF ablation to restore sinus rhythm in selected patients.

I - Atrioventricular Junction Ablation

Catheter ablation of the atrioventricular junction (AVJ) was first introduced in 1982 for the management of patients with drug refractory supraventricular tachycardia (1). Direct current, applied via catheter by a standard defibrillator was used, resulting in major complications including ventricular rupture and hemodynamic collapse (2, 3). With the advent of radiofrequency ablation in 1987, this technique became safer and the success rate increased. As our understanding of supraventricular arrhythmias and in particular atrial tachycardias and atrial flutter improved, curative procedures for these arrhythmias developed and AV junction ablation was relegated largely for patients with drug refractory atrial fibrillation.

Method

The procedure is performed by placing the ablating catheter across the tricuspid valve so that the largest His bundle potential is recorded. The catheter is then withdrawn to record atrial and ventricular signals of equal amplitudes. Application of RF energy at this location usually results in a burst of junctional tachycardia preceding complete AV block. In 10-20% of cases, complete AV block cannot be achieved from the right side and a left sided approach is needed. For left sided ablation, the ablating catheter is advanced retrograde across the aortic valve and RF energy is applied at areas showing the largest left

bundle or His bundle potential. The presence of a junctional escape rhythm after AV junction ablation is dependent on the site of RF applications. If the RF lesions damaged the compact AV node, a junctional escape rhythm often develops from the proximal His bundle. If the RF lesions damaged the His bundle itself or resulted in a right bundle branch block with a subsequent need for a left sided approach, the presence of a subsidiary pacemaker function is significantly impaired and the patient becomes pacemaker dependent.

Clinical Outcome after AV Junction Ablation

In general, the functional status, exercise tolerance, and symptoms improve after AV junction ablation and pacemaker implantation. In some patients with heart failure, left ventricular ejection fraction increases after 3-6 months, suggesting that the tachycardia itself might have been the cause of the left ventricular dysfunction (4-10). In contrast, some patients experience a decrease in left ventricular function (11) . In one series (11), up to 7% of patients developed worsening mitral regurgitation with a subsequent need for surgical intervention. This worsening in left ventricular function and mitral regurgitation is probably the result of the asynchronous ventricular activation that occurs during ventricular pacing. Another complication of this procedure is polymorphic ventricular tachycardia (VT), which has been reported in up to 6-7% of cases (12-14) . Usually this arrhythmia occurs in the first few days that follow the procedure and seems to be bradycardia and pause dependent. Geelen et al (13) have reported a decrease in the incidence of polymorphic VT down to 0% in patients paced at a rate of 90 bpm for 1-3 months after AVJ ablation. In summary, AVJ ablation is relatively safe and its benefits far outweighs its risks and complications, as long as short term rapid ventricular pacing is provided after the procedure.

Indications

It is generally accepted that an average heart rate above 90 bpm is likely to be detrimental, and that AVJ ablation with pacemaker implantation is required if drug therapy fails to reach this level of rate conrtol. In addition, patients with atrial fibrillation and life-threatening symptoms due to rapid ventricular response are best managed with this procedure. The advantages of this procedure include 1) improvement in symptoms, exercise tolerance and left ventricular function in some cases and 2) elimination of rate-controlling drug therapy. The main disadvantages are 1) rendering the patient pacemaker dependent for life 2) possible risk of sudden death and 3) the continued need for anticoagulation.

II - AV Node Modification

Since AVJ ablation results in complete AV block with a permanent need for pacemaker implantation, several investigators looked at modifying the AV node instead of ablating it as an alternative method to control the ventricular rate. Early attempts to modify the AV node targeted the fast pathway (anterior approach) but resulted in a low success rate and a high incidence of complete AV block. Subsequently, modification of the AV node was done targeting the slow pathway (posterior approach) resulting in a better success rate and a much lower complication rate.

Mechanism of atrial fibrillation rate control with slow pathway ablation

The mean ventricular response during atrial fibrillation has been shown to correlate with the AV node block cycle length and the AV node effective refractory period (ERP) (15, 16). In patients with dual AV node physiology, the AV node ERP is very much determined by the slow pathway ERP, which is shorter than the fast pathway ERP. Therefore, slow pathway ablation would be expected to increase AV nodal ERP and AV nodal block cycle length. Blanck and coworkers (17) showed a significant decrease in ventricular rate during induced atrial fibrillation in patients with atrioventricular nodal

reentrant tachycardia AVNRT following slow pathway ablation. This was accompanied by an increase in AV nodal effective refractory period. Several other studies also demonstrated that elimination of the slow pathway resulted in slowing of the ventricular response during spontaneous or induced atrial fibrillation. What remains unclear, is why a similar approach would be effective in patients with atrial fibrillation and no history of AVNRT or evidence of dual AV node physiology. Possible explanations include 1) elimination of posterior atrionodal connections which are a part of the normal physiology of the AV node, resulting in slowing of the ventricular response (18) and 2) direct damage to the compact AV node (19).

Method

Different techniques for modifying of the AV node have been described. Williamson et al (20) delivered radiofrequency ablation during steady state isoproterenol infusion. RF lesions were applied posteriorly near the coronary sinus ostium and advanced anteriorly until a target heart rate of 120 bpm was achieved. Feld et al (21) used the midseptal approach and gradually moved posteriorly until the ventricular rate fell below 100 bpm. Atropine (1mg) was then given and the procedure was considered to be successful if the ventricular rate was below 120 bpm. Today, most electrophysiologist start posteriorly and move anteriorly to minimize the risk of AV block. A ventricular rate less than 120 bpm following isuprel (4 ug/min) or atropine (1 mg) infusion is considered a success.

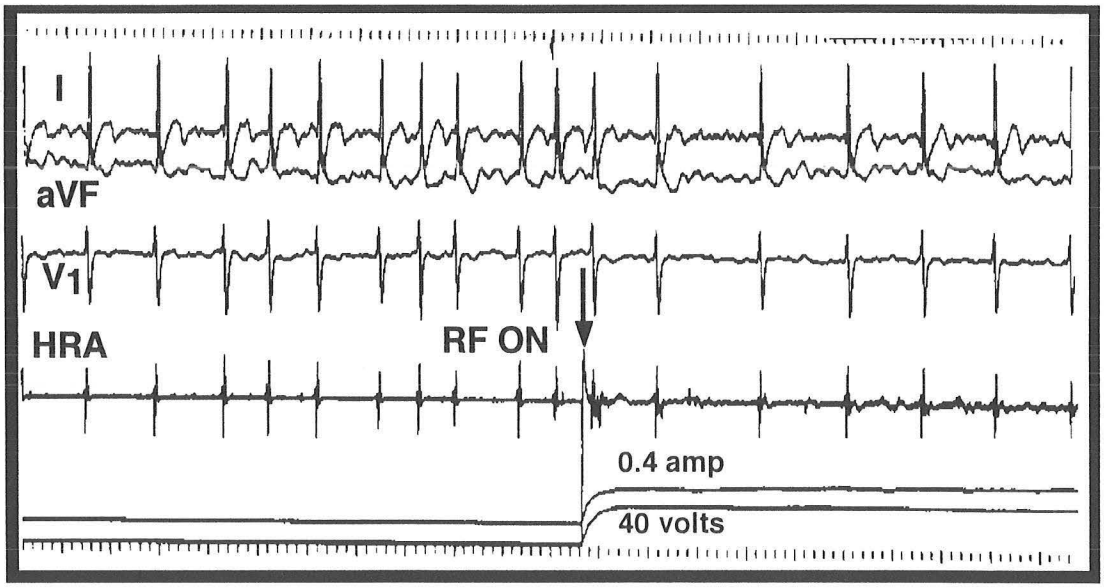


Figure 1 : Radiofrequency ablation applied in the posterior septal area resulting in slowing of the ventricular rate during atrial fibrillation.

Clinical Outcome After AV node modification

Patients with successful outcome show significant improvement in exercise tolerance, however, long term follow up data on large number of patients are not available. Even though the initial experience was promising, this procedure is not without its complications. The risks of AV nodal modification are significant. First, high degree AV block requiring permanent pacemaker implantation; AV block occurring during the first 72 hours have been reported in up to 16% of patients (19). Second, pause-dependent polymorphic VT has been reported in 3% of patients following this procedure (19); in our institution, sudden death occurred in one patient few weeks after slow pathway ablation. Third, symptom relief is not always achieved in these patients. In one study, almost 20%

of patients continued to have daily palpitations; this is not surprising since this procedure does not eliminate the irregular ventricular response that accompany atrial fibrillation.

Indications

With the incidence of polymorphic VT and sudden death noted above, the role of AV node modification in controlling ventricular rate in patients with atrial fibrillation remains unclear. Further studies are needed to elucidate the safety of this procedure. It is our opinion that this procedure should be avoided until its safety is demonstrated in prospective randomized studies.

III - Catheter Ablation Aimed at Restoring Normal Sinus Rhythm

It has long been documented that supraventricular tachycardia, atrial tachycardia, and atrial flutter may degenerate into atrial fibrillation. In these cases, radiofrequency catheter ablation aimed at the primary arrhythmia decreases the incidence of atrial fibrillation. However, in the majority of patients, atrial fibrillation is not the result of another tachyarrhtyhmia and the curative approach is more complicated. Indeed, the electrophysiologic approach to these patients is very much dictated by the postulated underlying mechanism.

The mechanism of atrial fibrillation seems to be heterogeneous. Today there appear to be at least two mechanisms: a rapidly firing focus degenerating into atrial fibrillation, and the multiple reentrant wavelets.

Mechanism 1 : Rapidly Firing Focus

It has long been demonstrated that a rapidly firing focus could produce atrial fibrillation. Cessation of pacing or ligation of the atrial segment where pacing was initiated lead to resumption of sinus rhythm in the remaining of the atria. Clinical counterparts of this model have been shown to exist.

Jais et al (22) first reported a focal source of atrial fibrillation in 9 patients that were treated with discreet radiofrequency ablation. All patients were free of structural heart disease and had frequent episodes of atrial fibrillation despite the use of at least 2 antiarrhythmic drugs. Atrial fibrillation was associated with runs of irregular atrial tachycardia or monomorphic atrial premature complexes. Electrophysiologic evaluation demonstrated that atrial fibrillation was the result of a rapidly firing focus located in the right atrium in 3 patients and in the left atrium in the remaining 6. Radiofrequency ablation targeted at these sites resulted in successful ablation. This study was the first to demonstrate that clinical atrial fibrillation in the absence of secondary arrhythmias can have a focal mechanism and that discrete radiofrequency ablation targeting these sites may result in atrial stabilization. Subsequently, Haissaguerre et al (23) studied 45 patients who had at least one episode of atrial fibrillation every two days and frequent isolated atrial etopic beats (more than 700 for 24 hrs). The spontaneous initiation of atrial fibrillation was mapped with the use of multielectrode catheters designed to record the earliest electrical activity preceding the onset of atrial fibrillation and associated with atrial etopic beats. Up to 4 foci per patient were identified and radiofrequency catheter ablation was applied at the earliest site of activation. These foci were predominantly located in the pulmonary veins, particularly in both the superior pulmonary veins. During a follow up period of 8 ± 6 months after ablation, 62% of the patients had no recurrence of atrial fibrillation. The authors concluded that the pulmonary veins were an important source of ectopic beats initiating frequent paroxysms of atrial fibrillation. These foci responded to treatment with radiofrequency ablation.

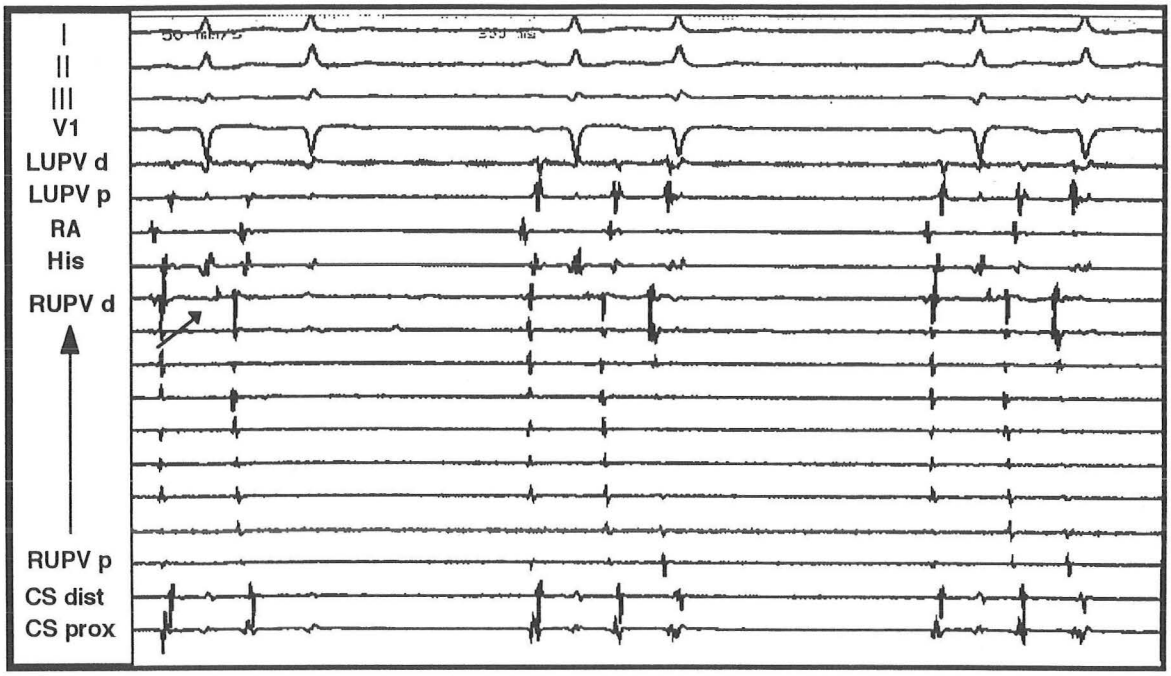


Figure 2 : Surface ECG showing atrial bigeminy. The earliest intracardiac electrogram preceding the ectopic P wave was recorded from the right upper pulmonary vein (RUPVd)

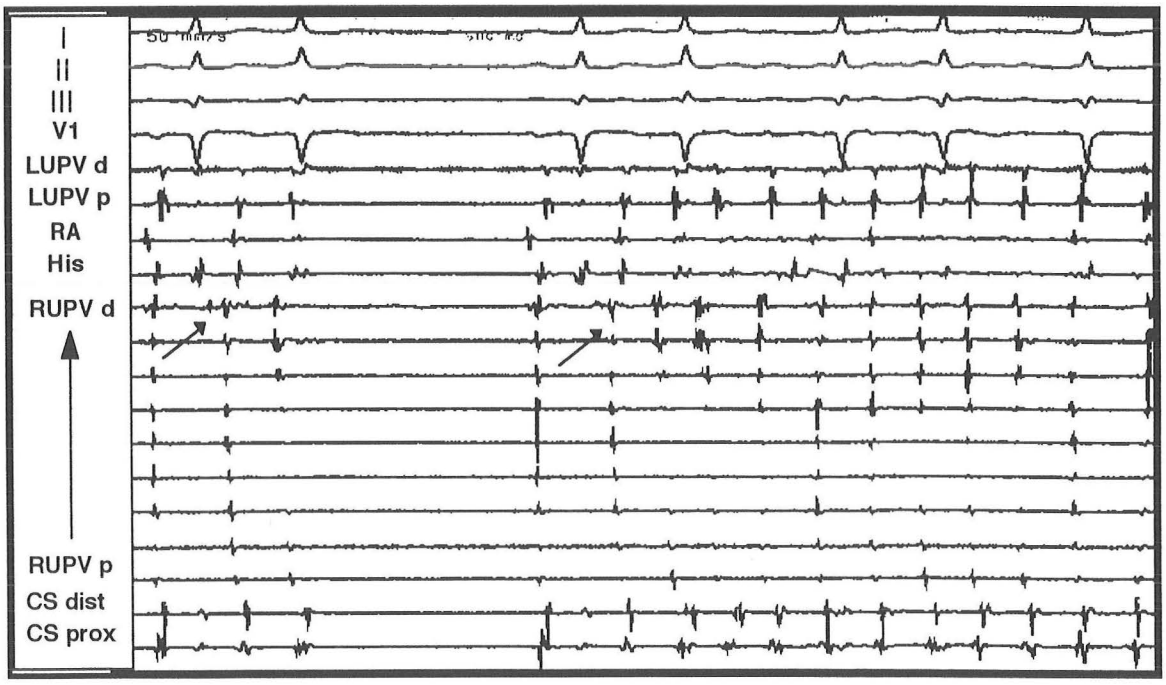


Figure 3 : Surface ECG showing an atrial premature beat preceding the onset of atrial fibrillation. Recordings from the right upper pulmonary vein (RUPVd) showed the earliest atrial activation. Radiofrequency ablation applied at this site resulted in elimination of atrial bigeminy and the paroxysms of atrial fibrillation.

The real question that remains unanswered is: "What proportion of patients have this form of atrial fibrillation?" In the early literature, these patients were reported to have a " normal" heart, frequent bursts of atrial tachycardia or atrial extrasystoles on 24 hour monitors and paroxysmal atrial fibrillation. In the most recent study, all patients had at least one episode of atrial fibrillation every 2 days and 39/45 patients had daily episodes. Therefore, if we were to apply these criteria, only a small fraction of patients with paroxysmal atrial fibrillation would qualify for this procedure. Moreover, once these patients are identified, successfully mapping of these foci can be quite challenging. As indicated in Haissagaire's paper, the firing of these atrial foci is quite unpredictable and is not necessarily responsive to programmed electrical stimulation or isoproternol infusion. In addition, should these extrasystoles degenerate to atrial fibrillation immediately at the onset of the study, mapping becomes difficult if not impossible. Some authors have found that creation of linear ablation lines in the right atrium and/or left atrium may render the arrhythmia pattern more organized, thus unmasking the single focus that triggered atrial fibrillation. Others are working on new mapping techniques that will allow detection of a focal origin of atrial fibrillation without the need for repeat cardioversion or the creation of linear lesions to organize the arrhythmia. Until these challenges are met, the diagnosis of patients with the so called " focal atrial fibrillation " and the successful ablation of the "trigger" site remain a challenge.

Mechanism 2 : Multiple wavelet hypothesis

In the late 1950s, Moe (24) performed a series of elegant experiments and proposed a model of atrial fibrillation suggesting that atrial fibrillation is the result or multiple wondering wavelets. Moe hypothesized that atrial fibrillation could only be induced when the refractory periods in some of the atrium were markedly different from the refractory periods in other nearby areas. This was later referred to as nonuniform recovery, nonuniform repolarization or dispersion of refractoriness. According to Moe, because of this uneven repolarization, an impulse is likely to propagate slower in areas with longer refractory periods and faster in areas with shorter refractory periods. He hypothesized that this would result in dissociation of the wave front into multiple reentrant circuits also called "wavelets". Moe's concepts remained theoretical until the mid 1970s when the technology allowed us to have simultaneous recordings of the atria during atrial fibrillation. These studies (25, 26) clearly demonstrated reentrant circuits during induced and spontaneous atrial fibrillation. Furthermore, the importance of the atrial orifices was pointed out during these experiments. It is now becoming increasingly recognized that the complexities of atrial anatomy, in addition to the heterogeneity of the electrophysiological properties, play an important role in defining the reentrant circuits. These anatomic obstacles include the pulmonary veins, the coronary sinus ostium, the eustachian valve, the cristae terminalis and the inferior and superior vena cava. Spatial dispersion in electrophysiological properties such as excitability and refractoriness may lead to local areas of conduction block and conduction delay, both contributing to the development of reentrant wavelets. In addition to the heterogeneity of both anatomic and electrophysiologic properties, atrial size is an important factor for the maintenance of atrial fibrillation. For atrial fibrillation to sustain, a minimum number of wandering wavelets must be present at any one time. Thus, the larger the atrium, the more likely that these reentrant circuits can coexist at a particular time. On the other hand, if the atria are debulked, or divided it into multiple segments, these wandering wavelets will extinguish, decrease in number, and will no longer allow atrial fibrillation to perpetuate. Several surgical techniques have been designed to ablate atrial fibrillation. These include the *left atrial isolation procedure*, the *corridor procedure*, and the *maze procedure.*

The *left atrial isolation procedure* (27) was designed to isolate the body of the left atrium from the remaining of the heart. This procedure was developed primarily for patients with automatic left atrial tachycardia in whom intraoperative mapping could not

localize the arrhythmogenic focus. The left atrial isolation procedure proved to be successful in some patients with atrial fibrillation as it confined the arrhythmia to the left atrium while the other chambers of the heart remained in normal sinus rhythm. Problems with this procedure are 1) the loss of synchrony between the left atrium and the left ventricle and 2) the persistent vulnerability of the patient to thromboembolism because of persistent left atrial fibrillation.

In 1985, Guiraudon and colleagues introduced the *corridor procedure* (28). In this procedure a strip of atrial tissue between the sinus node and the AV node is isolated from the rest of the atrial myocardium. The purpose is to allow the sinus node to drive the ventricles and to prevent the chaotic electrical activity of atrial fibrillation from reaching the AV node and the ventricles. However, with this procedure the atria may continue to fibrillate and the vulnerability to thromboembolism remains unchanged. Furthermore, because both atria are isolated from the ventricles, neither is synchronous with its respective ventricles postoperatively.

Because of all the limitations associated with the above techniques, Cox and associates designed the *maze procedure* (28-32). Cox demonstrated that by placing multiple strategically located transmural incisions on the atrium, macro-reentrant circuits responsible for atrial fibrillation could be reproducibly interrupted, thereby abolishing the ability of the atria to fibrillate. These incisions conform to the principle of a maze because they force the electrical activity of the atria to propagate from the sinus node through one circuitous route across the atria to the AV node, where it exits to the ventricles. This one true conduction route is also connected to multiple blind alleys of conduction that provide for the activation of all of the atrial myocardium, a prerequisite to restoring atrial contractile function postoperatively. Thus the electrical activity of the atria has one entrance (the sinus node), one exit (the AV node), one true conduction route between the two and multiple blind alleys of conduction. The purpose of the procedure was 1) to cure atrial fibrillation, 2) to restore AV synchrony, and 3) to restore atrial transport function. In light of the high success rate with the maze procedure, a considerable interest in the development of a transcatheter maze developed.

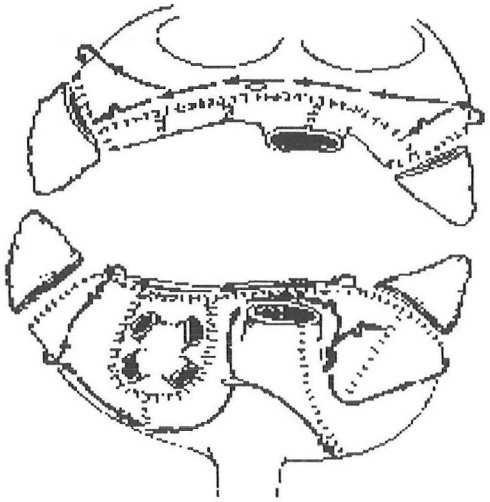


Figure 4 : The Maze procedure : The electrical activity of the atria has one entrance (the sinus node), one exit (the AV node), one true conduction route between the two, and multiple blind alleys of conduction, thereby conforming to the principle of a maze.

The Transcatheter Maze Procedure

Elvan and others (33) showed in animals that long lesions made with catheters may render atrial fibrillation noninducible. Mitchell et al (34) recently used multiple coiled electrode catheters to create temperature controlled radiofrequency lines at preselected atrial target sites in a dog model with atrial fibrillation. He showed that even though atrial fibrillation could no longer be initiated in 8 out of 9 dogs, the lesions themselves were incomplete and discontinuous. Two of 7 dogs had subsequently sustained atrial tachycardias. Although this study demonstrated that long linear lesions might reduce the susceptibility to sustain atrial fibrillation, it suggested the feasibility of achieving discontinuous long linear lesions. Discontinuous lesions may not only prove to be ineffective in preventing atrial fibrillation, but they are likely to be proarrhythmic in a manner similar to the surgical incisions created in patients with congenital heart disease.

In humans, Swartz and colleagues (35) first created linear lesions in both the right and left atrium in patients with chronic atrial fibrillation and showed progressive organization of atrial activity until sinus rhythm was restored. This procedure resulted in complications, however, including embolic events. The associated risks and the long fluoroscopy time made this technique unacceptable. Nevertheless his work suggested that a catheter based procedure for the cure of atrial fibrillation is achievable. In 1994 Haissaguerre (36) published a case of incessant episodes of atrial fibrillation treated with radiofrequency catheter ablation. In that case report, 3 linear lesions (2 longitudinal and 1 transverse that connected the two longitudinal lesions) were created in the right atrium. The final application interrupted atrial fibrillation that had been persistent for 55 minutes. The patient was no longer inducible in the laboratory and there were no complications. The same author subsequently published his experience with 45 patients with frequent symptomatic drug refractory episodes of paroxysmal AF (37). All 45 patients underwent right atrial ablation and 10 patients also underwent linear ablation in the left atrium. In patients with right sided lesions only, the success rate without drug therapy was around 13%. On the other hand, patients who underwent left sided ablations had a success rate of 40% without drug therapy. Success was defined as no more than one episode lasting 6 hours in a 3 month period. In addition to the above mentioned success rate, a significant number of patients showed improvement and responded to previously ineffective drugs. Another interesting finding of the study was that the creation of linear ablation lines in the atrium made the arrhythmia pattern more organized and allowed a single arrthymogenic focus to be observed in 12 patients. In these 12 patients, ablation of the single focus resulted in cure of the arrhythmia.

Despite these encouraging results, the transcatheter maze procedure remains investigational. Trombogenicity of left sided linear lesions, and their potential proarrhythmic effect, have to be seriously addressed before the procedure can be performed safely. Advances in ablation technology, including imaging techniques, energy type and catheter design, will certainly change the outcome of this procedure.

ATRIAL FLUTTER

Atrial flutter was first described by Jolly and Ritchie in 1911 (52) . The arrhythmia was reported to have typical saw-tooth configuration in the inferior leads. It was further clarified by Lewis in the 1920s (53) who nicely described the flutter wave morphology in the inferior leads (...the curve ascends sharply to a blunt summit and returns more gradually...the complexes are contiguous....as soon as one complex is complete the next starts....). The diagnosis of common atrial flutter continued to be based on Lewis' description and the presence of a very rapid rate (200 - 350 beats per minute). Based on vector analysis, Lewis was the first to show evidence of circus movement in human atrial

flutter. He showed a 360° revolution of the atrial electrical axis in each of the 3 orthogonal planes and concluded that the circus movement is occurring around the two cava, and the direction of movement is down the anterior right auricle and up the body of the left auricle. Several years later, vector analysis and use of intracavitary and esophageal electrograms in patients with common atrial flutter revealed that the direction of atrial activation was downward laterally and anteriorly and upward posteriorly. In 1970, Puech (54) classified flutter into several categories: 1) *typical* or the common form with a predominantly negative F-wave morphology in the inferior leads and a rate ranging between 250-320 beats per minute, 2) *atypical* form characterized by positive F-waves in the inferior leads, and 3) the impure flutter with a rate more than 320 beats per minute. Careful mapping using esophageal leads subsequently demonstrated that the sequence of activation during common atrial flutter is ascending on the septum and descending on the anterior lateral right atrial wall, thus propagating in a counterclockwise direction. In atypical flutter, the activation sequences was reversed to a clockwise direction with a high-low septal activation and low-high lateral wall activation. Therefore, the mechanisms of flutter was explained by a macroreentrant circuit localized to the right atrium with passive activation of the left atrium.

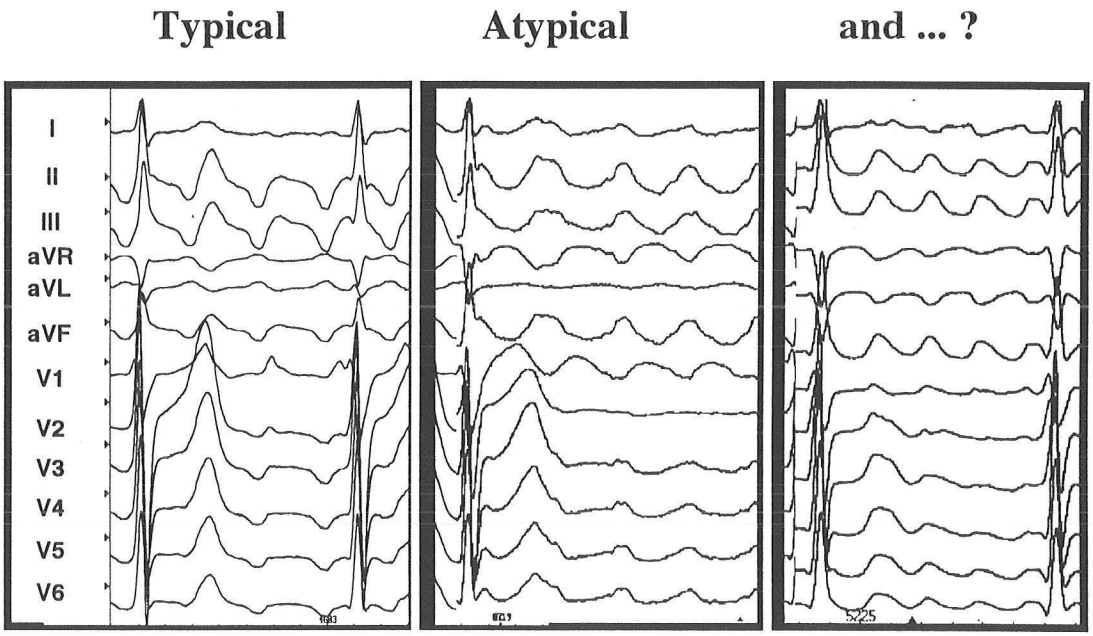


Figure 11 : *Left to right* : Typical atrial flutter with negative P waves in the inferior leads, Atypical atrial flutter with a positive P wave in the inferior leads and an Uncommon type of atrial flutter referred to as "true atypical flutter" or Type II flutter (see below). This uncommon type of flutter seem to have 1) heterogeneous EKG morphology, 2) cycle length shorter than that of flutter utillizing the commonly recognized circuit, 3) frequent transitions from and to atrial fibrillation and 4) are hard to entrain (adapted from *Kalman et* al, *JCE 1997*).

In 1977, Waldo et al (55) reported entrainment and interruption of atrial flutter with atrial pacing in patients who had undergone open heart surgery. Using entrainment technique, he subsequently demonstrated and localized the area of slow conduction in the reentrant circuit. Waldo's group subsequently divided atrial flutter into two types. *Type I* is slower, with a rate between 240 and 338 bpm. This type of flutter is always responsive to rapid atrial pacing with evidence of either entrainment, conversion to normal sinus rhythm, or conversion into atrial fibrillation. *Type II* atrial flutter is faster, with a rate

ranging between 340 to 433 bpm. This type of flutter is not influenced by rapid atrial pacing.

Type I Atrial Flutter

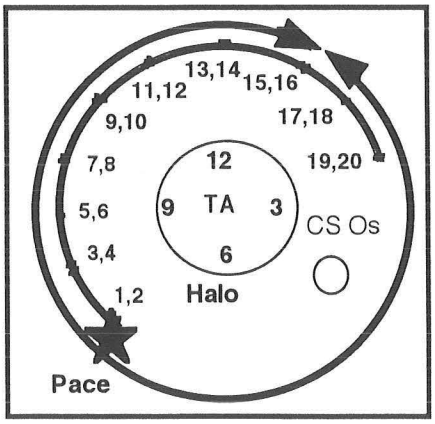
The importance of right atrial endocardial structures as barriers to conduction during atrial flutter was only recently demonstrated by Olgin et al (56). Previously, Cosio et al (57, 58) and others reported double potentials recorded along the lateral right atrium and that the area in the low right atrium between the tricuspid valve and the inferior cava is critical in atrial flutter. The exact anatomic correlate of these electrophysiologic events remained unresolved until recently when the use of intracardiac echocardiography and entrainment method helped define the anatomic features that delineate the boundaries of common flutter. The study by Olgin et al (56) demonstrated conclusively that the crista terminalis is the anatomic structure that bounds the flutter circuit posteriorly along the lateral right atrium and that the eustachian ridge is the posterior barrier to conduction in the inferior-posterior right atrium. Arribas et al have shown that the superior aspect of the circuit is activated along the anterior right atrium and not along the smooth right atrium below the superior vena cava orifice. Therefore it appears that during typical atrial flutter, the flutter wave activates the trabeculated right atrium in a cephalocaudal direction with the cristae terminalis acting as a posterior line of block, directing the impulse into a narrow isthmus of slow conduction between the eustachian ridge and the tricuspid annulus. The exit of this narrow isthmus is believed to be near the coronary sinus ostium. The wave then propagates either anterior or posterior to the coronary sinus ostium and proceeds to activate the interatrial septum in a caudocephalic fashion. The left atrium is activated passively. The question as to whether the conduction properties of the crista terminalis are different in patients with and those without history of atrial flutter was answered by Tai et al (59). The author showed that transverse conduction across the crista terminalis was significantly slower in patients with clinical atrial flutter. This finding suggests that poor transverse conduction across the crista terminalis may contribute to the occurrence and maintenance of clinical atrial flutter because it could effectively protect the reentrant circuit from short circuiting. Olgin et al (60) studied the mechanism of initiation of atrial flutter and clearly demonstrated that the direction of propagation of atrial flutter depended on the pacing site from which it originated. Pacing from the smooth right atrium induced counterclockwise flutter whereas pacing from the trabeculated right atrium induced clockwise flutter. The site of the unidirectional block during the initiation of either form of flutter was demonstrated to be in the low right atrium isthmus.

Radiofrequency ablation of atrial flutter

The above data suggest that successful ablation of atrial flutter can be achieved by applying radiofrequency catheter ablation between the tricuspid annulus and the eustachian ridge. Alternatively, ablative lesions can also be applied between the tricuspid annulus and the orifice of the inferior vena cava. Earlier studies using this anatomical approach clearly demonstrated flutter interruption with a high initial apparently successful rate. Long term recurrences, however, were reported to be as high as 44% (45, 61, 62). It was then clear, that flutter termination during RF ablation and the inability to induce flutter in the electrophysiology laboratory were not adequate end points. Potty et al (63) described a different end point during RF ablation of atrial flutter. In his study, evidence of conduction block at the IVC-TA isthmus was pursued despite noninducibility of atrial flutter. Conduction block was demonstrated in 11 out of 12 patients and during a follow-up of 9 ± 3 months, atrial flutter occurred only in 1 patient. This was the only patient that showed no conduction block after the procedure. The author suggested that demonstration of conduction block in the isthmus might be good predictor of long term success of atrial flutter ablation. Haissaguerre's group (64) also nicely demonstrated local bi-directional conduction block in the isthmus following successful atrial flutter ablation. Patients that came back with the recurrences always had evidence of recovery of conduction at least in

one direction in the isthmus. It was then clear that demonstration of bi-directional block is a better end point associated with a recurrence rate no more than 5-10%. Potty's group provided further insight into the various types of isthmus block (65). He nicely demonstrated that detailed multiple point low right atrial mapping is necessary to differentiate incomplete form complete isthmus block and that complete block is the best marker for long term success of atrial flutter ablation.

Unfortunately, despite the high success rate with atrial flutter ablation, a significant percentage of patients (up to 74%) may present with atrial fibrillation (61, 66). Roithinger et al (67) analyzed the relationship between atrial fibrillation and typical atrial flutter. The author studied the sequence of events present during conversion of atrial fibrillation to atrial flutter. He suggested that the anisotropic conduction properties in the right atrium and the anatomic barriers of the crista terminalis and eustachian ridge may play a role in the coalescence and annihilation of the multiple reentrant wavelets present during atrial fibrillation. As some of these wavelets organize, the presence of an excitable isthmus may allow a craniocaudal wavelet along the trabeculated right atrium to activate the septum and left atrium, which would ultimately lead to atrial flutter. Thus the presence of lines of conduction block in the right atrium and the excitability of the narrow isthmus between the inferior vena cava and the tricuspid annulus seem to be prerequisites for the organization of atrial fibrillation and the ultimate occurrence of atrial flutter. One can therefore argue that the creation of line of block in the IVC-TA isthmus would prevent the organization of many episodes of atrial fibrillation into atrial flutter. This may be an explanation for the occurrence of "de nouveau" atrial fibrillation in patients with previous history of atrial flutter who underwent successful radiofrequency ablation. The risk of atrial fibrillation following radiofrequency catheter ablation of atrial flutter was studied by several investigators. Philippon et al (61) have shown that 4 clinical variables were associated by univariate analysis with late occurrence of atrial fibrillation in patients who underwent catheter ablation for typical flutter. These were 1) the presence of structural heart disease, 2) history of atrial fibrillation before ablation of atrial flutter, 3) inducibility of sustained atrial fibrillation after ablation, and 4) a greater number of failed antiarrythmic drugs. By multivariate analysis, only the persistent inducibility of sustained atrial fibrillation predicted a later development of atrial fibrillation. Other authors have also shown that the presence of left ventricular dysfunction (EF < 50%) and history of spontaneous atrial fibrillation were the best predictors of future atrial fibrillation following RF ablation of atrial flutter (66). The presence of both these characteristics identified a high risk group with a 74% occurrence of atrial fibrillation. Patients with only 1 of these characteristics were at intermediate risk (20%) and those with neither characteristics were at lowest risk (10%).



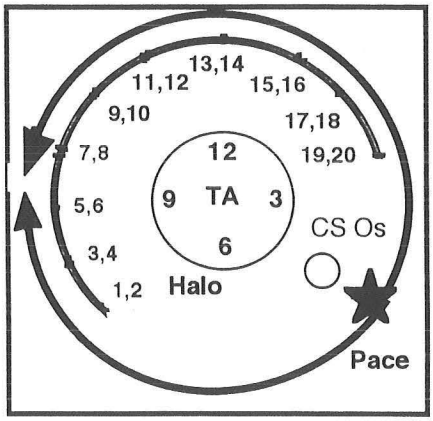


Figure 12 : Before RF ablation : *Left*, Pacing during sinus rhythm from the left lower RA results in 2 wavelets with opposite directions colliding on the septum. *Right* : Pacing duirng sinus rhythm, from the coronary sinus ostium results in 2 wavelets colliding on the lateral wall.

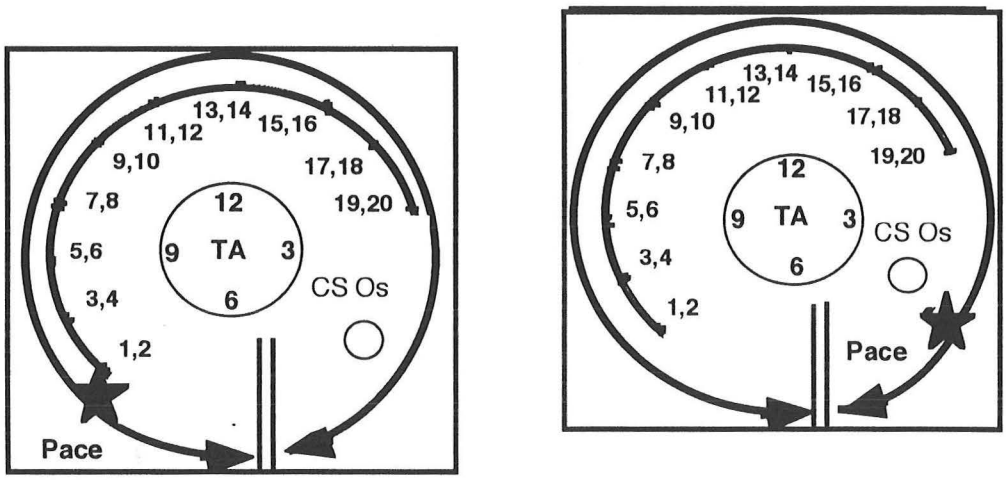


Figure 13 : After RF ablation and the creation of a line of block at the IVC-TA junction : *Left,* pacing from the low lateral RA results in activation of the tricuspid annulus in a clockwise direction. Right, pacing from the coronary sinus ostium results in counterclockwise activation of the tricuspid annulus.

Type II or "True Atypical" Atrial Flutter

While not as commonly encountered, other forms of atrial flutter are clinically recognized. These were referred to as *type II* flutter by Waldo's classification and others have called it *rare* or *uncommon* type of atrial flutter (45, 68, 69) . These type of atrial flutter seem to have 1) heterogeneous EKG morphology, 2) cycle length shorter than that of flutter utilizing the commonly recognized circuit, 3) frequent transitions from and to atrial fibrillation and 4) are hard to entrain, but when entrainment from the low right atrial isthmus is accomplished, it usually demonstrates surface fusion. On the basis of these findings, Kalman et al (69) report new classification for atrial flutter. He proposed to call typical atrial flutter any time the rotation is around the tricuspid annulus with the crista terminalis and eustachian ridge acting as posterior barriers, regardless of the direction of the rotation whether it's clockwise or counterclockwise. Any flutter that does not involve the typical flutter circuit would then be called true atypcial flutter. According to this classification, true atypical flutter shares the characteristics of type II atrial flutter reported by Waldo (55) i. e. inability to entrain, and the presence of a variable cycle length overlapping with typical flutter. In an era where radiofrequency ablation is curative for many atrial arrhythmias, it may be most useful to classify atrial arrhythmias according to the location of the circuit rather than the tachycardia rate.

The approach to "true atypical flutter" is difficult as the tachycardia circuit remains unidentified. These arrhythmias are unlikely to share one common circuit and therefore successful ablation requires extensive mapping and identification of the critical isthmus. The success rate with these arrhythmias is poor and awaits future studies involving a large number of cases.

ATRIAL TACHYCARDIA

The mechanisms of atrial tachycardia include enhanced automaticity, triggered activity, and reentry. Automaticity can be easily differentiated from reentry or triggered activity with the use of programmed electrical stimulation. By definition, automatic rhythms are not induced or terminated with programmed electrical stimulation, but are rather catecholamines sensitive. Macro-reentry around anatomical or functional obstacles is usually easily recognized, but the differentiation between a micro-reentrant mechanism and triggered activity can be more difficult . Micro-reentry can give rise to a focal activation pattern similar to what is seen with triggered activity. This is especially true if the inter-electrode distance of the recording catheter is large with respect to the diameter of the reentrant circuit. The response to short-lived rapid pacing can be useful since rapid pacing results in an increase in intracellular calcium and an increase in triggered activity. This tool is of limited use because reentrant arrhythmias may accelerate in response to rapid pacing. Regardless of the mechanism, tachycardias originating from a single focus can usually be ablated by targeting the earliest site of activation.

In the sections below, I will first discuss atrial tachycardias with focal activation pattern; in the second section I will discuss atrial tachycardia secondary to a macro-reentrant circuit.

I- Focal Atrial Tachycardia

Mechanism

Atrial tachycardia with a focal pattern, also referred to as "focal" or "ectopic" atrial tachycardia, can occur at all ages (38). In pediatric patients, most of the atrial tachycardias are automatic in nature. Furthermore, most of them are nonparoxysmal in onset and are located in the right atrium. Some of these tachycardias, however, are located near the orifice of the pulmonary veins and near the coronary sinus ostium. The presence of automatic tissues in the atrium might explains the predominance of an automatic mechanism in young patients. In the older patients, atrial tachycardias have more of a paroxysmal onset and are more likely to be nonautomatic. The decrease in automaticity with age may explain the predominance of nonautonomic mechanisms in the older patients. At all ages, the presence of cardiac disease is a predictor of multiple atrial tachycardia sites. This can be easily explained by the fact that cardiac diseases are frequently associated with extensive pathologic changes in the atria that can be arrythmogenic.

Radiofrequency Ablation

Atrial tachycardia, particularly when it occurs in young patients and is mediated by an automatic mechanism, may be incessant and lead to tachycardia related cardiomyopathy. The responses to drug therapy is frequently suboptimal and surgical therapy has not been uniformly successful. The conceptual foundation for catheter ablation of atrial tachycardia arose from the success of surgical ablative therapy. The largest initial experience with surgical ablative therapy was acquired in pediatric patients. Early reports from Gillette and coworkers (39, 40) described 16 patients with drug refractory atrial tachycardia, 13 of whom were successfully treated with map guided catheter ablation and also total atrial excision. The tachycardia sites in these 16 patients were right atrial in 10 and near the pulmonary vein ostia in the remaining 6. In patients with successful surgery, complete normalization of left ventricular function was observed in 9 out of 10 patients with suspected tachycardia related cardiomyopathy.

The initial attempts at radiofrequency catheter ablation for atrial tachycardia targeted the AV junction rather than the tachycardia focus itself. Successful catheter ablation of the natural tachycardia focus was first reported by Silka and coworkers (41) in a 10 year old patient. A 50 J DC shock terminated this incessant rhythm and patient remained in normal

sinus rhythm over the 10 month period of follow up. With subsequent recognition of the potential complications of DC catheter ablation, catheter based radiofrequency ablation was soon implemented and was proven to be safer and less arrythmogenic. Walsh and coworkers (42) produced the first series of patients with atrial tachycardia treated with radiofrequency catheter ablation. The procedure was successful in 11 of 12 patients and there were no late recurrences except in one patient. The selection of RF lesion sites was based on the earliest activation time during atrial tachycardia (also called *activation mapping*), and the response to "test burns". The range of activation times at successful ablation sites was 20-60 milliseconds (median 42) before the onset of the surface P wave. Another technique used for the ablation of atrial tachycardias is called *pace mapping*. With this technique, atrial pacing is attempted at different sites and the paced P-wave morphology is compared to the P wave morphology during tachycardia. This technique may be extended to what is called paced activation sequence mapping, introduced by Tracy and coworkers (43), where intracardiac atrial activation sequence during pacing is also compared to the sequence recorded during atrial tachycardia. Another innovation for atrial mapping was introduced by Kay et al (44), who used a double catheter technique in a number of patients with atrial tachycardia. With this technique, two catheters are sequentially moved with each becoming the reference for the other until the earliest activation time during tachycardia is found. Lesh et al, (45) among other authors, recorded fractionated electrograms during tachycardia at the successful ablation sites. It is now common practice to use the activation mapping technique with a search for fractionated electrograms in patients with ectopic atrial tachycardias. The success rate using the above methods is around 90%.

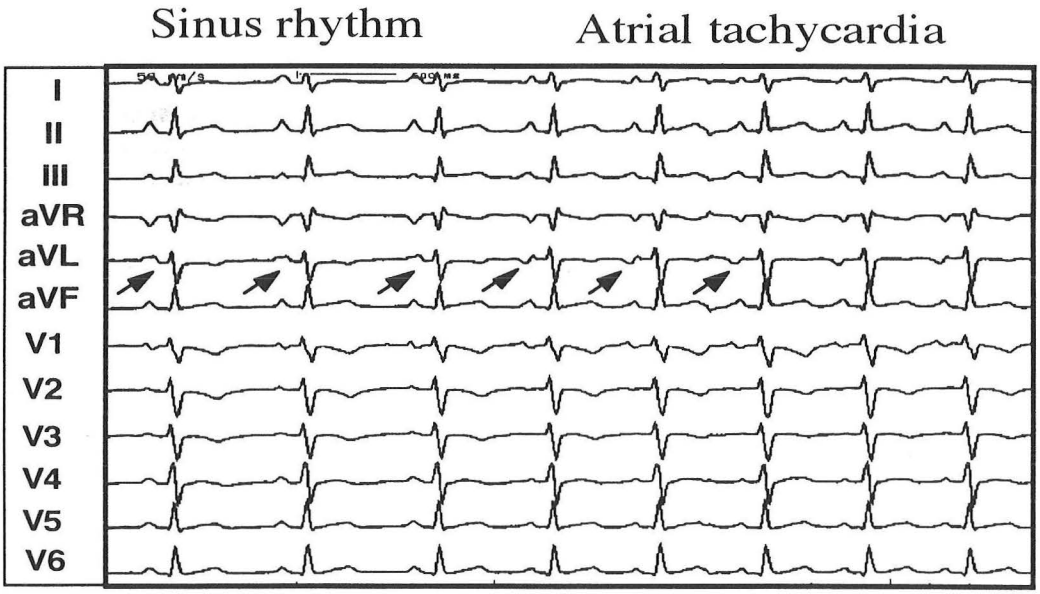


Figure 5 : Note the change in P wave morphology in lead aVL at the tachycardia onset.

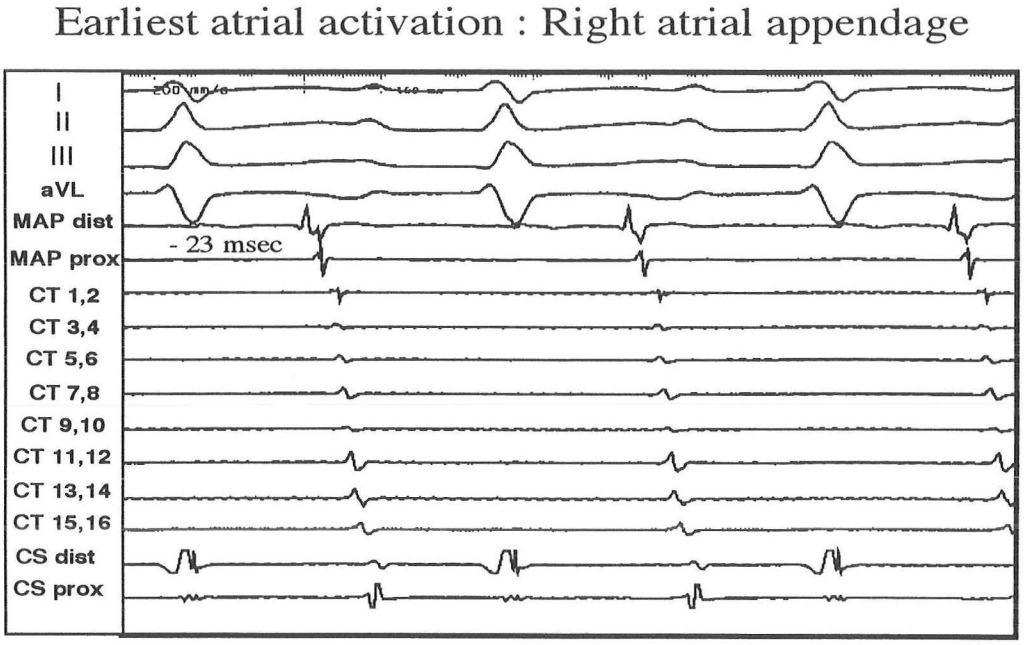


Figure 6 : Endocardial atrial mapping during tachycardia showed the earliest atrial activation on the MAP catheter located in the right atrial appendage (-23 msec before the P wave onset)

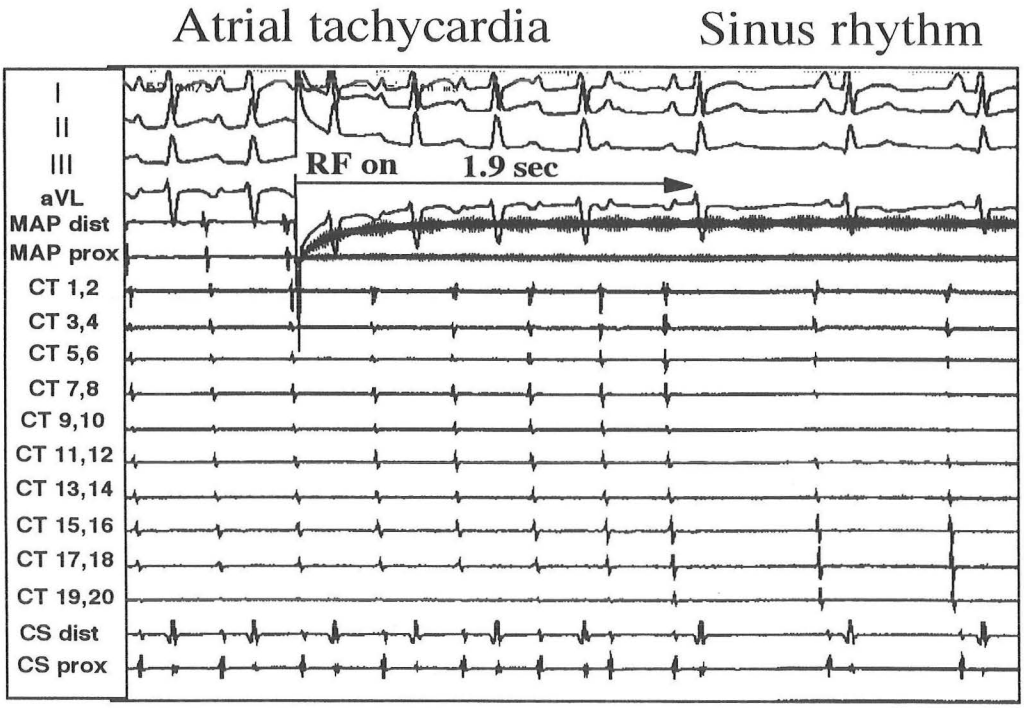


Figure 7 : Radiofrequency ablation applied at the site of earliest atrial activation (right atrial appendage) resulted in tachycardia termination.

Several authors have consistently found that most atrial tachycardias originate from the right atrium, in specific the area involving the cristae terminalis and the Koch' s triangle. Using intracardiac electrocardiography, Kalman et al (46) demonstrated that

approximately 2/3 of focal right atrial tachycardias occurring in the absence of structural heart disease arose along the cristae terminalis. Other common sites of focal atrial tachycardias include the pulmonary vein ostia. Recognition of this common distribution has facilitated mapping and ablation of these tachycardias. There are exceptions, where atrial tachycardias originate from atypical sites outside the cristae terminalis and the pulmonary veins. Mallvarapu et al (47) reported cases of left atrial tachycardias localized to the lateral left atrium adjacent to the mitral annulus. The P wave morphology during atrial tachycardia was negative in either leads aVL or lateral precordial leads.

II - Macro-reentrant Atrial Tachycardia

Mechanism

Macroreentrant tachycardias, also referred to as *intra atrial reentrant tachycardias* (IART), usually occur in patients with structural heart disease. The substrate for IARTs may be naturally-occurring anatomic barriers with areas of functional block, or may be the result of surgically created lines of conduction block. The approach to these arrhythmias is more difficult, as the success of radiofrequency ablation requires the identification of a critical isthmus of slow conduction. Identification of this site is usually accomplished with entrainment mapping and careful analysis of the paced P wave morphology and the return cycle length at the end of pacing (45, 48-50). In patients who have previously undergone corrective surgical procedures for congenital heart disease, IART may be a life threatening complication. Management of IART is especially problematic in these patients because the arrhythmia is often associated with a marginal hemodynamic status which may limit the choice of antiarrhythmic drug therapy. Furthermore, antiarrhythmic drugs may aggravate sinus node dysfunction and/or cause worsening of ventricular function. The factors responsible for the maintenance of IART have not clearly been identified, but several possibilities exist, including; 1) abnormal atrial anatomy associated with the congenital lesion itself, 2) atrial scarring caused by the surgical suture lines and pericardial inflammation, and 3) the presence of abnormal atrial wall stress in patients with abnormal hemodynamic states.

Radiofrequency ablation

It has been hypothesized and shown that successful ablations of these arrhythmias could be performed by targeting a protected isthmus of conduction bounded by natural and surgically created barriers, and that entrainment techniques could be used to identify these zones. Triedman et al (50) published his experience with 10 consecutive patients referred for treatment of recurrent IART after surgery for congenital heart disease. A total of 30 distinct IART circuits were defined during electrophysiologic evaluation. RF ablation was attempted in 22 of these circuits, targeting the presumed exit points from zones of slow conduction. RF ablation applied at these sites resulted in tachycardia termination in 77% of these cases. Concealed entrainment, however, was only demonstrated in 8 out of 22 tachycardias. During follow up, in 8 of 10 patients where at least one IART circuit was successfully ablated, 4 were free of clinical tachycardia and 3 showed improvement. There were no complications. Cruz et al (51) reported their experience with IART related to an atriotomy scar in 4 patients previously submitted to cardiac surgery for congenital heart disease. Radiofrequency energy was successfully delivered between the atriotomy scar and the IVC. After a 3 month follow up, none of these patients had recurrences. Kalman et al (49) described their experience in 18 patients with 26 IARTs complicating surgery for congenital heart disease (9 atrial septal defect repair, 4 Fontan, 2 Mustard, 2 Senning, and 1 Rastelli procedure). Entrainment mapping was done and RF ablation was only applied at sites that demonstrated concealed fusion with a post-pacing interval minus the tachycardia cycle length and the stimulant to P wave minus the activation time were less than 30 milliseconds. Anatomic barriers surrounding the critical isthmus of conduction were identified on anatomic grounds by the presence of split potentials signifying a line of block

or areas of electrical silence. With RF ablation applied at a protected isthmus of conduction bounded by natural and surgically created barriers, success was achieved in 15 patients with 21 arrhythmias. The author concluded that successful ablation of IART complicating surgery for congenital heart disease may be achieved by creating a line of block in a critical isthmus of conduction bounded by anatomic barriers.

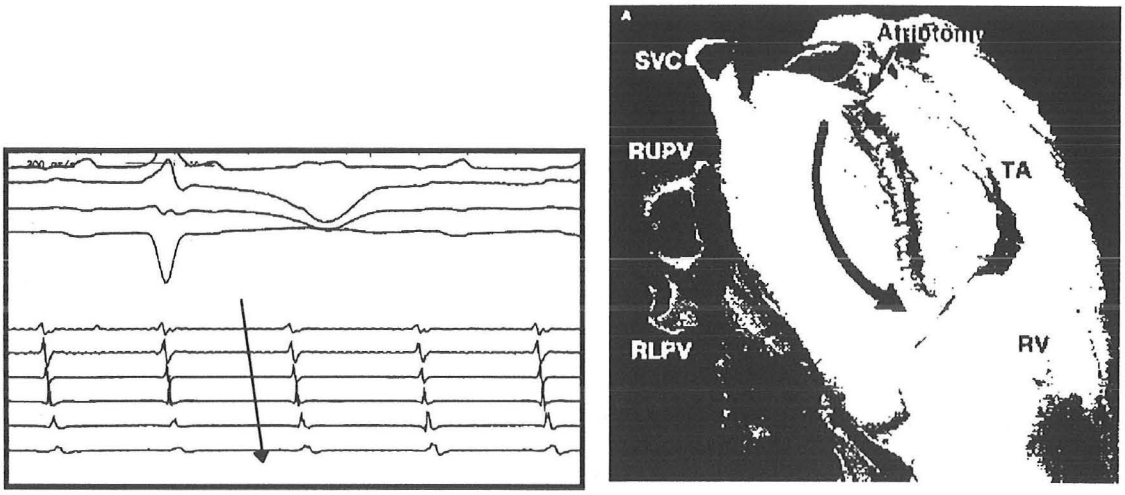


Figure 8 : Intracardiac atrial recordings from a catheter placed posterior to the atrial scar during atrial tachycardia. Endocardial activation indicates a high-low activation.

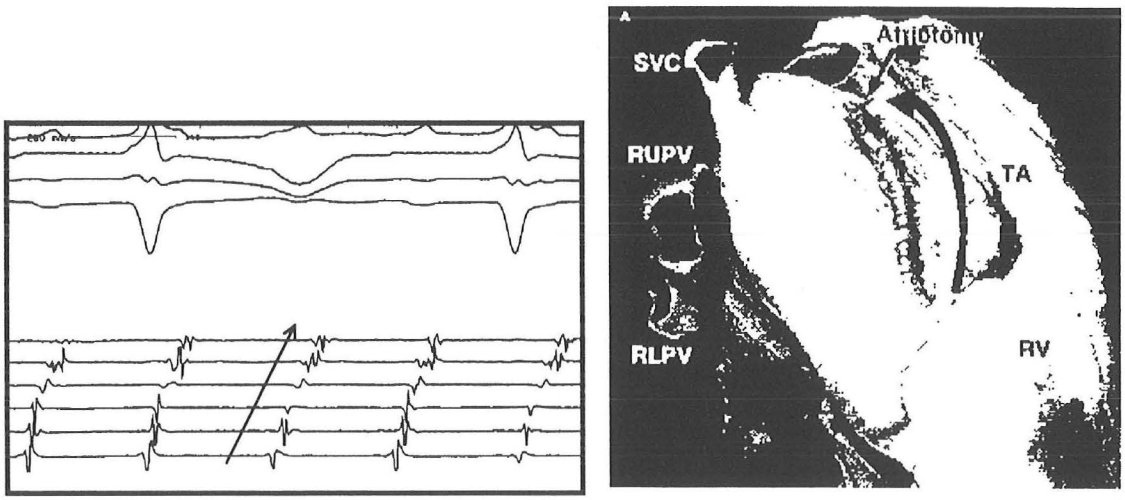


Figure 9 : Intracardiac atrial recordings from a catheter placed anterior to the atrial scar during atrial tachycardia. Endocardial activation indicates a low-high activation. Figures 8 and 9 suggest that the tachycardia circuit revolves around the surgical scar in a counterclockwise direction.

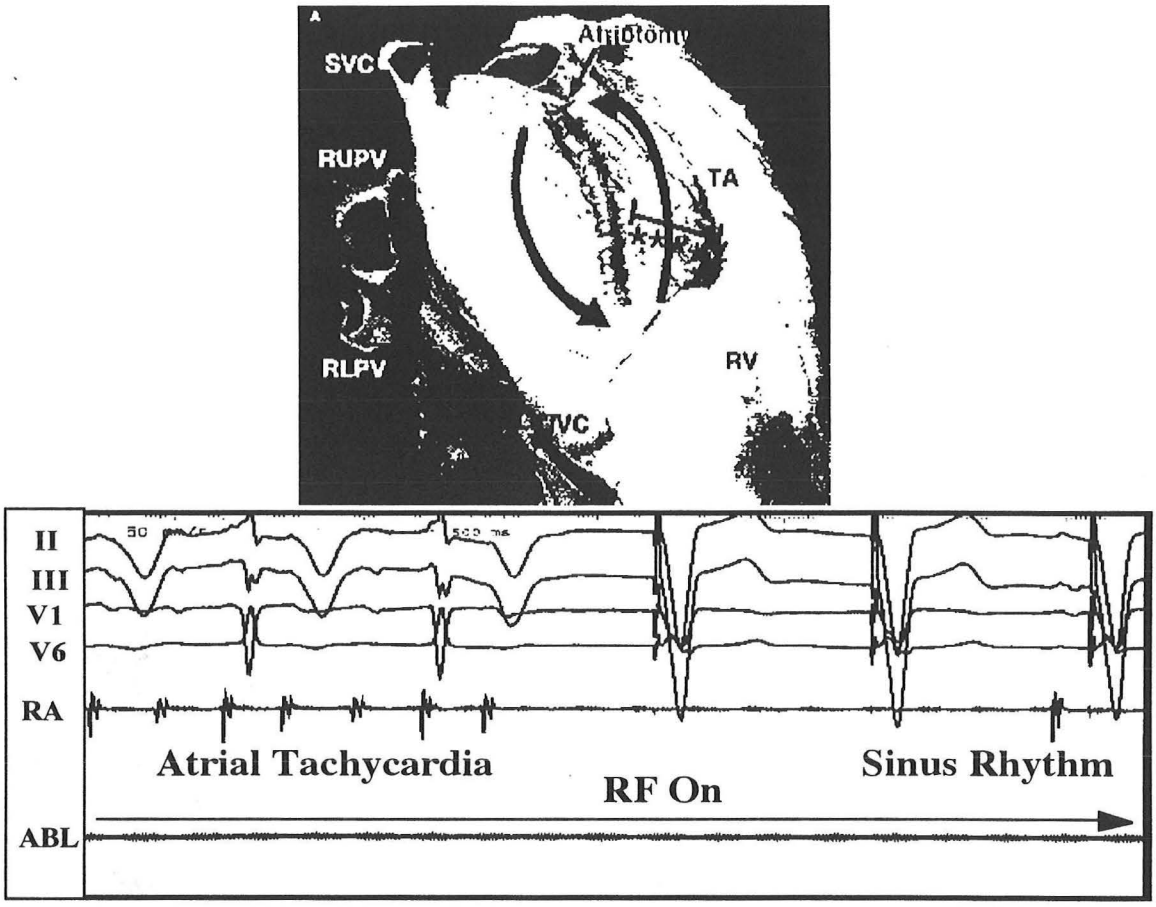


Figure 10 : Successful ablation of this "incisional" tachycardia was achieved by creating a line of block between the surgical scar and the tricuspid annulus.

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