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1. Introduction

Atrial Fibrillation (AF) is characterised by rapid and disorganised depolarisation of the atria resulting in uncoordinated atrial contraction. It is the most common cardiac arrhythmia encountered in clinical practice increasing in prevalence with age and the presence of heart disease. It is estimated that it affects 2.5 million people in the United States and 4.5 million in the European Union. The actual incidence of the arrhythmia may be much higher owing to undetected or asymptomatic patients within the population. Since it is an age-associated arrhythmia its incidence has steadily risen over the past decade and will continue to increase due to a growing population of the elderly in the western world. Contributing risk factors include hypertension, diabetes, coronary artery disease, valvular disease, electrolyte imbalance. Patients with atrial fibrillation have a higher risk for stroke, heart failure and death. Given the significant morbidity and mortality associated with AF and its associated economic burden, it is not surprising that there has been great interest in developing effective treatments for it.

Atrial fibrillation is a supraventricular arrhythmia where uncoordinated rapid atrial contractions produce an irregular ventricular response. The atria may discharge between 300-600 beats per minute but not all these impulses are conducted by the AV node. Ventricular response can be between 100-160 beats per minute in untreated patients with normal AV conduction. This chaotic rhythm disrupts normal movement of blood through the heart reducing cardiac output and increasing the risk for thromboembolism such as stroke as a consequence of stasis of blood in the atria.

The serious morbidity and mortality associated with AF are attributed to three detrimental consequences:

- Palpitations causing significant patient discomfort and anxiety
- Loss of the coordinated atrioventricular contraction and “atrial kick” compromising cardiac haemodynamics and can lead to varying levels of ventricular dysfunction
- Stasis and pooling of stagnant blood that can lead to intra-atrial thrombus formation and resulting in increased risk of thromboembolism

Medical therapy remains the most common and first line treatment for patients with AF but frequently ineffective at restoring sinus rhythm leaving the patient susceptible to cardiovascular morbidity and mortality. Therefore, the goal of pharmacological therapy is often shifted from rhythm control (maintenance of sinus rhythm) to rate control (slowing the ventricular response to AF).

The Atrial Fibrillation Follow-Up Investigation of Rhythm Management (AFFIRM) trial demonstrated no survival benefit of rhythm control strategy over a rate control strategy.
Although rate control slows the ventricular response to AF, preventing tachycardia induced cardiomyopathy, it does not reduce the rates of thromboembolism or congestive heart failure. The atria are still in fibrillation and there is loss of the atrial “kick” resulting in worsening of congestive heart failure and requires indefinite anticoagulation with warfarin to counter the risk for developing thromboembolism. Although warfarin reduces the annual risk of ischaemic stroke and systemic thromboembolism to approximately 2%, its use is associated with significant morbidity with a 2% annual risk of drug associated haemorrhage.\textsuperscript{8,9,10} Anticoagulation may reduce lifetime relative risk of stroke by 60% but does not eliminate it.\textsuperscript{11} Although the results of the AFFIRM trial demonstrated no long-term benefit of rhythm versus rate control, some patients may still have advantages of being in sinus rhythm. These include freedom from palpitations, increased exercise tolerance and prevention of atrial remodelling.\textsuperscript{12,13} Ineffective results with rate and rhythm control strategies have helped to encourage the development of new interventional catheter and surgical treatments. Although catheter ablation is an established therapeutic option, the highest success rates are typically seen in patients with paroxysmal AF and minimal structural heart disease.\textsuperscript{14} Therefore it is limited to a small number of patients treated by highly skilled electrophysiologists. On the other hand, almost all cardiac surgeons are capable of performing surgical ablation of AF.

2. Classification of atrial fibrillation

The terms used to classify and describe AF categorise it as paroxysmal, persistent, longstanding or permanent (figure 1). Paroxysmal AF is defined as at least two episodes of AF that terminate spontaneously within 7 days. If this is sustained beyond 7 days it is described as persistent. Another category of persistent AF includes longstanding AF, defined by duration of greater than one year. This usually leads to permanent atrial fibrillation in whom cardioversion has either failed or abandoned.\textsuperscript{15}

![Fig. 1. Classification of Atrial Fibrillation](https://example.com/fig1.png)
3. Electrophysiology of AF

In recent years there has been considerable progress in understanding the pathophysiology of AF. It was initially thought that AF was caused by random, multiple wavelets generated throughout the atria that propagated new wavelets to cause re-entry mechanism. In the 1980s, several procedures were developed aimed at curing atrial fibrillation. However, most of these were abandoned due to their inability to address all three detrimental sequelae of atrial fibrillation. It was discovered that electrical impulses were incapable of crossing areas of the heart that had been incised and sutured.16,17

This fostered the development of the Maze procedure, designed and first reported by Cox and colleagues in which multiple incisions were made and sutured in a manner that blocked the aberrant impulses within the atrium (figure 2). These incisions directed the SA node impulse to initiate and propagate throughout the atria to the AV node along a specified route created by the Maze incisional lesions18. However, recently studies have demonstrated that the initiation of paroxysmal but not necessarily persistent or permanent AF are generated by electrical waves from focal sources, particularly near the pulmonary veins. Haissaguerre et al.19 mapped the triggers of paroxysmal AF to originate around the orifices of the pulmonary veins in 94% of patients with atrial diameters of less than 5cm. These findings have directed the development of more precisely targeted procedures that can be performed less invasively with less destruction of normal tissue.

![Fig. 2. The pathway taken in the atria by the aberrant impulses in atrial fibrillation (arrows). The thick lines are the ablation lesions used in the Maze procedure. Key- SAN: sino-atrial node, AVN: atrio-ventricular node, RAA: right atrial appendage, LAA: left atrial appendage, SVC: superior vena cava, IVC: inferior vena cava, PVs: pulmonary veins.](www.intechopen.com)
A key concept in understanding the development of persistent atrial fibrillation is that the atria undergo electrical remodelling. Atrial electrical remodelling results in shortening of the atrial refractory period, myocyte calcium overload, decreased conduction velocity, dispersion of conduction and increased sensitivity to catecholamines. This phenomenon may be reversed after maintenance of sinus rhythm. The more a patient experiences AF, the more susceptible they are to continue fibrillating as a result of remodelling. Therefore AF can become a self-sustaining arrhythmia once atrial remodelling has occurred. Micro-reentrant triggers are then no longer necessary and limited to pulmonary venous impulse-triggering sites. Therefore, treatment for persistent or longstanding AF directed at the pulmonary veins only is likely to be unsuccessful.

4. Non pharmacological therapy

When pharmacological intervention is unsuccessful or contraindicated in patients, non-pharmacological therapy may be attempted. These include synchronised electrical cardioversion, catheter based ablation techniques or surgical intervention. Direct-current cardioversion differs from defibrillation whereby the shock is synchronised to the R wave in the patient’s ECG. The patient must be adequately anticoagulated during electrical cardioversion in order to prevent disruption of a pre-existing intra cardiac thrombus which will cause it to embolize to the brain or systemic circulation. The risks of electrical cardioversion include hypotension, bradycardia, pulmonary oedema, systemic embolization, skin burns and ventricular arrhythmias. Risks of emboli range from 0.5% to 3% and is further multiplied in patients who experience recurrence and treated with serial cardioversions.

Catheter based ablation techniques were initially developed following Cox’s pioneering work with the Maze procedure. These techniques were further influenced by research that demonstrated ectopic foci surrounding pulmonary veins. Isolation of the pulmonary veins remains the cornerstone of most AF catheter ablation procedures. Following heparinization, a percutaneous catheter placed into the femoral vein is advanced to the right atrium. The left atrium is accessed via an interatrial septal puncture. Lesions are created around the pulmonary veins using cryoenergy or radio-frequency energy. These techniques have shown a higher success in treating patients with paroxysmal AF compared to those with enlarged left atrium and persistent or permanent AF. Complications of catheter ablation include cardiac tamponade, atrioesophageal fistula and stroke.

5. Surgical treatment

Surgery is indicated in patients undergoing elective cardiac surgery that have symptomatic AF or those with asymptomatic AF with low operative risk. It is advised that patients with persistent or permanent AF scheduled for elective cardiac surgery should be considered for concomitant ablation procedure that may increase both short-term and long-term freedom from AF, in addition to lowering the risk of thromboembolism and improving long term survival and cardiac function. Surgery for lone AF may be considered in certain circumstances where patients have failed to respond to catheter ablation or in whom catheter ablation is contraindicated such as a mural thrombus. Patients that develop tachycardia-induced cardiomyopathy will also benefit from surgery. It results in atrial or ventricular dysfunction as a result of increased heart rates in an otherwise structurally normal heart. If left untreated it can lead to heart failure and is reversible if sinus rhythm is
restored. In addition patients in whom anticoagulation is contraindicated may also be suitable candidates that may benefit from surgical intervention following failure of catheter based ablation. Patients that continue to experience thromboembolic events despite adequate anticoagulation may also benefit from surgery. Several procedures were developed in the 1980s aimed at finding a cure to atrial fibrillation. However, most of these procedures were subsequently abandoned due to their inability to address all three of the detrimental sequelae of AF. Early attempts at surgical treatment of AF attempted to isolate and confine AF to a specific region of the atria and thereby stopping it from propagating its effects upon the ventricles. The left atrial isolation procedure developed by Williams and colleagues was successful in confining AF to the left atrium and thus restoring sinus rhythm to the rest of the heart. It also removed two of the 3 detrimental consequences attributed to AF namely, irregular heart rate and compromised haemodynamics. The latter was achieved because restoring sinus rhythm on the right side permitted a normal right-sided cardiac output that was delivered to the left side of the heart. The left ventricle responded to the normal cardiac output on the right side by delivering a normal cardiac output. Since the left atrium continued to fibrillate this procedure did not reduce the risk of thromboembolism. The ‘Corridor’ procedure was introduced in 1985 that isolated a strip of atrium that contained both the SA node and AV node from the rest of the atria to create a continuous pathway (corridor) directing the impulses from the SA node to the AV node to maintain sinus rhythm. Since parts of the right and left atrial were free to fibrillate it did not eliminate the risk of thromboembolism and nor did it restore atrioventricular synchrony.

Cox and colleagues described a series of experiments that attempted to cure AF in dogs. A single incision across both atria successfully prevented AF and atrial flutter. Further investigations by Cox and colleagues led to the Cox-Maze procedure in 1987. The procedure itself was based upon a cut and sew technique whereby multiple incisions were made in the atria. This created lines of scar that interrupted the conduction routes of the most common re-entrant circuits, thus preventing AF or atrial flutter by directing the sinus node impulses along a specified route. It was based around the concept of a maze and as a result was called the Cox-Maze procedure. In contrast to the previous surgical techniques, this was the first that addressed all three sequelae of AF and restored sinus rhythm, AV synchrony and thus significantly reducing the risk of thromboembolism and stroke. The original procedure, known as Cox-Maze I was complicated with a high incidence of heart block requiring pacemaker implantation. It also resulted in the late incidence of two problems. Firstly it led to the frequent inability of patients to generate an appropriate sinus tachycardia and secondly left atrial dysfunction. This was modified to the Cox Maze II procedure which despite decreased incidence of conduction system injury was technically difficult. It was therefore modified to the Cox-Maze III procedure that was associated with a higher incidence of sinus rhythm and improved long-term sinus node function and atrial transport function. In this procedure several dead-end “alleyways “create a maze-like pathway and permit the depolarization of all the atrial tissue. The Cox Maze III procedure can be performed both through median sternotomy as well as a partial lower sternotomy. The patient is fully heparinized and the surgeon cannulates the patient for cardiopulmonary bypass after dividing the sternum. Bicaval cannulation is achieved. The right atrial appendage is excised and a series of incisions are made to the right atrium including a cryolesion. The aorta is occluded preparing for the left atrial portions of the operation. Cold blood potassium cardioplegia is administered via retrograde perfusion of the coronary
sinus. The left atrium is exposed by an incision posterior to the interatrial groove close to the orifices of the right pulmonary veins. A number of incisions are made across the left atrium and the left atrial appendage is excised at its base. The incisions to the left atrium interatrial septum and right atrium are closed. Despite its complexity, the Cox-Maze III procedure became the gold standard for surgical treatment of AF. It has been performed in hundreds of patients and proven to be highly successful in ablating any form of AF irrespective of whether patients had concomitant heart disease or not. Although it adds to cardiopulmonary bypass and cardiac arrest time it does not increase the operative mortality. Sinus rhythm was reported in 97% at late follow-up and it was equally effective in patients with lone AF as those undergoing concomitant cardiac surgery. Similar results were reproduced by other institutions across the world.

Early postoperative AF is common following a maze procedure and usually abates by 3 months. In addition to restoring sinus rhythm the maze procedure is associated with additional clinical benefits for the patients. In those with mitral valve disease restoration of sinus rhythm improves survival. Risks for stroke, systemic thromboembolism and anticoagulant-related haemorrhage are also reduced. The freedom from late stroke is likely to be from restoration of sinus rhythm as well as excision of the left atrial appendage, an integral part of the maze procedure.

Despite the excellent results of the cut and sew maze procedure, few surgeons adopted the procedure due to its technical difficulty and is almost obsolete today. Advances in the understanding the pathophysiology of AF and newer ablation technologies fostered the development of novel strategies aimed at simplifying the procedure to make it more accessible to the average surgeon without compromising the results.

Use of ablative energy sources has enabled to replace most of Cox III incisions with a variety of energy sources including radiofrequency, cryoablation and high frequency ultrasound. The development of these technologies has rendered a technically difficult and time-consuming operation easy for all cardiac surgeons to perform. Ablation technologies have also helped foster the development of less invasive procedures through a small incision or port. In order to replace the incision in AF surgery, ablation technology must meet several requirements. It must reliably produce transmural lesion either from the epicardial or endocardial surface to ensure bidirectional conduction block. It should also be safe and render AF surgery simpler and less time consuming to perform. It would also need to be adaptable to minimally invasive approach. Melby and colleagues described procedure that replaced with cut-and-sew lesions with bipolar radiofrequency lines as the Cox-Maze IV. In this technique, the atrial septal lesion was not performed and an independent isolation of the pulmonary veins was made with a connecting lesion. Although bipolar radiofrequency may reliably produce transmural lines and applied minimally invasively for pulmonary veins, it does not permit secure performance of connecting lines in the left atrial isthmus or inside the right atrium.

Early follow-up suggests that the Cox-Maze IV procedure is similar in efficacy with 91% of patients having freedom from AF at 6 months. There was no operative mortality and the group had significantly shorter cross clamp time compared to the Cox Maze III group. Cox also suggested another simplified procedure to cure most patients of AF. This involved three essential lesions that include: 1. an incision encircling the pulmonary veins, 2. left atrial isthmus and companion coronary sinus lesions and 3. right atrial isthmus lesion. This modified Cox-Maze procedure has been shown to be nearly as effective as the Cox-Maze III.
6. Lesion sets for the surgical treatment of atrial fibrillation

Three general categories of lesion sets exist for the surgical treatment of AF in adults:

- Pulmonary vein isolation
- Left atrial lesion set
- Biatrial lesion set

Pulmonary vein isolation is only an ideal choice for those who have new onset paroxysmal AF. Lesions can be created using a variety of different approaches that include beating heart epicardial techniques or on pump endocardial approaches that use energy devices or the cut and sew technique. Pulmonary vein isolation can be achieved through either a single lesion encircling all pulmonary veins or two lesions encompassing the left and right pulmonary veins. Left atrial lesion sets are advised in patients with recent-onset or paroxysmal AF undergoing elective surgery with no justification to open the right atrium. This includes pulmonary vein isolation with the addition of linear lesions extending to the mitral annulus and left atrial appendage that is usually excised or excluded. This is because more than 90% of left atrial thrombi originate from the left atrial appendage in patients with non-rheumatic AF.

Biatrial lesion sets are the most effective treatment option for AF. Patients with longstanding or symptomatic AF, young patients or those undergoing right heart surgery would benefit from this procedure.

The Cox maze IV procedure is performed with the patient on CPB with bicaval cannulation. Using blunt dissection, the right and left PVs are dissected. If the patient is in AF, they are cardioverted. Pacing thresholds are obtained from all PVs. The bipolar ablation is performed around the cuff of atrial tissue surrounding the right and left pulmonary veins. Pacing is used to confirm block from both the superior and inferior PVs. Following PVI, the right atrial lesions are performed with the heart beating. An incision is created in the right atrial appendage as shown in figure 3. The bipolar device is used to make a right atrial free wall lesion. Following this a vertical right atriotomy is made around 2cm from the free wall ablation that extends from the crista terminalis toward the intra-atrial septum. The incision is then extended superiorly toward the AV groove. Two cryolesions are placed at the tricuspid annulus using cryoprobe. The bipolar clamp is used to create linear ablation lines from the SVC down to the IVC. The SVC ablation is made as laterally as possible to avoid damage to the SA node. The left sided atrial lesions are performed through a standard left atriotomy. This extends superiorly onto the dome of the left atrium and inferiorly around the orifice of the right inferior pulmonary vein. A lesion is made with the bipolar RF device to create a connecting lesion between the left atrial incision inferiorly to the ablation line encircling left inferior pulmonary vein. In atria greater than 5cm in diameter, a second connecting ablation is placed from the superior aspect of the incision into the left superior pulmonary vein. Finally, a bipolar radiofrequency ablation line is performed from the inferior aspect of the left atrial incision across the mitral valve annulus at a point between the circumflex and right coronary artery circulation. A cryolesion is placed at the mitral valve annulus. The left atrial appendage is amputated and a bipolar RF ablation is performed between the amputated left atrial appendage and superior PV. The left atrial appendage is oversewn. The aorta is unclamped and the right atrial incision is closed.

Patients with lone atrial fibrillation can choose between a catheter based approach or a minimally invasive surgical technique. Minimally invasive surgery to treat lone atrial fibrillation also can benefit patients who have a contraindication to warfarin, antiarrhythmic medications, or a history of cerebrovascular events. The procedure involves groin
Fig. 3. Cox-Maze IV procedure
cannulation to connect the patient to CPB. The right sided lesions can be completed on a
beating heart with or without cardiopulmonary support. After cross clamping the aorta, left
sided lesions are created in a similar pattern to the Cox-Maze II procedure, creating a box
lesion around all the pulmonary veins with a connecting lesion to the left atrial appendage
and to the mitral valve isthmus. The left atrial orifice is closed from the endocardial side.
When performed correctly, the results of the minimally invasive approach are excellent.\textsuperscript{61}
Minimally invasive surgical techniques have been an area of interest as an alternative to
catheter based pulmonary vein isolation. Bilateral thoracotomies or mini sternotomy can be
used to isolate the pulmonary veins\textsuperscript{62,63}. Its major advantage is that it can be performed in
the absence of cardiopulmonary bypass and in many cases left atrial appendage
disarticulation can also be offered. However, since AF does not always originate in the
pulmonary veins this is not suitable in all patients, particularly those with non-paroxysmal
AF.
Surgery for AF reduces medical costs successfully. When performed in conjunction with
elective cardiac surgery it is cheaper and more effective than medical or catheter based
therapy at a later time and cost effective in patients with a good prognosis\textsuperscript{64,65}. In summary,
surgery for AF has evolved during the previous 2 decades to include several different
approaches, lesion sets and energy sources. All patients undergoing concomitant cardiac
surgery with AF should undergo surgical treatment for their AF. Ablation technology has
simplified the procedure to make it easy for all surgeons to adopt and pave way towards
minimal access procedures.
7. References


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This book considers mainly the current perioperative care, as well as progresses in new cardiac surgery technologies. Perioperative strategies and new technologies in the field of cardiac surgery will continue to contribute to improvements in postoperative outcomes and enable the cardiac surgical society to optimize surgical procedures. This book should prove to be a useful reference for trainees, senior surgeons and nurses in cardiac surgery, as well as anesthesiologists, perfusionists, and all the related health care workers who are involved in taking care of patients with heart disease which require surgical therapy. I hope these internationally cumulative and diligent efforts will provide patients undergoing cardiac surgery with meticulous perioperative care methods.

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