Surgical treatment of atrial fibrillation: a review

James L. Cox*

Division of Cardiothoracic Surgery, Washington University School of Medicine, St. Louis, MO, USA

Submitted 25 May 2004; accepted after revision 24 June 2004

KEYWORDS
atrial fibrillation;
antiarrhythmic surgery;
maze operation;
high intensity focussed ultrasound;
surgical treatment of atrial fibrillation

Abstract  The optimal treatment of atrial fibrillation (AF) depends upon a proper understanding of the electrophysiological basis of its clinical manifestations. Whether AF is continuous (persistent) or intermittent (paroxysmal) depends on variable underlying electrophysiology and determines the choice of interventional treatment. The initiation of intermittent AF requires a “trigger”, often, though not always, located near the orifices of pulmonary veins. In contrast, continuous AF does not need a “trigger” to be re-induced repetitively and does not depend on the pulmonary veins or other abnormal automatic foci for its induction or perpetuation. Simple pulmonary vein encircling confines the trigger to the pulmonary veins and, if expertly performed, will cure the majority of patients with intermittent AF. On the other hand, continuous AF requires a Maze procedure or variant thereof to eliminate atrial macro-reentry while allowing sinus rhythm to activate the entire atrial myocardium and preserve atrial transport function. This article reviews the development of the surgical Maze procedure and its implications for the treatment of AF by percutaneous intracardiac or epicardial, minimally invasive techniques. High-intensity focussed ultrasound, a new energy source generating frictional heat, appears promising in the creation of focussed transmural lesions, while preserving the integrity of coronary arterial walls.

Introduction

The optimal treatment of atrial fibrillation (AF) depends upon a proper understanding of the electrophysiological basis of each of its clinical manifestations. It is fortuitous that in the case of AF, both its clinical manifestation and its proper interventional treatment are dictated by its underlying electrophysiology. AF presents clinically in one of two ways. The patient either has AF all of the time or not. If AF is present all of the time, it is classified as continuous (persistent). If it is not present all of the time, it is classified as intermittent (paroxysmal).
This simple classification correctly implies that there is a fundamental pathophysiological difference in the AF of a patient who presents with intermittent episodes versus one who presents with a continuous arrhythmia, and that all other differences are irrelevant from a perspective of interventional management. Indeed, the clinical presentations are different specifically because the underlying electrophysiology is different. This correlation explains why their interventional treatment must also be different.

**Intermittent atrial fibrillation**

It is intuitive that intermittent AF is initiated repetitively, and thus requires a “trigger” to induce each individual episode. In 1998, Haïssaguerre and colleagues suggested that this trigger is located in the orifices of the pulmonary veins in approximately 90% of patients [1], although more recent studies suggest a considerably lower percentage [2]. Regardless of the true prevalence of pulmonary vein triggers, the premature event originating within the pulmonary vein propagates into the left atrium and induces the formation of multiple macro-reentrant circuits involving both atria (Fig. 1). The multiple circuits cause the atria to quiver, a state known as AF (Fig. 2) [3].

Once induced, AF persists until the macro-reentrant circuits terminate spontaneously or are terminated by drugs or other interventions. If AF terminates spontaneously, normal sinus rhythm naturally returns (Fig. 3). Each subsequent episode of AF then requires another premature event to initiate the episode, the trigger again being the pulmonary veins in the majority of cases.

At least 10% of patients with intermittent AF have a triggering mechanism that does not involve the pulmonary veins (Fig. 4) [1,2]. AF can be induced episodically by the Wolff–Parkinson–White syndrome, atrioventricular (AV) node reentry, or the deterioration of atrial flutter [3,4], although, in most cases, non-pulmonary vein triggers, like their counterparts in the pulmonary veins, are automatic foci located elsewhere in the atria.

**Continuous atrial fibrillation**

If normal sinus rhythm is not re-established either spontaneously or by drugs, AF is no longer episodic and, once induced, the atria will continue indefinitely to fibrillate. Once AF is continuous, it no longer needs a “trigger” to be re-induced repetitively since the atria are fibrillating all of the time. Thus, continuous AF does not depend on the pulmonary veins for its induction or perpetuation.

AF becomes continuous because the macro-reentrant circuits in the atrial myocardium are capable of sustaining themselves. Allessie has attributed this to “atrial remodelling” [5].

---

**Figure 1** Induction of AF by a premature atrial complex (PAC) originating in the orifice of a pulmonary vein. These triggers may be caused by micro-reentrant circuits that occur in the cellular transitional zone between pulmonary vein endothelium and left atrial endocardium. However, these triggers are most likely automatic rather than reentrant, since recurrent atrial tachycardias are known to arise from automatic foci in other parts of the atria [11]. Reproduced with permission.

**Figure 2** Once induced, AF is characterized by the presence of multiple macro-reentrant circuits in the atria (posterior view). This electrophysiological basis of AF is unrelated to (1) how, or from where, it was induced, (2) whether it is intermittent or continuous, and (3) its duration. Reproduced with permission.

**Figure 3** Normal sinus rhythm naturally returns after a single episode of AF.

**Figure 4** AF can be induced episodically by the Wolff–Parkinson–White syndrome, atrioventricular (AV) node reentry, or the deterioration of atrial flutter.
This electrical remodelling may or may not be associated with anatomic remodelling, i.e., enlargement, hypertrophy or stretching of the atria. The essence of this concept, as Allessie points out, is that "atrial fibrillation begets atrial fibrillation". In other words, the more a patient develops atrial fibrillation, the more that patient is likely to re-develop it.

Figure 3  Intermittent AF. The patient is in normal sinus rhythm (NSR) some of the time. A premature atrial complex (PAC) induces AF until atrial macro-reentry can no longer sustain itself and ends spontaneously. Each episode of AF requires a PAC to trigger its induction. Reproduced with permission.

Figure 4  The trigger of intermittent AF was located in the pulmonary veins in 90% of patients and outside the pulmonary vein area in 10% of patients with normal or small-sized atria [1]. Reproduced with permission.
Interventional therapy

Simple pulmonary vein encircling confines the trigger to the pulmonary veins and, if expertly performed, will cure the majority of patients with intermittent AF (Fig. 5). Unfortunately, it is unsuccessful as an isolation procedure in a significant number of patients with intermittent AF because the trigger mechanism is not in the pulmonary veins. In addition, since continuous AF is not dependent upon the pulmonary veins for its maintenance, simple pulmonary vein encircling is not a rational treatment goal in patients with continuous AF (Fig. 6).

The only way to ablate atrial macro-reentry, and at the same time, leave the resultant sinus rhythm capable of activating the entire atrial myocardium and assure atrial transport function postoperatively is to perform the Maze procedure or a variant thereof. The Maze procedure is a classic ablation procedure which eliminates the macro-reentry responsible for AF. Unfortunately, since it is a major open-heart operation, it is too complex to be applied to all patients with AF and therefore, vigorous efforts are being made to develop a simplified version of the Maze procedure that can be applied more widely while retaining its efficacy.

Atrial lesions necessary to cure atrial fibrillation

All surgical interventions for cardiac arrhythmias can be classified as isolation or ablation procedures. Isolation procedures do not terminate arrhythmias but confine them and/or their trigger mechanisms to a desired region of the heart to minimise their adverse effects. Ablation procedures eliminate arrhythmias by destroying their trigger mechanism, or by altering or removing the substrate allowing their induction or sustenance.

We have learned empirically that several lesions created in the original surgical Maze-III procedure may not be essential to ablate AF. The Maze procedure was designed to preclude the development of macro-reentry anywhere and everywhere in the atria. There is growing evidence, however, that, although continuous AF is sustained by multiple macro-reentrant circuits, only a limited number of left atrial sites are capable of sustaining...
such circuits [6]. This is the theory behind the so-called “focal” AF that seems to respond to lesions critically created away from the pulmonary veins. It also supports the empirical observation of the elimination of continuous AF despite forgoing several lesions originally included in the Maze procedure (Fig. 7).

I have never been convinced that the septal lesion was essential to the efficacy of the Maze procedure and performed it primarily to gain

---

**Figure 6**  Failure of pulmonary vein isolation for continuous AF. Since the maintenance of continuous AF is not dependent upon triggers located in the pulmonary veins, simple pulmonary vein isolation is not a legitimate treatment. However, if a large amount of left atrium is ablated during prolonged catheter attempts to achieve "pulmonary vein isolation", it may eliminate continuous AF. This procedure is not a "pulmonary vein isolation" but an "ablation of the left atrium". Reproduced with permission.

**Figure 7**  The five left atrial lesions of the standard Maze-III surgical procedure for AF. Reproduced with permission.
better exposure of the inside of the left atrium. Reports of recent interventional and surgical therapy for AF suggest that the septal lesion is not necessary. In addition, the lesion extending out into the left atrial appendage was included in the Maze procedure in order to prevent the theoretically possible reentry around the base of the appendix. That reentrant circuit has never been documented and may not be a clinical entity. Therefore, that lesion may also be unnecessary.

If neither the septal nor the left atrial appendage lesions are critical to ablate AF, the essential left atrial lesions would be limited to the incision encircling the pulmonary veins and the lesion across the “isthmus”, between the inferior pulmonary veins and the mitral annulus. Experience with the Maze and other surgical antiarrhythmic procedures has amply confirmed that the left atrial isthmus lesion is extremely important in eliminating reentry responsible for AF. Indeed, this lesion, along with its companion cryolesion in the coronary sinus, is the “Achilles Heel” of the Maze procedure, in that every failure in our own series was associated with persistent conduction across this isthmus (Fig. 8) [7].

Besides the simplification of left atrial lesions, experimental observations by Allessie regarding differences in left versus right atrial refractory periods suggest that the right atrial lesions of the Maze procedure may be replaced by a simpler approach [8]. The duration of the local refractory period determines the minimum size of an atrial macro-reentrant circuit [9]. Since refractory periods are shorter in the left atrium, it can sustain the small macro-reentrant circuits that are characteristic of AF, whereas, in the right atrium, the longer refractory periods are probably not capable of sustaining AF, unless the atrium is pathologically enlarged. Thus, the treatment of AF can be focused on the left side, knowing that AF will not recur if macro-reentry can be prevented by lesions critically placed in the left atrium. In the right atrium, capable of sustaining only atrial flutter, a lesion in the isthmus between coronary sinus and tricuspid valve will disable a critical limb of the macro-reentrant circuit sustaining the vast majority of atrial flutters (Fig. 9) [10]. In summary, most patients with AF of either type could probably be cured by a “Mini-Maze Procedure” which includes (1) an incision encircling the pulmonary veins, (2) a left atrial isthmus and companion coronary sinus lesions, and (3) a right atrial isthmus lesion (Fig. 10).

Characteristics of the ideal operation for atrial fibrillation

Besides establishing this lesion pattern as an intelligent simplification of the original Maze

![Figure 8](image_url)  
**Left panel:** The lesion across the left atrial isthmus is essential to prevent the postoperative development of a macro-reentrant tachyarrhythmia (“slow left atrial flutter”) around the pulmonary vein lesions. However, this atriotomy alone does not preclude the development of this circuit and must be accompanied by the creation of conduction block in the coronary sinus. **Right panel:** The simplest technique for creating conduction block through the coronary sinus wall is by cryoablation. The cryolesion must be placed in the precise plane of the isthmus atriotomy to be effective. Reproduced with permission.
procedure, several years of clinical experience and vigorous industrial support have also defined the characteristics of the ideal technique by which this pattern should be applied. First, the procedure should be epicardial because of the greater safety of delivering the energy from epicardium to endocardium, instead of from the endocardium, where over-penetration may cause catastrophic complications (Fig. 11). Second, the energy source should be capable of penetrating fat as well as atrial muscle to eliminate the need to dissect away the epicardial fat prior to its application. Third, the procedure should be capable of ablating intermittent AF, continuous AF and atrial flutter. Fourth, cardiopulmonary bypass must be avoided. Fifth, the procedure should be endoscopic or minimally invasive, and be completed within \textless 60 min. Sixth, in absence of complications, patients should be able to leave the hospital no later than on the first postoperative day. In summary, the ideal AF procedure would be performed via a minimally invasive incision, endoscopically or robotically, off cardiopulmonary bypass, in \textless 1 h, with hospital discharge planned for the next morning.

**High-intensity focussed ultrasound (HIFU) — the ideal energy source?**

This ideal AF procedure demands the development of new energy sources capable of ablating tissue in critical areas, such as the left atrial isthmus, more rapidly, uniformly and safely than any energy source currently available. No energy source, including cryotherapy, uni- or bipolar radiofrequency, irrigated radiofrequency, microwave, or laser energy, is currently capable of creating the left atrial isthmus lesion from the epicardial surface because of the unavoidable penetration through...
the circumflex coronary artery to reach the left atrial wall near the posterior mitral annulus (Fig. 12). Therefore, the Mini-Maze procedure cannot be performed by an epicardial approach with any presently available energy source.

However, high-intensity focussed ultrasound (HIFU), a new energy source currently in clinical trials in Europe, seems to overcome most, if not all, the limitations identified thus far (Fig. 13). HIFU is capable of ablating efficiently atrial myocardium as well as epicardial fat. It creates tissue injury by causing cells to oscillate or “rub against each other”, generating frictional heat that destroys the cells. The absence of injury to the coronary arteries, perhaps the most important and unique characteristic of HIFU, is explained by the distance between circulating red blood cells, which prevents them from generating the heat of friction. Since the blood remains at body temperature, it protects the integrity of the arterial wall. This allows the ablation of myocardium and fat on both sides of a coronary artery without causing any injury to the vessel itself. Thus, HIFU can be used to create lesions across the left atrial isthmus from the epicardium without injury to the circumflex coronary artery, which no other energy source can accomplish (Fig. 14). HIFU also very rapidly creates uniform, contiguous transmural lines of tissue ablation, and is amenable to minimally invasive surgery without cardiopulmonary bypass.

**Closing comments**

Despite the promise of developing a simpler operation for AF that would be applicable in virtually all patients, it is noteworthy that many of the more recent concepts, particularly those relating to the induction of AF, are based on limited scientific data, on very few clinical cases, or on observations made in highly selected patients. The electrophysiology of AF is unforgiving and, despite our fondest wishes, the truth regarding AF and its treatment cannot be altered to fit preconceived notions. It is predictable that a significant number of patients with AF will continue to require the more extensive and complex surgical procedures. Nevertheless, these newer simplified surgical
approaches offer the possibility of curing patients who are currently doomed to live with their disease.

In summary, up to 90% of intermittent ("paroxysmal" or "persistent") atrial fibrillation can be cured by a properly performed isolation of the pulmonary veins. In order to attain a similar cure rate in patients with continuous ("permanent") atrial fibrillation, however, it is essential to add additional lesions in the left atrium, specifically the left atriotomy from the pulmonary vein encircling incision down to the mitral valve annulus posteriorly and ablation of coronary sinus conduction at the same point. Since this additional lesion is now becoming easier to perform, there is little reason not to add it in patients with intermittent atrial fibrillation as well.

References


