The first Maze procedure

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By the time the first Maze procedure was performed in 1987, we were already operating on approximately 120 patients a year for other types of cardiac arrhythmias. We began to focus on atrial fibrillation in a systematic fashion in our laboratory at Duke University in 1980 by first developing an animal model that was more clinically relevant than existing models. The main preconditions for the model were that no scars could be placed on either atrium and that the pericardium could not be entered because scars or pericarditis might obscure the characteristics of any atrial fibrillation caused by the model itself. The canine model included a sterile left thoracotomy and the introduction of a left atrial pressure catheter and a biopsy needle through a purse-string suture in the extrapericardial left superior pulmonary vein. Mitral insufficiency was created by transecting the chordae tendineae in a stepwise fashion until the left atrial pressure increased to approximately 20 mm Hg. The animals were allowed to recuperate for at least 3 months when atrial fibrillation could be easily induced. This new model allowed us to assess the effects of various surgical interventions without mapping them. The ability to map atrial fibrillation awaited the development of a computerized multipoint digital mapping system developed in the mid-1980s by Dr John P. Boineau, Dr Richard Schuessler, and Barry Branham in our laboratory at Washington University in St Louis.

One day in June 1986, I received a phone call from an airline captain from Cyprus named George Dheere. Captain Dheere was 37 years old and had experienced a severe transient ischemic attack as the result of new-onset atrial fibrillation. He was immediately anticoagulated and grounded, a personally devastating sequence of events. Captain Dheere had read an article that we had written in 1982 entitled “Surgical Management of Atrial Fibrillation,” in which we described how atrial fibrillation could be confined to the left atrium using the left atrial isolation procedure. Despite our discouraging Captain Dheere, he flew to St Louis a few days later and insisted that we perform some type of surgical procedure for his atrial fibrillation. During Dheere’s 1-week stay, he visited our research laboratories and discovered that we had also recently developed an operation called the “atrial transection procedure,” which was the first procedure designed specifically to abolish atrial fibrillation and had a cure rate of 100% in our atrial fibrillation animal model. He insisted on undergoing an atrial transection procedure, but we refused on grounds that it was not ready for clinical application. Disgruntled and disappointed, he returned to Cyprus in atrial fibrillation despite amiodarone therapy. In September 1986, however, Captain Dheere returned to St Louis and informed us that he was not going to leave until we did something surgically to alleviate his atrial fibrillation. After extensive discussions among the clinical and experimental cardiology and surgical teams, and after securing institutional review board approval from Barnes Hospital, we decided to proceed with an atrial transection procedure. Captain Dheere remained arrhythmia-free postoperatively for 5 months, but then viral pericarditis and recurrent atrial fibrillation developed. Fortunately, during the intervening months we had finally begun to accumulate sufficient critical data from our animal models, and from our mapping of patients with atrial fibrillation who were undergoing surgery for Wolff–Parkinson–White syndrome, we had begun to construct an accurate picture of the electrophysiologic basis of atrial fibrillation. Through the laborious work of Drs John Boineau and Richard Schuessler using their array of more than 250 bipolar electrodes experimentally and 156 electrodes clinically, and by using the computer programs developed by Barry Branham, the electrical events that were occurring in the atrium during atrial fibrillation were finally elucidated. Although the data were still incomplete, it was apparent that all atrial fibrillation, once established, was characterized by the presence of multiple large constantly shifting macro-reentrant circuits in the atria. Unfortunately, these maps also documented that the macro-reentrant circuits often remained in one location for only 200 ms, making map-guided surgery impractical. That left us with only one viable option. If we were going to treat atrial fibrillation surgically, it was now apparent that we had to develop a procedure that made it impossible for the atrium to fibrillate at all. The only way to do that would be to place incisions in the atria close enough together that macro-reentrant circuits could not form between them. Fortunately, the macro-reentrant circuits that we had mapped in both dogs and patients were physically relatively large, that is, more than 5 to 6 cm in diameter in the left atrium and much larger than that in the right atrium. Therefore, atrial incisions placed no more than 5 to 6 cm apart should theoretically prevent the development of macro-reentrant circuits anywhere in the atria. It was clear that if macro-reentrant circuits could not develop in the atria, then the atria could not fibrillate. The dilemma was how to place enough lesions on the atria to preclude the development of atrial macro-reentry...
(fibrillation) and leave behind an atrium that could be activated by the sinus node and still contract effectively.

That question was answered one Saturday afternoon while I was studying some of the Boineau–Schuessler experimental maps of atrial fibrillation in my office at Barnes Hospital. To better visualize the relationship between atrial anatomy and atrial electrophysiology, a rectangle was drawn to represent the entire mass of both atria 2-dimensionally. The rectangle was then arbitrarily partitioned into the left atrium (with its pulmonary vein orifices) and the right atrium (with the superior and inferior venae cavae orifices). Both atria had appendages. The atrial septum was placed between the right and left atria. The sinus (sinoatrial [SA]) node was placed at the top of the septum, and the atrioventricular (AV) node was placed at the bottom of the septum. This, then, represented all of the pertinent anatomy of both atria (Figure 1, A) on which known electrophysiology could be superimposed.

The pattern of activation during normal sinus rhythm that we had mapped numerous times in humans was then superimposed on this schematic rectangular diagram of the atria (Figure 1, B). We had also recorded several bouts of atrial flutter and atrial fibrillation, and those maps were also superimposed on the schematic rectangular atria. While studying this 2-dimensional representation of the atria with the superimposed electrophysiology of atrial fibrillation, I suddenly realized that everything necessary to abolish atrial fibrillation, while leaving the atrial activation and contraction intact afterward, could be accomplished by creating a pattern of lesions in the atria that was essentially that of a simple maze (Figure 1, C). The lesions could be placed close enough to prevent atrial macro-reentry, and if placed

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**FIGURE 1.** These diagrams demonstrate the concept of the Maze procedure first envisioned in early 1987. A, Two-dimensional representation of the anatomy of the atria with the right atrium harboring the orifices of the superior and inferior venae cavae, as well as the right atrial appendage. The left atrium has the pulmonary veins and left atrial appendage. The atrial septum divides the 2 with the SA node in the top of the right atrium near the septum and the AV node at the bottom of the septum connecting to the ventricles. B, Arrows depict the propagation of a normal sinus rhythm beat from its source (starburst) in the SA node to all of the atrial myocardium and then to its termination at the AV node. C, Heavy lines represent lines of conduction block created by lesions in the atria. By placing the lesions in the pattern of a maze, they can be placed close enough to prevent the development of macro-reentry anywhere in either atrium and still allow the sinus impulse to activate all of the atrial myocardium except the encircled pulmonary veins and excised atrial appendages (D). AV, Atrioventricular; IVC, inferior vena cava; LAA, left atrial appendage; SA, sinoatrial; SVC, superior vena cava; RAA, right atrial appendage.
in a maze pattern, the SA node could serve as the site of entry of electrical activity into the atria and the AV node as its site of exit from the atria. One contiguous “true route” of conduction would be left intact between the entrance and exit sites, and multiple “blind alleys” off this main conduction route would allow activation of all of the atrial myocardium, thereby preserving atrial contractility (Figure 1, D). One entrance, one exit, one true route between the two and multiple blind alleys … the pattern and principle of a maze.

Once I realized that atrial lesions placed in a maze pattern was the answer to the surgical treatment of atrial fibrillation, the next problem was to determine exactly where those lesions should be placed on the actual 3-dimensional atria. Remembering that John Boineau had several hundred canine hearts stored in formaldehyde in our research laboratory, I immediately went to our laboratory and dissected several complete atrial blocks (right atrium, atrial septum, and left atrium) completely away from the remaining portions of heart and great vessels. After drying them, a black dot was placed at the site of the anatomic SA node with a marking pen. Three intersecting lines were then placed around 3 sides of the SA node region to indicate the site of potential incisions. By leaving one side open, the sinus impulse could propagate only in that direction. By following this logic, lines were subsequently drawn close enough together to prevent the large macro-reentrant circuits from occurring (thereby precluding the ability of the atria to fibrillate) while at the same time “directing” the propagation of the imaginary sinus impulse in any desired direction. Once it was certain that the right atrium, the left atrium, and the septum would be activated in this manner, attention was directed toward preventing the impulse from “doubling back” on itself, thereby creating a single large macro-reentrant circuit. Finally, the ability of the sinus impulse to reach the AV node was ensured so that it could activate the ventricles as well (Figure 2).

Three such atrial “blocks” with their black lines were saved until the following Monday when the pattern was recreated on a fresh canine heart using atrial incisions rather than black lines. After suturing the incisions closed, the fresh atrial block was fixed in formaldehyde and used thereafter as a guide for the performance of this newly named “Maze procedure” in live animals (Figure 3). The Maze procedure was subsequently shown to abolish atrial fibrillation in our animal model, and long-term experimental studies proved that it was safe and permanent. Most important, we went to great lengths to document that atrial transport function remained intact postoperatively.

In the late summer of 1987, a cardiologist from Alton, Illinois, near St Louis, called about his cousin, who had severe atrial flutter/fibrillation. He had failed all medical therapy, including amiodarone. After a long, detailed workup and several intense discussions, the Barnes Hospital Institutional Review Board approved a “one-time” trial of the Maze procedure in this one patient, and on September 25, 1987, I performed the first clinical Maze procedure (Figure 4). The patient remained in sinus rhythm until the seventh postoperative day, when atrial fibrillation developed. Digoxin and procainamide were administered, and he quickly converted to sinus rhythm. One month after hospital discharge, the patient presented with a severe case of procainamide-induced lupus syndrome, diagnosed by Dr Bruce Lindsay, now the Chief of Electrophysiology at the Cleveland Clinic. The procainamide was discontinued. During the next month, he remained in sinus rhythm and his lupus syndrome relented. His digoxin was discontinued by Dr Lindsay the next month, and he remained in sinus rhythm. On his return outpatient visit at that time, the eminent electrophysiologist Dr Maurits Allessie from Maastricht, The Netherlands, was visiting our institution, and I felt privileged and fortunate to have him document that the patient was in normal sinus rhythm with no medications. The patient had no further episodes of atrial flutter or fibrillation, was not anticoagulated, and took no antiarrhythmic drugs. He eventually had a recurrence of atrial fibrillation just 1 month shy of the 20-year anniversary of his Maze procedure, but his arrhythmia is controlled on antiarrhythmic medication.

Having maintained weekly telephone contact with my office and with the first Maze patient himself during late 1987, Captain Dheere knew exactly what was unfolding.
in St Louis. Thus, after a few months, Captain Dheere insisted on having his previous atrial transection procedure “converted” to the Maze procedure. After waiting 5 months after the first Maze procedure, we performed the “conversion” from an atrial transection procedure to a Maze procedure on Captain Dheere in February 1988. This conversion resulted in a pattern of atrial lesions that was essentially the same as the iteration that we would later call the “Maze III” procedure. Unfortunately, on the eighth postoperative day, intractable atrial flutter developed in the patient. He remained in this rhythm until September 7, 1988, when he was finally documented to have the first example ever recorded of what would later be termed “atypical left atrial flutter.” This iatrogenic arrhythmia was using the coronary sinus as a critical “bridge,” allowing conduction to occur across the left atrial isthmus between the inferior pulmonary veins and the posterior mitral valve annulus. We interpreted this to mean that the cryolesion that had been placed on the coronary sinus at the time of his second surgery had failed. The circuit was interrupted by Dr Michael Cain, then Chief of Electrophysiology at Washington University and Barnes Hospital, using catheter fulguration. Captain Dheere

FIGURE 3. Photograph taken by Research Fellow Byung-Chul Chang, now the Professor and Chief of Cardiothoracic Surgery at Yonsei University in Seoul, Korea, in early 1987 of the author performing the first experimental Maze procedure in the Cardiothoracic Surgery Research Laboratories at Washington University in St Louis.

FIGURE 4. Two frames from a movie of the first Maze procedure performed clinically on September 25, 1987. A, Overhead view of the heart exposed via median sternotomy (the patient’s head is to the left) showing the multitude of wires extending from 3 silastic electrode arrays containing a total of 156 atrial electrodes. One bay of the electrodes is being connected to one of the computer cable connectors at the top of the table. B, One of the initial right atrial incisions of the first Maze procedure.
remained free of atrial arrhythmias for the remainder of his life with no medications (Figure 5). In addition, he ultimately was reinstated as a commercial airline pilot with his beloved Cypress Airways. One of my saddest days occurred in the summer of 2003, when I was called by one of Captain Dheere’s daughters in Cyprus and told that he had died suddenly of a heart attack just shy of his 54th birthday. I have often said that without the courage and persistence of this brave airline pilot from Cyprus, the development of the Maze procedure would have been delayed at least 10 years and perhaps would never have occurred at all. My personal experience with Captain George Dheere and the Maze procedure only strengthens my long-held belief that there are no courageous surgeons, only courageous patients.

**References**


**FIGURE 5.** Captain George Dheere running up the steps of the Curium Roman Amphitheatre in his native Cyprus. This photograph was taken by the author 20 months after Captain Dheere’s Maze procedure was performed. Captain Dheere was the first person to ever undergo a surgical procedure specifically designed to ablate atrial fibrillation, the second to undergo a Maze procedure, the first to undergo a re-do Maze procedure, the first to undergo the equivalent of the Maze III procedure, and the first to experience the iatrogenic problem of atypical left atrial flutter.