Atrial fibrillation is present in approximately 50% of all patients undergoing surgery for the treatment of mitral valve disease. For more than 40 years, cardiac surgeons have been forced to ignore this associated atrial fibrillation at the time of open mitral valve surgery because there has been no effective way to treat it. After the success of the maze procedure in treating atrial fibrillation, several surgeons began to add the maze procedure as an adjunct to mitral valve surgery to treat both problems. When properly performed, the results with this combined approach have been excellent, with no increase in perioperative morbidity or operative mortality and with documented long-term advantages over simply leaving patients in atrial fibrillation. Despite these results, adding the maze procedure to mitral valve surgery significantly prolongs and complicates the operation; therefore, most surgeons have understandably been reluctant to perform the combined procedure.

During the past 2 to 3 years, cardiologists have become extremely aggressive in their efforts to treat atrial fibrillation by modifications of the percutaneous radiofrequency catheters that have been so successful in treating other types of arrhythmias, especially the Wolff-Parkinson-White syndrome. It was only a matter of time until some surgeons used these same percutaneous endocardial catheters to treat atrial fibrillation intraoperatively when performing mitral valve surgery. This new surgical intervention was quickly recognized by medical device companies as a new market and, as a result, new radiofrequency devices have been adapted specifically for intraoperative use.

In this issue of the Journal, the group from Maastricht, The Netherlands, describes the use of intraoperative radiofrequency catheters to ablate atrial fibrillation in patients who required surgery primarily for mitral valve disease. This study is particularly important because it comes from perhaps the premier electrophysiology group in the world, especially in the area of atrial fibrillation. Clinical electrophysiology was born in the laboratory of Professor Dirk Durrer of Amsterdam and led to his becoming the first to perform an intraoperative electrophysiologic map of the human heart in 1957. In 1967, Dr Durrer’s brilliant protégé, Professor Hein J. J. Wellens, one of the authors of this article, described the technique of programmed electrical stimulation that provided a method for reproducibly inducing and terminating reentrant arrhythmias. This landmark achievement made possible the systematic characterization of most clinical arrhythmias and has remained largely unchanged since that time. Thus, it behooves us all to listen when this group speaks of electrophysiology matters!

The authors describe their experience with the use of intraoperative radiofrequency catheters to create linear lesions in both atria in an effort to ablate atrial fibrillation associated with other surgical heart disease in 122 patients, 108 of whom had associated mitral valve surgery. The pattern of lesions placed in the atria was described as “a modification of the maze III procedure.” Actuarial freedom from atrial flutter or atrial fibrillation at 39 months was 78.5% ± 5.1%.

These results are consistent with those obtained by Melo’s group in Portugal, Mohr’s group in Leipzig, Germany, and Alfieri’s group in Italy, all of whom also...
use radiofrequency energy to create the lesions in the atria. Similar results have also been attained by Stephan Schuler in Dresden, Germany,\textsuperscript{17} using microwave energy to create the atrial lesions. However, these studies raise an important question. Why are 20\% to 30\% of the patients not cured of their atrial fibrillation? Assuming that an 80\% cure rate is not acceptable to most cardiac surgeons, we are obligated to answer that question.

During the past several years, at least two major observations regarding atrial fibrillation have had a direct impact on these new intraoperative techniques to treat atrial fibrillation associated with mitral valve disease. The first is that the maze procedure, when properly performed, cures atrial fibrillation in nearly 100\% of patients with or without mitral valve disease.\textsuperscript{4-6} The second is that the majority of paroxysmal (intermittent) atrial fibrillation originates within the orifices of one or more of the pulmonary veins.\textsuperscript{18} Thus, to my knowledge, all of the techniques now being used to treat atrial fibrillation during mitral valve surgery incorporate some combination of the linear lesions of the maze procedure with isolation of one or more of the pulmonary vein orifices. Unfortunately, the scientific basis for these various approaches is somewhat less than completely sound.

A cardinal rule of scientific experimentation is that all variables in an experiment must be controlled except the one variable that is being studied. Thus, when attempting to modify a successful operative procedure, one should avoid introducing more than one variable if the final results of the modification are to be subject to scientific interpretation. Unfortunately, these recent attempts to ablate atrial fibrillation at the time of mitral valve surgery incorporate the precise pattern of the atrial lesions. The basic cut-and-sew technique was left unchanged. Again, when we made the first modification of the maze procedure (from the maze I to the maze II procedure),\textsuperscript{21,22} we were careful to introduce only one variable, a change in the pattern of the atriotomies, with the only variable that was introduced was a slight change in the precise pattern of the atrial lesions. The basic cut-and-sew technique was left unchanged. Again, when we made the second modification (from the maze II to the maze III procedure),\textsuperscript{21,22} we were careful to introduce only one variable, a change in the pattern of the atriotomies, with the modification. As a result, the efficacy of the maze procedure in curing atrial fibrillation remained unchanged from the maze I to the maze II to the maze III, but the undesirable side-effects of the first two procedures were eliminated by these modifications.

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The major problem with using radiofrequency, microwave, or laser energy to treat atrial fibrillation is that there is no way to document with certainty that a lesion is transmural at the time it is created. Studies have been performed to relate the depth of a lesion to the energy expended, time of application, and temperature of the probe tip,\textsuperscript{23-25} but the fact remains that there is simply no real-time method of being certain that a given lesion is transmural using these energy sources.

What, then, are the alternatives to creating the lesions with the old cut-and-sew technique that allow for real-
time documentation of the transmurality of the linear lesions? We now use cryosurgery for the creation of these linear lesions, rather than the cut-and-sew technique, because the surgeon simply has to look at the cryolesion to see when it has become transmural. Once the ice ball has become transmural, the cryoprobe is held in place for 120 seconds and then defrosted. The certainty of permanent transmurality with this method is based on scientific and clinical studies dating back over 25 years to when cryosurgery was first used intraoperatively to treat cardiac arrhythmias. In fact, we now perform the entire maze procedure using linear cryoprobes, rather than the cut-and-sew technique, whether or not it is performed in combination with mitral valve surgery.

**Pattern of Atrial Lesions**

The recent demonstration by Haissaguerre and associates that the majority of atrial fibrillation originates within the pulmonary veins was at once a magnificent revelation for basic electrophysiologists and a treacherous trap for unsuspecting interventional electrophysiologists and arrhythmia surgeons. This important contribution to the understanding of atrial fibrillation does not represent the holy grail for treatment purposes, but rather it is the remaining piece of the previously incomplete puzzle of atrial fibrillation. It fits with the other pieces in the following manner.

Under normal circumstances, the atria are activated by the sinus node, remain in sinus rhythm, and are extremely resistant to the development of atrial fibrillation, as evidenced by the fact that less than 2% of the general population is in atrial fibrillation. Because there is a transition zone between the endothelium of the pulmonary veins and the endocardium of the left atrium, this is the proper milieu for the development of micro-reentry circuits that can lead to premature atrial beats. These premature atrial beats, in turn, can stimulate the atria in such a manner that macro-reentrant circuits are set up in the atria that become self-perpetuating. This is atrial fibrillation. In other words, the atrial fibrillation is initiated by micro-reentrant circuits within the pulmonary veins and is sustained by macro-reentrant circuits in the atrial myocardium itself.

Usually, the atria retain their natural tendency to remain in a normal rhythm, so they will eventually spontaneously revert to a normal sinus rhythm. Later, the micro-reentry circuits in the pulmonary veins reinduce atrial fibrillation, and we recognize the clinical picture of paroxysmal (intermittent) atrial fibrillation. This type of atrial fibrillation can be cured by one of the following techniques:

1. Identifying the culprit pulmonary vein and ablating the micro-reentrant circuit within its orifice

This is the basis for the percutaneous endocardial catheter mapping followed by map-guided radiofrequency ablation within a single pulmonary vein orifice that is now performed by interventional electrophysiologists.

2. Identifying the culprit pulmonary vein and isolating its orifice from the remainder of the atrial myocardium

This also involves catheter mapping by interventional electrophysiologists but is followed by attempts at placing circumferential rings of block around the orifice of the culprit pulmonary vein using either radiofrequency, cryothermia, ultrasound, microwave, or laser energy sources.

3. Isolating the orifices of all four pulmonary veins from the remainder of the atrial myocardium

This is the most logical way for surgeons to treat this type of atrial fibrillation during mitral valve surgery because there is currently no way of identifying the culprit pulmonary vein intraoperatively.

Unfortunately, as the atrium begins to fibrillate more and more, it undergoes a process that has been described as “atrial remodeling” by Professor Maurits Allessie, one of Professor Wellens’ brilliant colleagues in Maastricht. Allessie describes the results of this electrophysiologic (and perhaps anatomic) remodeling as “atrial fibrillation begets atrial fibrillation.” In other words, the more the atrium fibrillates, the more it is inclined to fibrillate. Finally, in most patients, the paroxysmal atrial fibrillation evolves into chronic (continuous) atrial fibrillation because of this process of atrial remodeling.

From the treatment standpoint, it is critical to understand that once this has occurred, the pulmonary veins, and the latent or active micro-reentrant circuits that they may harbor, become irrelevant to the maintenance of the atrial fibrillation. In other words, not even complete isolation of all four pulmonary veins will ablate the atrial fibrillation at this point, and certainly anything less than their complete isolation cannot be expected to be effective. Indeed, the only known nonpharmacologic way to stop the atrial fibrillation at this point is to render the atria incapable of harboring the macro-reentrant circuits that are the basis of atrial fibrillation once it is established. This is what the maze procedure does. Unfortunately, no other pattern of atrial lesions has yet been described to accomplish this goal.

In summary, the attempts at ablating atrial fibrillation during mitral valve surgery using multiple energy sources that are easy to handle and quick to apply is to be strongly encouraged. However, surgeons should make every effort to assure that the lesions they create are transmural in every instance, or else the operation is doomed to a high rate of failure. Furthermore, until a less complex pattern of atrial lesions has been shown to be as effective as the maze procedure, I believe that the pattern of lesions applied during surgery should conform exactly, or nearly so, to the pattern of the maze procedure. With minor modifications, the maze
procedure can now be performed with the use of linear cryo-
probes to assure transmurality without substantially increasing
either the complexity or the duration of mitral valve
surgery and with the expectation that 99% of the atrial fib-
ribillation associated with the mitral valve disease will be
cured. That should be the goal of any other energy source
and of any other pattern of atrial lesions.

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