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## The central controversy surrounding the interventional-surgical treatment of atrial fibrillation

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In this issue of the *Journal*, Fasol and colleagues<sup>1</sup> from Vienna describe their technique for ablating atrial fibrillation in association with mitral valve surgery by creating a triangular pattern of lesions endocardially in the left atrium using irrigated unipolar radiofrequency. The triangular lesion pattern is described in the text as encompassing the orifices of the 4 pulmonary veins, with one corner of the triangle extending down to the mitral valve annulus. However, in Figure 1 the triangle does not include the orifices of the right pulmonary veins. In addition, in the legend for Figure 1, the authors state that the triangle also encompasses the closed orifice of the unresected left atrial appendage, although the figure itself clearly shows the closed appendage orifice to be outside the confines of the triangle. Thus, it is unclear where the triangle is located from reading the text and then observing Figure 1. Nevertheless, assuming that the triangle encompasses all 4 pulmonary vein orifices, regardless of whether it includes the closed orifice of the left atrial appendage, this lesion pattern is clearly simpler than that of the classic maze procedure, as the authors correctly state. However, several questions are left unanswered by the authors that affect the potential significance of their study.

The 10 patients in this study are described as having had “chronic atrial fibrillation.” Other than noting that the atrial fibrillation had been “. . . preexisting for longer than 6 months,” the authors do not clarify whether the term “chronic” refers to the duration of the arrhythmia or to the fact that it was continuously present in these patients. If “chronic” refers to the duration of the arrhythmia, and the patients were not in continuous atrial fibrillation, they were, by definition, having recurrent bouts of paroxysmal (intermittent) atrial fibrillation. In that case the authors had up to a 90% chance of curing the atrial fibrillation by means of pulmonary vein isolation alone, assuming that all of their lesions were transmural, because the individual episodes of atrial fibrillation are dependent on an induction trigger that is located within one or more of the pulmonary veins in as many as 90% of such patients.<sup>2</sup> In that case the 80% cure rate in this study would be about what is expected. If, on the other hand, the authors are using the term “chronic” in the traditional sense when applied to atrial fibrillation (ie, that the patients were in atrial fibrillation all of the time), then the 80% cure rate attained by the authors is better than expected. Because this success rate in patients with continuous atrial fibrillation cannot be attained with simple pulmonary vein isolation alone, it would confirm the importance of extending the triangle from the pulmonary veins down to the mitral annulus, a point that I<sup>3-5</sup> have emphasized in the past.

Perhaps the greatest importance of this article from the Vienna group is that it once again emphasizes the importance of describing the preoperative diagnosis accurately to interpret the results properly. This is the basis of the majority of the confusion that currently exists in the field of atrial fibrillation therapy. During the

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years 1987 through 2000, we evaluated approximately 4000 patients with atrial fibrillation. Roughly 60% of those patients had intermittent atrial fibrillation, and the other 40% were in atrial fibrillation continuously for an average of 8 years. Approximately 400 (approximately 10%) of those patients underwent the maze procedure, many of whom had intraoperative computerized mapping in the late 1980s and early 1990s in which more than 150 electrodes were placed on and inside the atria. We learned that atrial fibrillation, once it is established, exhibits exactly the same macroentrant pattern of activity in the atria during episodes of paroxysmal (intermittent) atrial fibrillation as it does during established permanent (continuous or chronic) atrial fibrillation. Unfortunately, despite our extensive experience in mapping human atrial fibrillation, we were never able to capture the spontaneous onset of a single episode of atrial fibrillation. As a result, although our mapping studies clarified the electrophysiologic basis of atrial fibrillation once it actually occurs, they revealed nothing about the spontaneous induction of atrial fibrillation.

Characterization of precisely how atrial fibrillation is induced awaited the studies of Haissaguerre and associates<sup>2</sup> in 1998, who reported that paroxysmal (intermittent) atrial fibrillation is usually induced by triggers located in the pulmonary veins (90%) and occasionally by triggers located outside the region of the pulmonary veins (10%). Although others have reported slightly different numbers, all have documented that each individual episode of paroxysmal (intermittent) atrial fibrillation is dependent on a trigger or triggers for induction and that most of the time those triggers are located in the pulmonary veins. Once this information became available, it was immediately clear that up to 90% of patients with paroxysmal (intermittent) atrial fibrillation could be cured by simple isolation of the pulmonary veins, which, through isolation of the triggers responsible for the arrhythmia, had the effect of stopping the arrhythmia. That knowledge spurred the rapid development of new tools and energy sources by the medical device industry to allow interventional cardiologists and cardiac surgeons to treat large numbers of patients with atrial fibrillation with a technique that was less complex than the maze procedure.

Unfortunately, the study of Haissaguerre and associates<sup>2</sup> was erroneously interpreted by many to mean that all atrial fibrillation could be treated satisfactorily by simple pulmonary vein isolation alone, including permanent (continuous) atrial fibrillation, which is found in 40% of all patients with the arrhythmia. That erroneous interpretation led many cardiologists and surgeons to make the subsequent mistake of using pulmonary vein isolation alone to treat continuous atrial fibrillation, apparently not recognizing the simple fact that atrial fibrillation that has been ongoing without interruption for many months or years does not need a trigger for its induction but only a driver to sustain it. The subsequent

dismal early results of treating permanent (continuous) atrial fibrillation with pulmonary vein isolation alone documented the irrationality of this approach.<sup>6</sup>

Several interesting things then happened. Some authors began reporting success rates in the 60% to 80% range with pulmonary vein isolation alone for all patients with atrial fibrillation.<sup>7</sup> At first glance, this seemed like a new and significant advance that was inconsistent with the established and generally accepted concepts of the induction<sup>2</sup> and maintenance<sup>3,8</sup> of atrial fibrillation. However, on closer inspection of these reports, it became apparent that their ultimate overall success rates were directly proportional to how many of their patients had paroxysmal (intermittent) atrial fibrillation: the more patients with intermittent atrial fibrillation, the higher the overall success rate for all patients with atrial fibrillation. That was compatible with existing theories because of the known efficacy of pulmonary vein isolation in the treatment of intermittent atrial fibrillation. It also became apparent, however, that many of those reports classified long-standing intermittent atrial fibrillation as chronic atrial fibrillation, thereby leading to the misconception that continuous atrial fibrillation was being cured by means of pulmonary vein isolation alone (a potential problem with the Vienna study in this issue of the *Journal*, as mentioned above).

As a result of the reporting of interventional-surgical results for both intermittent and continuous atrial fibrillation as if they were one entity and of the semantic problem caused by a lack of a proper classification system for atrial fibrillation, it is virtually impossible to determine the true success rates for interventional-surgical therapy. Two recent developments have now increased the confusion by an order of magnitude. First, several cardiologists decided that the triggers in the pulmonary veins were responsible not only for inducing individual episodes of intermittent atrial fibrillation but also for sustaining continuous atrial fibrillation, in which case they were referred to as drivers.<sup>9</sup> This notion was seemingly augmented by the identification of what was termed "pulmonary vein tachycardia," or a consistent rapid firing of foci (drivers) within the pulmonary veins in many patients with continuous atrial fibrillation.<sup>10</sup> In this scenario the maintenance of sustained (continuous) atrial fibrillation was attributed to continuous stimulation of the atria by this tachycardia emanating from the pulmonary veins. Second, some cardiologists have now reported that they can cure more than 80% of continuous atrial fibrillation with simple pulmonary vein isolation alone.<sup>11</sup> On the basis of our concept of the mechanisms involved in continuous atrial fibrillation that is simply not possible, although it would be if the pulmonary vein tachycardia hypothesis was correct. Thus, the crux of the controversy regarding the proper treatment of continuous atrial fibrillation is the identification of exactly what the drivers are that keep the atrial fibrillation

going indefinitely and where those drivers are located. The contemporary concept to which most interventional cardiologists subscribe is that the drivers are inside the pulmonary veins, and therefore pulmonary vein isolation alone is a rational treatment for continuous atrial fibrillation. Our concept is that the drivers are actually the established macroreentrant circuits in the atria that are capable of sustaining themselves because of the well-documented phenomenon described by Allessie<sup>12</sup> called “electrical remodeling” of the atria. In our concept the macroreentrant circuits in the atria would have to be abolished to cure continuous atrial fibrillation, and simple pulmonary vein isolation alone has little or nothing to do with its cure.

The first problem with the pulmonary vein tachycardia concept is that the tachycardia is not constant. That being the case, during the interludes when this pulmonary vein tachycardia is not present, what drives the atrial fibrillation, and why does the atrial fibrillation not stop at that point? No answers to these questions have been provided. The second problem is that if this hypothesis were correct, then the success rate for pulmonary vein isolation alone should be the same in patients with continuous atrial fibrillation as in patients with intermittent atrial fibrillation. In reality, the success rates, when precise and complete surgical lesions are used to encompass the pulmonary veins, are wildly different, being in the range of 90% for intermittent atrial fibrillation and 15% to 25% for continuous atrial fibrillation.<sup>2,6</sup> This simple observation alone is enough to refute the concept that continuous atrial fibrillation is maintained by drivers located in the pulmonary veins. But it does lend intrigue to the question of how a few cardiology centers can report success rates of more than 80% using pulmonary vein isolation alone to treat continuous atrial fibrillation.

It is important to remember that there is such a thing as surgical precision. When we surgeons report our results for pulmonary vein isolation alone, regardless of the energy source used, we are reporting the effects of placing a precise line of ablation around the pulmonary vein orifices. No such precision exists when cardiologists encircle the pulmonary veins with an endocardial catheter. After the advent of catheter ablation as a treatment for atrial fibrillation, I performed maze procedures on approximately 35 patients in whom prior catheter ablation procedures had failed, thereby providing direct observation of the inside of the left atrium after the attempted catheter ablations. The typical findings at the time of the operation in these patients was diffuse generalized scarring of the majority of the endocardium of the left atrium as a result of the previous catheter ablation attempts. I have never once seen a linear lesion of any type in any patient who has undergone a previous catheter ablation for atrial fibrillation. Moreover, I have yet to find a single surgical colleague who has ever seen a linear lesion inside the left atrium in any patient who has undergone a

catheter ablation attempt at treating atrial fibrillation. This is true whether the patient had undergone an attempt at pulmonary vein isolation alone with a catheter or the addition of other supposedly linear lesions that mimicked some or all of the maze procedure. This observation has profound implications in terms of what interventional cardiologists are really doing to the inside of the left atrium in patients with atrial fibrillation. Their patients are not receiving a simple pulmonary vein isolation procedure as one would commonly envision that operation, but rather virtually the entire inside of the patient’s left atrium is being obliterated, or at least it was in the 35 left atria that I observed. This is an entirely different interventional procedure in which so much of the left atrium is destroyed that it is incapable of fibrillating; that is, the atrial macroreentrant circuits have all been ablated because the atrial myocardium that harbored them has been obliterated. The successful ablation of atrial fibrillation in these patients is understandable because dead atria do not fibrillate. Unfortunately, the patients who are undergoing this procedure by cardiologists are almost certainly unaware of the level of destruction that is being created inside their left atria and that the lesions there bear no resemblance whatsoever to a simple line of scar around the pulmonary veins. Ideally, the interventional cardiologists performing these procedures are unaware of this fact as well. Regardless, this is the only way that 90% of continuous atrial fibrillation can be cured by what is billed as pulmonary vein isolation alone.

There are some simple steps that would correct this type of confusion and suboptimal clinical care, and they would also clarify the optimal method for treating patients with different types of atrial fibrillation. The first is to agree on the proper terminology, or classification, that would provide an accurate and meaningful diagnosis preoperatively. We have previously pointed out that although the new American Heart Association/American College of Cardiology classification of atrial fibrillation<sup>13</sup> is undoubtedly very helpful to physicians who treat atrial fibrillation with drug or pacing therapy, it is actually detrimental to the accuracy and understanding of treatment outcomes when used by physicians, surgeons, or both who treat atrial fibrillation with the creation of anatomic mechanical lines of conduction block (catheter ablation or surgical intervention).<sup>8</sup> The most important information for interventional cardiologists and surgeons is whether the patient has atrial fibrillation intermittently or continuously because that dictates the pattern of lines required for optimal results. The second issue on which we need to agree is that the triggers for intermittent atrial fibrillation are usually, although not always, within the pulmonary veins and that the drivers that sustain continuous atrial fibrillation are usually, although perhaps not always, the macroreentrant circuits in the atria. These 2 steps would allow the following simple, clear, and optimal interven-

tional-surgical approach to be taken in all patients with atrial fibrillation:

1. All patients with atrial fibrillation should have their pulmonary veins isolated.

2. Patients with intermittent atrial fibrillation should have additional lesions to preclude the development of atrial macroreentry in the 10% of patients with triggers outside the pulmonary veins unless the additional application of such lesions cannot be technically created or represents an additional hazard to the patient.

3. Patients with continuous atrial fibrillation should have additional lesions to ablate atrial macroreentry.

If interventional cardiologists and surgeons would follow these simple guidelines, their clinical results would improve. Our combined efforts could then be focused on determining what additional lesions are essential for success and how best to apply those lesions. Unfortunately for the patients, these additional lesions are difficult to apply through a catheter, and therefore interventional cardiologists are left with the choices of (1) obtaining suboptimal overall results, especially in the 40% of patients with continuous atrial fibrillation; (2) being more aggressive with their ablation attempts and damaging large portions of the left atrium; (3) redefining the basic mechanisms of atrial fibrillation so that the additional lesions appear to be less important than they actually are; or (4) sending their patients for surgical intervention. The most critical aspect in this field at the present time is the controversy surrounding the basic mechanism of how atrial fibrillation is induced and sustained, and our ultimate understanding of this issue has profound social, medical, and financial implications for the future.

## References

1. Fasol R, Meinhart J, Binder T. A modified and simplified radiofrequency ablation in patients with mitral valve disease. *J Thorac Cardiovasc Surg.* 2005;129:216-8.
2. Haissaguerre M, Jais P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med.* 1998;339:659-66.
3. Cox JL. The surgical treatment of atrial fibrillation: IV. Surgical technique. *J Thorac Cardiovasc Surg.* 1991;101:584-92.
4. Cox JL, Ad N. The importance of cryoablation of the coronary sinus during the Maze procedure. *Semin Thorac Cardiovasc Surg.* 2000;12:20-4.
5. Cox JL. Atrial fibrillation II: rationale for surgical treatment. *J Thorac Cardiovasc Surg.* 2003;126:1693-9.
6. Oral H, Knight BP, Tada H, et al. Pulmonary vein isolation for paroxysmal and persistent atrial fibrillation. *Circulation.* 2002;105:1077-81.
7. Melo J, Adragao P, Neves J, et al. Surgery for atrial fibrillation using radiofrequency catheter ablation: assessment of results at one year. *Eur J Cardiothorac Surg.* 1999;15:851-4.
8. Cox JL. Atrial fibrillation I: a new classification system. *J Thorac Cardiovasc Surg.* 2003;126:1686-92.
9. Morady F. Catheter ablation of supraventricular arrhythmias. *PACE.* 2004;27:125-42.
10. Oral H, Knight BP, Ozaydin M, et al. Mechanistic significance of intermittent pulmonary vein tachycardia in patients with atrial fibrillation. *J Cardiovasc Electrophysiol.* 2002;13:645-50.
11. Kanagaratnam L, Tomassoni G, Schweiker R, et al. Empirical pulmonary vein isolation in patients with chronic atrial fibrillation using a three-dimensional nonfluoroscopic mapping system: long-term follow-up. *Pacing Clin Electrophysiol.* 2001;24:1774-9.
12. Allessie MA. Atrial fibrillation-induced electrical remodeling in humans: what is the next step? *Cardiovasc Res.* 1999;44:10-2.
13. Fuster V, Ryden LE, Asinger RW, et al. ACC/AHA/ESC Guidelines for the Management of Patients With Atrial Fibrillation: Executive Summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to Develop Guidelines for the Management of Patients With Atrial Fibrillation) Developed in Collaboration With the North American Society of Pacing and Electrophysiology. *Circulation.* 2001;104:2118-50.

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