EDITORIAL COMMENT

Atrial Arrhythmias After Cardiac Surgery: Sisyphus Revisited?*

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The treatment of atrial arrhythmias after cardiac surgery has been an area of keen research interest over the last several years (1). Part of the motivation to find effective therapy is monetary. When atrial fibrillation or flutter (AFF) occurs postoperatively, patients remain in the hospital longer, and that costs money (2). When one considers the fact that cardiac surgery is the most common major surgical procedure in the U.S. and that AFF may be responsible for as much as a 30% increase in the real cost of the procedure, it is not hard to understand the attention that this problem has garnered (3). The arrhythmia causes symptoms, but even in patients with left ventricular dysfunction, these are not usually severe. Although there is the real risk of thromboembolic events in these patients, for whom anticoagulation carries an added hazard, the jeopardy period is relatively short, and so the amortized risk translates into a small number of patients who actually have a stroke or major hemorrhage (4). The risk period is brief because this is a self-limited disease in most patients; data from our institution indicate that over 95% of patients who develop paroxysmal AFF de novo after cardiac surgery are in normal sinus rhythm within two months after coronary artery bypass graft surgery (5). Physician concern about sending symptomatic patients home in AFF stalls the flow of patients out of the hospital. Hospital administrators, payers, patients and families prefer early discharge, and so the push for better treatment methods continues.

How does one go about treatment? Because physicians have no clear idea as to the mechanisms that generate the arrhythmia, treatment has been highly empiric. The most common scenario is to try to terminate the arrhythmia after it has occurred. There are several ways to acutely convert AFF to sinus rhythm that are electric as well as pharmacologic (6). The problem with either is a high relapse rate, especially in patients who are very ill and have high sympathetic tone, in whom all of the conditions for arrhythmia reinitiation are present. Thus, acute measures are often coupled with administration of oral antiarrhythmic drugs started either before or after cardioversion. To avoid the potential for antiarrhythmic toxicity, many clinicians have favored the conservative approach of rate control and anticoagulation over the term of the hospitalization, again with the knowledge that most patients will recover sinus rhythm spontaneously when they recuperate from surgery at home. The problem here is akin to the nonsurgical situation: there are no worthwhile trial data whatsoever that give a clear direction as to whether the strategy of rhythm control or rate control strategy is superior for attainment of any clinical end point.

Given the lack of a clear treatment directive, prophylaxis is a highly attractive goal that has been pursued since the inception of modern cardiac surgery. It has been learned from early experience that calcium channel blocking agents and digitalis are not particularly helpful, but that beta-blockade, with or without digitalis, reduces the incidence of the arrhythmia from about 40% for coronary surgery and 60% for valve surgery to 20% and 30%, respectively, for a 50% risk reduction (7). Still, with over 650,000 patients at risk, prevention methods that can reduce the occurrence even further may be worthwhile, and so the search for better methods of prophylaxis has continued. So far we have learned that membrane-active antiarrhythmic drugs have some activity, but it has been difficult to quantitate their benefit. Studies of older sodium channel blocking agents enrolled a relatively small number of patients with diverse clinical profiles, and the magnitude of treatment effect varied widely from study to study, in part because not all had applied proper background therapy (8). Careful quantification of benefit is particularly important in this realm, because these agents have strong potential for harm. Ventricular proarrhythmia is the most feared complication and mandates in-hospital observation during drug initiation. Perhaps more commonly, these drugs can cause malignant bradycardia and the need for cardiac pacing. A small number of these patients may end their hospitalization with a permanent cardiac pacemaker, which is not a good outcome when treating a disease that lasts only weeks and in many patients causes minimal symptoms.

There are several adequately designed and powered clinical trials, recently completed or in progress, that will examine the value of newer antiarrhythmic drugs for this indication, including propafenone, sotalol and amiodarone (9). The latter is the most intriguing candidate. Its track record of efficacy and safety in low doses for the treatment of AFF in other spheres is compelling (10). In addition, it has myriad electrophysiologic effects, some of which should be useful to control the heart rate response if AFF recurs. The oral form of the drug, used alone, does not lend itself

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well to this indication (11). It takes too long to achieve a satisfactory myocardial concentration of the drug. A prolonged period of oral loading preoperatively is necessary to achieve a therapeutic effect, a strategy that is not feasible in most patients, and that may expose patients to a small but finite operative pulmonary risk (12). Enter Guarnieri et al. (13), who reasoned appropriately that administration of the intravenous drug immediately postoperatively might be worthwhile. Although they proved benefit in reducing AFF incidence without substantial risk, the magnitude of the benefit was not large enough to translate into a reduced length of stay. Given the high cost of intravenous therapy ($500 to $700 per day), it is not a strategy to be recommended. However, the signal is clear, and protocols are being designed to extend the period of intravenous dosing and to couple it with oral therapy in some fashion, to maximize the chances of attaining an adequate myocardial concentration within two to five days after surgery, the period of maximum arrhythmia risk.

Is there a solution, or are cardiologists and cardiac surgeons damned to rock-rolling futility? It is unlikely that the problem will go away. Minimally invasive surgical techniques will not eliminate the problem; there now exists data to prove that the incidence of AFF in these patients, when corrected for disease severity, is the same as for those who have conventional sternotomy (14). Physicians are likely to see more attempts to identify drug strategies, perhaps using accessory methods to target patients at highest risk (15). For example, some have advocated the use of signal averaging techniques to measure p-wave duration, followed by more aggressive drug therapy in patients in the highest risk categories (16). Others have explored novel methods of drug delivery such as direct pericardial instillation, or even adding an antiarrhythmic drug to thewitch’s brew known as cardioplegia solution (17).

What of nondrug therapy? Tested in a relatively small number of patients, single or multiple site atrial pacing has not prevented AFF development (18). Specialists have recently learned of the potential value of an internal defibrillation method in which coiled epicardial, temporary wires, placed at the time of surgery, can be used to deliver small amounts of current with minimal discomfort and very good results (19). One can envision “hybrid therapy” in which patients are treated with an oral antiarrhythmic drug and the small number with a breakthrough episode of AFF have their arrhythmia pace terminated (if flutter), or cardioverted via epicardial wires (if fibrillation) as they prepare to leave the hospital (20).

Unfortunately, none of these novel ideas, as intriguing as they are, have been subjected to the rigors of an adequately controlled clinical trial. For now, what should our approach be? I believe that beta-adrenergic blocking agents should be used as soon as possible after surgery to maximize their benefit. When AFF develops, there needs to be a prompt decision about management, either rate or rhythm control, and drugs should be instituted promptly. In informal surveys in our hospital, stays have been prolonged in most cases by needless delays in instituting effective therapies, especially anticoagulation. Patients not only need excellent and careful in-hospital management, but they also need to be watched carefully after discharge. Policies of early dismissal have been associated with readmission rates approaching 40%, and half of these have been for AFF that occurred in its usual time frame but after a short hospital stay (21). Likewise, patients who are treated with antiarrhythmic drugs or anticoagulation need to be reassessed in the two to three months after surgery for their need for continued treatment, because the arrhythmia is likely to remit (5).

Unlike the case of Sisyphus, the king of Corinth notorious for his brigandage, condemned to Hades to have his boulder perpetually roll back to the bottom of the hill, there has been clear progress in the ability to help patients with AFF after cardiac surgery. But push on (and up) we must.

References


