Editorial Comment

High-Energy His Bundle Ablation
A Treatment of Last Resort

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A proportion of patients with atrial fibrillation and other supraventricular arrhythmias remain refractory to medical therapy and require definitive treatment. In some cases, the arrhythmia may be cured by catheter or surgical ablation. Techniques have recently been developed for disruption of the substrate for atrioventricular nodal reentry tachycardia1-3 and atrial flutter,4,5 and a potential surgical cure for atrial fibrillation6 has recently been described. These techniques cure the arrhythmia while allowing preservation of atrioventricular conduction. In many cases, however, the arrhythmia cannot easily be cured; under these circumstances, the physician may consider His bundle ablation, which entails destruction of part of the normal conduction system to achieve control of the ventricular rate. This results in the exchange of one problem, that of rapid ventricular rates, for pacemaker dependency and the continuing loss of the hemodynamic benefits of atrial activity. His bundle ablation is a major step, and the balance of benefit and potential risk needs very careful evaluation.

High-energy direct current (DC) ablation has been associated with a number of serious adverse effects. The electrical discharge results in gas formation and a high-pressure shock wave that may result in acute perforation and tamponade. In animals with initially normal ventricular function, high-energy DC shocks delivered in the ventricle frequently produced both early and late ventricular arrhythmias, some of which were refractory to defibrillation.7,8 Lower-energy arcing shocks were better tolerated but still showed a definite arrhythmogenic potential. A recent report demonstrated that repeated low-energy nonarcig shocks could produce focal necrosis of the same magnitude as that produced by fewer arcing shocks but with significantly less ventricular arrhythmogenesis.9 New spontaneous and inducible monomorphic ventricular tachycardia has also been documented after high-energy His bundle ablation.10

The impact of high-energy DC ablation on ventricular function is another important issue. Although some studies have demonstrated considerable improvement in general well-being and exercise tolerance after high-energy His bundle ablation,11,12 others have demonstrated impaired or deteriorating performance after ablation.13,14 High-energy discharge has recently been shown to result in acute deterioration15 in left ventricular function although the initial report by Gallagher et al16 showed no such change when assessment was made a few days after ablation. A recent report from Australia17 documented deterioration in ventricular function at 3 months postablation that was not evident at 48 hours. Regional wall motion abnormalities were most marked at the apexes of the ventricles, supporting the hypothesis that it was due to barotrauma. General anaesthesia is required, which may constitute a hazard in itself for patients with previously impaired ventricular or respiratory function. A major disadvantage of the traditional technique is the inability to titrate the energy delivery against the electrophysiological result, making accurate control difficult.

To counter these problems, radiofrequency and low-energy nonarcig DC ablation18 have been developed. Radiofrequency ablation produces small, discrete thermocoagulative lesions and thus allows fine control over the ablative process. It also has the advantage of not requiring general anesthesia. Low-energy DC ablation avoids the risks of barotrauma and allows multiple small shocks to be delivered to achieve similar or better results, and it may also be used safely within the coronary sinus.

Ventricular arrhythmias and sudden death after His bundle ablation have been described previously. The final results of the Percutaneous Cardiac Mapping and Ablation Registry (PCMAR)19 reported sudden death in 1.8% of 552 patients and new ventricular arrhythmias in 32 patients. The results of the prospective Catheter Ablation Register (CAR)20 presented in this issue of Circulation highlight the risks of arrhythmic events after His bundle ablation and have important implications for the future use of the technique. Of 136 patients undergoing high-energy DC ablation, eight (5.1%) died before discharge from the hospital. The majority of these

See p 1924
deaths were due to ventricular fibrillation, often with antecedent polymorphic ventricular tachycardia. The report identifies important risk factors for these catastrophic arrhythmic events. Arrhythmic events were associated with impaired left ventricular function, QT prolongation with or without class Ia antiarrhythmics, and electrolyte abnormalities.

There are two important considerations when reviewing the results of this report. First, in this series the mortality was surprisingly high. With the more widespread use of the technique, it is inevitable that it will be applied to patients with more severe underlying heart disease. In this study, 7% of patients had ejection fractions less than 20%, whereas in the PCMAR series, there was an 8% prevalence. However, data are available only for 29% of the PCMAR patients and in that series, 46% were free of structural disease compared with 32% in this series.20 The two series are therefore not strictly comparable. In the CAR report, four of the patients who died had severe heart failure or cardiogenic shock at the start of the ablation procedure; two of these were receiving inotropic support and one intra-aortic balloon counterpulsation. The very poor outcome of attempted resuscitation from ventricular fibrillation is also surprising. This might have been due to the severity of left ventricular dysfunction with the additional handicap of the absence of coordinated atrial activity.

The mechanism of death in most cases seems to be ventricular fibrillation secondary to polymorphic ventricular tachycardia. Such arrhythmias are frequently secondary to bradycardia and have been reported after interruption of conduction with radiofrequency energy.21 We are left to speculate as to whether the high death rate was due purely to interruption of atrioventricular conduction or to specific effects of the high-energy discharge leading to ventricular arrhythmogenesis. It is possible that high-energy ablation may be arrhythmogenic per se, presumably leading to increased triggered activity in patients already predisposed by QT prolongation, electrolyte abnormalities, and antiarrhythmic drugs in the presence of left ventricular dysfunction. Another possible contributory factor is the relative bradycardia when pacemaker rates are set at 50–60 beats/min.

The second consideration is the clinical implication of these results for future practice. The good safety record for the procedure in patients with structurally normal hearts is confirmed. However, in patients with impaired ventricular function, the risks and benefits must be considered very carefully. In the CAR report, the total mortality was 75% in those with very low ejection fractions. Further studies comparing continued aggressive medical therapy versus interruption of atrioventricular conduction using a variety of techniques would be of value in this difficult group of patients. There is limited data available from the CAR register on radiofrequency ablation that suggests a substantially lower efficacy (36% complete heart block) but fewer acute arrhythmic events.22 However, with increased experience, the introduction of larger tip electrodes,23 and tip temperature monitoring, substantially better results can be achieved. Results from two centers24,25 using low-energy nonarcing DC ablation have reported success, defined as complete heart block or modification of conduction to allow ventricular rate control with or without drugs, in 93–100% of a total of 54 patients. These results compare favorably with the 85% success rate of fulgurative DC ablation reported by PCMAR20 and CAR21 in which complete heart block was produced in 83%.

What should be our current policy toward interruption of atrioventricular conduction? Where possible, it should be avoided; specific curative ablation of the arrhythmia substrate should initially be considered. If this cannot be achieved, radiofrequency or nonarcing low-energy DC ablation might be considered first choices, especially in patients with structural heart disease. Given the evident risks and disadvantages associated with high-energy DC His bundle ablation, we would recommend its use only if radiofrequency or low-energy DC techniques have been unsuccessful.

If interruption of atrioventricular conduction (by whatever technique) is to be performed, it seems prudent to assess the QT interval before ablation. If prolonged, extra care should be taken after the procedure, particularly if there is a history of ventricular arrhythmias. Antiarrhythmic drugs, particularly those likely to prolong repolarization, should be withdrawn well before ablation and avoided afterward, if at all possible. Careful attention should be paid to electrolyte levels before and after the procedure. A ventricular pacing rate of at least 80 beats/min will help to reduce the chances of polymorphic ventricular tachycardia that may occur after ablation even using radiofrequency energy. A prolonged period of monitoring in the hospital should be considered for patients thought to be at risk of arrhythmic events. With scrupulous attention to these details, the potentially large mortality rate after His bundle ablation should be minimized.

The publication of this report20 comes at a time when the technique of high-energy His bundle ablation has already been partially overtaken by newer and potentially safer methods of energy delivery. These more modern techniques will need equally careful long-term evaluation.

This important report serves to reinforce the notion that catheter ablation is by no means a safe procedure. However, with careful patient selection, the adoption of less damaging ablation techniques, and careful attention to detail, the undoubted benefits of ablation can continue to be enjoyed by large numbers of patients without putting them at undue risk.

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