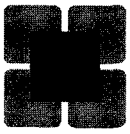

Catheter ablation: a 'charge' into the future

OPTIONS for the treatment of drug-refractory arrhythmias have expanded considerably in the past few years. In this issue of the *Cleveland Clinic Journal of Medicine*, Simmons and associates report excellent results with minimal morbidity in their experience with catheter ablation of the atrioventricular (AV) junction.

■ *See Simmons and associates (pp 223–228).*

Catheter ablation of the AV junction is part of the rapidly growing field of interventional electrophysiology. The first attempts to incise or ligate the His bundle were made in 1967. These techniques reliably produced complete AV block. Subsequent cryosurgical techniques were also very effective. The first report of catheter ablation of the AV junction in man came in 1979. It resulted from the accidental delivery of a direct current shock via an electrode catheter. By 1982, several investigators had reported creation of complete heart block in patients via catheter ablation. The early 1980s saw the rapid advancement of the use of catheter techniques for arrhythmia management. In 1983, the technique was successfully applied to management of ventricular tachycardia. Since then, there have been numerous reports of successful catheter ablation for a host of cardiac arrhythmias.[1]

The mechanism by which direct current (DC) catheter ablation modifies electrical substrates is not completely understood. A standard DC source is used to deliver a high energy electrical charge to the desired target. The charge is typically delivered as a damped sinusoidal wave form.[2] Determinants of the amount of tissue injury include electrode contact and the total energy delivered. Neither barotrauma nor thermal energy appears to play a major role in tissue injury,[3] but barotrauma is certainly responsible for some complications seen after catheter ablation.[2]

More important determinants of tissue injury include local current density and dielectric breakdown of the myocardial cells.[2,3] Histologically, successful AV nodal ablations reveal fibrosis (with or without cartilage formation) not only in the AV node, but also in the His bundle and proximal bundle branches.[4] This finding is suggested by Simmons and associates. The escape rhythm in their successful ablations was more likely to be wide complex and, presumably, infra-His in origin. The escape rhythm reported by Simmons and colleagues averaged 37 ±12 beats per minute; the average escape rate in the International Percutaneous Cardiac Mapping and Ablation Registry (PCMAR) was 46 ±12 beats per minute.[5]

The technique described by Simmons and associates is similar to others published. Because an adequate His signal may be difficult to achieve after the first shock, many laboratories routinely deliver at least two shocks to ensure successful ablation.[6]

The indications for AV nodal ablation have been reported in several large registries[7-12] and agree for the most part with those in the group treated by Simmons and associates. Simmons' patients had a higher incidence of ablation for ectopic atrial tachycardia (20.6%) compared to the other registries, which report 12 to 17%.[12] Simmons' patients were unresponsive to, or intolerant of, a mean of 4 ±1 antiarrhythmic agents (alone or in combination); other series reported inadequate responses to 3.5 to 4 drugs per patient.[3]

The PCMAR reports a success rate of 80% for His bundle ablation in only one session, using 50 to 500 J.[13] Simmons and coworkers report no significant differences with respect to number of shocks used or cumulative energy delivered between those in whom complete AV block was maintained and those in whom AV conduction resumed. These results are consistent with the PCMAR data. Simmons' hypothesis, that normal myocardium may require higher energy for successful ablation, is certainly intriguing. Although 47% of the

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patients in the PCMAR had no organic heart disease, it was not reported whether this population required additional shocks for successful ablation (or if AV nodal conduction was more likely to resume at a later time). As yet, the evidence is inadequate to recommend routine use of higher energies in the population with no organic heart disease. We certainly agree that this issue deserves further investigation.

The Simmons group identifies preablation electrophysiologic characteristics that predict a successful outcome. There was no significant difference in His amplitude in their patients with complete heart block compared to those who resumed AV conduction, but 81% of the patients with His potentials greater than 0.3 mV had successful ablation. Criteria that have been associated with a successful procedure are an amplitude of the His potential greater than 0.3 mV, energy delivery of greater than 3 J/kg, and a stable His bundle electrogram.[3] However, others have reported success with low-energy shocks (50 J or less).[14]

Although Simmons et al report a higher complete heart block rate, their overall results are similar to other published reports.[3] If one considers all successful outcomes (where complete heart block is induced, or AV conduction recurs but the patient is asymptomatic with or without antiarrhythmic therapy), several series report 82-86% good or excellent arrhythmia control.[9-11]

Complications of direct current AV junctional ablation include transient hypotension, cardiac perforation with tamponade, transient junctional and ventricular arrhythmias, sepsis, pericarditis, pneumothorax-hemothorax, subclavian vein thrombosis, pulmonary embolism, and late sudden death.[8] The Simmons group noted only deep vein thrombosis and short runs of ventricular tachycardia (one sustained episode of torsade de pointes was determined to be a proarrhythmic effect of quinidine) and problems associated with preexisting pacemakers. Their low complication rate is certainly a testament to the operators and their judicious practice of performing repeated shocks at a second or third ablative procedure.[1]

The pacemaker complications reported by the Simmons group were related to permanent pacemakers placed prior to the ablation procedure. Others have reported malfunction of existing pacemakers after the procedure,[15] but there is no consensus on how to handle the problem. General guidelines for safety with permanent pacemakers include complete pacemaker analysis prior to and after the procedure and a reliable temporary pacing system in the event of device malfunction. The pacemaker should also be reprogrammed to a slow rate and with a low amplitude. This permits access to the effects of ablation on the AV node and avoids pacing during the vulnerable period in the event of sensing failure. Ideally, the indifferent ablation electrode should be positioned so that an electric field orthogonal to the line joining the pacemaker active electrode to the generator is produced.[15] One of our colleagues has suggested explantation of the generator prior to ablation to protect it from damage. It could then be resterilized and reimplanted. This approach may be cost-effective, but we believe that the potential morbidity of routine explantation is unwarranted.

Simmons' long-term follow-up was similar to other reported series.[3] The PCMAR had a 1.6% incidence of late sudden death unrelated to pacemaker failure, which the Simmons group did not observe. This may be related to the longer follow-up in the registry group (23 ±18 months) compared to 14.6 ±15 months for the Simmons patients.

DC ablation of the AV junction in the treatment of supraventricular tachycardia has three important limitations. First is the requirement of (and often dependency on) permanent pacing. Second, even with successful AV junctional ablation, patients may still be symptomatic from the rapid atrial contractions occurring in AV nodal reentry tachycardia or atrial flutter.[16] The third limitation is the previously noted morbidity associated with direct current ablation.

Many of the limitations of DC ablation can be overcome with alternate energy sources. The use of radiofrequency energy promises to make catheter ablation a powerful tool in the management of supraventricular tachycardia. Ectopic atrial tachycardias and accessory pathways may be ablated.[17,18] AV nodal reentry tachycardia may be eliminated[19,20] (albeit with a small risk of complete heart block requiring permanent pacing). Complete AV block can be achieved in patients with drug-refractory atrial fibrillation and flutter. Multiple ablative lesions may be delivered with minimal patient morbidity.[21] As these techniques become more mainstream, they appear certain to diminish the need for pharmacotherapy and the indications for surgical intervention.

Surgical options for the treatment of supraventricular tachyarrhythmias are well summarized in a recent symposium.[22-25] Surgical approaches have the potential for morbidity and mortality associated with open chest procedures, but are potentially curative without the need for a permanent pacemaker.

Atrioventricular nodal reentry tachycardia has been

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treated successfully with cryosurgery. This technique has the advantage of eliminating the reentrant circuit without damaging antegrade AV nodal conduction; it therefore eliminates the need for permanent pacing. A cure rate of 100% has been reported.[22] Direct surgical dissection of the AV node is another option, with series reporting 84 to 100% cure rates.[23,24]

The Maze procedure (so called because it creates a "maze" of electrical conduction routes in the atria) has been investigated for the treatment of refractory atrial fibrillation. Two of the three primary conduction pathways between the sinoatrial (SA) and AV nodes are divided; macroreentry is theoretically prevented. Impulses may travel in only one direction, and conduction precedes antegrade via the AV nodal/His Purkinje system.[25] Although preliminary reports are very encouraging, we believe that it is too early to be certain of the procedure's overall antiarrhythmic efficacy and its ability to preserve atrial function.

The corridor procedure (which isolates a strip, or corridor, of atrial septum from the SA node to the AV node) abolishes only one of the three detrimental effects of atrial fibrillation, the irregular heart beat. The corridor procedure does not restore mechanical AV synchrony and therefore offers no beneficial hemodynamic effects. It leaves the atria fibrillating with the attendant risk of thromboembolism and offers no major advantages over His bundle ablation.[25]

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