Catheter ablation for treatment of tachyarrhythmias: present role and potential promise

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DELIERY OF high-energy direct-current shocks through electrode catheters for treatment of patients with cardiac arrhythmias was first introduced in 1981. Since that time, this experimental technique has become established as a therapeutic tool for selected patients. This essay will review the basic biophysical effects of such shocks, summarize the present clinical indications for this therapy, and speculate on the future directions for interventional electrophysiology.

Biophysical effects of catheter electroshocks. The delivery of 200 to 400 J of stored energy through an electrode catheter results in 2000 to 3000 V potentials at the electrode surface. If the discharge is delivered into a saline solution, an explosive flash develops. This electrical energy is at least partially converted to heat. In addition, the explosion produces concussion waves exceeding 1 to 2 atmospheres of pressure. Finally, the intense electrical charge may disrupt cellular integrity. Thus, local injury after delivered shocks may result from thermal electrocoagulation of tissue, strong concussive shock waves, or membrane damage due to the intense electrical field. One other factor should be emphasized. The structural integrity of the electrode catheter materials to withstand shocks of this magnitude must be assured. Most conventional direct-current defibrillators deliver capacitor discharge over 5 to 10 msec, with peak voltage achieved within 1 to 2 msec. Delivery of 1000 to 3000 V to one electrode of a multipolar electrode catheter may result in insulation breakdown with shunting of current to other electrodes and hence less delivered energy to the tissues. These factors will obviously influence the safety and efficacy of this approach.

The histologic effects of catheter-delivered shocks have been described and we have previously described the evolutionary histology after catheter-delivered shock in the normal canine heart. Anodal and cathodal discharge produce qualitatively similar spherical lesions that are 1 to 2 cm in diameter when total defibrillator energy is limited to 5 J/kg body weight or less. Histologic change is present within 20 min after the shock. The central foci show cellular deformation to heterogeneous globules with loss of cellular detail and pynknotic nuclei. The early lesions have interstitial red blood cells and edema but no inflammatory cell infiltration. Electron micrographs obtained 1 hr after shock show disrupted sarcomeres and numerous mitochondria containing electron-dense clumps within their intercrystall spaces. After 1 to 2 days, the injured regions are invaded by acute inflammatory cells and after 5 to 7 days, there is replacement of necrotic myocytes by an intense organizing reaction involving fibrocytic spindle cells, multinucleated histiocytes, and round cells. Lesions in atrial and ventricular myocardium are histologically identical.

Proarrhythmic effects of catheter discharge have been described at the time of shock and 12 to 48 hr later. In canines, tachyarrhythmias or bradyarrhythmias may occur immediately after shock. Jones et al. found a characteristic response to increasingly intense stimulation of cultured myocardial pacemaker cells. Threshold stimuli caused a single activation but stimulation intensities 24 times threshold values caused transient tachyarrhythmias. With stimuli 42 times threshold, there was arrest of rhythmic activity due to membrane depolarization to zero ("dielectric breakdown") and delayed repolarization. "Cellular fibrillation" or asynchronous contraction of sarcomere followed application of stimuli 80 times the threshold value. Lerman et al. reported spontaneous ventricular tachycardia 24 hr after catheter ablation of canine ventricular endocardium. These tachyarrhythmias were not lethal when delivered energy was 50 J or less. Programmed stimulation does not induce ventricular tachyarrhythmias 3 or more days after endocardial shock.

Clinical role of catheter ablation. To date, the largest available experience relates to use of catheter shocks to electrocoagulate the ativoventricular junction in patients with drug-refractory supraventricular tachycar-
This technique involves placement of an electrode catheter in close proximity to the His bundle. One or more shocks are delivered from the electrode showing the largest unipolar His bundle deflection (cathode) to a conductive plate (anode) positioned behind the left scapula. After an observation period of 24 to 48 hr confirming stable complete atrioventricular block, a permanent cardiac pacemaker is inserted. Since the initial clinical reports of Scheinman et al.\textsuperscript{1} and Gallagher et al.\textsuperscript{2} a wider experience has been accumulated from a worldwide voluntary registry.\textsuperscript{8} To date, approximately 250 attempted atrioventricular junctional ablation procedures have been reported to the registry and data have been analyzed for the first 209 consecutively enrolled patients. The procedure was found to produce chronic complete atrioventricular block in 70\% of patients; in 8\%, atrioventricular conduction resumed but drug therapy was not required for control of arrhythmias. In 13\%, atrioventricular conduction was sufficiently modified so that previously ineffective drugs proved effective in arrhythmia control while the procedure was proved totally ineffective in 9\%.

Immediate complications of the ablation procedure included development of transient ventricular arrhythmias in five patients. In two, direct-current shock was required for defibrillation, while the arrhythmia proved transient in three. Transient hypotension requiring pressors was described in four patients. Transient cardiac asystole followed by electromechanical dissociation occurred in one patient, and cardiac tamponade requiring emergency evacuation of pericardial blood was noted in another. Fortunately, no chronic sequelae have been reported from these complications. Chronic complications reported were similarly uncommon and largely related to either the catheterization procedures or insertion of a permanent pacemaker. One patient developed a large atrial thrombus and in another, hypotension requiring pressor support persisted for 3 days after ablation. A 2\% incidence of sudden death has been reported from 1 to 5 months after ablation. All of these patients had associated cardiac disease (either coronary artery disease or cardiomyopathy) and it is unclear whether the sudden demise was related to the procedure or was a consequence of the underlying heart disease. Catheter ablation of the atrioventricular junction has proved as effective as surgical ablation of the His bundle, with lower morbidity, mortality, and cost.\textsuperscript{10} The catheter procedure has essentially supplanted the need for His bundle ablative surgery in patients with drug-resistant supraventricular tachycardia.

Nonpharmacologic options in the treatment of drug-resistant supraventricular tachycardia. Almost two out of three of the atrioventricular junctional ablative procedures reported to the registry were attempted in patients with drug-resistant atrial fibrillation and flutter. In our practice, these patients are offered amiodarone therapy or catheter ablation of the atrioventricular junction. In patients with either atrioventricular nodal reentry or atrioventricular reciprocating tachycardia other nonpharmacologic options include antitachycardia pacing, cardiac electrosurgery, or catheter ablation of the accessory pathway (see below). Antitachycardia pacing is considered a suitable option if the arrhythmia can be readily and reproducibly terminated without induction of more serious atrial arrhythmias. In patients with drug-resistant atrioventricular nodal reentrant tachycardia, antitachycardia pacing, if effective, is thought to be more desirable than permanent ablation of the atrioventricular junction. In younger patients with drug-refractory atrioventricular reciprocating tachycardia, surgical or catheter ablation of the accessory pathway is the preferable approach since the chance of a cure is excellent and risks are acceptably low. In older patients or in those for whom surgery represents a significant risk, catheter ablation of the atrioventricular junction is an acceptable alternative provided the patient is not at risk for atrial tachyarhythmias with rapid antegrade conduction over the accessory pathway. In these patients, atrioventricular reciprocating tachycardia would be eliminated if the atrioventricular junction is a critical component of the reentrant circuit. These patients should receive permanent cardiac pacemakers since long-term reliability of atrioventricular conduction over an accessory pathway has not been established. Patients without accessory pathways undergoing ablation of the atrioventricular junction are pacemaker dependent, while in those with intact antegrade extranodal accessory pathway, the pacemaker serves as a backup should accessory pathway conduction fail.

Direct catheter ablation of accessory pathways. Only limited experience is available relative to the safety and efficacy of catheter ablation of accessory pathways. Fisher et al.\textsuperscript{11} were the first to attempt catheter ablation of left free wall accessory pathways via the coronary sinus. This approach seldom resulted in permanent modification of the accessory pathway and was associated with coronary sinus rupture and tamponade. Of the 15 attempted coronary sinus ablative procedures reported to the registry, four instances of coronary sinus rupture and one death have occurred. Based on this experience, catheter ablation via the coronary si-
nus does not appear an acceptable clinical technique, especially in view of the reported favorable surgical experience. Too few attempted right free wall ablative procedures have been reported to allow for definitive recommendations at this time.

In contrast to free wall accessory pathways, posteroseptal pathways are accessible to catheter ablation. This procedure involves insertion of a multipolar electrode catheter into the coronary sinus, positioning the distal electrodes just outside the os of the coronary sinus. The shock is delivered from the distal electrode pair (joined as cathode) to an external chest patch (anode). It must be emphasized that only patients with earliest retrograde atrial activation localized to the coronary sinus os are candidates for this procedure. In addition, great care should be taken to ensure that the electrical discharge is delivered just outside the coronary sinus os. It is our practice to visualize the coronary sinus os with injections of contrast medium to ensure proper positioning of the electrode catheter. Our preliminary results suggest that the catheter procedure may be preferable to surgery. An unsuccessful catheter ablative attempt does not preclude subsequent surgical intervention.

Catheter ablation of ventricular foci. Hartzer was the first to report attempted catheter ablation of ventricular tachycardia foci. Although a number of subsequent reports have been published, the experience to date is still quite limited. Ablation of ventricular tachycardia foci is technically the most demanding of the catheter ablative procedures. Endocardial mapping of both ventricles is required during ventricular tachycardia. The catheter is then positioned against the endocardial area showing earliest activation relative to multiple reference surface leads. Shocks are delivered from the electrode to an external chest wall patch. The largest experience has been reported by Fontaine et al. and their initial results have been excellent. Our own experience has been somewhat less favorable. In 12 consecutive patients with ventricular tachycardia undergoing ablation, only three have experienced long-term arrhythmia control without drugs. Six patients have experienced recurrent ventricular tachycardia that is well controlled with drugs that were previously ineffective, while the procedure has proven totally ineffective in three. Serious side effects reported to the registry include malignant ventricular arrhythmias within 1 week after ablation, low-output syndrome resulting in death, and one report of electromechanical dissociation and death after attempted ablation. Catheter ablation for patients with ventricular tachycardia foci must still be considered highly experimental. Suitable candidates might be those with frequent or incessant unimorphic ventricular tachycardia who are considered high-risk surgical candidates.

Future perspectives. The ideal catheter ablative technique involves ability to quickly and precisely localize the target area. The damage should be localized to this area with minimal destruction of surrounding normal myocardium, cardiac valves, or coronary vessels. Presently available techniques fall far short of the ideal in many respects. For atrioventricular junctional ablation, while current techniques appear adequate for precise localization of the atrioventricular junction, the damage induced by direct-current shocks results in damage to surrounding myocardium as well as to the support structures of the tricuspid and aortic valves. The long-term significance of these lesions is yet to be defined. Clearly many improvements are required before catheter ablation of ventricular tachycardia foci becomes clinically acceptable. Firstly, a better understanding of the ventricular tachycardia pathways is required. Current ablative techniques essentially produce a subendocardial lesion. While earliest activation over the subendocardium appears to be the rule in man, detailed animal studies have shown that ventricular tachycardia reentrant pathways are commonly localized to epicardial or intramural sites. Second, current techniques of endocardial mapping are laborious and lack the precision required for localization of ventricular tachycardia foci. Future advances in catheter ablative techniques will depend on a fruitful exchange between physicians and industry engineers and scientists. Physicians are in critical need of readily maneuverable multielectrode catheters designed to allow for rapid simultaneous endocardial mapping of numerous ventricular sites. In addition, catheters and energy delivery systems are required that will deliver quantifiable energy via catheters specifically designed for this purpose and suitable for local cardiac ablation without adverse remote effects.

In summary, the use of catheter ablative procedures for destruction of ventricular tachycardia foci or free wall accessory pathways is still considered highly experimental and the role of these procedures for patients with arrhythmias related to these lesions remains undefined. In contrast, catheter ablation of the atrioventricular junction and possibly posteroseptal accessory pathways are much closer to being recognized as accepted clinical tools for patients with drug-resistant supraventricular arrhythmias. Performance of catheter ablation of the atrioventricular junction is technically easy and involves equipment available in any cardiac catheterization laboratory. Perhaps more difficult is
deciding when the ablative procedure is superior to other available nonpharmacologic techniques. Sacrifice of the normal conducting system and induction of a pacemaker-dependent state should never be undertaken lightly. The art of medicine is the proper application of the physicians’ experience and knowledge. Catheter ablative techniques are still experimental and should be performed by physicians who are experienced both in the technical requirements and, more importantly, in evaluating the risks and benefits of all available therapeutic options.

References

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