OCCASIONAL REVIEW

'Map and zap' — electrode catheter techniques for treatment of supraventricular tachycardias

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Cardiac electrophysiologists around the world have become increasingly interested in the use of electrode catheters to ablate critical areas involved in the genesis and transmission of supraventricular tachycardias (SVTs). This interest has been stimulated by the demonstration that accessory atrioventricular (AV) connections in patients with Wolff-Parkinson-White (WPW) syndrome can be selectively damaged by radiofrequency energy (RF) delivered via catheters inserted percutaneously without general anaesthesia. The technique and results of its use were the main topics of interest at the 9th World Symposium on Cardiac Pacing and Electrophysiology held in Washington, DC, in June 1991. Effective cure of WPW syndrome entailing freedom from tachycardias without drug treatment has been reported in more than 90% of patients undergoing this procedure.[1] Not surprisingly, this has generated a great deal of excitement as previously these patients required either lifelong treatment with potentially toxic anti-arrhythmic drugs or open-heart surgery to ablate the accessory AV pathways responsible for the tachycardias.[2] The SVTs which are amenable to treatment with RF current include atrial fibrillation and flutter with uncontrollable ventricular response (AV node ablation), the WPW syndrome (accessory pathway ablation) and the extremely common form of SVT, AV nodal re-entrant tachycardia (AV node modification).

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Practical techniques for the control of SVTs by means of catheter ablation were first described in 1982.[3,4] High-energy DC shocks from a defibrillator were transmitted via an electrode adjacent to the His bundle. The aim was to induce complete heart block to allow control of the ventricular rate in patients with atrial flutter or fibrillation, otherwise uncontrollable by drugs. Successful induction of complete heart block required the implantation of a permanent pacemaker. General anaesthesia was required because of the pain and muscle spasm induced by the DC shock. The high energies (200 - 300 J) resulted in arcing at the catheter tip and vaporisation of blood that produced transient pressures up to 20 atmospheres.[5] The resulting barotrauma may have been responsible for ventricular dysfunction and the production of arrhythmogenic foci. Late sudden death has occurred in a small number of patients.[6] In addition, AV conduction recurred hours to months later in a sizeable number of patients, presumably because the AV node or proximal bundle of His had been stunned rather than destroyed.

Alternative energy sources were therefore developed. Modification of the output of the defibrillator allowed the use of much lower energies (20 - 50 J), avoiding the barotrauma while preserving the high-voltage heating effect of the shock.[7] This method is still in use, whereas high-energy DC shock has largely been abandoned. In 1987, the use of RF energy from standard surgical cautery machines was reported.[8,9] It was shown that small, discrete lesions could be produced, providing the catheter was in contact with the endocardium and successful ablation of the AV node could be achieved.[10] Because RF energy does not affect nerves or muscles, general anaesthesia is not necessary. The homogeneous lesions produced seem less likely to result in new arrhythmias. The safety of the technique was somewhat offset by a lower efficacy, probably because of the build-

up of resistance due to charring of blood at the electrode tip and because the smaller lesions required more precise positioning of the electrode. These problems have been addressed by the development of specially designed electrodes, which can be steered and have a larger surface area. These have improved the success rates.[11] Return of conduction and late arrhythmias and depression of cardiac mechanical performance have been virtually eliminated.

High-energy DC shocks were tried in patients with WPW syndrome, initially with limited success and significant complications, particularly in those with left-sided accessory pathways. However, more recently Warin *et al.*[12] reported impressive results in a large series of patients. They ablated left-sided pathways by placing the catheter in the left atrium or the left ventricle adjacent to the mitral annulus. Nevertheless, it was the advent of RF ablation that led to the current widespread use of catheter ablation to treat patients with WPW syndrome. Pathways in all locations around the AV ring can now be dealt with successfully. Left-sided pathways are approached by passing the catheter across the aortic valve into the left ventricle and curving it behind the posterior leaflet of the mitral valve; the tip is placed against the mitral annulus. Accurate location of the accessory pathway by electrophysiological mapping of the AV ring is essential and requires a fully equipped electrophysiology laboratory and an experienced electrophysiologist. Procedure times of 4 - 6 hours are usual and may be considerably longer, but a high success rate can be anticipated. A successful result means that the patient is effectively cured without the need for surgery or anti-arrhythmic drugs. This is both cost-effective and gratifying in a condition which results in significant morbidity and occasionally causes sudden death.[13] Complication rates appear to be low[1] and recurrences uncommon.

Where does this leave surgical treatment of WPW syndrome? Despite the high success rates reported, it is acknowledged that not all patients can be successfully treated by catheter ablation. The treatment of those with multiple accessory pathways and associated defects such as Ebstein's anomaly of the tricuspid valve is more likely to fail. So far, RF catheter ablation of accessory pathways has been attempted in 14 patients with WPW syndrome in South Africa and was successful in 8; 2 were successfully operated on after catheter ablation failed, 1 of whom had Ebstein's anomaly with two accessory pathways. There is undoubtedly a learning curve with reported initial success rates around 50%[14] and improved results are anticipated with greater experience. This, together with a relatively low complication rate, augurs well for the future.

The other common cause of paroxysmal SVT is reentry within the AV node. While this usually responds well to agents which delay AV nodal conduction (β-blockers, verapamil, digitalis), it occasionally becomes a major clinical problem and fails to respond even to the more toxic anti-arrhythmic agents such as disopyramide, propafenone or amiodarone. It is now possible to modify the upper part of the AV node by RF energy via a catheter so that the retrograde pathway is selectively damaged.[15] This prevents further episodes of tachycardia while leaving AV conduction intact. However, there is a risk of about 10% that antegrade transmission will also be damaged, resulting in AV block and the need for a permanent pacemaker. It is to be hoped that refinements in technique will reduce this risk so that the procedure can be offered to more patients.

While these 'high-tech' solutions may seem inappropriate in the economic and social climate of present-day South Africa, it should be remembered that the people affected by these conditions are usually young and economically active. A procedure which offers freedom from daily medication and the fear of breakthrough tachycardias has much to recommend it.

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