Ventricular tachycardia: is it a burning issue?

The advent of radiofrequency (RF) ablation has transformed electrophysiological practice in the past 10 years. RF ablation is now considered first line treatment for many supraventricular arrhythmias. Buoyed by this success the eager ablator has turned to ventricular tachycardia (VT).

The first ventricular arrhythmias to fall convincingly to RF ablation were the focal VTs occurring in patients without evidence of structural heart disease; 70% arise in the ventricular outflow tract, usually the septal surface of the right ventricular outflow tract. This arrhythmia is typically catecholamine dependent, adenosine sensitive, and probably caused by cyclic AMP mediated triggered activity.

The usual clinical picture is of a young woman with symptomatic ectopic beats of left bundle and inferior axis morphology and with non-sustained VT on exercise (fig 1). Success rates for RF ablation in this condition are more than 90%.

Occasionally, VT may arise in the left ventricular outflow tract or the epicardial right ventricular outflow tract and must be approached differently. Left septal (fascicular) VT (fig 2) arises in relation to a portion of the specialised conducting tissue, usually the left posterior fascicle. Like idiopathic outflow tract tachycardia it is exercise related, but it is reentrant in mechanism, verapamil sensitive, and of right bundle morphology.

Successful ablation of any arrhythmia requires adequate mapping and subsequent delivery of energy to produce a lesion. In focal VT where there is radial spread from a point source, localisation of the source can be performed by finding the site of earliest ventricular activation or reproduction of an identical 12 lead ECG by pacing from the site. Both of these techniques are robust guides to ablation in VT in structurally normal hearts but, even including the occasional patient with bundle branch reentry, such patients will contribute less than 10 procedures per year at most electrophysiology centres.

The dominant cause of VT is myocardial infarction and the options for mapping and radiofrequency ablation for these patients are very different. Postinfarction VT is caused by reentry within complex three dimensional structures of electrically inert scar and functioning myocardium. Within the scar region there may be several potential...
circuits with different exits into the surrounding myocardium yielding different 12 lead morphologies. Ablation at sites guided merely by conventional activation or pace–mapping is rarely successful.

The use of pacing manoeuvres during VT at sites in or close to the critical reentry circuit is very helpful for detailed mapping of postinfarct VT circuits. Such entrainment mapping identifies critical zones (entrance, slow conduction zone, exit) within the reentry circuit that can be targeted by RF ablation (fig 3). The need to perform activation and entrainment mapping in VT means that patients eligible for RF ablation of VT are those with haemodynamically well tolerated VT. Such stable VT patients are only about 5% of the total population with postinfarction VT. For them, RF ablation is an attractive curative management option. The other 95% of patients are arguably those with most to gain by curative ablation procedures. Their prognosis is worse than those with tolerable VT and, by definition, they are more severely symptomatic. It is difficult to perform conventional mapping if VT is haemodynamically unstable, although multipoint mapping systems may identify an RF target location from a single provoked episode or even a spontaneous beat of identical morphology.

Schilling et al have reported their experience of a novel non-contact multipoint mapping system to guide RF ablation of VT. They report remarkable results that herald a major advance in the management of these difficult patients. Even without this technology, fast VTs can occasionally be mapped conventionally by slowing the arrhythmia pharmacologically; however, drug administration may temporarily mask unsuspected but potentially important arrhythmias.

Developments in mapping approaches are being accompanied by advances in ablation technology. Catheters that can produce large lesions by microwave, ultrasound, cryo-therapy, and cooled tip RF energy are all under development and will contribute to the growing success of VT ablation. Even with these catheters, some technique for localising the arrhythmia is crucial. The ability to produce larger amounts of myocardial damage will also bring with it the challenge of control to prevent damage to essential structures.

A different but very attractive approach for poorly tolerated VT is to map and ablate without the arrhythmia being present. The target zone is then defined by a combination of anatomical features, sinus rhythm electrogram characteristics, and pace–mapping. Preliminary results are encouraging but questions remain. How do we select suitable patients? What are the risks? Is it a practical procedure that can be performed in any well equipped ablation centre? Is the cure permanent? Will back up implantable cardioverter defibrillator treatment be necessary in every patient?

What role does RF ablation play in patients with VT? In the few patients who have VT without structural heart disease, enormous symptomatic benefit can be derived from a procedure that offers cure with a success rate comparable to any other ablation procedure. RF ablation for postinfarction VT that is well tolerated is very effective, and for the small group with incessant VT it is the treatment of choice. But for the majority, it remains experimental and is often an adjunctive procedure. Whether RF ablation has a role in most patients with scar related VT we are burning to discover.

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