Understanding atrial arrhythmia mechanisms by mapping and ablation

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Received 14 September 2012; accepted after revision 21 September 2012

This editorial refers to ‘Catheter ablation of scar-related atypical atrial flutter’ by J.O. Coffey et al., on page 414

Coffey et al.1 report in this issue of the Journal the results of mapping-based ablation for atypical atrial flutter in 91 patients, 59 (65%) of whom had prior catheter ablation for atrial fibrillation (AF), 11 (12%) a surgical maze procedure for AF, and 14 (15%) other type of cardiac surgery. Only seven patients (8%) had ‘idiopathic scars’, i.e. areas of low voltage of unknown origin, as the substrate for the arrhythmia.

It is perhaps pertinent to note the limitations of the electrocardiogram diagnosis. The term ‘flutter’ is used to describe rapid atrial tachycardias (AT) with an undulating waveform without isoelectric baseline; however, this has been overlooked by many groups reporting left AT that, as in Coffey’s paper, show rather long cycle lengths, around 300 ms, and longer isoelectric baselines between P-waves. Since the term ‘flutter’ is so closely related to cycle lengths, around 300 ms, and longer isoelectric baselines, it tends to suggest a macroreentrant mechanism, and the term ‘atrial tachycardia’ would appear more appropriate, as it does not pre-judge the mechanism.2 In fact, around one-third of the mechanisms described in this study were focal discharges.

The mapping technique, leading to ablation of the critical isthmus or the focus responsible for the arrhythmia, is remarkable because it creates excellent activation maps on the virtual atrial anatomy and allows, with some limitations, better understanding of the arrhythmia mechanism. Reduction of pacing manoeuvres to the minimum necessary to confirm the role of critical areas of the circuit minimizes the chances of altering activation sequences, a significant concern in left atrial macroreentrant AT, in which complex combinations of anatomic and functional obstacles can support multiple reentrant circuits and/or focal mechanisms. The average number of AT mechanisms encountered was 1.9 per patient, and 21 patients had three or more AT mechanisms, underlining the importance of this method.

Another point of interest is the extremely low voltage recorded at some of the critical isthmuses that would suggest that activation could course over thin epicardial layers of atrial myocardium over endocardial scars. The interpretation of these local electrograms would be very difficult in the absence of the full activation maps supported with critical entrainment runs to confirm participation of adjacent areas in the circuit. The complexity and low amplitude of the electrograms would make quite impossible to reliably measure local post-pacing intervals at these critical isthmuses.

Atrial tachycardia mechanisms were based in both atria and one-third were called septal. Two-thirds of the AT had a macroreentrant mechanism, and about one-third were focal. It is not clear from the paper if combinations of focal and macroreentrant mechanisms were found in the same patient and if more than one AT could be localized at distant sites in the same patient, but the report does give the general impression of dealing with severely diseased (remodelled! injured!) atria. The worse performance with ‘septal’ AT raises questions about the ability to precisely locate foci or critical isthmuses in this anatomically complex area. This seems particularly relevant because fragmented electrograms are often recorded from the septal atrial walls3 and this would make these areas a target for ablation.

The fact that atriotomy scars can become the centre of a macroreentrant arrhythmia is well known. Electrophysiologists have helped the surgeons learn to bring one of the ends of an atriotomy to an anatomic obstacle (inferior vena cava, mitral annulus) to prevent activation rotating around scars, and hopefully this may have prevented at least some post-operative macroreentrant AT. However, the world turns around and today catheter ablaters often feel free to create ill-defined lesions with or without connections to fixed obstacles, under the assumption that fragmented electrograms may represent the presence of rotors sustaining electrical activity.4 Lines may also be ablated across the left atrial roof or between the left inferior pulmonary vein and the mitral valve in an effort to prevent macroreentry.5 The arrhythmogenic potential of these ablation lesions has been well shown.6,7 Experimental observations have disclosed the potential for the creation of slow conducting pathways between necrotic areas.8 Over two-thirds of patients in this study had one or more AF ablation
procedures. The report does not allow a conclusion as if macro-
reentrant mechanisms were related to wider myocardial ablation
strategies.

The high prevalence of focal mechanisms merits comment, as in
Coffey’s cases it probably originated from damaged myocardium,
after pulmonary vein reconnection was ruled out or corrected.
We know very little about the potential of ablation lesions to
promote focal activity and, in contrast to macroreentrant circuits
which could be prevented by transmural lesions appropriately con-
ected to fixed obstacles, we have no clues as to the ablation strat-
egies to prevent the appearance of focal activity. A significant
question is to what extent the focal mechanisms found in these
patients are the result of previous ablation or of the persistence
of myocardial abnormalities underlying AF that have not been cor-
rected by the ablation procedure(s).

Patients with ‘idiopathic scars’ had the worst prognosis, which is
in keeping with our lack of data on the significance of those low-
voltage areas, called ‘scars’, that could be just a local manifestation
of a diffusely diseased atrial myocardium.9 Atrial tachycardia post-
catheter ablation of AF had the next worse prognosis, which may
be related to the absence of well-defined design of ablation lines,
points, or areas, in fragmented electrogram ablation. Patients
with surgical incisions fared best, perhaps indicating a ‘cleaner’
design of the scars produced by incisions and/or cryoa blation.

Finally, the clinical results in terms of maintenance of sinus
rhythm after multiple ablation procedures inevitably have to
make us think again if we can keep pursuing a cure of AF exclusive-
ly through catheter ablation. Coffey et al. feel rather satisfied with
their ‘long-term’ results (16 ± 12 months); however, the Kaplan–
Meier curve in Figure 4 may leave room for thought, as (few?)
patients followed-up for 3 years approached a recurrence rate of
50%. Perhaps in many cases of AF we should start thinking of ab-
lation as adjunctive palliation, rather than cure, and this approach
could give the patients and the doctors a more realistic view.
Perhaps our clinical, long-term results would be better if we sys-
tematically used a multi-pronged approach that would not
exclude antiarrhythmic drugs in the appropriate patients. Early, vig-
orous intervention on the pathogenetic factors underlying AF
should be an essential aspect of this multi-disciplinary approach.10
This would not exclude an earlier application of ablation proce-
dures in those cases of AF with better perspectives for a favour-
able response.

Conflict of interest: none declared.

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