Editorials

Incessant ventricular tachycardia: a lost case or new hope?

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Ventricular tachycardia remains a difficult problem in spite of all the recent advances. Anti-arrhythmic drugs continue to fail in more and more trials, and it is unlikely that we will have effective (and safe) drugs based on new principles in the near future. In contrast, the results of implantable cardioverter/defibrillator therapy remain very encouraging, in particular in patients with ventricular tachycardia. Furthermore, implantable anti-arrhythmic devices are being continually improved, and implantable cardioverters/defibrillators now resemble the shape of the early pacemakers. Therefore, it is very tempting to skip anti-arrhythmic drug treatment, and to proceed immediately to implantable cardioverter/defibrillator therapy. However, the paper of Cao and Gonska in this issue is an illustration of the fact that such an approach is not always the right one.

If implantable cardioverters/defibrillators give a shock, and terminate a sustained tachycardia, they do what we expect them to do. However, ventricular tachycardia does not always behave as we would like, and when such an arrhythmia becomes 'incessant' this poses a major problem with respect to survival. On such occasions implantable cardioverters/defibrillators usually give multiple shocks or intervene with antitachycardia pacing, but fail to prevent immediate recurrences.

We expect anti-arrhythmic drugs to prevent recurrences but the clinical status of those patients with incessant tachycardia will often deteriorate because of the myocardial depressant or vasodilating effect of the therapy. Drugs are even incriminated in the pathogenesis of the incessant mechanism because they slow conduction, and enhance re-entry, sustaining arrhythmias which would otherwise have been short and sometimes even asymptomatic. This pro-arrhythmic mechanism is well known for drugs such as procainamide, disopyramide, flecainide, propafenone, cibenzoline, and probably all other sodium-channel-blockers. We have also observed several patients who were referred for treatment only after episodic tachycardia became incessant in the course of amiodarone loading, causing severe left ventricular dysfunction. This rather specific pro-arrhythmic property of amiodarone might be the most important one from the clinical point of view. Furthermore, it is now well established that amiodarone increases the defibrillation threshold, and this corresponds with earlier clinical observations. This may be another reason to avoid the use of amiodarone in the setting of incessant tachycardia. Therefore, even amiodarone, a drug that we have used for about 20 years in Europe, has no definite place in the treatment of incessant ventricular tachycardia. It takes (even in the view of those who seem to be believers) at least 4 days to control only about 60% of 'critically ill' patients with recurrent sustained ventricular arrhythmias, before high-dose oral amiodarone controls the arrhythmia (most often in combination with other drugs). This does not address the specific situation of incessant ventricular tachycardia, but highlights the difficult setting in which this kind of patient survives. It also underscores the high frequency with which the combination of anti-arrhythmic drugs is used. Combining drugs is a well-known recipe for torsades de pointes, which can lead to haemodynamic deterioration, or may require repeated defibrillation and can ultimately result in death.

If there is a strategy involving drugs in incessant tachycardia, it seems clear that the first line consists of β-blockers. These drugs are of the highest importance for limiting the number of shocks in patients with an implantable cardioverter/defibrillator, and also for terminating incessant tachycardia. Drugs such as lidocaine remain (not only in my opinion) the second choice when serious arrhythmias occur and have only limited pro-arrhythmic effects. When a situation is not controlled with β-blockers, lidocaine or diphenyl-hydantoine, sedation has to be considered, as it has to be assumed that a third line (amiodarone) will be fully effective only after at least 72 h.

It is then that other timely measures have to be taken in order to prevent major complications (renal failure, pulmonary oedema . . .). A direct intervention on the arrhythmia substrate is always preferable but has to be safe and acceptable with respect to survival. Cox reported that anti-arrhythmic surgery is
associated with increased mortality in the setting of incessant tachycardia. This was also the experience of more recent investigators using surgery. Other investigational approaches never fulfilled their initial promise.

Therefore, the work of Cao and Gonska is very important. It confirms earlier reports on sustained ventricular tachycardia, and proves not only that it is possible to localize the area of slow conduction and interrupt re-entry in patients with ischaemic heart disease (and possibly with large aneurysms), but also that this can be achieved very successfully in critically ill patients; the complication rate is surprisingly low. The fact that there are recurrences of tachycardia during the follow-up is no surprise, but as implantable cardioverters/defibrillators are available these situations can be managed. These results are so encouraging that a more aggressive approach may be possible — also as regards paroxysmal sustained tachycardia — before proceeding to the implantation of devices. Briefly, the paper by Cao and Gonska is the herald of more work in the cath lab in the foreseeable future.

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References


