Amiodarone for the treatment of stable ventricular tachycardia – has the Resuscitation Council got it wrong?

Dear Sirs,

Amiodarone has been the UK Resuscitation Council’s recommendation for the treatment of haemodynamically stable ventricular tachycardia (VT) since 2000. It is my opinion that the evidence in support of amiodarone in this setting is poor and that superior agents may exist.

In the last six years, three retrospective studies have been published showing a dismal success rate when amiodarone is used to treat patients with stable VT. Marill et al reported that eight out of 28 (29%) patients cardioverted using a dose of 150 mg. Tomlinson et al looked retrospectively at stable VT patients given a dose of 300 mg and reported that six out of 41 (15%) patients cardioverted after 20 minutes. A further multicentre retrospective chart review also by Marill looked at 53 patients given amiodarone – of these just 25% cardioverted.

These studies are low level evidence – retrospective chart reviews with small numbers and less than perfect methodology. Nonetheless they appear to be the best evidence we have in support of amiodarone in the treatment of stable VT.

So what are the alternatives? A single trial in favour of sotalol was published in 1994. Lignocaine and sotalol went head-to-head in a randomised crossover trial looking at the treatment of spontaneous, sustained stable VT. The study was methodologically sound with sensible and pragmatic inclusion criteria, comparable groups at baseline, effective double-blinding and intention-to-treat analysis. Over two-thirds (11/15 (69%)) of patients given sotalol as an initial treatment cardioverted compared with only 3/17 (18%) who received lignocaine. Numbers are small and the trial may be inadequately powered but the success rate for sotalol appears convincing.

Procainamide was studied in a randomised crossover trial in comparison with lignocaine. In response to initial drug infusions, 12/15 (80%) of patients reverted to sinus rhythm compared to 21% of patients given lignocaine. A similar success rate of 76% was reported in a recent Japanese case-series at odds with these results is the 2010 chart-review by Marill et al who reported a 30% success rate – but only a minority of these patients received procainamide first-line.

VT is thought in most cases to be due to a re-entrant arrhythmia arising in an area of scarred myocardium. Drugs prolonging the refractory period in this area of scarring would be predicted to terminate the arrhythmia.Whilst procainamide and sotalol lengthen the myocardial refractory period acutely, amiodarone does not appear to do so and may be predicted to work poorly on theoretical grounds alone.

Many argue that amiodarone remains the drug of choice for safety reasons. In patients given amiodarone, direct-current cardioversion (DCCV) was required in 6-19% due to cardiovascular collapse, which is similar to rates reported for procainamide and sotalol (7-13% and 10%, respectively). Numbers are small and there is no standardised event reporting across the studies, but this is the best evidence we have from real patients, and amiodarone does not appear to live up to its reputation as a safer agent.

The Resuscitation Council has the difficult job of producing a guideline, which is simple, pragmatic and of benefit to the majority. However, it could be anticipated that resuscitation guidelines will be most useful to the non-specialist seeing a patient who has presented acutely with VT. For such patients, amiodarone does not appear to work well.

It is without doubt that UK physicians have great experience and familiarity with amiodarone, but this should not be an excuse to use it as a ‘comfort-blanket’ if better drugs exist. It is also true that sotalol and procainamide are not always readily available as intravenous preparations but, again, this is no justification for using an inferior drug.

When next confronted with a patient in VT, my inclination will be to reach for the defibrillator. However if this were unavailable, I would like the option of sotalol or procainamide in my armamentarium!

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References
Chest pain - troponin and athletes

Dear Sirs,

We read with great interest the article by Magdy and Dubrey1 on the subject of chest pain and troponin elevation in athletes and, in particular, the case of an Afro-Caribbean athlete who presented in this manner. This was accompanied by the finding of T-wave inversion in leads V4-V6 on the resting electrocardiogram (ECG), which normalised on exercise, and left ventricular hypertrophy on echocardiography. In our experience of over 900 Black athletes, the incidence of T-wave inversion in the lateral ECG leads is only 4.1%.2 During a mean follow-up period of 70 months, one Black athlete experienced aborted sudden death, whilst two were diagnosed with hypertrophic cardiomyopathy (HCM). All three of these athletes had T-wave inversions in the lateral leads. In contrast, our experience of Black HCM patients is that lateral T-wave inversion is an extremely common finding (77%).2 We would also point out that normalisation of T-wave inversion on exercise is a non-specific finding which may also be observed in HCM.3

We note that the athlete in question demonstrated a ‘mean left ventricular wall thickness of 15 mm’, suggesting that the maximal wall thickness may have been in excess of this figure. Our experience of Black athletes has demonstrated a maximal left ventricular wall thickness greater than 15 mm in only 1.5% of cases, with none greater than 16 mm.4

We agree with the authors that troponin elevation in athletes must be interpreted with caution. However, an athlete of any ethnicity demonstrating repolarisation anomalies in the inferior or lateral ECG leads requires detailed investigation including cardiac magnetic resonance imaging and familial evaluation, particularly in the presence of cardiovascular symptoms. Even in the absence of marked left ventricular hypertrophy, such anomalies may reflect early HCM, which may not manifest fully until many years later, and hence long-term follow-up is also essential.

These assertions are supported by recent European Society of Cardiology guidelines which state that ‘T-wave inversion in inferior and/or lateral leads are uncommon even in black athletes and warrant further investigation for an underlying heart disease’.5

Conflict of interest
None declared.

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References