

doi:10.1016/j.ehj.2004.08.028

### Myocardial infarction after coronary revascularization: role of cardiovascular magnetic resonance oedema imaging – Reply

Dear Editor,

We find the recent study by Abdel-Aty et al. [1] very interesting and we thank them for their comment. In our study [2], we applied strict criteria in order to exclude patients with previous myocardial infarctions. The finding that the higher the biochemical marker level the greater the amount of infarcted tissue, supported our assumption of a causal connection between peri-operative myocardial infarction and post-operative elevation of biochemical markers. However, contrast-enhanced magnetic resonance imaging may distinguish between viable and non-viable regions throughout infarct healing [3], but does not differentiate between acute and chronic infarction [4]. Since not all patients with previous infarctions have a history of myocardial infarction or evidence of infarction on the ECG, echocardiography or ventriculography, we believe that the newly described imaging approach [1] may be a valuable contribution for the future assessment of patients with elevated cardiac markers after coronary revascularization.

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Available online 11 November 2004

doi:10.1016/j.ehj.2004.07.042

### Risk of decompression illness among 230 divers in relation to the presence and size of patent foramen ovale

Dear Sirs,

Sandra Rea Torti et al., in the June issue of the *Journal*,<sup>1</sup> report on a group of divers, examined for patency of the foramen ovale (PFO), and claim to be able to calculate the relative risk for decompression illness (DCI) when a diver has a PFO.

Where it is true that in large samples with a low occurrence of a disease in proportion to a risk factor, the Odds Ratio may approach the value of the Relative Risk, in this series that claim is unjustified.<sup>2</sup>

First of all, the authors undertook a retrospective study. Relative Risks can only be reliably calculated from prospective analyses. This is the reason why we ourselves refrained from calculation of the RR in our publication<sup>3</sup> we did however (in spite of what Torti et al., pretend) calculate the Odds Ratio, which we found to be 5.6 for 'undeserved cerebral decompression sickness' in divers with Grade 2 PFO.

Secondly, the subject selection is not detailed: was it a random sample out of the sports divers federation, or were divers selected upon their response to a call for participation? If so, what measures were taken to ensure that divers would not present on the basis of previously experienced symptoms that might have been attributable to DCI? Such a selection bias is most likely, as is the fact that these divers would not 'admit' they had had DCI.<sup>4</sup> In the 'no-PFO' group there were 10 out of 167 divers (5.9%) who had experienced serious DCI. It is easy to calculate how, if only 10 more divers would have

'self-selected' on the basis of previous DCI symptoms, the Odds Ratio would be halved, with 95% confidence intervals of 1.4–6.0.

The article by Torti, and most likely the way the abstract was written, has caused quite some concern in sports diver's lay literature. In these articles, it is concluded that PFO is a major risk factor and that closure of PFO should be considered in all sports divers. Torti et al., mention only in brief terms near the end of their report, that diving with a PFO can be as safe as diving without a PFO, if the dive profile is conservative enough not to produce venous nitrogen bubbles upon ascent and after surfacing. It is the inappropriateness of the currently used dive profiles, not the PFO, which is the cause of decompression illness. The Divers Alert Network Europe, an international diving safety organization, is conducting studies aimed at developing adapted dive profiles towards 'low-bubble' decompression.<sup>5</sup>

Finally, we were delighted to find a bibliography reference pointing towards a possible "opening" of the PFO in saxophone players.<sup>6</sup> Although we have proposed this mechanism to be possible in sports divers already in 2002,<sup>7</sup> we have not been aware of medical literature backing up this hypothesis in other groups of people. Imagine our disappointment when the cited reference did not contain any mention of PFO!

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Available online 11 November 2004

doi:10.1016/j.ehj.2004.08.022

### Prevention of atrial fibrillation after cardioversion: results of the PAFAC trial

To the Editor,

I read with interest the recent publication on the use of sotalol, quinidine and verapamil in the prevention of atrial fibrillation (AF) following electrical cardioversion.<sup>1</sup> I was, however, surprised to see no mention of the several recent randomised trials favouring a rate control strategy in the management of AF. Nevertheless my principal concern lies with the choice of anti-arrhythmic agent following cardioversion.

AF is common in ageing patients with ischaemic and structural heart disease. Data from this study suggests 20% of patients suffer angina (some of who had suffered a previous MI) and 42% have NYHA class II or above heart failure symptoms. The use of verapamil, quinidine and sotalol in such patients is not without significant risk.

Safety and efficacy are important considerations in choosing an antiarrhythmic drug for the treatment of AF. In patients with cardiac failure amiodarone, digoxin and dofetilide are the only drugs with studies demonstrating a neutral effect on survival. The SWORD trial established that d-sotalol was associated with increased mortality in patients with left ventricular dysfunction or previous MI.<sup>2</sup> A different study comparing sotalol to bisoprolol in preventing AF recurrence showed equivocal efficacy but a superior safety profile (less pro-arrhythmic events) for bisoprolol.<sup>3</sup> The current study describes 10 epi-

sodes (one fatal) of *torsade de pointes* tachycardia, all associated with sotalol use.

The concern regarding quinidine ('quinidine syncope') stems mainly from a meta-analysis of quinidine trials which demonstrated more deaths in patients receiving quinidine for maintenance of sinus rhythm than placebo.<sup>4</sup> The vagolytic action of quinidine and its inherent risk of accelerated arrhythmias necessitate its use with AV nodal blocking agents; in this case verapamil. Based on the Cardiac Arrhythmia Suppression Trial data, class I agents should probably be avoided in post-MI patients.

The Canadian Trial of Atrial Fibrillation (CTAF) together with the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) substudy have shown amiodarone's superiority to sotalol and class I anti-arrhythmics, although side-effects were once again common.

As regards alternatives to anti-arrhythmic drugs in 'high risk' populations; there is growing evidence for the prevention of occurrence and recurrence of AF in patients with systolic dysfunction, by the use of 'up-stream' therapy. Both angiotensin converting enzyme (ACE) inhibitors and angiotensin-receptor blockers (ARB's) can lead to regression of atrial remodelling and restoration of atrial effective refractory periods (ERPs). Chronic beta-blockade is also associated with prolongation of action potentials and ERPs in human atrial cells. Evidence from clinical trials, albeit mainly retrospective data, indicates a decrease in the incidence of AF with ACE-inhibitors,<sup>5</sup> ARBs,<sup>6</sup> and beta-blockers.<sup>7</sup> Therefore, aggressive treatment of heart failure is likely to result in a reduction in the incidence of AF, and a reduction in the recurrence rate of AF following restoration of sinus rhythm.

If anti-arrhythmic agents are required for maintenance of sinus rhythm, their safety profile, together with individual patient characteristics, should be of utmost concern.

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Available online 11 November 2004

doi:10.1016/j.ehj.2004.09.027

### Prevention of atrial fibrillation after cardioversion: results of the PAFAC trial – Reply

To the Editor,

We share the concerns of Dr. Shelton concerning the safety of antiarrhythmic drug therapy. The intention of this trial, planned some years ago, was indeed to assess the efficacy and safety of antiarrhythmic drugs in maintaining sinus rhythm after electrical cardioversion. Therefore, we did not discuss the important and presently much debated issue of which strategy to prefer – rate control or rhythm control.

We agree that amiodarone and dofetilide are now the only drugs for which studies have demonstrated a neutral effect on survival in heart failure. However, our population was not a heart failure population. His statement that 42% had NYHA class II or above heart failure symptoms is correct but misleading since only 4% had NYHA III and none IV. Putting it differently, 50% of patients were in class I or II whereas 38% did not report any heart fail-