LETTERS TO THE EDITOR

Scope
Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation
Letters should be:

- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors

They may contain short tables or a small figure. Please send a copy of your letter on disk. Full instructions to authors appear in the January 1998 issue of Heart (page 106).

Human stress cardiomyopathy mimicking acute myocardial syndrome

Sir,—Pavin and colleagues' report of “human stress cardiomyopathy” is additional evidence of the probable catecholamine base of the characteristic T wave change. Their figures 1B, 1C, and 2B neatly fit the ECG criteria established by us in a prospective investigation of 100 patients' with a long follow-up (mean, eight years). Since the response is diagnostically non-specific, we termed it “global T wave inversion” and speculated that it was a catecholamine effect. As with Pavin and colleagues, the complete (global) response was almost never on the initial ECG and it disappeared in a matter of days. Both their patients were women, as were 81 of ours. My only criticism of their report concerns fig 1D. It is not normal: P2, P3 (but not PAVF), and PV4–6 are all inverted. The ST segments are so elongated (global) response was almost never on the initial ECG and it disappeared in a matter of days. Their patients had a permanent pacemaker implanted because of sick sinus syndrome—most probably a defect in bradycardia rhythms. In those patients, an attempt at AAI pacing could have proven effective, perhaps with combinations of both pacemaker lead and β-thromboglobulin in the normal (control) range. This could have clinical and practical implications regarding the choice of pacemaker when significant bradycardia rhythms are the primary reason for pacemaker implantation.

As the only single chamber mode of pacing was in the ventricle, there is the possibility that single atrial pacing would be as beneficial (from a haemostatic aspect) and as effective (from a haemodynamic aspect) as DDD(R). It is a pity to have missed an opportunity to establish whether it is asynchrony that causes increased platelet activation (as the authors claim) or whether single chamber pacing (be it atrial or ventricular) is the culprit.

Moreover, the increase in platelet activation could be attributed to pacing in the ventricle and, maybe, if patients were paced in the atrium only, concentrations of platelet activity would be in the control range. Such clarification could have a significant impact both on policy making and economics considering the difference in price for single (AAI, VVI) versus dual chamber pacemakers.

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Effect of atrioventricular asynchrony on platelet activation

Sir,—I read with great interest the article on the effect of atrioventricular asynchrony on platelet activation by Lau, et al. The authors conclude that “loss of atrioventricular synchrony related to single chamber ventricular pacing is a major cause of increased platelet activation.” However, the tables and text indicate that in both modes of DDD pacing, platelet activation is increased by approximately 160–200% compared with the control group. Twelve of their patients had a permanent pacemaker implanted because of sick sinus syndrome—most probably a defect in bradycardia rhythms. In those patients, an attempt at AAI pacing could have proven effective, perhaps with combinations of both pacemaker lead and β-thromboglobulin in the normal (control) range. This could have clinical and practical implications regarding the choice of pacemaker when significant bradycardia rhythms are the primary reason for pacemaker implantation.

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NOTICES

11th annual congress of the European Society of Intensive Care Medicine (joint meeting with the ninth annual congress of the European Society of Paediatric Intensive Care) will be held in Stockholm, Sweden from 6–9 September 1998. For further information please contact ESICM/ESPC Congress Secretariat, 40 Avenue Joseph Wybren, B-1070 Brussels, Belgium (tel: +32 2 527 58 29; fax: +32 2 527 00 62; email: esicm@pophost.eunet.be).

Interventional electrophysiology in the management of cardiac arrhythmias

This international symposium will be held in Salzburg, Austria from 23–26 September 1998, under the auspices of the North American Society for Pacing and Electro physiology and the European Heart Institute. For further information please contact Prof. Dr. B Lüderitz, University of Bonn, Department of Medicine–Cardiology, Sigmund Freud–Str 25, 53105 Bonn, Germany (tel: +49 228 287 5217; fax: +49 228 287 6823).

State of the art in congestive heart failure

Current concepts and controversies in aetiology, diagnosis and economic issues. This international symposium will be held in Brussels, 23 and 24 October 1998. For further information, please contact Dr R Gunzenberg, Niellenstraat 14, 2600 Berchem, Belgium (fax +32 3 240 20 40).