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 figues. **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'[1] report of "human stress cardiomyopathy" is additional evidence of the probable catecholamine basis 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[2] with a long follow up (mean, eight years).[3] Since the response is diagnostically non-specific, we termed it "global T wave inversion" and speculated that it was a catecholamine effect.[2] As with Pavin and colleagues, the complete (global) response was almost never on the initial ECG and it disappeared in a matter of days.[2 3] 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 as to resemble those in hypocalcaemia. Can the authors explain those findings? This is not to criticise, but rather to augment a nice report.

D H SPODICK
Saint Vincent Hospital,
25 Winthrop Street
Worcester, MA 01604–4593, USA

1 Pavin D, Le Breton H, Daubert C. Human stress cardiomyopathy mimicking acute myocardial syndrome. *Heart* 1997;**78**:509–11.
2 Walder L, Spodick DH. Global T wave inversion. *J Am Coll Cardiol* 1991;**17**:1479–85.
3 Walder L, Spodick DH. Global T wave inversion: long-term follow-up. *J Am Coll Cardiol* 1993;**21**:1652–6.

This letter was shown to the authors, who reply as follows:

Dr Spodick makes a pertinent remark about fig 1D, which is not normal. This ECG shows abnormalities of the P wave that seem to be isodiphasic (±) rather than inverted in leads D2, D3, V5, and V6. Unfortunately we cannot explain these findings. Indeed, this ECG was done by the patient's physician during a routine visit late after the events described in the report and we have no information regarding metabolic or neurohumoral state at this time. We cannot exclude atrial conduction disturbances. However, I don't think it is of major importance in this report, as this ECG was shown to illustrate the normalisation of QRS and T wave morphology.

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*.[1] 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 impulse generation with or without accompanying tachyarrhythmias. In those patients, an attempt at AAI pacing could have proven effective, perhaps with concentrations of both platelet factor and β thomboglobulin in the normal (control) range. This could have clinical and practical implications regarding the choice of pacemaker when significant bradyarrhythmias 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 chamber 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.

B MAZOUZ
Psagot, DN Mizrach Binyamin
Israel 90–624

1 Lau C-P, Tse H-F, Cheng G. Effect of atrioventricular asynchrony on platelet activation: implication of thromboembolism in paced patients. *Heart* 1997;**78**:358–63.

This letter was shown to the authors, who reply as follows:

We thank Dr Mazouz for his interest. We agree that in patients with sick sinus syndrome, AAI pacing is as effective and beneficial as DDD pacing. In fact, a long term follow up, randomised, prospective trial[1] demonstrated that AAI pacing in patients with sick sinus syndrome is associated with significantly lower cardiovascular mortality ($p = 0.022$) and fewer thromboembolic events ($p = 0.028$) than VVI pacing. We found that all patients with pacemakers have evidence of increased systemic platelet activation,[2] which is likely to relate to contact of platelets with an artificial surface. More importantly, with the patients acting as their own controls, VVIR pacing was associated with a significantly higher degree of platelet activation than DDD or DDDR modes.

AAI mode was not tested in our study for the following reasons. First, our study population included some patients with complete heart block and some with sick sinus syndrome, thus AAI mode could not be used in all of them. Second, all patients had a dual chamber pacemaker, so even when they are paced in AAI mode the effects of an additional ventricular lead on platelet activation could not be assessed. Finally, if we only compare platelet activation during VVI versus AAI mode, we could not establish whether it was the atrioventricular asynchrony or ventricular pacing itself that causes increased platelet activation.

Our results demonstrated that there was no relation between platelet activation and the frequency of ventricular pacing, suggesting that ventricular pacing itself did not increase platelet activation. Future studies comparing platelet activation in AAI versus VVI mode in patients with sick sinus syndrome may provide further insight into the mechanism of reduced thromboembolism associated with AAI pacing.

1 Andersen HR, Nielsen JC, Thomsen PEB, *et al*. Long-term follow-up of patients from a randomised trial of atrial versus ventricular pacing for sick-sinus syndrome. *Lancet* 1997;**350**:1210–16.
2 Lau CP, Tse HF, Cheng G. Effects of atrioventricular asynchrony on platelet activation: implication of thromboembolism in paced patients. *Heart* 1997;**78**:358–62.

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/ESPIC Congress Secretariat, 40 Avenue Joseph Wybran, B-1070 Brussels, Belgium (tel: +32 2 529 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 Electrophysiology 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 6423).

State of the art in congestive heart failure. Current concepts and controversies in aetiopathogenesis, diagnosis and economic issues. This international symposium will be held in Brussels, 23 and 24 October 1998. For further information, please contact Dr R Gunzberg, Niellonstraat 14, 2600 Berchem, Belgium (fax +32 3 240 20 40).