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 July 1996 issue of Heart (page 93).

Circadian variation of left ventricular diastolic function in healthy people

Sir,—In their study of healthy people, Voutilainen et al found a nocturnal decrease and a daytime increase in the rate of left ventricular relaxation, which they tentatively attributed to sympathoadrenal activity.1 Neurobiological features are suggested by reports that link dysregulation of brainstem and head cardiovascular control and cardiovascular reactivity in challenging tasks with dopamine abnormalities lateralised to the right hemisphere. This hypothesis is supported by the importance of dopamine in the control of wakefulness manifested by a reduction of reaction time and gap frequency, optimal response organisation at intermediate dopamine tone in a medial-frontal-striatal activation system, and inhibition of the right hemisphere promoting left-hemisphere dominance associated with cardiac arrhythmia and vasocostriction.2 It seems reasonable to assess these states can be identified during the twenty-four hours after a myocardial infarction.

We also showed that the magnitude of the fall in cholesterol after myocardial infarction seemed to be related to the severity of the episode. We were therefore surprised that Carlson et al did not supply data comparing the size of infarction in their thrombolytic groups with their non-thrombolytic groups. This, not minor differences in the timing of the initial sample as they suggest, seems to us to be the most likely explanation of the differences in cholesterol that they showed between the groups.

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Effect of percutaneous fenestration of the atrial septum on protein-losing enteropathy after the Fontan operation

Sir,—We read with interest the paper by Mertens et al. They reported that in patients who had undergone the Fontan operation, fenestration of the intracardial septum should be considered before proceeding to Fontan take-down or heart transplantation.

We recently treated a boy with hypoplastic left heart syndrome who had had a Norwood operation as a neonate and hemi-Fontan operation at the age of 6 months. The Fontan (TCPC) operation was completed when he was one year old and he was discharged on 57th postoperative day. The patient was immediately resistant to protein-losing enteropathy which was resistant to medical treatment and finally we decided to construct a percutaneous fenestration of the intracardial septum made with a Gore-tex graft. A 6 French long sheath was introduced into the intra-atrial venous chamber from internal carotid vein, a Brockenbrough needle was introduced through the long sheath. A Blalock-Park blade catheter and 10 mm ballloon catheter were introduced and a 10 mm diameter hole was made. Low cardiac output improved immediately. Transosophageal echocardiography immediately after the procedure showed a 7 mm diameter hole with significant right-to-left shunt in the Gore-tex baffle. The patient looked well and ascites and pleural effusion were reduced. However, the pleural effusion and ascites gradually returned within three weeks. Because he showed clinical signs of severe low cardiac output we decided on a Fontan take-down 41 days after the transcatheter fenestration. The patient died from multi-organ failure three days after the take-down operation. At operation we found that the fenestration in the Gore-tex graft had closed. The Gore-tex graft had thickened up to 2 mm and the hole was completely closed and covered by endocardium.

Mertens et al reported successful treatment of a patient whose native intracardial septum was fenestrated by means of a Brockenbrough needle. Our case suggested that early closure of the fenestrated hole in the Gore-tex baffle is likely.

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

Sir,—We read with interest the case described by Satomi et al in which they confirmed the effectiveness of a secondary fenestration as a treatment for protein-losing enteropathy (PLE) after Fontan operation. We share their experience of seeing a significant haemodynamic and clinical improvement in patients immediately after the fenestration was created. However, in our experience with cases we are aware of in other centres where this technique has been applied, Satomi et al observed a strong tendency for the procedure to revert to close. The three fenestrations we created in Fontan patients, all became considerably

Serum lipids four weeks after acute infarction are a valid basis for lipid lowering intervention in patients receiving thrombolysis

SIR,—As long as ago as 1971 we first suggested that patients with myocardial infarction should be screened for hyperlipidaemia on the first morning after admission.1 We were disappointed to note that Carlson et al failed to acknowledge this,2 quoting our 1971 paper in support of the statement that "serum lipid concentrations are usually not assessed after myocardial infarction" when we in fact concluded that "hyperlipidaemic states can be identified during the twenty-four hours after a myocardial infarction."

We thought it important to point out that Carlson et al did not supply data comparing the size of infarction in their thrombolytic groups with their non-thrombolytic groups. This, not minor differences in the timing of the initial sample as they suggest, seems to us to be the most likely explanation of the differences in cholesterol that they showed between the groups.

Coronary Palmaz-Schatz stent implantation in acute myocardial infarction

Sir,—Neumann et al are to be applauded for reporting that coronary stenting is an effective safe adjunct to direct percutaneous transluminal coronary angioplasty (PTCA) for acute myocardial infarction.1 This finding has far-reaching implications. Their pilot study clearly lacks power to assess the clinical impact of pre-existing target vessel thrombus on recollusion. It is, however, residual thrombus after balloon PTCA (seen in 36% of their cases) that gives greater concern. Moreover, without coronary ultrasound or angiography, it may sometimes be difficult to determine whether such residual thrombus is due to covert dissection, intimal disruption, or is a reflection of a highly thrombogenic milieu despite seemingly optimum dilatation and flow. When the latter is thought to apply, we are naturally hesitant to stent, even though we would routinely use adjunctive intra-aortic balloon counterpulsation to optimise coronary perfusion.2 Like others, we sometimes resort to a period of intra coronary thrombolysis using an infusion catheter, but the results are unpredictable.3 In our experience, the most thrombogenic patients tend to be those undergoing not primary PTCA but rescue PTCA, particularly if they seem to be resistant to several doses of intravenous thrombolysis which may have induced a procoagulant state.4 It remains to be seen whether the platelet glycoprotein IIb/IIIa receptor antibody (c7E3 Fab) will have a major role in this difficult situation.

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

Sir,—As Dr Lim and Dr Norell correctly point out, coronary thrombus after balloon PTCA is a problem, particularly when a stent is thought to be needed. Clearly, our study does not have sufficient power to disprove their suggestion that residual thrombus before stent placement increases the risk of subsequent stent thrombosis in acute myocardial infarction. In fact, the trend we found points towards an increased risk. Nevertheless, our data show that stenting in the presence of residual thrombus does not carry a prohibitive risk of subacute stent thrombosis. Even with the help of coronary ultrasound it may be difficult to distinguish between a primarily thrombogenic milieu and intimal disruption as the major mechanism for coronary thrombus formation. Our findings suggest that a coronary stent should be implanted in any case if needed and, although we cannot provide hard data to support our recommendation, we believe that adjunctive antplatelet therapy should be given. We agree with Lim and Norell that the newly developed platelet glycoprotein IIb/IIIa receptor antagonists deserve serious consideration for this purpose.

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Significance of perfusion of the infarct related coronary artery for susceptibility to ventricular tachyarrhythmias in patients with previous myocardial infarction

Sir,—Huikuri et al highlighted a very important aspect of current cardiology—that is, risk assessment for sudden death after a myocardial infarction (MD).1 The quest for a single test with a high predictive power has been the holy grail of cardiology for the past 10 years. The risk factors assessed so far, including reduced heart rate variability, baroreceptor sensitivity, signal averaged electrocardiography (ECG), and the ventricular arrhythmia power, are poor predictors when used alone but were additive in combination. Farrell et al found that heart rate variability and signal averaged ECG offered the best sensitivity and specificity.2 Even in this “high risk” group between 70% to 85% of patients will be event-free over several years of follow up, hence the need for a single test with a high predictive power.

The study of Huikuri et al implies that revascularisation of the infarct related artery will reduce ventricular arrhythmias. However, we are not told of the number of previous infarctions in the groups or whether a ventricular aneurysm was present: revascularisation would only prevent unfavourable ventricular tachycardia (VT) in the presence of a large myocardial scar (O'Rourke).3 Although the time elapsed after myocardial infarction is comparable in Huikuri et al's patients and our samples are skewed and the use of the median and non-parametric tests might have shown that the groups were not comparable. The emphasis placed on epidemiological studies is not justified because most studies suggest that this is a poor predictor of sudden death in uncomplicated infarctions. Kowey et al in a meta-analysis found no difference in arrhythmic events between those who had inducible VT and those who did not.4 Vetterott et al showed that the best predictor of low amiodarone. This reduction in the number of potential candidates could also be achieved by angioplasty with a balloon size of 1.5—1.7 mm, which is beyond the period of myocardial salvage.5 Hohnloser et al showed that this benefit translates into event-free survival.6 In their study patients underwent revascularisation if they had objective evidence of ischaemia. The most powerful predictors of arrhythmias were a closed artery, an anterior location of the infarct, and left ventricular dysfunction (LVEF ≤ 35%). The reasons why these three risk factors were summed they had a positive predictive power of only 50%. Undertaking coronary angiography and revascularisation has tremendous implications for costs and time. A better cost benefit approach may be to use a less sensitive test but treat those at risk with amiodarone. This is the basis of the eagerly awaited European and Canadian trials.

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