

## 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).

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

SIR,—As long ago as 1971 we first suggested that patients with myocardial infarction should be screened for hyperlipidaemia on the first morning after admission.<sup>1</sup> We were disappointed to note that Carlsson *et al* failed to acknowledge this,<sup>2</sup> 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 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 Carlsson *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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- 1 Fyfe T, Baxter RH, Cochran KM, Booth EM. Plasma lipid changes after myocardial infarction. *Lancet* 1971;ii:997-1001.
- 2 Carlsson R, Lindberg G, Westin L, Israelsson B. Serum lipids four weeks after acute myocardial infarction are a valid basis for lipid lowering intervention in patients receiving thrombolysis. *Br Heart J* 1995;74:18-20.

### 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.<sup>1</sup> Neurobiological features are suggested by reports that link dysregulation of brainstem 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 vasoconstriction. These observations prompt a multidisciplinary approach involving neuropharmacology and cardiovascular physiology<sup>2</sup> in evaluating the pathophysiological importance of diurnal changes<sup>1</sup> and the time course of speech induced ischaemia.<sup>3</sup> This method is supported by the correlation of the frequency and duration of speech hesitation pauses with a sixfold increase in the prevalence and incidence of coronary heart disease and mood, respectively, which reflect properties of neuronal activity and firing, and by the correlation of anxiety with the sixfold increase in fatal coronary heart disease, in particular, sudden cardiac death. It is also supported by the reduction in blood pressure associated with longer, less recurrent pauses<sup>4</sup> that is predictive of a response to neuropharmacological intervention,<sup>5</sup> and by the association of pause rate and variability in pause duration with the left and right hemisphere, respectively<sup>6</sup>—hence the need to tailor interventions to asymmetrical brain functions.

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- 1 Voutilainen S, Kupari M, Hippelainen M, Karppinen K, Ventila M. Circadian variation of left ventricular diastolic function in healthy people. *Heart* 1996;75:35-9.
- 2 Friedman EH, Mehta PM, Grainger TA, Lust RM, Movahed A, Terry J, *et al*. Effect of cocaine on left ventricular function (letter and response). *Circulation* 1996;93:199.
- 3 Ahlawat SK, Legault SE. Pathophysiology and time course of silent myocardial ischaemia during mental stress (letter and reply). *Heart* 1996;75:101.
- 4 Friedman EH, Coyle CP, Santiago MC. Neurobiology of depressive symptoms (letter and reply). *Arch Phys Med Rehabil* 1995;76:1179.
- 5 Ranelli CJ, Miller RE. Behavioral predictors of amitriptyline response in depression. *Am J Psychiat* 1981;138:30-4.
- 6 Blonder LX, Pickering JE, Heath RL, Smith CD, Butler SM. Prosodic characteristics of speech pre- and post-right hemisphere stroke. *Brain Lang* 1995;51:318-35.

### 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*.<sup>1</sup> They reported that in patients who have a poor clinical result after Fontan operation, fenestration of the interatrial 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 six months. The Fontan (TCPC) operation was completed when he was one year old and he was discharged on 57th postoperative day. The postoperative course was uneventful and he was doing well at home for a month, before he returned to hospital with right heart failure. An intra-atrial venous route was created by using 10 mm Gore-tex graft. Right heart failure progressed to protein-losing enteropathy which was resistant to medical treatment and finally we decided to construct a percutaneous fenestration of the interatrial 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 balloon catheter were introduced and a 10 mm diameter hole was made. Low cardiac output improved immediately. Transophageal 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*<sup>1</sup> reported successful treatment of a patient whose native interatrial 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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- 1 Mertens L, Dumoulin M, Gewillig M. Effect of percutaneous fenestration of the atrial septum on protein-losing enteropathy after the Fontan operation. *Br Heart J* 1994;72:591-2.

This letter was shown 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. Also like us and experience with cases we are aware of in other centres where this technique has been applied, Satomi *et al* observed a strong tendency of the percutaneous fenestration to close. The three fenestrations we created in Fontan patients, all became considerably

smaller within six months. This has led us to consider using stents to maintain the patency of the fenestration. In an experimental study on mongrel dogs we showed that this was technically feasible.<sup>1</sup> Meanwhile stenting of an atrial fenestration has been performed in another centre with a good clinical result (Dr De Giovanni; Birmingham; personal communication).

We consider creating a percutaneous fenestration as a temporary treatment in Fontan patients with PLE who have no treatable cause for systemic venous hypertension (conduit stenosis, pulmonary artery stenosis, arrhythmia, ventricular failure) and when conventional medical treatment (diuretics, steroids) is ineffective. It does not appear to offer a long-lasting solution but buys time to improve the patient's general condition before Fontan take-down or heart transplantation is performed.

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### 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.<sup>1</sup> This finding paves the way to a randomised evaluation. Their pilot study clearly lacks power to assess the clinical impact of pre-existing target vessel thrombus on reocclusion. 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.<sup>2</sup> Like others, we sometimes resort to a period of intracoronary thrombolysis using an infusion catheter, but the results are unpredictable.<sup>3</sup> 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 pro-coagulant state.<sup>4</sup> It remains to be seen whether the platelet glycoprotein IIb/IIIa receptor antibody (c7E3 Fab) will have a major role in this difficult situation.<sup>5</sup>

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- 1 Neumann F-J, Walter H, Richardt G, Schmitt C, Schömig A. Coronary Palmaz-Schatz stent implantation in acute myocardial infarction. *Heart* 1996;75:121-6.  
2 Ohman EM, George BS, White CW, Kern MJ, Gurbel PA, Freedman RJ, *et al* for the

Randomized IABP Study Group. Use of aortic counterpulsation to improve sustained coronary artery patency during acute myocardial infarction. Results of a randomized trial. *Circulation* 1994;90:792-9.

- 3 Gulba DC, Daniel WG, Simon R, Jost S, Barthels M, Amende I, *et al*. Role of thrombolysis and thrombin in patients with acute coronary occlusion during percutaneous transluminal coronary angioplasty. *J Am Coll Cardiol* 1990;16:563-8.  
4 Fitzgerald DJ, Catella F, Roy L, Fitzgerald GA. Marked platelet activation *in vivo* after intravenous streptokinase in patients with acute myocardial infarction. *Circulation* 1988;77:142-50.  
5 The EPIC Investigators. Use of a monoclonal antibody directed against the platelet glycoprotein IIb/IIIa receptor in high-risk coronary angioplasty. *N Engl J Med* 1994;330:956-61.

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 antiplatelet 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 (MI).<sup>1</sup> 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 electrocardiogram (ECG), and ejection fraction, 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.<sup>2</sup> 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 suppress 50% of inducible ventricular tachycardias (VT) in the presence of a large myocardial scar (O'Rourke<sup>3</sup>). Although the time elapsed after myocardial infarction is comparable in Huikuri *et al*'s two patient groups, the samples are skewed and the use of the median and of non-parametric tests might have shown that the groups were not comparable. The emphasis placed on electrophysiological 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.<sup>4</sup>

Vatterott *et al* showed that the best predictor of late potentials on a signal averaged ECG was a closed artery; the next best predictor was a previous MI.<sup>5</sup> This reduction in the number of late potentials could also be achieved by angioplasty between days 6 and 15—that is, beyond the period of myocardial salvage.<sup>6</sup> Hohnloser *et al* showed that this benefit translates into event-free survival.<sup>7</sup> In their study patients underwent revascularisation if they had objective evidence of ischaemia. The most powerful predictors of arrhythmias were a closed artery  $P < 0.0002$ , left ventricular dysfunction  $P < 0.003$ , and late potentials  $P < 0.04$ . Even when 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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- 1 Huikuri HV, Koistinen MJ, Airaksinen KEJ, Ikäheimo MJ. Significance of perfusion of the infarct related coronary artery for susceptibility to ventricular tachyarrhythmias in patients with previous myocardial infarction. *Heart* 1996;75:17-22.  
2 Farrell TG, Bashir Y, Cripps T, *et al*. Risk stratification for arrhythmic events in post-infarction patients based on heart rate variability, ambulatory electrocardiographic variables and the signal averaged electrocardiogram. *J Am Coll Cardiol* 1991;18:687-97.  
3 O'Rourke RA. Role of myocardial revascularisation in sudden cardiac death. *Circulation* 1992;85(1 suppl):112-7.  
4 Kowey PR, Taylor JE, Rials SJ. Does programmed stimulation really help in the evaluation of patients with nonsustained ventricular tachycardia? Results of a meta analysis. *Am Heart J* 1992;123:481-5.  
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6 Boehrner JD, Glamann B, Lange RA, *et al*. Effect of coronary angioplasty on late potentials one to two weeks after acute myocardial infarction. *Am J Cardiol* 1992;70:1515-9.  
7 Hohnloser SH, Franck P, Klingenhoben T, *et al*. Open infarct artery, late potentials, and other prognostic factors in patients after acute myocardial infarction in the thrombolytic era. A prospective trial. *Circulation* 1994;90:1747-56.