

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.¹ 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.¹ 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.² Like others, we sometimes resort to a period of intracoronary 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 pro-coagulant 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.⁵

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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 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).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 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.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 suppress 50% of inducible ventricular tachycardias (VT) in the presence of a large myocardial scar (O'Rourke³). 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.⁴

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.⁵ 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.⁶ Hohnloser *et al* showed that this benefit translates into event-free survival.⁷ In their study patients underwent revascularisation if they had objective evidence of ischaemia. The most powerful predictors of arrhythmias were a closed artery $P < 0.00002$, 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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