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. Meanwhile stenting of an atrial fenestration has been performed in another centre with a good clinical result (Dr De Giovanni, Birmingham; personal communication).

We considered creating a percutaneous fenestration as a temporary treatment in Fontan patients with PLE who have no treatable cause for systemic venous hypertension (conduit stenosis, coronary 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.

LUC MERTENS
MARC GEWILLIG
Paediatric Cardiology,
University Hospital Gasthuisberg,
Herestraat 49,
B 3000 Leuven,
Belgium

1 Gewillig M, Mertens L, Stockx L. Percu-
taneous fenestration of the atrial septum with a stent. Eur Eur Europ Caribbean

Coronary Palmsz-Schatz stent implanta-
tion in acute myocardial infarction

Sir,—Neumann et al are to be applauded for reporting that coronary stenting is an effec-
tive safe adjunct to direct percutaneous transluminal coronary angioplasty (PTCA) for acute myocardial infarction.1 This finding is based on randomized trials. Their pilot study clearly lacks power to assess the clinical impact of pre-existing tar-
gent 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 dis-
section, 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 coro-
nary perfusion.2 Like others, we sometimes resort to a period of intra coronary thrombo-
ysis 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 determined whether the platelet glycoprotein IIb/IIIa receptor antibody (c7E3 Fab) will have a major role in this difficult situation.

RICHARD LIM
MICHAEL S NORELL
Cardiology,
Hull Royal Infirmary,
Hull HU3 5JZ

2 Ohman EM, George BS, White CW, Kern MJ, Gurbel PA, Freedman RJ, et al for the Randomized IABP Study Group. Use of aor-
tic counterpulsation to improve sustained coronary artery patency during acute myocardial infarction. Results of a random-
3 Galba DC, Daniel WG, Simon R, Jost S, Bartheils M, Amende I, et al. Role of throm-

5 The EPIC Investigators. Use of a monoclonal antibody Fab against the platelet glyco-

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 sufficient power to dis-
prove their suggestion that residual throm-
bus 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 meca-

nism 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.

FRANZ NEUMANN
HANNA WALTER
ALBERT SCHOMIG
1 Medizinische Klinik und Poliklinik rechts der Isar
Im Anzinger Straße 22,
81675 Munich,
Germany

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 impor-
tant 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 diagnostic accuracy and speci-
ficity.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 previ-
ous infarctions in the groups or whether a ventricular aneurysm was present: revascu-
larisation would only be possible if there were un-
cific ventricular tachycardias (VT) in the presence of a large myocardial scar (O'Rourke). 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 sud-
den death in uncomplicated infarctions. Koyew et al in a meta-analysis found no dif-
ference in arrhythmic events between those who had inducible VT and those who did not.3 Vatterott et al showed that the best pre-
dictor of late potentials on a signal averaged ECG was a closed artery; the next best pre-
dictor was a previous MI.4 This reduction in the number of late potentials could also be achieved by angioplasty (Lange et al15 and 16)—that is, beyond the period of myocardial salvage.5 Hohnloser et al showed that this benefit translates into event-free survival.6 In the study patients underwent revascularisa-
tion if they had objective evidence of ischaemia. The most powerful predictors of arrhythmias were a closed artery P < 0-00002, left ventricular dysfunction P < 0-0003, and late potentials P < 0-004. Even when these three risk factors were summed they had a positive predictive power of only 50%. Obtaining 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 tri-
als.

K K RAY
West Green Clove,
Edgbaston,
Birmingham

1 Huikuri HV, Koistinen MJ, Airakinen KJ, Ikhaheimo MJ. Significance of perfusion of the infarct related coronary artery for suscepti-
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infarction patients based on heart rate vari-
ability, ambulatory electrocardiographic variables and the signal averaged electrocar-
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Coronary Palmaz-Schatz stent implantation in acute myocardial infarction.

R. Lim and M. S. Norell

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