Coronary Palmez-Schatz stent implantation in acute myocardial infarction

Stir—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. This finding is to be welcomed.

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. Like others, we sometimes resort to a period of intracoronary thrombolysis using an infusion catheter, but the results are unpredictable.

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. It remains to be seen whether the platelet glycoprotein IIb/IIIa receptor antibody (c7E3 Fab) will have a major role in this difficult situation.

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

Stir—Huuriki et al highlighted a very important aspect of current cardiology—that is, risk assessment for sudden death after a myocardial infarction (MD). 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 correlation in sensitivity and specificity. 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 Huuriki 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 of truly treatable ventricular tachycardias (VT) in the presence of a large myocardial scar (O'Rourke). Although the time elapsed after myocardial infarction is comparable in Huuriki et al's two patient groups, the 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 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.

Vatterott et al showed that the best predictor of low potential on a signal averaged ECG was a closed artery; the next best predictor was a previous MI. This reduction in the number of low potential could also be achieved by angiographically normal, 0-1—15—that is, beyond the period of myocardial salvage.

Hohloser et al showed that this benefit translates into event-free survival. In their study patients underwent revascularisation if they had objective evidence of ischaemia. The most powerful predictors of ischaemia 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%. 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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This letter was shown to the authors, one of whom responds as follows:

Sir,—It is true that the positive predictive accuracy of all the available tests for predicting the arhythmic death in patients after myocardial infarction is low and that more specific tests are needed. Because our study was cross sectional it cannot give information on the predictive accuracy of an occluded infarct-related artery. However, it was the first study to show the beneficial effects of perfusion of an old infarct scar on the electrophysiological substrate. We agree that in this type of cross sectional comparison it is difficult to achieve 1:1 matching of all the factors that may influence arrhythmogenesis. None the less the study was specifically designed to match the patient groups for ejection fraction, wall motion abnormalities (including presence of ventricular aneurysm), and number of previous infarctions¹ (tables 1 and 2).

No conclusions about the benefits of revascularisation can be based on the data of our study,¹ but we hope that a randomised prospective trial that is underway will give insights into the potential beneficial effects of angioplasty of the occluded infarct on the arrhythmic substrate.

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Non-surgical ablation of the ventricular septum for the treatment of hypertrophic cardiomyopathy

Sir,—We read with interest Professor Oakley's erudite review of the natural course and treatment of hypertrophic cardiomyopathy.¹ Sadly, she regards the development of outflow tract obstruction of the ventricular septum at our hospital as an ingenious but unimportant endeavour. She observes that symptoms, gradients, and outlook are unrelated in hypertrophic cardiomyopathy; that surgical relief of outflow tract obstruction does not improve outcome and may impair overall left ventricular function; and finally that the natural course of hypertrophic cardiomyopathy is towards a reduction in outflow tract obstruction with time as left ventricular impairment and dilatation progress.

Undoubtedly, the degree of obstruction of the outflow tract does not correlate well with either symptomatic status or outlook within populations of patients with hypertrophic cardiomyopathy. None the less, when an individual patient has a large outflow gradient and symptoms that correlate with such obstruction—namely, exertional angina, dyspnoea, and syncope—an association between outflow tract obstruction and symptoms seems beyond any doubt. Furthermore, there is evidence that these symptoms are improved by manoeuvres that reduce the obstruction, including our new technique.¹ As there is no prospective randomised evidence to suggest that surgical relief of outflow tract obstruction either prolongs or shortens life, it is important that both surgical and non-surgical myocardial resection are performed for the palliation of symptoms. We have not suggested that surgical benefit accrues from non-surgical septal reduction—although we hope it does.

In any cardiomyopathic process, maintaining as many normally functioning myocytes as possible is clearly desirable, but the evidence that myomegaly significantly improves overall left ventricular function is slim. In the series mentioned in the editorial,¹ the evidence for such impairment was a rise in end diastolic diameter from a mean of 4-5 cm to 5-1 cm over mean follow up of 8-9 years. Fractional shortening was unchanged (41% ± 39%). The evidence quoted from Spirito et al's study¹ that the natural course of hypertrophic cardiomyopathy is a progressive, inevitable decline in overall left ventricular function, with a consequent reduction in gradient, is not robust: in Spirito's series of patients with severe hypertrophic cardiomyopathy, those who had normal ejection fractions at baseline (n=54) had a mean rise of just 1 mm in end diastolic diameter over follow up and none developed clinical heart failure. The 13 patients with ejection fractions of less than 50% had a scarcely impressive rise of 5 mm in end diastolic diameter, and only one patient in the series had a definite reduction in gradient with time. We cannot rely on time and the natural course of the disease to rid all of our patients of their worrisome and incapacitating left ventricular outflow tract obstruction.

The primary goal in the treatment of hypertrophic cardiomyopathy is clearly the development of strategies known to prolong life and prevent sudden death, but the provision of symptomatic relief for patients can not be ignored. Professor Oakley concludes that "the extreme clinical and genetic heterogeneity of the disease has prevented any prospective randomised trials to assess the effect on outcome of most forms of treatment". We hope that she recognises that this clinical heterogeneity encompasses a minority of patients with large outflow gradients and corresponding disabling symptoms. We feel our efforts to provide symptomatic relief for this subgroup by means of non-surgical septal reduction are worthwhile, even though the long-term effects on outcome may not be known for many years.

CHARLES KNIGHT
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This letter was shown to the author, who replies as follows:

Sir,—I thank Dr Knight and Dr Sigwart for summarising my editorial so well in the first paragraph of their letter and of course I agree that progressive decline in overall left ventricular function is not inevitable. Some have mild disease and some die early but others suffer gradual loss of systolic efficiency. Only a small increase in residual volume heralds the onset of low output failure because of the marked diastolic impairment in these patients whose ventricles do not readily dilate. The natural progression of myocyte fall-out, whether due to the myopathy or to ischaemia, will have been accelerated by "myocardial reduction" in the name of therapy. We shall see.

CELIA M OAKLEY
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A wide health remit for aspirin

Sir,—It is widely recognised that aspirin helps to reduce the risk of certain cardiovascular diseases. More recently, good evidence has indicated that aspirin can also help to reduce the risk of certain gastrointestinal cancers.¹ I write to ask whether cardiologists who frequently prescribe aspirin have any "dormant" data on the risk of cancer. I am also interested in collaborating with any colleagues who might be conducting randomised trials of aspirin intervention. I would be able to advise on the measurement of wider health gains relating to a reduced risk of cancer.

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NOTICE

An international workshop on Recent Developments in Cardiac Surgery (video assisted demonstrations on left ventricular reduction and minimally invasive coronary surgery) will be held on the 3 and 4 October 1996 at the Hilton National Hotel, Bristol, United Kingdom. For further information please fax: +44-117-929737 (Mrs N J Merrell).