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 arrhythmic 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 infarctions[1] (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 artery on the arrhythmic substrate.

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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.

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 non-surgical ablation 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,[2] including our new technique.[3] 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 reduction are performed for the palliation of symptoms. We have not suggested that survival 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 myomectomy significantly impairs overall left ventricular function is slim. In the series mentioned in the editorial,[4] the evidence for such impairment was a rise in end diastolic diameter from a mean of 4·5 to 5·1 cm over mean follow up of 8·9 years. Fractional shortening was unchanged (41% *v* 39%). The evidence quoted from Spirito *et al*'s study[5] that the natural course of hypertrophic cardiomyopathy is a progressive, inevitable decline in overall left ventricular function, with a consequent reduction in gradient is also 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.

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1 Oakley CM. Non-surgical ablation of the ventricular septum for the treatment of hypertrophic cardiomyopathy. *Br Heart J* 1995; **74**:479–80.
2 Maron BJ, Merrill WH, Freier PA, Kent KM, Epstein SE, Morrow AG. Long-term clinical course and symptomatic status of patients after operation for hypertrophic subaortic stenosis. *Circulation* 1978;**57**:1205–13.
3 Sigwart U. Non surgical myocardial reduction for hypertrophic obstructive cardiomyopathy. *Lancet* 1995;**346**:211–3.
4 Seiler C, Hess OM, Schoenbeck M, Turina J, Jenni R, Turina M, *et al.* Long-term follow-up of medical versus surgical therapy for hypertrophic cardiomyopathy: a retrospective study. *J Am Coll Cardiol* 1991;**17**:634–42.
5 Spirito P, Maron BJ, Bonow RO, Epstein SE. Occurrence and significance of progressive left ventricular wall thinning and relative cavity dilatation in hypertrophic cardiomyopathy. *Am J Cardiol* 1987;**59**:123–9.

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.

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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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1 Morgan GP. NSAIDs and the chemoprevention of colorectal and oesophageal cancer. *Gut* 1996;**38**:646–8.

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: +44-117-9299737 (Mrs N J Merrell).