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

Sr—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 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 is very strong in our experience. 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 resection—although we hope it does.

In any cardiomyopathic process, maintaining as many normally functioning myocytes as possible is clearly desirable, but the evidence that myocyte count significantly impairs 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 to 5.1 cm over mean follow up of 8-9 years. Fractional shortening was unchanged (41% \( \pm \) 3%). The evidence quoted from Spirito et al’s study1 of 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 encapsulates 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.


A wide health remit for aspirin

Sr—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 We 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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