4 Kienzle MG, Ferguson DW, Birkett CL, Myers GA, Berg WJ, Mariano DJ. Clinical, haemodynamic and sympathetic neural correlates of heart rate variability in congestive heart failure. *Am J Cardiol* 1992;69:761–7.

Dipyridamole and dobutamine for myocardial perfusion imaging

SIR,—Kumar and colleagues conclude that dipyridamole is better than dobutamine during thallium myocardial perfusion tomography.[1] We also prefer to use a vasodilator routinely (we use adenosine) and we reserve dobutamine for patients who are unable to exercise and in whom adenosine is contraindicated.[2 3] Kumar and colleagues give several reasons to justify their conclusion, including greater stress perfusion scores in the lateral wall and apex of the left ventricle when dipyridamole is used. In the light of a recent editorial pointing out the importance of rigorous statistical methods in biomedical research,[4] we question whether the conclusion and hence the title of the paper is valid.

Without a prior hypothesis of regional differences it is not appropriate to make multiple statistical comparisons of individual segments. Analysis of variance is the preferred test statistic, using a nonparametric method (Kruskal-Wallis) given the discontinuous nature of the scoring system, followed by an appropriate post hoc test for individual segments only if there is evidence of heterogeneity. Any regional differences detected in this way should then be tested prospectively in a separate group of patients. The claimed segmental difference between the two forms of pharmacological intervention is unlikely to be real because there is no plausible reason why these segments should differ from the remainder of the myocardium. Kumar *et al* make no attempt to explain this anomaly.

They also claim a better correlation of perfusion score with a score derived from the *x* ray angiogram when dipyridamole rather than dobutamine is used. There is no description of the statistical methods used in this analysis, and therefore the validity of this claim cannot be judged from the data provided.

We therefore suggest an alternative conclusion: that the null hypothesis of equivalence in efficacy for dipyridamole and dobutamine cannot be rejected, and that practical matters such as cost and duration of protocol should determine which is used in individual circumstances.

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1 Kumar EB, Steel SA, Howey S, Caplin JL, Aber CP. Dipyridamole is superior to dobutamine for thallium stress imaging: a randomised crossover study. *Br Heart J* 1994; 71:129–34.
2 Pennell DJ, Underwood SR, Swanton RH, Walker JM, Ell PJ. Dobutamine thallium myocardial perfusion tomography. *J Am Coll Cardiol* 1991;18:1471–9.
3 Pennell DJ, Underwood SR, Ell PJ. Safety of dobutamine stress for thallium-201 myocardial perfusion tomography in patients with asthma. *Am J Cardiol* 1993;71:1346–50.
4 Altman DG. The scandal of poor medical research. *Br Med J* 1994;308:283–4.

This letter was shown to the authors, who reply as follows:

SIR,—We sought statistical advice from a professional statistician on the methods used in our paper. It was suggested that Student's *t* test was appropriate for comparison of perfusion scores obtained with the two pharmacological stressing agents. The original hypothesis was that there was no difference in the effect of the two agents on myocardial perfusion and we therefore expected no difference in perfusion scores. We did do multiple analysis and agree with Dr Underwood and Professor Wood that this may have been the cause of the segmental differences.

Because dobutamine and dipyridamole induce abnormalities of perfusion by different mechanisms there may be a "true" difference in their ability to produce segmental hypoperfusion. The correlation of perfusion score with angiographic score was performed by linear regression according to the methods in Draper and Smith (*Applied regression analysis*. 2nd ed. New York: Wiley, 1981;84).

Our study undoubtedly showed that studies with dipyridamole are cheaper, better tolerated, and less time consuming than dobutamine studies, without any loss in the ability to detect abnormal myocardial perfusion.

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Long-term results of the corridor operation for atrial fibrillation

SIR,—The corridor operation for atrial fibrillation[1] is an ingenious operation in which the surgeons isolate the left and right atrial free walls from the atrial septum, leaving a corridor of contiguous tissue between the sinus and atrioventricular nodes, thereby permitting chronotropically responsive atrioventricular conduction.

Unfortunately, because both atria remain in fibrillation, the corridor procedure fails to address the two major consequences of atrial fibrillation—namely, the loss of atrial transport function and thromboembolism. Therefore, it seems that the corridor procedure has no advantage over His bundle ablation and currently it is a major cardiac surgical procedure. I note that in the series of 36 patients reported by van Hemel *et al* His bundle ablation was performed and a pacemaker implanted in five patients "in whom the corridor operation was unsuccessful".

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1 van Hemel NM, Defauw JJAMT, Kingma JH, Jaarsma W, Vermeulen FEE, de Bakker JMT, Guiraudon GM. Long-term results of the corridor operation for atrial fibrillation. *Br Heart J* 1994;71:170–6.

This letter was shown to the authors, who reply as follows:'

SIR,—We welcome the opportunity to respond to Professor Cheng's concern.

According to the Frank-Starling law, right atrial contraction and right filling pressures are the main determinants of cardiac function[1]; in addition, diminished or absent left atrial contraction is not associated with alteration of cardiac function.[2] The normal heart acts as a suction pump during normal diastole. So, at least in the normal heart, the atrial contribution to cardiac function is negligible.[3]

Because chronotropic sinus node function is the main determinant of increasing cardiac output during exercise,[4] preservation of the physiological chronotropic response is one of the aims of the corridor concept. The maintenance of native chronotropic sinus function prevents impaired exercise tolerance and avoids the lifelong dependency on a pacemaker that is one of the consequences of His bundle ablation.[5] Our long-term results show that sinus node function remained undisturbed in most of our patients.

During the corridor operation the left atrial appendage, which is commonly the origin of atrial thrombosis in patients with atrial fibrillation, is excised. Possibly, resection of the left atrial appendage alone could prevent systemic emboli in patients with lone atrial fibrillation. This measure has not been tried in patients in whom atrial fibrillation continues after catheter ablation of the His bundle for rate control of drug refractory atrial fibrillation. Their risk of thromboembolism is not negligible.

Though corridor surgery did not suppress atrial fibrillation in all our patients, postoperative atrial fibrillation occurred only in the left atrium and never de novo in the corridor. The operation was sometimes unsuccessful because we failed to create a persistent conduction block between the left atrium and the coronary sinus. This is why some of our patients needed His bundle ablation. Such surgical failures require technical improvement, but they do not detract from the corridor concept.

Currently, it is impossible to make a valid comparison between a non-selective and less invasive procedure such as His bundle catheter ablation[6] and selective surgery for atrial fibrillation.[7] This is because of the differences in selection criteria, patient population, and the end point of treatment. The comparison is not even scientifically valid, because atrial fibrillation is a multifactorial protean disease.[8] In the face of excellent long-term results, we strongly believe that surgical procedures for atrial fibrillation will become a well-established treatment in some subgroups of patients—for example, those who do not want to be dependent on a pacemaker and those who need cardiac surgery for other reasons.

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1 Robinson TF, Factor SM, Sonnenblick EH. The heart as a suction pump. *Scientific American* 1986;254:84.

2 Williams JM, Ungerleider RM, Lofland GK, Cox JL. Left atrial isolation: new technique for the treatment of supraventricular arrhythmias. *J Thorac Cardiovasc Surg* 1980; **80**:373–80.
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4 Atwood JE. Exercise hemodynamics of atrial fibrillation. In: Falk RH, Podrid PJ, eds. Atrial fibrillation, mechanisms and management. New York: Raven Press, 1992; 145–63.
5 Hayes DL. Pacemaker complications. In: Furman S, Hayes DL, Holmes DR Jr, eds. A practice of cardiac pacing. Mount Kisco, New York: Futura, 1986;253–71.
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7 Cox JL, Boneau JP, Schuessler RB, Ferguson TB Jr, Cain ME, *et al.* Successful surgical treatment of atrial fibrillation. *J Am Med Ass* 1991;**266**:1976–1980.
8 Guiraudon GM. Surgical treatment of atrial fibrillation. *Herz* 1993;**18**:51–9.

Neurostimulation and myocardial ischaemia

SIR,—I read with interest the recent report by de Jongste *et al* and the editorial by Mulcahy *et al* on neurostimulation and the treatment of intractable angina.[1 2] de Jongste *et al* provide further evidence that neurostimulation does not simply abolish chest pain but also affects myocardial ischaemia, reducing the frequency and duration of transient ischaemic episodes during ambulatory monitoring.[1] They propose that the anti-ischaemic action of spinal cord stimulation may be the result of an increased oxygen supply to the heart caused by a redistribution of coronary blood flow.

I and coworkers showed that transcutaneous electric nerve stimulation (TENS) can increase resting coronary blood flow.[3] We studied the effect of TENS in 34 patients with syndrome X (group 1), 15 patients with coronary artery disease (group 2), and 16 heart transplant recipients (group 3). Coronary blood flow velocity (CBFV) (mean (SD)) in the left coronary system was measured with a Judkins-Doppler catheter at rest and after stimulation. There was a significant increase in the resting CBFV in group 1 (from 6·8 (4·1) to 10·5 (5·7) cm/s, $P < 0.001$) and group 2 (from 6·8 (4·1) to 10·5 (5·7) cm/s, $P < 0.001$). However, there was no significant change in the resting CBFV in group 3. There were no significant changes in the coronary arterial diameters as a result of neurostimulation, suggesting that the mechanism of action of TENS is at the microcirculatory level. This is the first study to show that neurostimulation can increase coronary blood flow. This may explain its anti-ischaemic effects, which have been reported by several studies.[1 4 5]

I agree with the conclusion of Mulcahy *et al* that TENS and spinal cord stimulation are effective in the treatment of intractable angina and should be considered before the patient is subjected to a less tried treatment.[2] Certainly, TENS treatment may provide a useful, non-invasive, and a safe alternative in the treatment of patients with intractable angina. Indeed, it may also provide a means of selecting patients who are more likely to benefit from spinal cord stimulation, a more invasive method of pain relief.

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1 de Jongste MJL, Haaksma J, Hautvast RWM, Hillege HL, Meyler PWJ, Staal MJ, *et al.* Effects of spinal cord stimulation on myocardial ischaemia during daily life in patients with severe coronary artery disease: a prospective ambulatory electrocardiographic study. *Br Heart J* 1994;71:413–8.
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3 Chauhan A, Mullins PA, Thuraisingham SI, Taylor G, Petch MC, Schofield PM. Effects of transcutaneous electrical nerve stimulation on coronary blood flow. *Circulation* 1994;**89**:694–702.
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5 Mannheimer C, Eliasson T, Andersson B, Bergh C-H, Augustinsson LE, Emanuelsson H, *et al.* Effects of spinal cord stimulation in angina pectoris induced by pacing and possible mechanisms of action. *BMJ* 1993; **307**: 477–80.

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

SIR,—Several non-randomised clinical studies showed that, in addition to its analgesic effect, neurostimulation reduced myocardial ischaemia assessed by electrocardiography during exercise testing. We confirmed these findings in a randomised study.[1] Though myocardial ischaemia, when present, does not seem to be concealed by the neurostimulation, a placebo effect is likely to some extent. However, only a prospective mortality study can establish definitively that the treatment is safe. Furthermore, the mechanism for the anti-ischaemic action is not clear. Chauhan and coworkers provide us with valuable evidence of an increase in resting coronary blood flow velocity only after 5 minutes of neurostimulation.[2] Their finding accords with the study by de Landsheere *et al*,[3] who used positron emission tomography (PET) during epidural spinal cord stimulation, and Mannheimers' recent article on the beneficial influence of spinal cord stimulation on impaired left ventricular function.[4] De Landsheere *et al* found that ST segment depression was significantly reduced during neurostimulation and that regional myocardial blood flow was increased at rest. However, they did not see a significant increase in myocardial blood flow during exercise. This latter finding may relate to the method or to a long-lasting carry over effect of neurostimulation. We showed, however, in our PET study after dipyridamole stress testing in nine patients, that the perfusion ratio in the ischaemic region increased more than the ratio in the non-ischaemic region. This indicates a redistribution phenomenon.[5] Whether the presumed anti-ischaemic effect of neurostimulation is related to alterations in myocardial oxygen supply or in demand is not yet known.

Because neurostimulation is thought to trigger many interactions of neurohumoral compounds involved in neuronal networks, molecular biology may help us to determine the mechanism of action of neurostimulation.

Finally, before neurostimulation becomes generally accepted as an additional treatment for patients with severe angina, many technical problems remain to be overcome, such as lead dislocations and optimal stimulation characteristics, and strategies are needed to establish which patients need what kind of stimulation.

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1 De Jongste MJL, Hautvast RWM, Hillege HL, Lie KI. Efficacy of spinal cord stimulation as adjuvant therapy for intractable angina pectoris: a prospective randomized clinical study. *J Am Coll Cardiol* 1994; **23**:1592–7.
2 Chauhan A, Mullins PA, Thuraisingham SI, Taylor G, Petch MC, Schofield PM. Effects of transcutaneous electrical nerve stimulation on coronary blood flow. *Circulation* 1994;**89**:694–702.
3 De Landsheere C, Mannheimer C, Habets A, Guillaume M, Bourgeois I, Augustinsson LE, *et al.* Effect of spinal cord stimulation on regional myocardial perfusion assessed by positron emission tomography. *Am J Cardiol* 1992;**69**:1143–9.
4 Kujacic V, Eliasson T, Mannheimer C, Jablonskiene D, Augustinsson LE, Emanuelsson H. Assessment of the influence of spinal cord stimulation on left ventricular function in patients with severe angina pectoris: an echocardiographic study. *Eur Heart J* 1993;**14**:1238–44.
5 Hautvast RWM, de Jongste MJL, Staal MJ, Blanksma PK, Lie KI. Spinal cord stimulation in myocardial perfusion in patients with refractory angina pectoris as assessed by positron emission tomography. International Neuromodulation Society: Proceedings of the second international congress Gothenburg, 1994.

Simon Dack

SIR,—May I clarify an editorial adjustment made to the appreciation of Simon Dack (*British Heart Journal*, August 1994, page 104)? In the course of editing the manuscript an error crept in which I only saw in the published version. Once Dr Dack had retired from the Editorship of the *Journal of the American College of Cardiology*, Dr Parmley enlisted him as the outside consultant editor for articles emanating from Dr Parmley's own institution, the University of California, San Francisco, and not, as appears, the Mount Sinai Hospital. I only take the trouble to point this out as the purpose was to maintain the highest possible standards of peer review, to which Dr Dack was devoted: the idea that Dr Parmley had was to continue to use his services so that there would be completely independent editorial assessment of contributions submitted to the *Journal of the American College of Cardiology* from the University of California, San Francisco.

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