

- 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.
- 3 Sonnenblick EH, Braunwald E, Williams JF Jr, et al. Effects of exercise on myocardial force-velocity relations in intact unanesthetized man: relative roles of changes in heart rate, sympathetic activity, and ventricular dimensions. *J Clin Invest* 1965;44:2051.
- 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.
- 6 Scheinman MM, Evans-Bell T. Catheter ablation of the atrioventricular junction: a report of the percutaneous mapping and ablation registry. *Circulation* 1984;70: 1024-9.
- 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.

ANOOP CHAUHAN
Department of Cardiology,
Royal Infirmary of Edinburgh,
Lauriston Place,
Edinburgh EH3 9YW

- 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.
- 2 Mulcahy D, Knight C, Stables R, Fox K. Lasers, burns, cuts, tingles and pumps: a consideration of alternative treatments for intractable angina. *Br Heart J* 1994;71: 406-7.
- 3 Chauhan A, Mullins PA, Thuraingham SI, Taylor G, Petch MC, Schofield PM. Effects of transcutaneous electrical nerve stimulation on coronary blood flow. *Circulation* 1994;89:694-702.
- 4 Mannheimer C, Carlsson CA, Emanuelsson H, Vedin A, Waagstein F, Wilhelmsson C. The effects of transcutaneous nerve stimulation in patients with severe angina pectoris. *Circulation* 1985;71:308-16.
- 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 neuro-

stimulation 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.

MIKE J L DE JONGSTE
Department of Cardiology, Thoraxcenter,
University Hospital of Groningen,
The Netherlands

- 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, Thuraingham 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, Blankens 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.

D M KRIKLER
Past Editor *British Heart Journal*