Dipyridamole anddobutamine for myocardial perfusion imaging

Sr.,-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 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 techniques in biomedical research,3 we question whether the conclusion and hence the title of the paper is valid.

Without a priori 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 scheme 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 significant 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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Long-term results of the coroild operation for atrial fibrillation

Sr.,—The corridor operation for atrial fibrillation is an ingenious operation in which the surgeon isolates 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 for a pacemaker implanted in five patients “in whom the corridor operation was unsuccessful”.

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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. They propose that the anti-ischaemic effect 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 electrical 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 12 healthy, matched 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 6·9 (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

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 referred 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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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 of the anti-ischaemic effect 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, concordant with the study of De Lange et al.,3 who used positron emission tomography (PET) during epidual spinal cord stimulation, and Mannheimsers' 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 to resting level.5 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 finding indicates a redistribution phenomenon.6 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.


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, I erred in reformatting 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, and 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 an independent editorial assessment of contributions submitted to the Journal of the American College of Cardiology from the University of California, San Francisco.

D M KRKLER
Past Editor British Heart Journal