Lead specificity of the maximum ST/heart rate slope response

Sir,

Once more correspondence columns have borne witness to the confusion caused by the Leeds exercise test. Yet again Professor Linden and his colleagues have been able to claim that their protocol has not been adhered to—and of course on this occasion they are correct in that respect.^{1,2} They have, however, chosen to ignore other studies that have followed their protocol meticulously and that have, nevertheless, failed to reproduce their results.^{3,4}

This has surely now reached a point for real concern to be expressed. A considerable amount of time and money, much of the latter from charitable sources, has been spent trying to confirm the perfect accuracy of this test and this has proved a wild goose chase. This might be a cause for some amusement were the competition for scarce research funds not so intense. Nevertheless, the alleged usefulness of this test has been widely and uncritically accepted in many countries. There is reason to believe that the management of many patients may have been largely determined by the result of a Leeds exercise test, and of course this could well result in quite inappropriate decisions with regard to further investigation and treatment in individual cases.

The fact is that the test works only in Leeds. Nobody else, using the unmodified Leeds protocol, has been able to reproduce the results, and surely there-

fore the test must now either be abandoned or else all patients undergoing it must be sent to Professor Linden's unit since that would appear to be the only place where it can be properly performed and interpreted.

It is of interest that the recent letter from Professor Linden and Dr Mary seems to indicate that the test, even in Leeds, is no longer perfect so perhaps the time has come for it to be abandoned.

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References

- 1 Beattie JM, Seibert GB, Blomqvist CG. Lead specificity of the maximum ST/heart rate slope response [Letter]. *Br Heart J* 1985; 53: 349.
- 2 Linden RJ, Mary DASG. Lead specificity of the maximum ST/heart rate slope response [Letter]. *Br Heart J* 1985; 53: 688.
- 3 Balcon R, Brooks N, Layton C. Correlation of heart rate ST slope and coronary angiographic findings. *Br Heart J* 1984; 52: 304-7.
- 4 Quyyumi AA, Raphael MJ, Wright C, Bealing L, Fox KM. Inability of the ST segment/heart rate slope to predict accurately the severity of coronary artery disease. *Br Heart J* 1984; 51: 395-8.

Management of primary pulmonary hypertension

Sir,

Dr Oakley's interesting editorial on the management of primary pulmonary hypertension (*Br Heart J* 1985; 53: 1-4) contains an error. The sequence of the vascular lesions that is described ends with fibrinoid necrosis and the "plexiform arteriopathy" of Wagenvoort and Dr Oakley goes on to say "but the therapeutic dilemma is how to get hold of patients at a stage of medial hypertrophy before these irreversible changes occur." Plexiform lesions develop after fibrinoid necrosis. "Plexiform arteriopathy" does not exist. The term, plexogenic arteriopathy was devised by a World Health Organisation committee¹ to describe a morphological pattern of which the plexiform lesion is the final stage. There-

fore, plexiform lesions and other irreversible changes need not to be present in plexogenic arteriopathy. The condition, at least in association with congenital heart disease, is reversible in its early stages and not only when medial hypertrophy alone is present.² The primary form of plexogenic arteriopathy is also likely to be reversible if an effective treatment can be used at an early stage of disease.

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References

- 1 Hatano S, Strasser T. *Primary pulmonary hypertension. Report of a WHO meeting*. Geneva: World Health Organization, 1975: 14-16.
- 2 Wagenvoort CA, Wagenvoort N, Draulans-Noë Y. Reversibility of plexogenic pulmonary arteriopathy following banding of the pulmonary artery. *J Thorac Cardiovasc Surg* 1984; 87: 876-86.

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

Sir,

I am grateful to Professor Wagenvoort for his semantic correction. "Plexogenic" presumably means giving rise to "plexiform". If only the arteriopathy

were "plexogenic" rather than "plexiform" when these patients are referred then we might produce some therapeutic benefit with vasodilator drugs. Very occasionally death from primary pulmonary hypertension occurs when the arteriopathy is still only "plexogenic" and one feels that in these rare patients the disease might have been amenable to treatment. We need to see patients when their arteriopathy although apparently "plexogenic" is not yet "plexiform".

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Cardiac transplantation

Sir,

I was interested to read the letter in which Dr Evans (1985; 53: 472) compared the survival statistics of the Stanford University Cardiac Transplantation Program with the survival rates of medically treated patients with dilated cardiomyopathy in the United Kingdom. Dr Evans states that medical treatment of dilated cardiomyopathy carries a one year mortality rate of 34.7% and five year mortality rate of 59.4%. He compares this with the 37% one year mortality rate and a 61% five year mortality rate at Stanford. He then takes exception to the statement "that cardiac transplantation prolongs life". Fortunately, the current data upon which we base our justification for cardiac transplantation in the United States do not agree with Dr Evans's contention for several reasons.

The first and foremost criterion for cardiac transplantation in the United States is the presence of New York Heart Association class IV heart failure. In patients with class IV congestive heart failure, including those with coronary artery disease, one year mortality is 50% and three year mortality is 80%.¹ Since the quoted Stanford data include both patients with ischaemic heart disease and primary dilated cardiomyopathy the data do indeed appear promising. The Stanford statistics quoted comprise data accumulated before the introduction of cyclosporin immunosuppression. Comparison of one year and three year mortality rates in the registry maintained by the International Society for Heart Transplantation,² shows a 15% one year mortality

rate and a 25% three year mortality rate in patients who have had cardiac transplantation and immunosuppression with cyclosporin. Therefore, the most recent data indicate that cardiac transplantation does indeed prolong survival in those patients with New York Heart Association class IV heart failure. The problem in 1985 is not one of the therapeutic efficacy or of satisfactory long term immunosuppression but rather that of donor heart availability. The efforts of the medical community must be directed towards reducing the high early mortality rate of patients who presently are not surviving long enough to undergo cardiac transplantation.

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References

- 1 Califf RM, Bounous P, Harrell FE, *et al.* The prognosis in the presence of coronary artery disease. In: Braunwald E, Mock MB, Watson JT, eds. *Congestive heart failure and current research and clinical applications*. New York: Grune and Stratton, 1982: 31-40.
- 2 Kaye MP, Elcombe SA, O'Fallon WM. The International Heart Transplantation Registry: the 1984 report. *J Heart Transplant* 1985; 3: 290-2.