Chelation therapy for ischaemic heart disease

I read with interest the review by Dr Len Brake of *Chelation Therapy for Ischaemic Heart Disease – A Randomised Controlled Trial* (Knudston et al., JAMA 23rd January 2002, Vol 287 No 4 pp481–6 – reviewed in *NZFP* Vol 29 April 2002 p131).

The review states: *'Patients were randomly assigned either an infusion of 40 mg/kg EDTA or a placebo infusion twice weekly for 15 weeks.'*[1]

However, although this statement appears in the abstract of the research, it is in fact far from correct.

The methodology clearly states that the comparison was between (1) EDTA 40 mg/kg + 750 mg magnesium sulphate + 5 grams of ascorbic acid + 5 grams of sodium bicarbonate + 80 mg of Lidocaine versus (2) 750 mg of magnesium sulphate + 5 grams of ascorbic acid + 5 grams of sodium bicarbonate + 80 mg of Lidocaine.

A placebo, by definition, is an inert substance. However, the control group in this study did not receive a placebo – they received an intravenous infusion of vitamin C and magnesium (an infusion which is known to be vasoactive).

Despite a small sample size (after screening 3 140 patients, 84 were enrolled), the EDTA (case) group showed a statistically significant improvement in ETT time to ischaemic change ($p<0.001$). The 'control' group, who received the so-called 'placebo', also showed a significant improvement in ETT time to ischaemic change ($p<0.001$).

Thus there was no significant difference between the two groups. The authors concluded that *'...there is no evidence to support a beneficial effect of chelation therapy in patients with ischaemic heart disease, stable angina, and a positive treadmill test for ischaemia.'*

Since, however, the 'control' group received a vasoactive infusion, the logical conclusion is that EDTA chelation therapy produced a significant improvement, and that treatment with a series of intravenous infusions of vitamin C and magnesium also produced a significant improvement.

The study published in JAMA has several flaws. However, since the 'placebo' was not a placebo, the study is not worthy of further consideration.

This is not the first time that a prominent medical journal has published a RCT showing a negative result for EDTA chelation therapy. The Van Rij study[2] was flawed by the same defect as the JAMA study, and (not surprisingly) led to the same conclusion.

Practitioners are entitled to rely on the editorial boards of the world's most prominent medical journals (and on reviewers of papers) to be rigorous. Publication of research should be restricted to soundly-based studies of the highest quality, which have been reviewed by persons knowledgeable in the subject. Anyone with knowledge of EDTA chelation would have rejected the JAMA paper out of hand because of the non-placebo 'placebo'.

In reviewing the Knudston article, therefore, it is important to correct the conclusions drawn in the article.

Dr Jon Richardson

References

1. Knudston ML, Wyse DG, Galbraith PD, Brant R, Hildebrand K, Paterson D, Richardson D, Burkart C, Burgess E. JAMA 2002; 287:481–6.
2. Van Rij AM, Solomon C, Packer SG, Hopkins WG. Chelation therapy for intermittent claudication: a double blind, randomised, controlled trial. Circulation 1994; 90:1 194–1199.