Treatment of hypertrophic obstructive cardiomyopathy with verapamil

Sir,

We read the article by Kaltenbach *et al.*¹ with interest.

The principle of the treatment is based on the interference with calcium availability in the super-normal contracting ventricle of hypertrophic cardiomyopathy. Though the idea is appealing and the reported results impressive there is a potential problem in the use of verapamil.

Haemodynamic studies with verapamil² have shown a rise in the left ventricular end-diastolic pressure and a concomitant drop in dP/dt, evidence for a depressant effect of the drug on myocardial function. Such an action would be of benefit to subjects with hypertrophic cardiomyopathy.

A more recent report,³ however, showed clearly that, because of the potent vasodilator effect of the drug, cardiac output increases, despite the decreased contractility.

In patients with hypertrophic cardiomyopathy, the pronounced vasodilator effect of verapamil could increase the intraventricular obstruction, thereby precipitating a potentially dangerous situation. It is of interest that the authors found that left ventricular pressure gradients did not show the same consistent improvement as did subjective symptoms. In some of their patients the gradients, in fact, increased after verapamil.

This potential problem should certainly be considered when using this drug in these patients.

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References

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- ²Lewis BS, Mitha AS, Gotsman MS. Immediate haemodynamic effects of verapamil in man. *Cardiology* 1976; **60**: 366-76.
- ³Ferlinz J, Easthope J, Aronow WS. Effects of verapamil on myocardial performance in coronary disease. *Circulation* 1979; **59**: 313-9.

This letter was shown to Dr Kaltenbach and Dr Hopf who reply as follows:

Sir,

Calcium antagonists have actions on the smooth muscle of blood vessels as well as on the myocardium. With different substances either the central or the peripheral effect may be more pronounced. The vasodilator activity of verapamil can best be seen after intravenous administration as a bolus injection. Theoretically, patients with hypertrophic cardiomyopathy may be made worse by this effect. In fact, however, after 10 mg verapamil intravenously there was no change in intraventricular gradient. On the other hand, after giving the substance as intravenous infusion, Rosing *et al.*¹ found, in patients with hypertrophic cardiomyopathy, a significant decrease in the intraventricular gradient from a mean of 85 to 50 mmHg, as well as a decrease in the gradient provoked by the Valsalva manoeuvre or amyl nitrite.

Our own studies with verapamil administered orally into the coronary circulation show negative inotropic and chronotropic activity. The changes seen after high-dose oral administration are comparable to the intracoronary administration.

Thus the vasodilator activity of verapamil after oral administration seems not to counteract the therapeutic benefit on the myocardium. It is, however, important to note that the beneficial therapeutic activity of this substance seen in patients with hypertrophic cardiomyopathy, therefore, can only be attributed to verapamil and not necessarily to other calcium antagonistic substances.

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Reference

- ¹Rosing DR, Kent KM, Borer JS, Seides SF, Maron BJ, Epstein SE. Verapamil therapy: a new approach to the pharmacologic treatment of hypertrophic cardiomyopathy. *Circulation* 1979; **60**: 1201-13.