Echocardiographic, haemodynamic, and angiographic correlations in hypertrophic cardiomyopathy

Sir,

Chahine *et al.* in a recent paper (*British Heart Journal*, 1977, 39, 945-953) conclude that there is no pathognomic finding in idiopathic hypertrophic subaortic stenosis; furthermore, there is no such disease as idiopathic hypertrophic subaortic stenosis but rather a spectrum of pathology. I doubt whether on the basis of an ill-defined group of 14 patients one could seriously question the classical concept of asymmetrical septal hypertrophy. In their series, 8 patients with intraventricular gradients and cavity obliteration had no echocardiographic sign of idiopathic hypertrophic subaortic stenosis, neither asymmetrical septal hypertrophy nor systolic anterior movement of the mitral valve. I suggest three possible explanations for this discrepancy.

(1) Organic subvalvar aortic stenosis, which was not excluded in the present study, sometimes presents with the same clinical and haemodynamic signs as idiopathic hypertrophic subaortic stenosis.

(2) Cavity obliteration on the angiogram is not generally accepted as a pathognomic feature of idiopathic hypertrophic subaortic stenosis, but is a nonspecific sign of left ventricular hypertrophy and hypercontractile states.

(3) Could the echocardiograms have been misinterpreted? I wonder if the two echocardiograms in Fig. 5 were obtained from the same patient? I suggest that levels A and B show concentric hypertrophy without evidence of idiopathic hypertrophic subaortic stenosis, while level C is a typical idiopathic subaortic stenosis pattern with both asymmetrical septal hypertrophy and systolic anterior movement of the mitral valve. The EF slope on levels A and B is normal while on level C it is definitely reduced. In my experience such variation of the EF slope does not occur.

Maria Lengyel,
Hungarian Institute of Cardiology,
1450 Budapest, P.O.B. 88,
Hungary.

This letter was shown to Chahine and his co-authors who reply as follows:

Sir,

We appreciate Dr Lengyel's interest in our recent paper and are grateful for the opportunity to clarify further some of the points we discussed.

Our patients were carefully chosen on the basis of well-accepted criteria: typical clinical picture supported either by the echocardiographic findings of asymmetrical septal hypertrophy and systolic anterior movement or the haemodynamic demonstration of an intraventricular pressure gradient. These are the most usual diagnostic features, as they appear in the current textbook to which we referred in our paper.

In our series of patients only 1 (not 8) had neither asymmetrical septal hypertrophy nor systolic anterior movement of the mitral valve on the echocardiogram: 5 had both asymmetrical septal hypertrophy and systolic anterior movement, 6 had asymmetrical septal hypertrophy without systolic anterior movement, and 2 had systolic anterior movement without asymmetrical septal hypertrophy, as clearly shown in the Table in our paper.

In answer to the 3 possible explanations for the discrepancies seen by Dr Lengyel:

(1) None of the patients included in this paper had any haemodynamic or angiographic evidence of any form of fixed aortic stenosis (valvar, subvalvar, or supra-valvar). If such a diagnosis were suspected, all necessary studies were performed and the patient was excluded from this series.

(2) We make no claim in this paper that cavity obliteration is a pathognomic finding in idiopathic hypertrophic subaortic stenosis. Dr Lengyel clearly notes that we do not think that there is a pathognomic feature. We only recognise that this angiographic finding is common in idiopathic hypertrophic subaortic stenosis, but it can occur in other conditions, as we have recently reported (Raizner *et al.*, 1977).

(3) The echocardiographic records in Fig. 5 are taken from the same patient and serve to illustrate the variability of the echocardiographic findings recorded at different levels and the need for M-mode scans for appropriate diagnosis. Dr Lengyel's concern that the two echocardiograms appear to be

from different patients further enhances our point.

We have listed in our paper several references reporting cases of idiopathic hypertrophic subaortic stenosis without asymmetrical septal hypertrophy and asymmetrical septal hypertrophy in conditions other than idiopathic hypertrophic subaortic stenosis. We do not doubt that 'idiopathic hypertrophic subaortic stenosis' exists; rather, we question its homogeneity. We are pleased to note that since we have submitted this paper several other publications have appeared focusing on atypical aspects of idiopathic hypertrophic subaortic stenosis (Come *et al.*, 1977; Falicov and Resnekov, 1977). Such reports lend support to our concept that patients presenting clinically as idiopathic hypertrophic subaortic stenosis may represent a spectrum of pathology rather than a single well-defined disease.

Robert Chahine, Albert E. Raizner,
Tetsuo Ishimori, and Alfredo C. Montero,
Baylor College of Medicine,
V.A. Hospital,
Houston,
Texas,
U.S.A.

References

- Come, P. C., Bulkley, B. H., Goodman, Z. D., Hutchins, G. M., Pitt, B., and Fortuin, N. J. (1977). Hypercontractile cardiac states simulating hypertrophic cardiomyopathy. *Circulation*, **55**, 901-908.
- Falicov, R. E., and Resnekov, L. (1977). Mid ventricular obstruction in hypertrophic obstructive cardiomyopathy. New diagnostic and therapeutic challenge. *British Heart Journal*, **39**, 701-705.
- Raizner, A., Chahine, R., Ishimori, T., and Awdeh, M. (1977). Clinical correlates of left ventricular cavity obliteration. *American Journal of Cardiology*, **40**, 303-309.