transposition. Yet all the patients that they studied with atrioventricular discordance also had ventriculo-arterial discordance. As far as we are aware (and we are supported by Metcalfe and Somerville) this combination is the one usually referred to as congenitally corrected transposition. It is quite clear that our comment was made specifically in the context of atrioventricular discordance. Thirdly, they accuse us of raising the equiplanar insertion of the atrioventricular valve leaflet to the status of a pathognomonic sign of an inlet ventricular septal defect. We do not know how they reached this conclusion. This statement is not to be found anywhere in our writings. Indeed, in the introduction to the paper they refer to, we stated—alluding to the valve attachments being at the same level—"This finding is of course characteristic of a double inlet atrioventricular connexion, but in this case its importance is trivial compared to the other abnormalities present." There is another equally important reason why we would not make this statement. The offsetting of the atrioventricular valve leaflets and the presence of a muscle bar projecting downwards from the atrioventricular junction to roof a defect clearly differentiates an inlet muscular defect from a perimembranous inlet defect.

We are grateful to take this opportunity to clarify our figure legends in the four instances in which pathological specimens were shown with the corresponding echocardiograms. As these stand in the text they could lead to some confusion. In Fig. 3 and Fig. 4 the pathological specimens were not from the patient whose echocardiogram is shown but were sections from similar patients drawn from the cardiopathological collections of the three centres concerned. In Fig. 9 and Fig. 10 the pathological specimens were from the patient whose echocardiogram is shown.

Finally we wish to defend the principle of using corresponding anatomical specimens to illustrate echocardiographic stop frame images. This has now become a widely adopted form of presentation, and such correlative papers have appeared in the majority of leading cardiac journals. Indeed a large number of such papers have undergone review and publication in the British Heart Journal. We feel strongly that such a correlative presentation leads to a greater appreciation of the information which can be derived from echocardiographic images obtained from patients with complex heart disease. We would further suggest that Foale et al are in the minority in voicing criticisms of what is now a widely accepted form of correlative presentation.

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Reference

Myocardial disorganisation in hypertrophic cardiomyopathy

Sir,

"Whatever is only almost true is quite false, and among the most dangerous of errors, because being so near truth it is the most likely to lead astray."

I was reminded of the words of Henry Ward Beecher when reading Dr Maron's editorial in the July issue of this journal (1983;50:1–3). In his rebuttal of our work I Dr Maron makes errors not only in misunderstanding what we had to say but also in pointing in the wrong direction when complaining that others have generated a controversy regarding the role of myocardial disorganisation (or disarray). Indeed, Dr Maron does not hesitate to extrapolate from his interpretation of the works of others, including the paper by Becker and Caruso, that those investigators lack any meaningful experience with hypertrophic cardiomyopathy.

Regarding the issue at stake it may be necessary to briefly reiterate the most salient points. Firstly, the rather loose use of terms such as "characteristic," "typical," and "specific," when referring to myocardial disorganisation in cases with hypertrophic cardiomyopathy, has led to the misconception that this particular histological feature is a useful diagnostic
marker in a clinical setting. The problem started after
the excellent article by Ferrans and associates,2 con-
cerned in particular with the pathogenesis of the dis-
eease, stating that disarray was “unique” for hyper-
trophic cardiomyopathy. The statement implied that
disorganisation was a pathognomonic feature and
hence could serve as a diagnostic marker. Subse-
quently, the Bethesda group—with Dr Maron quite
often in the lead—gradually shifted their stand using
phrases such as “not absolutely specific”3,4 and “not
specific and commonly found in other forms of
myocardial hypertrophy.”5 They presently claim that
myocardial disarray is a highly sensitive and specific
marker for hypertrophic cardiomyopathy when sites
of naturally occurring “disorganisation” are excluded.
Their diagnostic sections are taken midway through
the ventricular septum or from the affected left ven-
tricular free wall, in through and through fashion,
and then microscopic sections are sliced in a transverse
plane.6–8

In the light of these statements we have simply
questioned the relevance of myocardial disarray as a
diagnostic marker in a clinical setting.1 This possi-
Bility has not passed unnoticed to these distinguished
investigators, since they also had reached the conclu-
sion that small pieces of myocardial tissue are of
limited value in diagnosing hypertrophic car-
diomyopathy.9,10

In the light of the above it is sad that Dr Maron in
his paragraph “cardiac muscle cell disorganisation as a
marker for hypertrophic cardiomyopathy” fails to
point out that the quantitative assessments to which
he refers are based on sections encompassing the full
thickness of the ventricular septum or the left ven-
tricular free wall. His statement, therefore, that
myocardial disarray distinguished hypertrophic car-
diomyopathy from other lesions producing left ven-
tricular hypertrophy should be considered with this
restriction in mind. It is here that Dr Maron comes
quite close to the truth but for reasons unknown has
decided not to go all the way. This is the more regre-
table since we agree that full thickness sections will
usually show disarray as the leading histological fea-
ture. As far as I am aware all other workers in the field
will agree with this statement. The point we made,
and which Dr Maron apparently has not appreciated,
is that small biopsy specimens taken from the left
ventricular endomyocardium are unlikely to be diag-
nostic for hypertrophic cardiomyopathy since in that
circumstance myocardial disarray is a poor marker.
On that basis we question whether myocardial disor-
ganisation has a place in the clinical decision making
when a patient is suspected of having this disease.

Dr Maron has apparently not appreciated the fact
that we studied normal hearts purposely in order to
see whether or not disorganisation occurred in the
setting of the normal heart and to what extent it could
affect the interpretation of tissue samples of normal
myocardium. Since our study showed that “disorgan-
isation” could be produced by changing the orienta-
tion of the plane of section we cautioned against an
overenthusiastic diagnosis of myocardial disorganisa-
tion, with all its implications, based on routinely pro-
cessed histological sections. We had the impression
that this was all clear and straightforward, not giving
rise to further bias. To our surprise Cardiology 1983
contains a similar misinterpretation of our work.11 Be
that as it may, in our paper1 nowhere did we extro-

tax our findings to hypertrophic cardiomyopathy so
as to suggest that myocardial disarray is not a feature
of that disease. I challenge Dr Maron to quote from
our paper,1 without distorting the context, any sen-
tence that even suggests this implication. It seems,
that, this interpretation is based on pure inference
on the part of Dr Maron rather than on any implica-
tion by ourselves.

As far as I am aware none of the workers quoted by
Dr Maron, including Becker and Caruso, has claimed
that myocardial disorganisation is not a feature of
hypertrophic cardiomyopathy. Indeed, when Dr
Maron reads and digests our paper fully he will
undoubtedly encounter a crisp sentence: “The above
discussion should not be construed as suggesting
that myocardial disarray is not a feature of hypertrophic
cardiomyopathy” (p 536). It is ironic, therefore, that I
agree wholeheartedly with Dr Maron’s final conclu-
sion that further study of myocardial disarray may be
beneficial in understanding the natural history of
hypertrophic cardiomyopathy.

I am left with the overall conclusion that Dr Maron in
his editorial has in no way aired another viewpoint.
Instead, he has produced still further obfuscation
with his “straw man.”

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References

1 Becker AE, Caruso G. Myocardial disarray. A critical
2 Ferrans VJ, Morrow AG, Roberts WC. Myocardial
ultrastructure in idiopathic subaortic stenosis. A study of
operatively excised left ventricular outflow tract muscle
3 Maron BJ, Ferrans VJ, White RI. Unusual evolution of
acquired infundibular stenosis in patients with ventricu-
Correspondence

10 Isner JM, Maron BJ, Roberts WC. Comparison of amount of myocardial cell disorganization in operatively excised septectomy specimens with amount observed at necropsy in 18 patients with hypertrophic cardiomyopathy. Am J Cardiol 1980; 46: 42-7.

Notices

"Congenital heart disease made simple"

A course on the major congenital cardiac anomalies is to be held from 8 to 11 October 1984 at the Institute of Child Health, London. Further information may be obtained from: Dr S G Haworth, Institute of Child Health, 30 Guilford Street, London WC1N 1EH.

British Cardiac Society

The Autumn Meeting in 1984 will be held on 3 and 4 December 1984, and the closing date for receipt of abstracts will be 15 August 1984.