Correspondence

Br Heart J 1981; 45: 230-1

Mechanism of influence of PR interval on loudness of first heart sound

Sir,

I would like to make some comments about the article by Leech and coworkers (*Br Heart J* 1980; 43: 138-42).

The authors studied the relation between the intensity of first heart sound and the interval Q mitral valve closure. In Fig. 2, we see this relation plotted but no information is given about its statistical significance and so we have to assume that it was not significant. In Fig. 4, the relation is again plotted, this time in semilogarithmic form; and here "the correlation was highly significant ($r=0.65$; $p<0.001$)". It is on this basis that the authors state: "we have confirmed the fact that closure of the mitral valve from the wide open position produced a loud sound, and from a semi-closed position a soft sound".

I would like to point out that two variables can show a relation to each other without a cause-effect relation between them; they might be related to a third variable. The authors have shown in Fig. 4 only the existence of a relation between $\log M1/A2$ and Q-MC. Since the correlation coefficient is 0.65, the explained variance is 42.25 per cent of the total, so that only less than one-half of the variance of the data is explained by the calculated relation. It is clear from that figure that, for Q mitral closure intervals restricted to 45 to 60 ms, the range of intensity of the first heart sound covers practically the whole plotted scale.

Thus, the data cannot be interpreted as showing that the timing of mitral closure is a primary determinant of the amplitude of the first component of the first heart sound in normal subjects.

Another fact against this is that "there was no overall correlation between closing velocity (of mitral leaflets) and $M1/A2$ ratio". It is true that a delayed closure of the mitral valve occurs, "at a point where the rate of rise of the ventricular pressure pulse is steeper"; it is also true that "the closing velocity of the anterior mitral leaflet is obviously faster in the subject with a short PR interval"; therefore, it is surprising that the loudness of the first heart sound showed no significant correlation

with the closing velocity unless other major factors are also involved (despite the mention by the authors of technical difficulties, at least the mean closing velocity of the anterior leaflet is easily measurable).

In conclusion, it is my impression that the presented data are not sufficient evidence for the alleged role of the mitral valve in the mechanism of production of the first heart sound.

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This letter was shown Drs Brooks, Leech, and Leatham who reply as follows:

Sir,

Dr Portaluppi questions the significance of the relation between the timing of mitral valve closure and the amplitude ratio $M1/A2$. Since the relation shown in Fig. 1 is obviously non-linear, it was not analysed as such, but in semilogarithmic form. The basic data of the two relations are the same: the significance of each, one curvilinear and one linear, is therefore identical. That the basic Q-MC-PR relation is not linear in no way detracts from our hypothesis; indeed it is tempting to point out that the curve it inscribes resembles a left ventricular pressure pulse which initially rises slowly and then increases in steepness up to the onset of ejection.

It is true that the correlation, though highly significant, is not very close. Considering the relatively crude method used to assess the loudness of the first sound this is not a source of great surprise. We discussed some of the more obvious shortcomings of our technique in the paper. Furthermore, we did not state, as Dr Portaluppi implies, that the timing of mitral closure is the sole ("primary") determinant of the loudness of the first sound, only that it is a cause of variations in loudness associated with different PR intervals.

Finally, Dr Portaluppi proposes that our explana-

tion is discredited by inability to show a correlation between first sound amplitude and closing velocity of the mitral valve; he suggests that "at least the mean closing velocity of the anterior leaflet is easily measurable". Closure of the mitral valve has two components, the result of two independent factors; an initial phase caused by atrial contraction and relaxation, and a terminal phase caused by the onset of ventricular contraction, so the "mean velocity" would be totally meaningless.

For these reasons, we feel no inclination to retract any of our conclusions.

The scepticism of Dr Luisada and his colleagues over the valvular origin of the first heart sound is well known, and their inability to produce a plausible explanation to account for the variations in loudness associated with different PR intervals has been a major weakness in their alternative theory.

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Notice

Conference on stroke

The Chest, Heart and Stroke Association Annual Conference on stroke will be held at The Bloomsbury Centre Hotel, London WC1, on Wednesday 24 June 1981.

Further information from Miss Hilda V Walsh, Assistant Director, Tavistock House North, Tavistock Square, London WC1H 9JE.