Regional Left Ventricle 
Performance

To the Editor:

In their article on regional dyskinesis in postinfarct patients (Circulation 48: 679, 1972) Feild et al. have convincingly demonstrated a relationship between disorders of wall motion and overall ventricular function. Our only regret is the use of their theoretic spherical model to predict the functional state of the nonakinetie myocardium.

The principal error lies in their assumption that a ventricle with an akinetic segment would have a normal ejection fraction (EF) provided the nonakinetie segment functions normally and serves as the total circumferential length. This is implied in the derivation of equation 6 in the appendix. Given these conditions, the following a priori circumstances could evolve. A spherical ventricle with 50% of its hemisphere rendered akinetic would have its nonakinetie circumferential length equal to 50% of total length. If there were no change in the contractility of the nonakinetie myocardium, the stroke volume would be reduced by half whereas the end-diastolic volume derived from half the original circumferential length (4/3π [0.5 La/2π]3) would be reduced by 87.5%. This would produce a ventricle ejecting almost three times its end-diastolic contents which is clearly absurd.

Furthermore, the authors go on to derive a formula which is supposed to predict the EF of the akinetic ventricle assuming the nonakinetie segment is normal.

This is equation 7 which is reproduced as follows:

\[
\frac{SV_A}{4/3\pi (La/\pi)^3} = 0.67 \left(1 - \frac{AS}{100}\right)^3
\]

where \(SV_A\) = stroke volume of the ventricle with akinesis, \(La\) = total circumferential length, and \(AS\) = akinetic segment expressed as a percent of total length.

Using our hypothetic ventricle with \(AS = 50\)% it is clear that the left-hand expression would be \(0.67 \times 0.5 = 0.34\) whereas the right-hand expression would be \(0.67 \times (0.5)^3 = 0.084\). The latter expression seriously underestimates the actual EF of the model and yet it was precisely this formula that was employed to predict the EF of the akinetic ventricles with presumably normal nonakinetie segments.

The authors then applied the above model to their data and found a curiously good fit (fig. 4). They mistakenly surmised that these patients had normal nonakinetie myocardial segments when in fact the predicted ejection fraction derived from the model was seriously underestimated. If their figures are correct, we would reinterpret the findings to indicate several possibilities: (1) the nonakinetie segments were depressed in function; (2) despite the use of biplane analysis, contiguous areas of akinesis in other planes were present but not observed; or (3) there was significant paradoxical motion of the akinetic segments accounting for an overall reduction in ejection fraction.

We submit, however, that unless a more appropriate geometric model is developed, it would be hazardous to use the data derived from quantitative regional akinesis to predict the functional state of the nonakinetie myocardium.

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The Authors reply:

To the Editor:

We appreciate the comments of Dr. Fallen and Mr. Herscovitch, who have however, incorrectly interpreted the derivation of our spherical model equation. They incorrectly state that we assumed, in equation 6 of our appendix, that “a ventricle with an akinetic segment would have a normal ejection fraction provided the nonakinetie segment functions normally and serves as the total circumferential length.”

The derivation can best be understood by first considering equation 2 of our appendix. This is a general equation which states that the total end-diastolic circumferential length of a ventricle with akinesis (La) can be divided into two components: the
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