creased. This would have been the case had this been upright exercise, although this may not have been the case in the usual posture of these subjects.

In relation to the foregoing, the protocol indicates a work load increase every 3 minutes with a mean duration of exercise of 8.9 minutes. Their figure 2 suggests that at the first 3-minute point the heart rate was 90 beats/min, the next 110 beats/min, jumping finally (third data point during exercise) to the 152 beats/min peak rate. Was this the actual sequence?

The third omission which may or may not be significant is that the protocol makes no mention of the position of the legs at the onset of exercise. Were the legs raised to the level of the pedals during the control period or only afterward? This factor can affect control stroke volume.

These considerations are not intended to detract from, but rather to clarify, a well-done and valuable report.

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References


The authors reply:

To the Editor:

We thank Dr. Spodick for his comments on our manuscript. We are pleased to address the issues raised in his letter.

With regard to ejection rate, we measured Vcf by the method of Hirshleifer et al. As implied by Dr. Spodick, this method involves measuring ejection time from the echocardiogram. While we agree that increased ejection rate is likely a manifestation of increased inotropy in the absence of the Frank-Starling effect, the rise in cardiac output early in exercise is unlikely to be attributable to increased ejection rate independent of increased heart rate, as stroke dimension remains constant during these early levels of exercise, not increasing significantly until peak exertion. Thus, heart rate alone appears to account for increased cardiac output during pre-peak levels. We are certainly in agreement, however, that the progressive increase in Vcf suggests a progressive increase in ejection rate.

As to the time course of change in stroke dimension, this index increased abruptly at peak exercise levels, remaining relatively constant before that time. As stated in our manuscript, work load was increased every 3 minutes. In figure 2, the heart rates of 90, 110, and 152 beats/min are displayed irrespective of the time in exercise at which they occurred. Thus, 90 beats/min did not necessarily occur at 3 minutes in exercise.

Finally, the position of the legs during the pre-exercise control period was identical to that during exercise. The legs were raised to the level of the pedals during the entire study.

We thank Dr. Spodick for his valuable comments, and hope we have clarified the issues raised.

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Detection of Ischemia by Exercise Echo

To the Editor:

In a recently published article Mason and co-workers evaluated the change of systolic wall thickness (SWT) in relation to severe stenosis of one main coronary artery. They stated that reduction of SWT during exercise might be useful as a reliable tool for demonstrating coronary artery disease (CAD). Though it is appreciated that remarkable reduction of coronary blood flow results in a diminished or absent SWT and an abnormal wall motion, their examples (figs. 1 and 2) do not support the usefulness of echocardiography in identifying patients with CAD. In figure 1 the right septal endocardium is recognized arbitrarily. The fine echo just posterior to the thick one (cords from the tricuspid-valve apparatus?) can be taken as endocardium as well. In this case SWT would be almost identical at rest and during exercise. Furthermore, the systolic septal motion has an absolutely normal pattern strongly arguing against a severe reduction in coronary blood supply.

In figure 2 the echocardiograms at rest and during exercise show two different areas of the septum. At rest septal motion reveals a pattern from the area just beyond the mitral leaflets. During exercise maximum septal SWT occurs after maximum posterior wall thickness, i.e., after the closure of the aortic valve. This pattern resembles the hinge zone between the anterior aortic root and the left ventricular cavity. Maximal "systolic" wall thickness, as it is measured by Mason, seems really to be a diastolic one.

Echocardiography is appreciated as a useful method in noninvasive cardiology. Despite the numerous trials in introducing echo as a new approach for detecting CAD, its usefulness has not been established. The patient’s history together with ECG tracings at rest and during exercise obviously provide a better tool in noninvasive cardiology for uncovering symptomatic CAD.

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References


The authors reply:

To the Editor:

We appreciate Dr. Menne’s critical reading of our paper and his thought-provoking comments. We agree that the interpretation of which lines on an echocardiogram represents the endocardium is occasionally difficult. Nevertheless, each of our subjects underwent a thorough echocardiographic examination and only the data from those subjects in whom both sides of the septum and posterior wall
Detection of ischemia by exercise echo.
H D Menne

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