LETTERS TO THE EDITOR
Letters to the Editor will be published, if suitable, and as space permits. They should not exceed 1,000 words (typed double spaced) in length, and may be subject to editing or abridgment.

Effect of Posture on Exercise Performance
To the Editor:
In the July issue of this journal Drs. Spodick and Quarry-Pigott studied the influence of postural attitude on exercise performance measured by systolic time intervals. Recently we performed identical tests on five male volunteers between 27 and 32 years of age. However, in our schedule, the individuals were subjected to maximal tolerance tests in steps of 25 Watts during 4 min periods. Simultaneous registration of the carotid pulse tracing, ECG, and phonocardiograms were made every 2 min during the test and every minute during the recovery period.

We found similar results (table 1) as Dr. Spodick; however, in our study there was a significant increase of LVETc and ETI in the supine position. Although this increase was not as important as the one which we obtained in the sitting position, the change in position accounts for about half of it. Since LVETc and ETI are merely influenced by changes of stroke volume,2 our results suggest that this volume does increase during supine exercise.

Using direct invasive methods, Bevegård and Shephard34 mentioned an increase of stroke volume during supine exercise of 10 to 20% vs 50% while sitting upright.

In the second phase of the tolerance test, we did not find any further change in the systolic time intervals either in sitting or in supine position.

We fully agree with Dr. Spodick's conclusion that this noninvasive technique is an appropriate means for determination of the cardiac response during exercise.

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References

The authors reply:
We are grateful to Dr. Guy De Backer for his informative study which amplifies the exercise findings utilizing noninvasive polycardiography. We would make two observations on some of their results: 1) Although we calculated ETc in our subjects, subsequent studies have made us uncertain of its theoretical as well as practical applicability. 2) It is difficult to understand some of the protocol of the study cited; the heart rate increments cited were indeed equivalent for both postures, but the endpoints could not have been maximal: even at Level II, a heart rate of 139 is close to 40 beats below maximal for 30 year old men. Apart from these minor observations, the results of this study are indeed of interest and value and we thank the author for citing them.

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Coronary Heart Disease Prevention
To the Editor:
Dr. Epstein's endorsement of coronary heart disease (CHD) prevention programs seems overly cautious . . .

Table 1
Systolic time intervals with supine and upright exercise

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
<th>Level I</th>
<th>Exercise</th>
<th>Level II</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mean ± SD</td>
<td>% change</td>
<td></td>
<td>mean ± SD</td>
<td>% change</td>
</tr>
<tr>
<td>HR (b/min)</td>
<td>S.</td>
<td>71 ± 8</td>
<td>111 ± 8</td>
<td>+ 56 *</td>
<td>133 ± 15</td>
</tr>
<tr>
<td></td>
<td>U.</td>
<td>77 ± 14</td>
<td>119 ± 9</td>
<td>+ 64 *</td>
<td>139 ± 17</td>
</tr>
</tbody>
</table>
| LVETc (msec) | S. | 291 ± 15 | 261 ± 12 | - 10 | 235 ± 20 | - 9 *
|         | U. | 244 ± 21 | 235 ± 14 | - 4.5 Ns | 215 ± 17 | - 8 Ns |
| ETI     | S. | 376 ± 13 | 393 ± 7 | + 5 * | 394 ± 7 | 0 Ns |
|         | U. | 357 ± 5 | 373 ± 7 | + 11 | 381 ± 8 | + 2 Ns |
| LVETe   | S. | 316 ± 16 | 352 ± 9 | + 12 | 347 ± 14 | - 2 Ns |
|         | U. | 273 ± 7 | 326 ± 10 | + 20 | 325 ± 11 | - 1 Ns |
| PEP     | S. | 99 ± 20 | 65 ± 17 | - 34 | 56 ± 15 | - 13 Ns |
|         | U. | 116 ± 10 | 66 ± 10 | - 43 | 62 ± 14 | - 6 Ns |

Abbreviations: HR = heart rate; LVET = left ventricular ejection time; ETI = ejection time index; LVETe = corrected ejection time; PEP = pre-ejection period; S = supine; U = upright; SD = standard deviation; *P < 0.05; Ns = not significant.
“an all-out campaign must await conclusive data from intervention studies” (Circulation 48: 185, 1973). One intervention study has been with us for half a century... the marathon run. This powerful tool for life-style change has made “acquired ectomorphs” of all middle-aged men who trained for the 42 kilometer race. Morris found that vigorous exercise of a moderate degree gave approximately 66% protection against CHD (Lancet 1: 333, 1973). When the level of exercise is raised to that of a marathon runner the protection appears to be absolute. The American Medical Joggers Association (AMJA) has been unable to document a fatal myocardial infarct among marathon runners of any age. (Lancet 2: 711, 1972). Rehabilitation centers have begun using distance running for patients who have recovered from one or more infarcts. Seven such patients finished the Boston Marathon this year. They ran with their cardiologist, Terrence Kavanagh, from the Toronto Rehabilitation Centre. (JAMA 224: 1580, 1973) If marathoning continues to protect cardiac patients from myocardial infarction, there is no excuse for the rest of us to “await conclusive data.” With half of all physicians eventually dying from CHD (JAMA 223: 1391, 1973) the members of the AMJA have adopted the hobby of marathoning in an effort to join the “protected” group. Thus far, there have been no repeat MI’s among Kavanagh’s marathoning heart patients or the marathoning physicians. As long as this protection holds we have a very simple tool for preventive cardiology... one that requires no great investment of time or money; just an average of an hour a day of social running.

And most of the AMJA physicians actually seem to enjoy their new hobby!

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Dr. Epstein replies:

Dr. Bassler may well be right that endurance sports like marathon running provide significant protection against coronary heart disease. However, acceptable evidence in support of his claim is lacking. Moreover, it is open to question whether marathon running, even under careful medical supervision, is protective. The reference regarding such patients which Dr. Bassler quotes (JAMA 224: 1580, 1973) is merely a news item referring to only seven men observed for only five years; no information whatever on their history or course in terms of personal, clinical, or laboratory findings is given.

The marathon run does not constitute, as Dr. Bassler states, an “intervention study” unless one uses the term to include undocumented statements referring to highly self-selected individuals, using for a control group simply the assertion that “half of all physicians eventually die from coronary heart disease,” quoted from a short letter by Dr. Bassler to the Editor of the JAMA (223: 1391, 1973). This letter and another by himself in the Lancet (2: 711, 1972) provide no information other than the one given in the present communica-

Echocardiography of Mitral Valve Prolapse

To the Editor:

The echocardiogram of a patient with posterior mitral leaflet chordal rupture in a report in Circulation (46: 550-586, 1972), is represented as being specific for mitral insufficiency due to chordal rupture, as opposed to mitral insufficiency of other etiologies, and to indicate a flail posterior mitral leaflet as shown in the accompanying line diagrams. The retention of characteristic movements of anterior and posterior mitral valve leaflets in diastole, even though the initial diastolic movement of the posterior leaflet is anterior, is not typical of flail posterior mitral valve leaflets. There are also hazards, as noted by the author, in interpreting echoes anterior to the posterior left atrial wall. The surgical and pathological findings are not discussed.

We recently observed a 63-year-old man with acute mitral insufficiency and congestive failure resistant to medical therapy. Echocardiography of the mitral valve was similar to that in the above article. Angiography revealed ballooning of the posterior mitral leaflet into the left atrium with retention of chordae support at the free leaflet margin. At surgery, he was found to have rupture of one chordae within two millimeters.