LETTERS TO THE EDITOR

"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 "wait conclusive data." With half of all physicians eventually dying from CHD (JAMA 223: 1391, 1971) 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 MIs 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-

tion. Thus, there is no factual support for his thesis. Comparing recurrence rates in a small group of marathon runners (JAMA 224: 1580, 1973) with the "usual" recurrence rates for myocardial infarction is likewise meaningless unless the numbers and the clinical characteristics of the survivors are known.

Data in favor of a protective effect of exercise are not easy to interpret because people who exercise habitually are probably not similar in most other pertinent respects to their more sedentary counterparts so that the observed lower disease frequency is not necessarily ascribable to greater physical activity. A randomized preventive trial, intervening on physical activity only, would get around this problem but is not likely to be done in the foreseeable future. The very high rate of coronary heart disease among heavy laborers in rural Finland is the best single piece of evidence that more exercise will not by itself reduce the toll from coronary heart disease in the population. This does not mean that for some individuals a program such as marathon running might not make the difference between life and death. By presenting as fact something which, at best, is based on conjecture, Dr. Bassler does a disservice to his own cause and the many others who believe that physical activity is, indeed, protective and struggle to collect better supportive evidence. His own enthusiasm and efforts are admirable but does he really think that I have been "overly cautious" in not having advocated an immediate, all-out national campaign to make marathon runners of us all and now . . . ?

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Echocardiography of Mitral Valve Prolapse

To the Editor:

The echocardiogram of a patient with posterior mitral leaflet chordae rupture in a report in Circulation (46: 580-586, 1972), is represented as being specific for mitral insufficiency due to chordae 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.
of the leaflet connection to each of the mitral leaflets. However, as observed at the operating table, there was prolapse of both leaflets rather than flail leaflets. We suggest that, despite chordae rupture, the echocardiographic picture is more compatible with mitral valve prolapse rather than with a flail posterior mitral valve leaflet.

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1. FEIGENBAUM H: Echocardiography. Philadelphia, Lee
and Febiger, 1972, p 62, figs 4-21, 4-22
2. FEIGENBAUM H: Echocardiography. Philadelphia, Lee
and Febiger, 1972, p 153, figs 9-11

The authors reply:

As suggested by Drs. Sasse and Lightfoot, it may be difficult to distinguish prolapsing mitral valve leaflets due to ruptured chordae tendineae from prolapse due merely to long, redundant chordae and/or ballooning mitral valves. In two recent papers on the subject,
2 we have described those findings which most commonly distinguish these two types of prolapse.

In all the cases of ruptured chordae tendineae to the posterior leaflet confirmed at surgery, we have seen a paradoxical anterior movement of this posterior leaflet in early diastole. This abnormality is often associated with a hammock-like posterior bulging of the posterior leaflet during systole.
2 When this posterior leaflet is recorded throughout diastole, it remains primarily in an anterior position and moves posteriorly only in late diastole when it does so in a sharp manner. Probably because of increased flow across the mitral valve, the anterior leaflet tends to have a slightly increased magnitude and velocity of opening. As reported,
2 these echo characteristics were present in eight surgically confirmed cases of ruptured chordae tendineae to the posterior mitral leaflet and subsequent to that report in seven other cases operated at our institution.

We also agree with Drs. Sasse and Lightfoot that there may be hazards in interpreting echoes immediately anterior to the posterior left atrial wall. Currently, we identify posterior mitral leaflet as seen within the left atrium only if clearly defined valvular motion is recorded. More recently, we have found distinct valvular structures can occasionally be recorded during systole in the anterior portion of the left atrium near the posterior wall of the aorta. Upon changing the transducer position, this echo is found to be continuous with the paradoxically moving posterior mitral leaflet in early diastole.

Prolapsing posterior leaflets without ruptured chordae tendineae may also show a posterior sagging of the CD segment during systole and wide separation from the anterior leaflet echo. Recognition of this sagging in multiple echocardiographic views is considered mandatory in making this diagnosis in order to eliminate artificial "false-positive" diagnoses. Also of note is that the echoes from the anterior mitral leaflet oftentimes show a rounded E point, forming a "roller coaster" appearance. In the absence of ruptured chordae, we have never seen either paradoxical anterior movement of the posterior leaflet echo at the onset of diastole or the leaflet within the left atrium. It should be recognized, however, that in some cases in which there is clear echo evidence of systolic prolapse of the mitral valve, it may be difficult to determine if there is a accompanying rupture of mitral chordae tendineae.

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References

Wenckebach Periods and His-Purkinje Conduction

To the Editor:

Halpern and co-workers' demonstration of Wenckebach periods of alternate beats was thought provoking.
1 We accept the authors observations and interpretations in individual cases. We would like to question their over-all conclusion that AW are primarily a manifestation of depressed conduction in the His-Purkinje system (HPS). Their conclusion was based on 1) The occurrence of intraventricular blocks in all five cases with AW, 2) Pathological observations in one of their cases, with demonstration of lesions in the His-Purkinje system. 3) His bundle recording in one of their cases, with demonstration of AW distal to the His bundle. 4) Experimental production of AW with surgical trauma to the HPS.

Our thoughts regarding this evidence are as follows:
1) The presence of intraventricular conduction defects also implies a significant incidence of associated A-V nodal dysfunction.
2, 3 In our experience, progression of A-V nodal disease in patients with bifascicular block is not uncommon. Thus, the occurrence of AW in a patient with intraventricular block is consistent with AW in either the A-V node, HPS, or both. 2) The demonstration of pathological lesions in the HPS does not exclude the occurrence of major conduction block at the A-V node.
3 The latter may be functional, without an apparent anatomic abnormality.
3) In regard to the one case with His bundle recordings, we have recently seen a patient with somewhat different findings. Our patient had chronic bifascicular block and digitalis intoxication. The patient had

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