The authors reply:

To the Editor:

We very much appreciate Dr. Chatterjee’s response to our article and confess that we overlooked his previously published data when reviewing the medical literature for our own manuscript. The average values for the mean normalized systolic ejection rate in his normal and cardiomyopathy groups are remarkably similar to the values in our patients with normal and depressed ventricular function, respectively. It is of interest that the values he derives from single plane cineangiograms are in close accord with those which we calculated from biplane studies. It should be noted, however, that our methodology is somewhat different; we estimate the ejection time from the cine framing rate assuming an arbitrary 50 millisecond duration for the prejection period, whereas Chatterjee derived the ejection rate by “drawing a tangent on the ejection portion of the volume curve.” Dr. Chatterjee’s data, we believe, further validate the use of ejection phase indices, and in particular the mean normalized systolic ejection rate, to assess myocardial performance in man.

Kirk L. Peterson, M.D.
David Skloven, M.D.
Philip Ludbrook, M.D.
John Uther, M.D.
John Ross, Jr., M.D.
University of California
San Diego, California

Jogging and Coronary Heart Disease

To the Editor:

Dr. Epstein, in responding to Bassler’s enthusiasm for jogging, has misquoted the evidence bearing on occupational exercise and coronary heart disease as revealed by studies of the Finnish lumberjacks. The study of Karvonen et al. (Table 1) clearly shows that the physically active lumberjacks have a lesser prevalence of ECG evidence of coronary disease than do other Finns despite similar unfavorable risk factors in those men. This evidence has been so often misused by the dietary faddists that those who do not look at the original data are misled. While there may be other factors than fitness protecting the lumberjacks they were surely experiencing less CHD than Finns in other occupations. A skeptic might not wish to accept this as evidence for exercise as a preventive for CHD but he surely cannot use it as evidence to discount exercise, as Epstein has done. While I agree with Epstein that “an all-out campaign (on CHD) must await conclusive data from intervention studies,” I also think the same requirement should be applied to the zealots who promote dietary preventive measures which have not been substantiated by any of the extensive clinical trials.

George V. Mann, M.D.
Vanderbilt University
Nashville, Tennessee

Mitral Valve Reopening

To the Editor:

In their study relating mitral valve motion to first sound intensity in patients with complete heart block, Burrgraf and Craige 1 note a discrepancy between their work and that of Shah et al. 2 concerning the subject of mitral valve reopening following atrial systole. The authors also note some variability within their own patients as to the time of mitral valve reopening. Several authors have shown conclusively that atrial systole, regardless of its timing in ventricular diastole, usually results in mitral valve opening followed by mitral valve closure. 3, 4

On the basis of our own investigation of mitral valve motion with various arrhythmias, we would make the following suggestion regarding mitral valve reopening following atrial systole. The timing of valve reopening and perhaps the velocity of reopening is related to the extent of ventricular filling which has occurred at the time of atrioventricular closure. If the atrial contraction in question follows completion of the rapid filling period, mitral valve reopening tends to be delayed, although this is not invariable. When atrial contraction occurs very early in diastole, it in effect interrupts and abbreviates the rapid filling phase. In this instance the valve promptly reopens following atrial contraction and ventricular filling presumably resumes. 5

Thus, without a change in P-R interval small changes in the S-T-P interval secondary to minor alterations in heart rate can determine whether reopening occurs following atrioventricular closure. We have observed this phenomenon in a patient with marked first degree A-V block and sinus arrhythmia. Similarly, when two atrial contractions occur in the same diastole during third degree A-V block, the second will be followed by a prolonged period of mitral valve closure, presumably because ventricular filling is complete.

We would further suggest that variations between patients in the timing of mitral valve reopening following atrial contraction, given similar timing of the P wave in diastole, may relate to different patterns of ventricular filling secondary to a variety of hemodynamic factors probably including ventricular compliance. Such an hypothesis would explain the finding of Shearn et al. 6 that mitral valve reopening is more common in younger subjects.

William D. Towne, M.D.
Ramesh Patel, M.D.
Kamal K. Chawla, M.D.
Cook County Hospital,
Chicago, Illinois 60612

Table 1


<table>
<thead>
<tr>
<th></th>
<th>Lumberjacks</th>
<th>Other occupations</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG abnormal (rate/1000)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Q and QRS</td>
<td>8*</td>
<td>39</td>
</tr>
<tr>
<td>Axis deviation</td>
<td>13*</td>
<td>41</td>
</tr>
<tr>
<td>ST-segment abnormality</td>
<td>16*</td>
<td>62</td>
</tr>
<tr>
<td>T wave abnormality</td>
<td>84*</td>
<td>131</td>
</tr>
<tr>
<td>Cholesterol (mgm %)</td>
<td>207</td>
<td>266</td>
</tr>
<tr>
<td>Blood pressure (mm Hg)</td>
<td>147/90</td>
<td>149/91</td>
</tr>
<tr>
<td>Cigarette use (%)</td>
<td>never</td>
<td></td>
</tr>
<tr>
<td></td>
<td>8.4*</td>
<td>17.3</td>
</tr>
<tr>
<td></td>
<td>&gt;20/d</td>
<td>35</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td></td>
</tr>
</tbody>
</table>

*P < 0.05.
The authors reply:

To the Editor:

We wish to thank Dr. William D. Towne and his associates for their comments concerning our paper. We would agree that the effect of atrial systole on mitral valve motion is dependent upon the phase of the cardiac cycle during which it occurs. When atrial systole occurs during the very early rapid filling period there is no effect on mitral valve motion. As we noted in our results, P-R intervals from 0.2 to 0.5 seconds were associated with closure of the valve without reopening in most subjects. With P-R intervals in this range atrial systole would have occurred at a time when most of diastolic filling was completed. The one exception was an eight-year-old boy with congenital heart block in whom reopening occurred with this P-R interval range. We would agree with their suggestion that this variation is related to different patterns of ventricular filling and that this is more common in younger subjects.

We would agree that any maneuver that changes the temporal relationship between atrial systole and the phases of filling of the left ventricle, be it complete atrioventricular dissociation or simply variations in heart rate with respiration, will alter the patterns of mitral valve motion.

G. W. Burggraf, M.D.
Queen’s University
Kingston, Ontario

Ernest Craigie, M.D.
North Carolina Memorial Hospital
Chapel Hill, North Carolina

Serum Levels in Evaluating Antidysrhythmic Therapy

To the Editor:

In the recent paper by Jelinek et al. (Circulation 49: 659, 1974) on the effectiveness of procainamide and quinidine in sporadic ventricular arrhythmias, serum levels were given but only scarcely discussed by the authors. However, table 5 (p 664) of their paper illustrates an important point about the significance of serum levels for the evaluation of antidysrhythmic therapy.

If one tries to correlate the serum levels with the observed therapeutic results, the mean levels (and ranges), expressed in mcg/ml, read as follows:

<table>
<thead>
<tr>
<th></th>
<th>Effective</th>
<th>Not effective</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procainamide</td>
<td>6.55 (2.9–11.5)</td>
<td>4.90 (2.2–8.5)</td>
</tr>
<tr>
<td>3.0 g/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6.0 g/day</td>
<td>8.97 (7.0–10.9)</td>
<td>9.96 (4.4–16.5)</td>
</tr>
<tr>
<td>Quinidine</td>
<td>3.13 (2.7–3.4)</td>
<td>2.77 (0.2–5.6)</td>
</tr>
<tr>
<td>1.2 g/day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.8 g/day</td>
<td>5.25 (3.2–8.6)</td>
<td>4.58 (2.7–9.7)</td>
</tr>
</tbody>
</table>

It is obvious that neither for procainamide nor for quinidine do the levels show a significant difference whether or not the dysrhythmia is controlled (Wilcoxon test, P > 0.05). Likewise no correlation can be found between the serum levels and the presence or absence of toxic effects.

These data confirm our own findings with several antidysrhythmic drugs in patients with chronic ventricular dysrhythmias.1,2 In these studies we have shown that the concept of therapeutic and toxic serum or plasma levels6 has to be interpreted cautiously. Indeed, our results indicate that for a group of patients, the plasma level values show a poor correlation with the observed effect.

The plasma level of a drug is obviously not the only determining factor for effectiveness or toxicity. Our results and those of Jelinek et al. suggest that for antidysrhythmic drugs the sensitivity of the individual patient towards each drug is perhaps the most important factor.

Jean-Pierre Van Durme, M.D.
Marc G. Bogaert, M.D.
University of Gent
9000 Gent, Belgium

References


“Silent” Mitral Prolapse

To the Editor:

The excellent article by DeMaria et al. concerning the echocardiographic manifestations of the prolapsed mitral valve (Circulation 50: 33, 1974) stimulated us to review our own records. Before reading the article, we had already diagnosed 75 cases out of a total of approximately 600 patients echocardiographed. Afterwards, we reviewed the records of other patients who had been referred because of clinical suspicion of prolapse, and in whom we had initially rendered a “normal” interpretation to the echocardiogram. In every instance evidence of prolapse was found, and we so advised the referring physician of our revised interpretation.

Which brings me to the point: What kind of a “molehill” has echocardiography uncovered, and are we making a “mountain” out of it? Granted that somewhere in this spectrum the potential for sudden death (and endocarditis) exists. Nevertheless when over 12% of patients referred to a community hospital’s echocardiographic laboratory supposedly have one entity, has that entity indeed been properly defined? Moreover, are we as physicians justified in

Circulation, Volume 50, December 1974
Mitral Valve Reopening
WILLIAM D. TOWNE, RAMESH PATEL and KAMAL K. CHAWLA

Circulation. 1974;50:1283-1284
doi: 10.1161/01.CIR.50.6.1283-b

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/50/6/1283.3.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not
the Editorial Office. Once the online version of the published article for which permission is being
requested is located, click Request Permissions in the middle column of the Web page under Services.
Further information about this process is available in the Permissions and Rights Question and Answer
document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/