over 1 h, using a beat-to-beat QT and RR interval measurement, may not apply to the next hour in the same patient. This may occur because RR-QT data collected during the course of the day tend to show considerable scatter because of the normal fluctuations in autonomic tone. With this in mind, one really wonders whether correcting the QT interval makes any sense at all?

JAGMEET SINGH, MD, DM
*Department of Cardiovascular Medicine*
*John Radcliffe Hospital*
*Oxford OX3 9DU, England, United Kingdom*

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***Reply***

We thank Sarma et al. and J. Singh for their comments on our recent publication in the Journal (1) The inaccuracy of Bazett's method for adjusting QT intervals has caused bias in research and confusion in clinical practice. The popularity of the Bazett equation is based on its simplicity, not on its fit. The newer improved but complex methods have largely been ignored. Our goal was to create a simple and accurate method to compare QT intervals of *rest electrocardiograms* (ECGs) without violating the electrophysiologic principles derived from action potential studies (2).

Sarma et al. derived an exponential equation using our data. Their equation produces an average mean-squared residual value of 304, whereas our nomogram method gives the value of 291 in young men (Table 3 [1]). Notwithstanding the possible advantages at very low heart rates, the use of an exponential equation is problematic in clinical practice, which is why we avoided presenting one. The major advantage of the nomogram method is that it is empiric and has no limiting preassumptions. The disadvantage of all regression equations is that the same rule is applied over the entire range of heart rates.

As pointed out by J. Singh, the nomogram works at steady state only. This was ensured in our study by recording ECGs at rest, as stated in the report's title. Estimating the patient's QT interval at a different heart rate, a question raised by Sarma et al., is really another matter, as shown in Figure 7 in our study (1).

Sarma et al. write that there is an insurmountable limitation to our method for estimating the QT interval in patients using class III antiarrhythmic drugs, whereas J. Singh reminds us that the nomogram presented may be inapplicable in disease states. These are important issues, similarly applicable to all equations for adjusting QT intervals. If the goal is to evaluate the effect of antiarrhythmic drugs or a disease process on the QT interval, then the Holter method should be chosen (3,4). In the Holter method the need for heart rate adjustment is avoided by measuring the QT intervals at the same spontaneous heart rates before and after intervention.

J. Singh asks whether there is any sense at all in correcting the QT interval. Because there is no such thing as a single correct QT interval, we preferred the term *adjusted QT interval*. Although the dynamic changes in the QT interval and the dispersion of repolarization may be of greater importance in arrhythmogenesis, in clinical practice one should be able to evaluate the QT interval in the rest ECG reliably. Moreover, the QT interval in the rest ECG is the key to understanding QT dynamics. When the most reliable QT interval-adjusting tools for rest ECGs for different populations of patients under medication are needed, they should be constructed for the particular population using the principle presented, for example. When the effects of medication or the disease process on the QT interval in an individual patient have to be evaluated, we recommend the Holter method (3).

MATTI VIITASALO, MD
MATTI MÄNTTÄRI, MD
VESA MANNINEN, MD
*First Department of Medicine*
*Helsinki University Hospital*
*00290 Helsinki, Finland*

JOUKO KARJALAINEN, MD
*Central Military Hospital*
*PL 50, 00301 Helsinki, Finland*

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## Atrial Function Can Only Be Assessed by Combined Use of Volume- and Pressure-Assessing Noninvasive Methods

In the recent study of Manning et al. (1) a new index for assessing the atrial contribution to diastolic performance was introduced. Using Newton's second law of motion and variables derived from two-dimensional imaging and Doppler echocardiography, the authors attempted to introduce a more accurate mode of assessing atrial function in 29 patients after elective cardioversion for atrial fibrillation. When Newton's law is replaced by echocardiographic variables, the "atrial ejection force" is proportional to peak A velocity squared and varies directly with mitral orifice area. Although the atrial ejection force was found to be significantly reduced in the patients compared with a small group of normal control subjects, this new index does not represent an assessment of "atrial function" or "atrial contribution" in these patients with coronary and hypertensive heart disease because there are several problems with the interpretation of this major finding by the authors.

Manning et al. mention that, according to data of Choong et al. (2), "peak A wave velocity is over a physiologic range relatively independent of ventricular preload." However, they do not emphasize another important finding, namely that in patients with clearly abnormal diastolic function and elevated ventricular filling pressures, peak A velocity is predominantly, if not exclusively, dependent on the degree of elevation of end-diastolic pressure. Obviously, the patients in the Manning et al. study could be expected to have at least a moderate

increase in end-diastolic pressure. Using simultaneous Doppler echocardiographic and apexcardiographic recordings, we have previously reported (3,4) that in patients with coronary and hypertensive heart disease, a decreased peak A flow velocity is most frequently associated with a significantly elevated apexcardiographic A wave height. According to our data, atrial systolic function, which is given by the "generation of pressure" and assessed by the relative height of the apexcardiographic A wave, is not decreased but significantly increased in such patients. Consequently, the decrease in flow through the mitral valve during atrial contraction in this clinical setting does not reflect a diminished power of atrial contraction; rather, it is the result of elevated ventricular filling pressures only. Thus, "true atrial function" can be evaluated only by an additional assessment of the power of "pressure generation" and the resulting "atrial kick" by the use of apexcardiographic recordings. These alternative fundamental pathophysiologic aspects, which have been extensively analyzed by many investigators using various techniques, were entirely neglected by Manning et al. By excluding these important data from the interpretation and discussion of their findings, the authors arrived at false conclusions about the nature and definition of atrial function itself.

A combined Doppler echocardiographic and apexcardiographic A wave index would probably help greatly in providing a clinically useful evaluation of "true atrial function." Such an index could be, for example, the ratio of the relative A wave to total height of apexcardiogram and the peak A flow velocity; the former provides information about the force of "pressure generation" and the latter about the "change in flow" during atrial contraction.

We hope that our previous work using both Doppler (flow) and apexcardiographic (pressure) A waves will stimulate the development of such combined indexes for accurately evaluating the "true atrial function and performance," which can only be assessed when both parts of the equation are given.

JAN MANOLAS, MD
*Diagnostic and Therapeutic Center of Athens "HYGEIA"*
*Kifisias and 4 Erythrou Stavrou Street*
*151 23 Marousi, Athens, Greece*

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### *Reply*

In our report (1) we define atrial ejection force as "that force exerted by the left atrium in propelling blood into the left ventricle during atrial systole . . . and should not be misinterpreted as an assessment of 'total' atrial force." Total atrial force would be the vector sum of all forces acting within the atrium. Utilizing echocardiographic variables, atrial ejection force is proportional to peak A velocity squared.

We agree that peak A velocity is frequently elevated among patients with heart disease. Because peak A velocity may be increased among patients with coronary and hypertensive heart disease, one would then have expected atrial ejection force to be increased in the study group (compared with control subjects), yet it was significantly depressed after cardioversion and continued for at least 1 week after cardioversion. With each patient used as their own control, atrial ejection force significantly increased during the succeeding period of observation. To explain our findings on the basis of changes in filling pressure alone, one would have to hypothesize that left atrial filling pressure increased during the month after cardioversion. It is more likely that filling pressures declined (2). Thus, through the use of longitudinal data, we are comfortable in affirming the validity of atrial ejection force as an index of atrial systolic function. We are unaware of serial apexcardiographic data among patients undergoing cardioversion and cannot be certain how this variable would change. Because the height of the apexcardiographic A wave is more closely related to ventricular stiffness, end-diastolic pressure and the volume of atrial systolic flow, concordance between it and atrial ejection force may be limited.

We fully appreciate that "transmitral Doppler data alone do not fully reflect changes in ventricular compliance and . . . a less compliant ventricle might present greater resistance to transmitral inflow and result in a depressed peak A wave velocity" (1). Better models are indeed needed, but because of the complexity of left ventricular diastolic and left atrial systolic function, one must carefully identify which components of cardiac performance are being assessed.

WARREN J. MANNING, MD, FACC
PAMELA S. DOUGLAS, MD, FACC
*Cardiovascular Division*
*Beth Israel Hospital*
*330 Brookline Avenue*
*Boston, Massachusetts 02215*

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## Heparin and Aspirin in Unstable Angina: Insufficient Sample Size May Lead to Erroneous Conclusions

In their article, Holdright et al. (1) address the interesting question whether, in patients with unstable angina, heparin combined with aspirin is more effective in preventing transient myocardial ischemia than aspirin alone. The authors attack the current standard of practice in the United States, which is to use both aspirin and heparin (2). The authors, therefore, have the burden of proof.

Holdright et al. conclude that "combined therapy with heparin and aspirin compared with aspirin alone makes no difference in the development of [transient myocardial ischemia]." Strikingly, their data shown in Table 2 (1) suggest just the opposite. The number of patients with at least one episode of transient myocardial ischemia was 25% less in the heparin plus aspirin group than in the aspirin alone group, 18% vs. 24% of patients, respectively. Even more strikingly, this pattern was consistent in every single variable presented by the authors. The total number of episodes in the heparin plus aspirin group was less by 35%, the median duration of episodes shorter by 16%, the total duration of