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?

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### References

1. Karjalainen J, Viitasalo M, Mäntäri M, Manninen V. Relation between QT intervals and heart rates from 40 to 120 beats/min in rest electrocardiograms of men and a simple method to adjust QT interval values. J Am Coll Cardiol 1994;23:1547–53.
2. Bexton RS, Vallin HO, Camm AJ. Diurnal variation of the QT interval: influence of the autonomic nervous system. Br Heart J 1986;55:253–8.
3. Franz MR, Swerdlow CD, Leim BL, Schaefer J. Cycle length dependence of human action potential duration in vivo: effects of single extrastimuli, sudden sustained rate acceleration and deceleration, and different steady state frequencies. J Clin Invest 1988;82:972–9.

***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).

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### References

1. Karjalainen J, Viitasalo M, Mänttäri M, Manninen V. Relation between QT intervals and heart rates from 40 to 120 beats/min in rest electrocardiograms of men and a simple method to adjust QT interval values. J Am Coll Cardiol 1994;23:1547–53.
2. Franz MR. Time for yet another QT correction algorithm? Bazett and beyond [editorial comment]. J Am Coll Cardiol 1994;23:1554–6.
3. Viitasalo M, Karjalainen J. QT intervals at heart rates from 50 to 120 beats per minute during 24-hour electrocardiographic recordings in 100 healthy men: effects of atenolol. Circulation 1992;86:1439–42.
4. Karjalainen J, Viitasalo M. Fever and cardiac rhythm. Arch Intern Med 1986;146:1169–71.

## 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