with this point, the primary focus of our report was the incremental benefit of TEE in the evaluation of patients with *S. aureus* bacteremia rather than the management of patients with endocarditis.

In conclusion, we respect the points raised by Guzzo and Simpson; however, we feel that the use of a validated diagnostic reference standard, the probable underestimation of endocarditis among excluded patients and the clinically logical conclusions in our study emphasize the need to consider TEE early in the evaluation of patients with *S. aureus* bacteremia.

VANCE G. FOWLER, JR., MD
Duke University Medical Center
P.O. Box 3824
Durham, North Carolina 27710
E-mail: fowle007@mc.duke.edu

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Propagation Velocity of Left Ventricular Filling Flow Measured by Color M-Mode Doppler Echocardiography

I read with great interest the report by Duval-Moulin et al. (1) regarding to the application of color M-mode Doppler to assessing left ventricular diastolic function. The report indicated that left ventricular diastolic dysfunction during coronary angioplasty could be assessed by the propagation velocity of left ventricular early filling flow using color M-mode Doppler technique and that this index showed good correlation with an invasive variable, tau. Their method was very similar to, but not the same as, our method, which was reported in the Journal in 1996 (2). In their discussion, they introduced our method as follows: "Takatsuji et al. used a derived technique by measuring the interval between the maximal rate of increase of action potential upstroke (Vmax) at the mitral level and 70% Vmax in the apical region." There are several inappropriate notations in this sentence. In our report, the propagation velocity was defined as follows:

> By changing the first aliasing limit sequentially at intervals of 2 cm/s with the use of the baseline shift, a flow velocity higher than the aliasing velocity could be displayed in blue within red filling flow signals. First, we located the point of maximal velocity around the mitral orifice in early diastole, which was obtained at the center of the minimized aliasing area. Next, we changed the first aliasing limit to 70% of the maximal velocity and located the point nearest to the apex on the aliasing boundary (which is usually obtained in the mid-left ventricle). The distance/time ratio, that is, the upward slope of the line connecting these two points, was measured and defined as the rate of propagation of peak early filling flow velocity.

First, the authors used the terms "action potential" and "Vmax," which were not used in our report, and this terminology might mislead the readers and prevent appropriate understanding of our method. Second, we did not measure the "interval" but the "distance/time ratio"; and third, the second measurement point is not "in the apical region" but "in the mid-left ventricle." We have some evidence that the propagation velocity measured by our method is more accurate than that measured at the wavefront of filling flow, which was used in their report. Therefore, if they carefully traced our protocol, I believe that they would obtain better correlation between propagation velocity and tau. Nevertheless, we appreciate the authors' results because their study enhances the usefulness of color M-mode Doppler for evaluating diastolic function in many clinical settings.

AKIRA KITABATAKE, MD, FACC
Department of Cardiovascular Medicine
Hokkaido University School of Medicine
Kita-15, Nishi-7, Kita-ku
Sapporo 060, Japan

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Reply

We have taken into account the different points emphasized by Kitabatake about the report by Takatsuji et al. (1). As Kitabatake described, color M-mode Doppler allows remarkably effective observation of diastolic flow as a function of time and of space.

The color M-mode Doppler method has been developed by several groups, including Takatsuji et al. (1), Stugaard et al. (2) and ourselves (3,4). The technique shows correlation of the propagation of early diastolic flows in the left ventricle with hemodynamic data of relaxation, especially the index tau.

With regard to the report of Takatsuji et al. (1) we were naturally interested in their methods and results, which analyze the rate of propagation of peak early filling flow. However, their study was published in the Journal during the review process of our study. Consequently, we added this new information in the revised version of our manuscript, which may explain why we did not discuss all the aspects of the interesting report of Takatsuji et al.

Nevertheless, we would like to point out that in contrast to Takatsuji et al. (1) and Stugaard et al. (2), we preferred to analyze the flow front wave at the beginning of filling rather than the later events of the propagation of peak early filling flow to better evaluate the relaxation process.

Finally, it is obvious that together we agree that color M-mode Doppler is a new and interesting noninvasive method for the evaluation of diastolic function.

ANNE MARIE DUVAL-MOULIN, MD
Service de Cardiologie
Hopital Henri Mondor
51 Avenue du Marechal De Lattre de Tassigny
94010 Creteil, France

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Is Pharmacologic Cardioversion of Atrial Fibrillation Really Preferable to Electrical Cardioversion?

Harjai et al. (1), in a nonrandomized study, conclude that patients undergoing electrical cardioversion of atrial fibrillation (AF) display a "greater degree and longer duration" of mechanical atrial dysfunction than those who convert pharmacologically or spontaneously. The authors strongly imply that this finding is a result of the mode of cardioversion. However, examination of their data reveals that this conclusion is unsupported and, indeed, that an absence of effect is more likely.

There are several flaws in the authors' analyses and logic. We would first question whether it is valid, in such a small study of cardioversion of atrial fibrillation, to include patients who spontaneously revert to sinus rhythm. Spontaneous conversions often occur in the first week of the arrhythmia (2). In a group with a median duration in excess of 1 month, inclusion of spontaneous convertors with pharmacologic converters may bias the data in favor of the latter because, as the authors point out, a duration of AF <7 to 14 days has been previously shown to be associated with better postconversion atrial recovery. Indeed, reference to Table 1 indicates that almost twice as many patients in the nonelectrical converter group had an arrhythmia duration <28 days—a difference that fails to reach statistical significance only on the basis of the very small numbers in the former group.

The arbitrary division of AF duration into those with a duration <28 or >28 days is also problematic. Because previous studies have demonstrated that an AF duration <14 days is associated with less depression of postreversion A wave height (3), the authors should have analyzed the effects of a shorter arrhythmia duration on A wave recovery. Indeed, the use of AF duration as a continuous, rather than a dichotomous, variable would have better elucidated the role of duration of AF on atrial function.

Multivariate analysis is generally accepted as the "gold" standard in determining whether a variable is truly associated with an outcome. When adjusted for several other clinical variables (AF duration, left atrial size and ejection fraction), the authors state that the mode of cardioversion was not associated with recovery of atrial electromechanical function, yet they seem to ignore this finding and conclude from "bivariate analysis" that "only the mode of cardioversion was seen to have any impact on the recovery of atrial function."

Finally, it is in our opinion, inaccurate to categorize this study as a comparison between patients undergoing either pharmacologic or electrical cardioversion. Presumably, many of the patients who subsequently underwent electrical cardioversion had been prescribed an antiarrhythmic agent either in an attempt to convert the arrhythmia or to maintain sinus rhythm after cardioversion. If this is so, then the study is predominantly a comparison of patients who responded to pharmacologic agents with those in whom pharmacologic conversion failed. Seen in this light, attributing postreversion atrial stunning to the mode of reversion is inaccurate.

Whether pharmacologic cardioversion of AF produces less atrial mechanical dysfunction than electrical cardioversion is an interesting question that may have some bearing on postconversion risk of thrombus formation. However, the answer to this question will require a randomized trial of immediate, drug-free electrical conversion compared with pharmacologic conversion. Failure to convert, either electrically or pharmacologically, will have to be treated as failure to recover mechanical atrial function. To retain a high likelihood of pharmacologic conversion, entry should probably be limited to those patients with a short (<7 or <14 days) arrhythmia duration. Only the results of such a study can give meaningful answers to the question of whether the atrium really cares how sinus rhythm is restored (4).

RODNEY H. FALK, MD, FACC
JEANNE DECARA, MD
VIVIAN ABASCAL, MD
Division of Cardiology
Boston University Medical Center
D8 East Newton Campus
1 Boston Medical Center Place
Boston, Massachusetts 02118

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Reply

We agree with Falk and colleagues that a randomized trial of electrical versus pharmacologic cardioversion would be the best way to assess the impact of mode of cardioversion on postcardioversion atrial mechanical dysfunction. Although our study (1) was not a randomized trial, it represents the only attempt so far to address this issue after multivariate adjustment for other clinical variables that could potentially influence postcardioversion atrial function. Of all the variables tested, only the mode of cardioversion was seen to have any influence on the recovery of atrial function. It is noteworthy that the delay in mechanical recovery of atrial function that was associated with electrical (vs. nonelectrical) cardioversion was significant after adjustment for pa-