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.

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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).

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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-

tient age, underlying cardiovascular disease or use of antiarrhythmic drug therapy, but not so after adjustment for left ventricular ejection fraction, left atrial diameter or duration of atrial fibrillation. We feel that our results justify the conclusion that "patients who undergo electrical cardioversion display a greater degree and longer duration of mechanical atrial dysfunction than those who convert pharmacologically or spontaneously" (1).

We agree with Falk et al. that some of the patients who underwent electrical cardioversion may have had attempts at pharmacologic cardioversion in the past. By the same token, some of the patients who had pharmacologic cardioversion in our study had also undergone previous attempts at cardioversion. Previous history of cardioversion attempts was not specifically monitored in our study; hence, we are unable to comment on the actual number of study patients who had had previous attempts at cardioversion. To say that "the study is predominantly a comparison of patients who responded to pharmacologic agents compared with those in whom pharmacologic cardioversion failed" is therefore erroneous.

To the best of our knowledge, there is only one previous study that showed that duration of atrial fibrillation <14 days (vs. >6 weeks) is associated with faster recovery of atrial function (2). In that particular study, no multivariate analysis was performed to account for differences between groups of patients who had atrial fibrillation <14 days versus >6 weeks. For this reason, we are not convinced that atrial fibrillation duration <14 days should be a standard reference division point for all future studies with regard to atrial fibrillation. Until more definitive information is available on the impact of duration of atrial fibrillation on atrial function recovery, any dichotomization of this variable will have to be "arbitrary." The division of atrial fibrillation duration in our study is identical to the inclusion criterion used by Falk et al. in their study addressing postcardioversion atrial function (3). Due to uncertainty about the exact duration of atrial fibrillation in many cases, it is not feasible to use duration of atrial fibrillation as a continuous variable.

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Before Predicting Survival in Children With Pulmonary Hypertension and Congenital Heart Disease . . .

In the recent article by Clabby et al. (1), the authors provide very interesting data on the predictive value of hemodynamic data with respect to survival in pediatric patients with primary and secondary pulmonary hypertension. In primary pulmonary hypertension, such data have been reported earlier (2,3). In patients with plexogenic arteriopathy and congenital heart disease, these data are scarce (4).

The design of this study has led to the pitfalls of highly selected patient population and small sample size in relation to the used analysis techniques, and consequently the results have to be interpreted with great caution. The most serious problem with the study design is the composition of group 2. This group includes patients with repaired and unrepaired heart defects, which leads to two comments on the interpretation of the data:

1. In patients with persistent pulmonary hypertension after adequate surgical repair of a heart defect, hemodynamic variables are comparable to those in patients with primary pulmonary hypertension. In contrast, in patients with unrepaired heart defects or residual shunts, hemodynamic variables cannot be interpreted in the same way. The authors suggest that the variable "mean right atrial pressure (mRAP) × pulmonary vascular resistance (PVR)" represents the pressure load on the right ventricle and indicates how well the right ventricle handles that pressure. However, in children with large intracardiac communications, with or without transposition of the great arteries, increased PVR is not simply a pressure load for the right ventricle and ventricular dysfunction does not necessarily lead to increased mRAP.

2. More important, the authors conclude that survival in children with pulmonary hypertension in the presence of congenital heart defects, as predicted by the suggested hemodynamic variables, can be used to determine the optimal timing of lung transplantation. However, before predicting survival or scheduling lung transplantation, the question to be addressed is whether the pulmonary arteriopathy has progressed to the irreversible stage. Pulmonary plexogenic arteriopathy in children with left to right shunts is a reversible disease until a so-called "point of no return" is reached, at which the process has become irreversible, and progression will occur even if the heart defect is corrected (5). In the reversible stage, the treatment of choice will obviously be surgical correction of the heart defect. It can be speculated that the pulmonary vascular disease might have been still reversible in some of the study patients: In 80% of group 2 patients, the heart defect was unrepaired or palliated, or residual defects were present, whereas right to left shunting was present in only 29%. Hemodynamic or histologic evaluation is currently used to assess the progression of pulmonary vascular disease. However, criteria to predict in which patients pulmonary vascular disease will progress despite surgical correction have a broad gray zone (5,6). Therefore, before including these children in a study to predict survival in patients with irreversible pulmonary vascular disease, we should be able to determine the reversibility of this disease process more accurately.

In other words, one does not want to predict the optimal timing for lung transplantation in children who may be cured by surgical correction of their heart disease.

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