will set a precedent for human ventricular function studies by other groups.

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Reply to the Editor:

Chaturvedi and associates note the limitation of the volumetric methods to which the conductance catheter has been compared. We agree. However, confidence in the conductance catheter volumes can hardly be bolstered by arguing that the validation methods are inadequate. This only increases the uncertainty of any derived data. A method with known and quantifiable behavior (linear or not) is preferable to a method with uncertain and unpredictable error. The primary concern that we wished to express about this method relates to the level of validation that is needed before an invasive technique makes the transition to clinical use in human beings. The absence of adequate methods of validation certainly contributes to this concern.

Chaturvedi and associates argue that their data¹ were acquired over a sufficiently narrow range to avoid nonlinear behavior in the conduction catheter. Although the data collected during each inferior vena caval occlusion may have been limited to a 10-ml absolute volume (as indicted by the authors), this is really not the point. For the prebypass and postbypass data to be compared, the behavior of the catheter should be linear over the full range of both data collections. The only data presented in this article relevant to this issue are those of Fig 1, where the prebypass volume range appears to be 12 to 16 ml, whereas the postbypass range is 27 to 37 ml. If a valid comparison is to be made between the 2 data collections, the *total* range over which linearity must be assumed is therefore 25 ml. The article by Applegate, Cheng, and Little² certainly demonstrates significant nonlinearities for data collected over a volume range of less than 20 ml (Figs 5 and 7 in their article). Of the other three articles that Chaturvedi's group references in this regard,³⁻⁵ only the article by Cassidy and Teitel³ actually examined how conductance volumes compare with other methods. In the latter study, 2 scatter plots of cross-sectional data comparing angiographic volumes with conductance catheter volumes are presented (their Figs 2 and 3). Cassidy and Teitel³ performed no residual analysis or other statistical tests designed to detect nonlinear behavior, but it is clear from both of these figures that data in the lower data range fell preferentially below the linear regression line, as would be expected for a nonlinear relation. The absolute data ranges for these 2 figures were about 25 ml (Fig 2) and 20 ml (Fig 3), not dissimilar to the range for the data from Chaturvedi and colleagues. Thus it is not unreasonable to assume that the nonlinearities that have been described in the conductance catheter would affect the comparison of prebypass and postbypass data presented by Chaturvedi and coworkers.

There is at least one important point that we attempted to make in our remarks, which Chaturvedi and colleagues seem to have missed. They note again the conclusion made by Applegate, Cheng, and Little² that volume data derived from the conductance catheter are sufficient to detect changes in contractile state. However, as we noted, in this study² the response to inotropic stimulation was assessed by comparison of pressure-volume data collected over a similar pressure

and volume range before and after intervention. In contrast, Fig 1 from Chaturvedi and coworkers indicates *no* overlap between the pressure and volume data before and after bypass. From a statistical point of view, comparison of slopes collected from nonoverlapping data is fundamentally invalid, because it relies on extrapolation. Although Fig 1 shows a line drawn across the entire figure, the only range for which the derived slope value can be assumed to be valid is the narrow range over which the data were collected. We do not know what the prebypass pressure-volume relation was for the pressure range observed after bypass, because no data were collected in this range. Under these circumstances, the nonlinearities of both the measuring system and the index become far more significant. This is a general and insufficiently recognized issue for slope models and for parameters derived from curve fits.

It is not clear why Chaturvedi and coworkers believe we "confuse the slope of the $dp/dt_{\max}$ -end-diastolic volume relationship" with the ratio of $dp/dt_{\max}$ to maximal developed pressure, as used by them. In fact, we specifically noted that they had taken a different approach from the more common preload adjustment of $dp/dt_{\max}$. However, they are correct that we failed to note that they reported R^2 rather than r values, and we apologize for this. Finally, whether the analysis of contractility represents "conflicting results" will no doubt remain contentious. The results for the three indices of contractility were certainly not the same. The debate as to which of these various indices is "better" is surely also unsettled. It is precisely this uncertainty that leads us to recommend further experimental verification of this method before widespread use in human beings.

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"This" is topped again

To the Editor:

On April 6, 1976, I implanted a 31 mm Hancock model 242 porcine aortic heterograft (serial No. A28481) and implanted saphenous vein grafts to four coronary arteries (anterior descending, diagonal, circumflex marginal, and posterior descending) for a 59-year-old man with calcific aortic stenosis and coronary disease. He has had no symptoms of cardiac disease since and has had both legs amputated and a carotid endarterectomy for atherosclerotic arterial problems. An echocardiogram on March 21, 1998, showed "mild" aortic regurgitation and a peak systolic gradient across the aortic valve of 9 mm Hg.

I examined him on June 12, 1998, 22 years 2 months since replacement of the aortic valve and could hear neither aortic diastolic or systolic murmurs.

This valve has lasted at least 1 year longer than those reported,¹⁻³ and the clock is still running.

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