profile is insonated by the ultrasound beam. The central regions of flow are interrogated by the beam, but the lateral areas of flow are not accounted for because the Doppler beam width cannot encompass the entire area of flow. Unless the flow profile through the region of interest is flat, a significant amount of error in the Doppler frequency profile and power spectrum will be incurred by this nonuniform insonation of flow (14,15).

Given the pitfalls of continuous wave Doppler, it is intriguing that MacIsaac et al. found good correlation between regurgitant fractions measured at cardiac catheterization and the regurgitant fraction estimated by the backscattered signal intensity analysis. A possible explanation for their results may be a fortunate cancellation of repeatedly sampled flow volumes in the mitral inflow with those in the aortic outflow region. In fact, given the continuous wave Doppler considerations described above, this must be the case. Although this method may be potentially useful in the patient population described, caution should be exercised when interpreting such data because there are theoretic concerns about the observed outcome, and, depending on anatomy and flow conditions the fortunate cancellations may not always occur.

How can these concerns be overcome? One possible solution is the application of pulsed wave Doppler. The essential properties of backscattered signal intensity should be equally applicable to pulsed wave Doppler ultrasound (16). Pulsed wave Doppler satisfies the first sampling requirement by allowing the user to focus at a specific location. However, the problem of adequate beam width still remains when pulsed wave Doppler is applied to intracardiac flow. This limitation may be addressed by one of two possible approaches: 1) Assume velocity profiles across the cross-sectional area of flow; or 2) utilize special hardware that is capable of insonating a wide cross-sectional area of flow, similar to pulsed wave dual-beam Doppler echocardiography (17).

The benefit of using backscattered signal power to assess flow ratios is that this method may be applied without knowledge of the cross-sectional area of flow. Although beam width would need to be adjusted for the approximate annulus size so that the beam area is greater than or equal to the flow area, precise beam width would not be a requirement if other sites of flow were avoided during the Doppler interrogation.

In summary, applications of Doppler backscatter theory to estimations of flow ratios using continuous wave Doppler are limited by two fundamental pitfalls. Conventional pulsed wave Doppler overcomes one of these limitations, and the availability of beam width adjustment should overcome the other, so that the full potential of this promising theory can be explored.

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### *Reply*

We thank Ritter and colleagues and Tacy and Cape for their comments about the theoretic background to our report. Although Ritter and colleagues agree that the power of the Doppler signal is proportional to the volume of insonated blood in motion, they argue we should not have used the power of the Doppler signal but its first moment to calculate flow. This latter approach, often referred to as "intensity weighted mean frequency," generally involves the use of a pulsed Doppler system to define a (relatively short) sample volume that encompasses the flow cross section. The first moment of the Doppler spectrum is calculated and integrated over time to yield an estimate of flow. The method is based on the idealization that the Doppler system is sampling an infinitely short cross section of the flow stream. Therefore, the signal power at each Doppler shift frequency is proportional to that portion of the cross-sectional area over which the corresponding velocity value is to be found. Calculation of the first moment is then analogous to multiplying each velocity value by the corresponding area and summing all such contributions to estimate flow. In a variant of this concept, the "attenuation compensated flow meter" uses a narrow second beam to calibrate the relation between Doppler signal power and cross-sectional area.

Our approach differs fundamentally from that just described. It uses continuous wave Doppler, which provides a signal whose power is proportional to the *entire volume* of moving blood within the region insonated by the ultrasound beam. Our hypothesis is that this volume is proportional to the rate of flow through the corresponding valve orifice. As indicated in our report, a full theory of the anterograde flow through a valve orifice has yet to be developed, and hence there is no accepted model against which we can rigorously test this assumption. As a "first-order" argument, the cross-sectional area of the flowing stream of blood may be proportional to the area of the valve orifice, whereas the length of the stream of flow may be proportional to the speed at which blood flows through that orifice.

Both letters express concern regarding the Doppler beam width

relative to the valve orifice. We thank Ritter et al. and Tacy and Cape for drawing this issue to our attention. The beam width of the continuous wave Doppler transducer used in our study was ~8 to 14 mm at depths between 8 and 12 cm. To the extent that this beam width is less than the orifice area, it provides a potential source of error. It is one of a number of issues that require further attention.

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## Pathogenesis of Stroke After Nonanterior Myocardial Infarction

In their recent retrospective study, Bodenheimer et al. (1) found stroke to be as frequent after nonanterior myocardial infarction as after anterior myocardial infarction, during a follow-up period of up to 52 months. Bodenheimer et al. quote our study finding (2) of a similar incidence of stroke in anterior and nonanterior myocardial infarction, but unlike their study our follow-up period was 1 month. The causes of "early" stroke in the first month after myocardial infarction (3) are different from those of "late" stroke after myocardial infarction (4). "Early" stroke has been shown in several studies (3) to be more frequently associated with anterior than nonanterior myocardial infarction and is usually thought to be due to embolism from thrombus resulting from left ventricular apical akinesia. In patients with "late" stroke it is more difficult to identify a single pathogenesis for stroke. In a series of 94 strokes occurring at least 3 months after myocardial infarction, both cardiac and noncardiac causes of stroke were identified (4). In addition to an akinetic left ventricular segment, 21% of patients had significant carotid artery disease, 12% had probable lacunar infarctions, and 12% had atrial fibrillation. A population-based study (5) found a significant difference between the observed and expected probability of stroke only within the first 2 months after myocardial infarction, suggesting that "late" stroke does not relate directly to myocardial infarction but to associated risk factors. If this is true, then the location of the myocardial infarct is not of pathogenetic importance for "late" strokes.

A proportion of "early" strokes after myocardial infarction are associated with nonanterior myocardial infarction. In a study of 445 patients with myocardial infarction in a population with a high prevalence of diabetes (2), we found a similar frequency of anterior (10 [53%] of 19) and nonanterior (9 [47%] of 19) infarct location in 19 patients with stroke within 1 month after myocardial infarction. Three patients with nonanterior myocardial infarction had evidence of probable embolism. Of the nine patients with nonanterior myocardial infarction, seven were diabetic, five (all diabetic) had severe hypotension (a reduction in mean arterial pressure >25%), and in two (both diabetic) the onset of "early" stroke was associated with severe hypotension. This suggests that poor left ventricular function in diabetic patients may predispose to "early" stroke in nonanterior myocardial infarction. Global left ventricular dysfunction may thus be important in the pathogenesis of stroke associated with nonanterior myocardial infarction. Further research is needed to determine whether anticoagulation would reduce the risk of stroke in patients with nonanterior myocardial infarction who are diabetic or who have poor left ventricular function.

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### *Reply*

Pullicino appears to agree with our essential finding, namely, that the similar incidence of stroke in patients with anterior and nonanterior myocardial infarction implies the presence of multiple mechanisms. That these may vary dependent on the time after infarction is entirely possible. Indeed, it is conceivable that myocardial infarction is an epiphenomenon. Thus, although Pullicino quotes Johannesen et al. (1) as supporting embolus as an important mechanism early in the period after myocardial infarction, in their study two of five patients with a stroke had no echocardiographic evidence of left ventricular thrombus, suggesting that even in this early period other causes may need to be considered.

Martin et al. (2) deal only with strokes that occurred a minimum of 3 months after a myocardial infarction. They found a multiplicity of potential causes, both cardiac and noncardiac, supporting our contention that the mechanism of stroke after infarction is often not cardiac. Indeed, in only 14% of their patients was an akinetic left ventricular segment the lone risk factor that they could identify.

In contrast to Pullicino et al. (3) we found no relation between left ventricular function as measured by ejection fraction and stroke (4). Their study "investigated the effect of diabetes on stroke after myocardial infarction" (3). Interestingly, in nondiabetic patients, they could not identify any risk factor for stroke (3).

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