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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