flow underestimates the LAD graft flow by more than 11%.
Moreover, the data during the LAD graft occlusion seems to be mis-plotted in figure 4. Figure 2 was mislabeled.

Hiroe Nakazawa, M.D.
Keio University
Tokyo, Japan

References

The authors reply:
To the Editor:

We are pleased to learn that Dr. Nakazawa shares our enthusiasm for the potential usefulness of the regional thermodilution method to estimate left anterior descending (LAD) blood flow and appreciate the opportunity to clarify some areas of possible confusion.

The first concerns interpretation of the great cardiac vein (GCV) flows recorded during LAD graft occlusion. Nakazawa and colleagues have shown that blood sampled near the GCV origin reflects perfusion almost entirely from the LAD, whereas more distal samples reflect more heterogeneous perfusion. These studies and Dr. Nakazawa's reference to finding that 70% of LAD graft flow is included in the GCV flow refer to studies done in dogs. While human postmortem studies demonstrate some vascular pathways from the septal region to the GCV, the proportion of blood flow draining from this region to the GCV remains to be determined in man. Also, it would be more accurate to state that the contribution of flow depends upon the location of the total or subtotal occlusion rather than the site of the graft to LAD, as noted by Dr. Nakazawa. In each of our cases, the LAD occlusion was proximal (i.e., before or involving the origin of the first septal perforating branch) and the region between the occlusion and the vein graft was opacified when the vein graft was restudied. Therefore, it would not be appropriate to subtract the flows recorded during transient occlusion from the GCV flow. Furthermore, the flows measured in the GCV during transient graft occlusion (5-12 ml/min) were recorded after hypothermic cardioplegic arrest, cardiopulmonary bypass, construction of one or several vein grafts and during a transient occlusion after a variable period of perfusion through the graft, etc. We did not feel that it was appropriate to attempt to interpret the pattern of drainage observed during transient occlusion and therefore, we did not comment about the basis for these flows in the manuscript.

The second area of concern relates to the values summarized in figure 4. These data are correctly plotted. These data represent the absolute change in flow observed when the graft was occluded (i.e., flow before minus flow during occlusion). This is stated in the methods and headings for table 2 where the raw data appear (first and third columns from the right) in addition to the figure legends. These data should not be confused with the actual blood flow recorded during the occlusion (i.e., 5-12 ml/min GCV flow) as Dr. Nakazawa may have. Figure 2A should be labeled figure 2B and vice versa.

Carl J. Pepine, M.D.
J. Mehta, M.D.
Wilmer W. Nichols, Ph.D.
Department of Medicine
Division of Cardiology
University of Florida
Gainesville, Florida 32610

References

The author replies:
To the Editor:

I appreciate the questions raised by Dr. Shubrooks in regard to my paper. The first question is in regard to the statement, "The left ventricular region supplied by the coronary artery with the most marked narrowing was closely related to the region defined by the electrocardiographic leads with ST-segment elevation." To some, patient 4 appears to contradict this statement, as he demonstrated both spontaneous and ergonovine-induced changes in the anterior descending artery, but developed ST-segment elevation inferiorly.

Several points require clarification. In each of the seven patients, episodes of ST-segment elevation that occurred spontaneously or after ergonovine were remarkably similar in regard to coronary

Ergonovine and Variant Angina

To the Editor:

The recent paper by Curry et al. addresses the important question of whether ergonovine-provoked episodes of coronary spasm in patients with variant angina are similar to those that occur spontaneously. They conclude, on the basis of a limited experience of seven patients, that "the left ventricular region supplied by the coronary artery with the most marked narrowing was closely related to the region defined by the electrocardiographic leads with ST-segment elevation." This is, in fact, not supported by their data.

Their patient 4 was reported to have had ST elevation in inferior leads during spontaneous and provoked episodes of pain, yet developed near-occlusion of the left anterior descending artery with only 40% narrowing of the right coronary artery, a degree of narrowing unlikely, on the basis of present knowledge, to cause ST-segment deviation. Their statement that the right coronary artery was involved in three patients, the left anterior descending in three and both in one is therefore also in error. It seems likely that this patient's exertional angina was due to his fixed 70% left anterior descending lesion and that the inferior ST elevations during angina at rest may have been caused by severe right coronary artery spasm; however, this was not demonstrated angiographically during either spontaneously or induced pain.

Thus, the question of consistency of the relationship of ergonovine-induced spasm to electrocardiographic changes and to the anatomic location of ischemia remains unresolved. This study again emphasizes for both authors and journal reviewers the need for accuracy in the reporting and interpretation of data to be published in the medical literature.

Samuel J. Shubrooks, Jr., M.D.
Assistant Professor of Medicine
Harvard Medical School
Boston, Massachusetts

Reference