Phenylephrine and Myocardial Ischemia in Patients Undergoing Carotid Endarterectomy. II.

To the Editor—We read with great interest the article by Smith et al. concerning the effects of phenylephrine on myocardial ischemia in patients undergoing carotid endarterectomy.1 In their concluding statement, the authors made a broad clinical recommendation regarding the use of phenylephrine in patients at risk for myocardial ischemia. Since this recommendation may become accepted in the medical community, it is imperative that all concepts be clearly defined and agreed upon. While Smith et al. clearly defined the ECG criteria for ischemia, they unfortunately failed to do so for the echocardiographic measurements. Nevertheless, one gets the impression that decreases in regional myocardial function were equated with ischemia.

According to our understanding, regional myocardial function is determined by regional preload, regional afterload, and regional contractility. Ischemia can indeed decrease a segment’s contractility (i.e., its capacity to perform work) and, at equal loads, this would be manifested by reduced function. When loading conditions change, however, the effect of these changes must be evaluated before drawing conclusions about contractility or ischemia.

In normal subjects, a wide variability in quantitative regional function has been documented.2 This variability increases after repeated ischemic insults. Since most of the patients in the study by Smith et al. were reported to suffer from angina or to have had previous myocardial infarctions, one would suspect that they exhibited considerable heterogeneity in regional function. It was, therefore, not surprising that as ventricular load was altered with the various pharmacologic regimens, a nonuniform response to the load alterations was observed and that some segments displayed a greater reduction in function than others.

Differentiating between ischemic changes in regional function and load-dependent changes in function is certainly not easy. However, the explanation by Smith et al. that some contraction was present at baseline and that, as a result, “the worsening of regional function was not due to changes in loading, per se,” is inadequate. It ignores the growing body of evidence related to intraventricular mechanical interaction and the importance of regional loading in the determination of regional function.3-5

Since alterations in regional function are not necessarily manifestations of acute ischemia, it would be essential to confirm the presence of ischemia by independent techniques prior to recommending changes in medical management.

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REFERENCES


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In Reply—The letter by Herschman and Thys raises a point about loading conditions and the diagnosis of myocardial ischemia by echocardiography. While we certainly appreciate their point, when regional wall motion abnormalities are accompanied by thickening abnormalities and EKG changes that indicate myocardial ischemia, myocardial ischemia may be present. In this effort, one should say, “Is the benefit of using the drug worth the probable risk that myocardial ischemia is occurring?” It is clear that all alterations in regional function are not necessarily manifestations of acute ischemia, but some undoubtedly are, and the occurrence of wall-thickening changes at the same time as a 2° change in wall motion, and at the same time as electrocardiographic changes indicating ischemia led us to believe the context of our statement, “The data do not support the routine use of phenylephrine to maintain blood pressure in patients at high risk for development of myocardial ischemia.” We think a key word here is routine, as none of these patients demonstrated an indication for using phenylephrine that was other than routine, and in each patient there was an equally good alternative method of causing the same effect as that achieved by the use of phenylephrine.