Volume 75, No. 3
September 1991

THE JOURNAL OF Anesthesiology
THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

EDITORIAL VIEWS

Anesthesiology
75:385–387, 1991

Acute Ventricular Wall Motion Heterogeneity

A Valuable but Imperfect Index of Myocardial Ischemia

Acute regional wall motion abnormalities (RWMA) have been increasingly investigated as sensitive indicators of myocardial ischemia. The clinical usefulness of RWMA in the detection of ischemia depends directly, though not exclusively, on their specificity.* The specificity of RWMA has been questioned. In this issue of ANESTHESIOLOGY, Buffington and Coyle report the effect of the postischemic state on regional ventricular performance.[1] Using sonomicrometry, the authors measured regional wall thickening in postischemic and nonischemic myocardium under a variety of loading conditions. The postischemic myocardium has previously been demonstrated to have normal perfusion.[2] Thus, by definition, acute RWMA in this condition must represent the nonspecificity of RWMA for ischemia.

In these experiments, Buffington and Coyle measured ventricular wall thickening under control conditions and after 10 min of occlusion plus 1 h of reperfusion. Recordings were obtained both from a test zone subjected to ischemia and reperfusion and from a remote zone supplied by another coronary artery and not subjected to ischemia and reperfusion. Measurements were made at nine different combinations of left atrial pressure (used as an index of preload) and mean arterial pressure (used as a surrogate of afterload). Loading conditions were tightly and independently controlled. Systolic wall thickening was expressed both in absolute values and as percentages of diastolic wall thickness.

Systolic ventricular wall thickening increased with increasing left atrial pressure under control conditions, whereas it did not change with increasing left atrial pressure in the postischemic myocardium. The authors concluded that the postischemic myocardium had a diminished response to increases in preload.

The magnitude of systolic wall thickening decreased with increasing afterload in both the postischemic test zone and in control myocardium. When systolic wall thickening was plotted against mean arterial pressure, the negative slope of the line was the same for control and postischemic myocardium, suggesting similar sensitivity† to increasing afterload. Expressed another way, a given increase in afterload resulted in the same relative decrease in percent systolic shortening in postischemic and control myocardium, despite a smaller absolute increase in wall thickness in postischemic myocardium. The authors interpreted these findings to suggest that the response to afterload was unchanged by ischemia and reperfusion. Furthermore, the authors compared the ratio of test-zone wall thickening to remote-zone wall thickening (rather than the ratio of test-zone and remote-zone thickening to their respective controls). They found that under control conditions, the test-zone wall thickening was 130–140% of remote-zone wall thickening for all mean arterial pressures. They attributed this finding to heterogeneity of regional contraction.

Buffington and Coyle concluded that both the test zones and the remote zones were equally affected by increases in afterload before ischemia and reperfusion. However, after ischemia and reperfusion, the thickening of the remote zone unexpectedly increased with increasing after-

Accepted for publication May 30, 1991.

Address reprint requests to Dr. Lowenstein: Anesthetist-in-Chief, Department of Anesthesia and Critical Care, Beth Israel Hospital, Boston, Massachusetts 02215.

Key words: Heart, ischemia: wall motion abnormality.

* Specificity in this context is defined as no change in wall motion or thickening when ischemia or infarction is not present.

† Sensitivity is defined as abnormal wall motion or thickening when ischemia or infarction is present.

load, whereas thickening in the test zone decreased. Thus, the ratio of percent test-zone thickening to percent remote-zone thickening decreased. The authors concluded that with increasing afterload, the proximity of ever more weakly contracting postischemic myocardium to strongly contracting myocardium can mimic an ischemic RWMA even when ischemia is presumably no longer present.

It is the interpretation of this finding that is so intriguing. Initially, it appears to be (as the authors suggest) an example of acute reversible ventricular wall motion heterogeneity that would be detectable by echocardiography. Since acute reversible RWMA is considered by some to be pathognomonic of myocardial ischemia, this would be an example of a false positive finding, since myocardial perfusion is known to be normal in the acute postischemic state.

Many clinical echocardiographic researchers require greater changes in wall motion before they consider the finding compatible with a new abnormality. Leung *et al.*, for example, describe four worsening grades of RWMA (mild hypokinesia, severe hypokinesia, akinesia, and dyskinesia) and require a two-grade worsening in wall function before designating the change a new RWMA.[3] The absolute differences in wall thickening described by Buffington and Coyle are quite small. The difference in thickening between the postischemic test zone and remote zone is never more than 1.2 mm. The difference in thickening between the postischemic test zone and the nonischemic test zone is never more than 1.6 mm under the same loading conditions. It is unlikely that changes of this magnitude would, in fact, fulfill the echocardiographic criteria for acute RWMA with currently available technology.

The authors analyzed their data in an unconventional manner. Rather than comparing thickening in the postischemic test zone to its own preischemic control and the remote zone to its own control, ratios of test zone thickening to remote zone thickening were calculated under control conditions and after ischemia and reperfusion. These calculations emphasized the differences between the test zone and the remote zone, rather than changes in a given region over time; they are comparable to clinically observing an isolated echocardiogram for the first time and examining it for heterogeneity of contraction. However, because it does not compare data after ischemia and reperfusion to data of the same region prior to ischemia, the analysis may be misleading. This is particularly important because there were differences between zones at control that were nearly as great as those postischemic differences that form the basis of the authors' conclusion. Other investigators have been careful to evaluate RWMA by comparing segments to their respective controls.[4] Such a comparison is critical in the clinical setting in which preexisting RWMA are common, and the goal may be to identify changes in wall motion after an intervention rather than relative regional differences at any given moment in time.

We have replotted the authors' data in a way that compares wall thickening in the postischemic test zone and remote zone to that of the same region in the control state (fig. 1). With increasing afterload, the performance of the test zone after ischemia and reperfusion relative to control conditions declines. If the decline is statistically significant (which cannot be determined from the data given), postischemic myocardium might indeed be more sensitive to afterload than is normal myocardium, in contrast to the authors' conclusions. Compared to its own control, the wall thickening of the remote zone actually increases with increasing mean arterial pressure. This analysis is consistent with and in fact emphasizes the authors' point that the remote zone thickens better than the test zone with increases in afterload. They both do thicken, however. Neither zone becomes akinetic, dyskinetic, or even severely hypokinetic. Whether a difference of this degree would fulfill the criteria for presence of a RWMA echocardiographically in humans remains unclear.

Despite these reservations, the questions raised by these investigators are pertinent. Severe ischemia has been known since 1935 to cause abnormal wall motion.[5] There are abundant data to suggest that RWMA are sensitive indicators of ischemia as well. A decrease as small as 10% in coronary blood flow causes a decline in regional performance detectable by sonomicrometry.[6] Unfortunately,

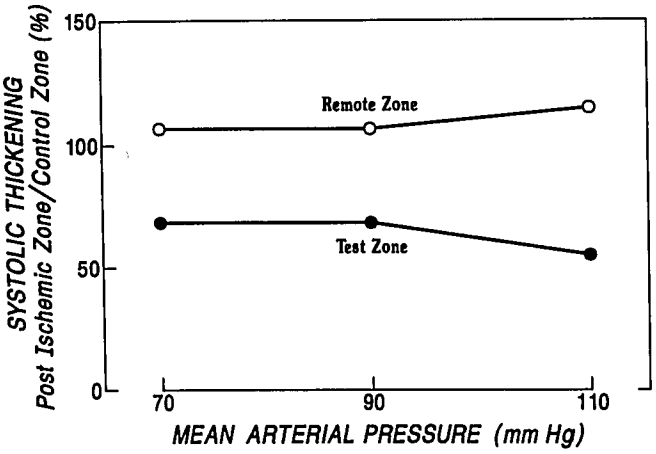

FIG. 1. Systolic wall thickening of the remote zone and the test zone after ischemia and reperfusion are compared to their respective controls. The relative systolic thickening (postischemic zone thickening as a percentage of its control state) is plotted against mean arterial pressure (afterload). With increasing afterload, the performance of the test zone declines. This may indicate that the postischemic myocardium is more sensitive to afterload than is normal myocardium. In contrast, the performance of the remote zone improves with increasing afterload. Data are given in table 1.

regional changes may occur clinically in areas not visualized by echocardiography or any method of detection that does not image the entire ventricle.

The question of the specificity of RWMA remains. How often are the RWMA observed clinically caused by factors other than ischemia? Numerous causes have been documented. For example, left ventricular wall motion normally is not uniform throughout the ventricle. In fact, contractility decreases from apex to base and may even differ around the minor axis, as the authors' control data suggest.[7,8] Acute RWMA have been described after reinstitution of coronary flow postbypass in cardiac surgical patients. Leung *et al.* have suggested that new RWMA observed clinically after bypass by echocardiography are ischemic in origin.[3] It is possible that blood flow actually may be optimal but that contractility has been depressed by other associated factors. Although this does not cast doubt on the association of new RWMA with adverse cardiac outcomes, it may warrant a reexamination of the postulated mechanism.

Regional dysfunction has been documented previously to be caused by changes in loading conditions.[9] However, no study before that of Buffington and Coyle has so systematically examined the effect of loading in the postischemic myocardium. This and other studies underscore the point that all regional heterogeneity in ventricular function is not ischemic in origin. With the current interest in this area and advancement in technology, we hope that these issues will be addressed and that the elusive goal of a specific and sensitive clinical index of myocardial ischemia will be achieved.

EDWARD LOWENSTEIN, M.D.
J. MICHAEL HAERING, M.D.

Department of Anesthesia and Critical Care,
Beth Israel Hospital
Department of Anaesthesia, Harvard Medical School

PAMELA S. DOUGLAS, M.D.
Cardiovascular Division, Beth Israel Hospital
Department of Medicine, Harvard Medical School
Boston, Massachusetts

References

1. Buffington CW, Coyle RJ: Altered load dependence of postischemic myocardium. ANESTHESIOLOGY 75:464–474, 1991
2. Jeremy RW, Stahl L, Gillinov M, Litt M, Aversano TR, Becker LC: Preservation of coronary flow reserve in stunned myocardium. Am J Physiol 256:H1303–H1310, 1989
3. Leung JM, O'Kelly B, Browner WS, Tubau J, Hollenberg M, Mangano DT: Prognostic importance of post bypass regional wall-motion abnormalities in patients undergoing coronary bypass graft surgery. ANESTHESIOLOGY 71:16–25, 1989
4. Leung JM, O'Kelly BF, Mangano DT, the SPI Research Group: Relationship of regional wall motion abnormalities to hemodynamic indices of myocardial oxygen supply and demand in patients undergoing CABG surgery. ANESTHESIOLOGY 73:802–814, 1990
5. Tennant R, Wiggers CJ: The effect of coronary occlusion on myocardial contraction. Am J Physiol 112:351–361, 1935
6. Vatner SF. Correlation between acute reduction in myocardial blood flow and function in conscious dogs. Circ Res 47:201–207, 1980
7. Clements FM, de Bruijn NP: Perioperative evaluation of regional wall motion by transesophageal two-dimensional echocardiography. Anesth Analg 66:249–261, 1987
8. Diedericks J, Leone BJ, Foex P: Regional differences in left ventricular wall motion in the anesthetized dog. ANESTHESIOLOGY 70:82–90, 1989
9. Ross J: Assessment of ischemic regional myocardial dysfunction and its reversibility. Circulation 74:1186–1190, 1986