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Transesophageal Echocardiography and Intraoperative Monitoring of Left Ventricular Function

RECENT studies have suggested that intraoperative transesophageal echocardiography (TEE) provides monitoring of left ventricular wall motion that is superior to electrocardiography and pulmonary capillary wedge pressure in the assessment of ischemia.[1,2] In addition, Leung *et al.* have demonstrated that such perioperative wall motion abnormalities in patients may have prognostic significance: 6 of 18 patients with a postbypass wall motion abnormality, *versus* 0 of 32 without an abnormality,[3] had adverse outcomes.

In the study reported by Leung *et al.*[4] in this issue of ANESTHESIOLOGY, TEE, electrocardiography, and hemodynamics were continuously monitored perioperatively in order to study the relationship between wall motion abnormalities and indices of myocardial oxygen supply and demand. The authors observed that regional wall motion abnormalities most often were not triggered by acute increases in myocardial oxygen demand, suggesting that a primary decrease in myocardial oxygen supply is the important mechanism.

The authors note that without an absolute reference standard, not all regional wall motion abnormalities are necessarily indicative of acute myocardial ischemia; they discuss other causes, including "stunned" or "hibernating" myocardium, changes in loading conditions, previous infarction, and temperature inhomogeneity. Although these caveats are important, there is, nevertheless, an extensive experimental and clinical literature supporting the view that, in the setting of coronary disease, a new wall motion abnormality is most likely due to acute ischemia or infarction. For example, canine studies using sonomicrometers and transthoracic two-dimensional echocardiography (TTE) have validated the dependence of wall thickening on perfusion. In addition, when sonomicrometers and TTE were compared in the detection of ischemia induced by severe coronary stenosis, both techniques demonstrated dyskinesis (systolic wall expansion or thinning).[5,6] In humans, new wall motion abnormalities observed immediately after exercise suggest the presence of coronary artery stenosis.[7] The improved sensitivity (76 *vs.* 36%) of echocardiography over exercise electrocardiography has been demonstrated in the detection of single-vessel coronary disease. Furthermore, the development of an exercise-induced wall motion abnormality remote from an infarcted area is sensitive for the presence of multi-vessel coronary artery disease.[8] The mechanism of such exercise-induced wall motion abnormalities is presumed to be increased oxygen demand.

On the other hand, experimental animal studies of ischemia and infarction have not been performed with TEE. Because of the medial position of the heart, the canine left ventricle does not image well by TEE.[9] Thus, although the extensive literature on *TTE* monitoring of wall motion abnormalities in the assessment of ischemia most likely is applicable to *TEE* as well, this remains an assumption not yet supported by extensive data.

Several other considerations apply to the use of TEE monitoring of left ventricular function:

1) Repeated imaging for left ventricular function assessment requires reproducible data acquisition and analysis. This, in turn, requires careful attention to probe position and orientation. Smith *et al.* have shown that this

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is feasible: repositioning the TEE probe at the midpapillary level after withdrawing or inserting the probe 2 cm yielded no difference in end-diastolic or systolic areas and no greater than one grade of wall motion abnormality difference.[1]

2) Comprehensive high-quality scans of both ventricles are needed for complete, accurate detection of wall motion abnormalities.[10] In the current study by Leung *et al.*,[4] wall motion evaluation by TEE was limited to short-axis views at the midpapillary muscle level. Although perfusion beds of the three main coronary arteries usually are imaged at this level, basal and apical wall motion abnormalities may go undetected;[11] this may be part of the explanation why Leung *et al.* did not detect wall motion abnormalities in some patients who did show electrocardiographic abnormalities. The use of multiple imaging planes (mitral and papillary short axis and four chamber views), permits identification of all segments.[12] New transducers may permit biplane imaging of the ventricle, with more complete scanning of the entire ventricle.

3) Criteria for the diagnosis of infarction and ischemia affects the sensitivity and specificity of the technique. Infarctions that do not produce dyskinesis in dogs tend to be very small (≤4% left ventricular mass at 20 min after coronary occlusion and ≤ 6% left ventricular mass at 2 days).[13] Easing the criterion by including hypokinesis may improve sensitivity, but at the expense of specificity. In studies comparing two-dimensional wall motion abnormalities to morphologic evidence of infarction in 20 autopsied patients, Weiss *et al.* demonstrated that hypokinesis could not distinguish between subendocardial myocardial infarction and uninjured myocardium.[14] The lack of specificity of hypokinesis in the detection of ischemia was evident in a study by Pandian *et al.*[15] Wall thickening was measured in 12 normal subjects; multiple short-axis views of the left ventricle were obtained. Wall thickening varied from 0 to 150% (average 54%), indicating that considerable inhomogeneity of contraction occurs in normal individuals at rest. Myocardial segments in the lower end of this wide range easily might be considered "hypokinetic" and consequently ischemic, a conclusion that would be incorrect in the normal subjects studied in that investigation. Requiring akinesis of dyskinesis as the criterion for ischemia would be more specific, since systolic wall thinning occurred in fewer than 1% of normal segments studied by Pandian *et al.*,[15] but inevitably would be less sensitive.

Leung *et al.*, in the current study, have adopted a relatively conservative criterion, defining a "TEE episode" suggestive of ischemia as a new regional wall motion abnormality that worsens two or more grades (*e.g.*, normal becoming severely hypokinetic, or hypokinetic becoming akinetic or dyskinetic) and lasting at least 1 min. These criteria are reasonable and yet still need validation, since the specificity of any TEE thickening criterion for ischemia remains undetermined. In addition, the need to determine sensitivity is emphasized by the finding in the current study that only 8 of the 18 episodes (44%) of ischemia detected by ECG were identified by TEE.[4]

4) Echocardiography is an interactive examination that depends heavily on the skill of the operator.[10] Leung *et al.* have an established record of experience and skill in using the technique[3] and come from an institution that pioneered its use.[1,9] Anesthesiologists who wish to begin using TEE for intraoperative monitoring must understand regional cardiac anatomy (with tomographic views) and physiology prior to attempting the identification of abnormal ventricular function.[16] Seward *et al.*[16] recommend 6–8 weeks of training in basic echocardiography under the tutelage of an experienced echocardiographer before independent performance of TEE.

In summary, intraoperative TEE permits qualitative assessment of wall motion abnormalities; their presence perioperatively may indicate ischemia and poor outcome in patients undergoing myocardial revascularization. Currently, however, the relationship of abnormal wall motion to ischemia is validated only incompletely and is based on the TTE literature. Further validation and quantitative wall motion analysis should enhance applications of TEE.

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