Is Coronary Artery Disease Equivalent to Ischemia?

To the Editor.—In a recent commentary, Fleisher stated that even if ST segment trend monitoring has identified accurately a significant ST segment change, ST segment depression may not represent myocardial ischemia in younger individuals or in those with a low probability of coronary artery disease (CAD).1 In support of this contention, the argument is made that structural CAD usually is absent in these patients when tested postoperatively.2 This argument seems to equate the presence of structural CAD with myocardial ischemia, which we define for this discussion to be inadequate myocardial blood flow or oxygen delivery to meet metabolic demands. Although it may be true that ST segment depression does not indicate the presence of CAD in low-risk patients and, therefore, represents a false positive predictor of CAD, it may not be possible to extrapolate these data to state that a myocardial oxygen imbalance or myocardial ischemia does not exist.

Even if CAD is unlikely in the obstetric population, insufficient evidence exists to dismiss ST segment depression during cesarean section as physiologically unimportant because hemodynamic perturbations known to affect myocardial perfusion frequently coexist during these clinical occurrences. It is common for the combination of diastolic hypotension and tachycardia to coexist immediately after delivery when rapid infusions of oxytocin are administered. It is possible that the combination of hypotension and tachycardia may decrease myocardial perfusion, even in the absence of CAD. Similarly, preoperative smoking was associated with ST segment depression in patients with a low risk of coronary artery disease,3 and the incidence of ST segment depression increased with indices of cardiac work and intraoperative carbon monoxide concentrations. This indicates that metabolic poisoning caused by smoking may be responsible for markers of ischemia such as ST segment depression in patients with a low risk of CAD. In addition, the possible contribution of endothelial dysfunction to occurrences of ST segment depression may be nearly impossible to establish during an intraoperative event. Even in the absence of angiographically demonstrable CAD, endothelial dysfunction may produce ischemia in response to what are normally vasodilator stimuli in the normal vessel.4,5 Endothelial dysfunction is an emerging concept that may explain symptoms of ischemic heart disease in angiographically normal patients.

Although it may be difficult or impossible to prove their contribution in any individual patient in a clinical perioperative setting, these metabolic, hemodynamic, or endothelial-related etiologies may potentially contribute to intraoperative ST segment depression. Although the existing literature has shown that, in low risk populations, intraoperative ST segment depression may not indicate structural CAD, the literature does not support the contention that myocardial ischemia does not exist in these situations, nor has it been shown that ST segment depression should go untreated or ignored in patients who have a low risk of CAD and may have other etiologies for myocardial oxygen imbalance.

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References


In Reply.—The comments by Drs. Woehlck, Cinquegrani, and Connolly are appreciated. The authors have highlighted an important issue with regard to any diagnostic test, i.e., the correlation between a positive result and a specific disease. In the case of the interpretation of ST segment on an electrocardiogram, it is the correlation of ST segment changes with structural coronary artery disease, not myocardial ischemia, that has been used in the stress test literature to define a positive test. Therefore, I agree with the authors of the letter that the presence of myocardial ischemia cannot be ruled out in surgical and obstetric patients without structural disease. In fact, short-term ST segment changes after induction of general anesthesia in patients with normal coronary arteries has led to a myocardial infarction, most likely the result of coronary spasm.1 However, ST segment depression is not pathognomonic for myocardial ischemia. It is conceivable that ST segment depression changes observed during elective cesarean section or general surgery in low-risk patients reflect supply–demand mismatches in patients without known structural coronary disease, but many of these changes did not correlate with the hemodynamic effects or the medication usage outlined by the authors.2–4 Additionally, regional wall motion abnormalities, a more sensitive marker of myocardial ischemia, were not correlated with the electrocardiographic changes.6,7 Ideally, coronary sinus lactate measurements would be required to determine the etiology, but they have not been studied in these cases. Therefore, I agree with the authors and appreciate their comments that ST segment depression perioperatively in these patients may reflect myocardial ischemia from supply–demand mis-
matches, and treatment of hemodynamic derangements should be implemented. However, further evaluation or treatment beyond control of heart rate and blood pressure is warranted rarely in the absence of other signs or symptoms.

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References


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