Detection and Treatment of Intraoperative Myocardial Ischemia

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Despite the potentially protective effects of anesthetics, myocardial ischemia can occur at virtually anytime during the perioperative period. Prompt diagnosis and treatment of ischemia is important for three reasons. First, all myocardial infarctions are preceded by myocardial ischemia, i.e., the period (usually hours) before irreversible myocardial damage has occurred. Timely therapeutic measures may reduce the size of the infarction or actually abort it. Second, when large areas of myocardium become ischemic, ventricular function is compromised and cardiogenic shock may ensue. Third, even small areas of myocardial ischemia may give rise to potentially life-threatening dysrhythmias. In these lecture notes, I will review clinical methods used to detect and treat intraoperative myocardial ischemia

Diagnosis

The electrocardiogram (ECG) is the principal tool used for the diagnosis of myocardial ischemia. Normally, the ST segment of the ECG is isoelectric, that is, the ST segment lies on the same level as the subsequent TP interval (the interval between the next T wave and P wave). During myocardial ischemia, the ST segment is depressed 0.1 mV or more and its contour is usually horizontal or down sloping. Although some drugs (especially digitalis), electrolyte abnormalities, and pericarditis may also cause ST changes, when horizontal or down sloping ST depression $>$ 0.1 millivolt (mV) accompanies anginal chest pain, myocardial ischemia is almost certainly present. Other ECG abnormalities, such as T wave inversion, bundle-branch block, and dysrhythmias, are far less specific for myocardial ischemia.

Unfortunately, a number of aspects of the operating room environment hinder the ECG diagnosis of myocardial ischemia. First, many operating room monitors display a highly filtered ECG signal. Low frequency filtering reduces the wandering of the ECG signal up and down the display screen sometimes seen in association with respiration or movement. Unfortunately, low frequency filtering may distort the ST segment and render it unusable for the diagnosis of ischemia. The owner's manual for your monitor will usually list the frequency range of the ECG. Newer monitors allow the user to defeat excessive low frequency filtering and thereby regain the diagnostic capabilities of the monitor.

Second, appropriate calibration of the ECG signal is essential. Standard calibration is 1 cm/mV. At this calibration, 1 mm of ST depression equals 0.1 mV. However, a 1-mm change is very difficult to see on a monitor, and therefore I recommend that your monitor's ECG signal be doubled to 2 cm/mV. Remember, at this calibration, a 2 mm change in the ST segment is necessary before the diagnosis of ischemia is likely. If you don't know your ECG's calibration, you really can't be certain what an ST segment change or lack of change means!

Third, not all leads of the ECG are equally valuable for the detection of ischemia: lead V_5 is the most sensitive single lead. When two leads can be monitored simultaneously, lead II is the best compliment to lead V_5 because it significantly improves the sensitivity for ischemia and usually reveals the p wave for dysrhythmia diagnosis (1). If your ECG system has only three electrodes, you can obtain a modified V_5 by setting the monitor to display lead I, and then placing the left arm electrode in the fifth intercostal space at the anterior axillary line, the left leg electrode on the left shoulder (this electrode's placement isn't crucial), and the right arm electrode on the right shoulder just below the middle of the clavicle.

Fourth, even when ST segment change occurs, anesthesiologists frequently miss it, because their attention is required for many other diagnostic and therapeutic tasks (2). Fortunately, reliable automated ST segment analysis has arrived and been incorporated into many monitors. Multiple leads (usually 2 or 3) of the ECG can be simultaneously, continuously, and automatically monitored. Artifact elimination is not perfect, and therefore, the anesthesiologist must confirm the validity of the detected ST segment deviation.

Even with automated analysis, ECG monitoring for ischemia is far from perfect. Unless the ischemia is severe and/or prolonged (at least a few minutes), the ECG is unlikely to change. In contrast, within seconds

after the interruption of myocardial blood flow, normal inward motion and thickening of the affected myocardium ceases. No other pathophysiologic process produces such acute changes in segmental myocardial contraction. Until the advent of transesophageal echocardiography (TEE), anesthesiologists could not detect these changes because they had no way of directly monitoring myocardial contraction.

Smith et al. (3) used TEE in 50 patients undergoing coronary artery or major vascular surgery. At predetermined intervals, echocardiograms and multilead ECGs (limb leads, augmented leads, and V5) were recorded, both of which were evaluated by "blinded" observers. Intraoperatively, six patients had ST segment changes diagnostic of myocardial ischemia (>0.1 mV deviation), whereas 24 had new SWMAs diagnostic of myocardial ischemia. No patient experienced an ST segment change before or in the absence of a new SWMA. In three of the six patients who experienced ST segment change, the SWMA occurred minutes before the ECG change. Three of the 50 patients suffered intraoperative myocardial infarctions, and all had a SWMA develop and persist until the end of surgery in the corresponding area of myocardium. Only one of these patients had intraoperative ST segment change diagnostic of ischemia. Ten patients without coronary disease were also studied and none of these patients had SWMAs. In the 50 patients with coronary disease, 97% of the echocardiograms were analyzed, but inadequate resolution or an inappropriate cross section prevented analysis in the other 3%. In contrast, only 86% of the ECGs were analyzed because of the onset of bundle-branch block or paced rhythm.

In a similar study, in which continuous TEE and 2-lead Holter recordings were utilized inpatients undergoing coronary artery surgery, Leung et al. (4) found comparable results. In neither study were hemodynamics predictive of ischemia or postoperative cardiac complications. Thus, myocardial ischemia can be detected more frequently with TEE than with ECG, and when new SWMAs persist through the conclusion of surgery they should be viewed as prognostic signs for cardiovascular complications.

Van Daele et al. (5) confirmed the insensitivity of hemodynamics, specifically pulmonary capillary wedge pressure (PCWP) monitoring, for the detection of myocardial ischemia. In 98 patients anesthetized with large dose narcotics, 12-lead electrocardiography and TEE identified 14 patients experiencing ischemia before the start of coronary artery surgery (all 14 had TEE-detected SWMAs and 10 also had ST segment changes). Concomitant with the onset of ischemia, PCWP increased 3.5 ± 4.8 mm Hg. However, many patients without ischemia experienced similar increases in PCWP. In fact, an increase of 3 mm Hg in the PCWP had a sensitivity of 33% and a positive predictive value of 16% for detection of myocardial ischemia in this patient population.

Limitations of TEE in the detection of ischemia also should be recognized. When an area of myocardium is clearly in view, segmental contraction can be difficult to evaluate if the heart rotates or translates markedly during systole, or if discoordinated contraction occurs due to bundle branch block or ventricular pacing. Consequently, a valid system for SWMA assessment first must compensate for global motion of the heart (typically by using a "floating" frame of reference), then evaluate both regional endocardial motion and myocardial thickening. A worsening of segmental wall motion and wall thickening (in the absence of similar global changes) of at least two classes is required to make the diagnosis of ischemia; less pronounced changes are not consistently interpreted, even by experts.

Interpretation of septal motion is the most problematic because it often is confounded by discoordinated contraction patterns. However, a simple rule applies: when the septum is viable and nonischemic, it thickens appreciably during systole, although its inward motion may begin slightly before or after the inward motion of the other ventricular segments. Thus, new SWMA can be detected during bundle-branch block, ventricular pacing, and marked global movements of the heart, but not by assessment of endocardial motion alone; wall thickening also must be assessed. Because not all hearts contract normally and not all parts of the normal heart contract to the same degree, not all SWMA are indicative of myocardial ischemia. For example, myocardial infarction, myocardial stunning and myocarditis can cause SWMA. However, a sudden, severe decrease or cessation of segmental contraction is almost certainly attributable to myocardial ischemia.

Treatment

Myocardial ischemia must be viewed with the same sense of urgency as hypoxemia or hypotension. All three are associated with an imminent risk of death. No ideal treatment strategy for the management of intraoperative myocardial ischemia has been established. I offer you my own approach for your consideration. Because myocardial ischemia can be a manifestation of inappropriate anesthetic management, I first evaluate the adequacy of ventilation, oxygenation, and anesthetic depth. Next, I turn to the control of hemodynamics, then to direct antianginal agents, and finally to the institution of invasive measures such as intraaortic balloon counterpulsation or angioplasty.

In the control of hemodynamics, management of heart rate takes priority. Increases in heart rate not only increase myocardial oxygen demand, but also

decrease myocardial oxygen supply because the duration of diastole is shortened by increases in heart rate and it is during diastole that coronary blood flow occurs. Heart rate can be controlled by addition of a small dose of narcotic such as fentanyl but may also require the use of a β blocker (6). Esmolol is a cardioselective β-adrenergic antagonist. It is rapidly metabolized in blood and liver by hydrolysis and has a much shorter duration of action than other available β blockers.

Next, I control blood pressure. Clearly, knowing the etiology of a blood pressure change is the key to determining the appropriate therapy. Hypovolemia is best managed with volume, and inappropriate vasodilation best managed with a vasoconstrictor. Next, I attempt to control filling pressure. Remember that coronary perfusion pressure equals the aortic diastolic pressure minus the left ventricular diastolic pressure. Thus, a normal coronary perfusion pressure would be approximately 75 mm Hg. However, in the presence of a severe coronary obstruction, the pressure distal to that obstruction may be much lower than aortic diastolic pressure. Therefore, the effective coronary perfusion pressure may be a fraction of normal, and increases in left ventricular diastolic pressure may virtually abolish coronary perfusion. Subsequently, I will discuss some of the theoretical advantages in using nitroglycerin to control left ventricular diastolic pressure. However, a final hemodynamic consideration is to reduce myocardial contractility. Thiopental, inhalation anesthetics, calcium channel blockers, and β blockers all accomplish this purpose. Unfortunately, it is almost impossible in clinical practice to assess myocardial contractility and therefore these therapies are difficult to titrate effectively.

In contrast, IV nitroglycerin is easily titrated because of its very rapid onset and short duration of action. It produces marked venodilation with limited arterial dilation. Thus, left ventricular filling volume and pressure are usually reduced to a much greater degree than arterial blood pressure. Obviously, this is of a substantial advantage in enhancing effective coronary perfusion pressure. In addition, nitroglycerin dilates larger coronary arteries and even the residual lumen within coronary constrictions. Because of these facts, IV nitroglycerin is usually the first pharmacologic agent I choose for control of intraoperative myocardial ischemia after basic anesthetic management and hemodynamics have been optimized. If nitroglycerin induces hypotension, I add phenylephrine to restore the blood pressure unless I suspect left ventricular failure (7–9). If nitroglycerin fails to alleviate the myocardial ischemia, I will consider additional pharmacologic agents such as β blockers or calcium channel blockers but also at this time I consider changing anesthetic agents. Because anesthetics may have highly variable effects on cardiovascular physiology, I would recommend changing anesthetic strategies if all of the above efforts fail to relieve ischemia. Finally, if myocardial ischemia still persists or is accompanied by left ventricular failure, I will request the placement of an intraaortic balloon pump, coronary angioplasty, and/or coronary thrombolysis. An interventional cardiologist will be needed for such endeavors, but failure to treat persistent myocardial ischemia or delay in its treatment may result in unnecessary loss of myocardium, cardiac reserve, or viable cardiac function. Such a multidisciplinary approach is more complex and difficult to orchestrate, but represents state-of-the-art care. Recently, one of my patients experienced profound myocardial ischemia during a major orthopedic procedure. Despite the efforts outlined above, his condition deteriorated into cardiogenic shock. After our surgeons rapidly completed surgery, we took the patient to the cardiac catheterization laboratory where our cardiologists confirmed that he had clotted the stent in his left anterior descending coronary artery. They reopened the stent and he recovered rapidly without significant myocardial injury.

Summary

Anesthesiologists have powerful tools for the diagnosis and treatment of intraoperative myocardial ischemia. When myocardial ischemia develops, the basics of anesthetic management must be secured first, and thereafter, pharmacologic and mechanical interventions may be necessary. Intraoperative myocardial infarctions will occur no matter how vigilant and how skilled we become; however, within our reach is the prospect that proper anesthetic management, in its broadest meaning, will reduce the risk that our patients with ischemic heart disease face in the perioperative period.

References

1. London MJ, Hollenberg M, Wong MG, et al. Intraoperative myocardial ischemia: localization by continuous 12-lead electrocardiography. Anesthesiology 1988;69:232–41.
2. Roy WL, Edelist G, Gilbert B. Myocardial ischemia during noncardiac surgical procedures in patients with coronary-artery disease. Anesthesiology 1979;51:393–7.
3. Smith JS, Cahalan MK, Benefiel DJ, et al. Intraoperative detection of myocardial ischemia in high-risk patients: electrocardiography versus two-dimensional transesophageal echocardiography. Circulation 1985;72:1015–21.
4. Leung JM, OKelly B, Browner WS, et al. Prognostic importance of postbypass regional wall-motion abnormalities in patients undergoing coronary artery bypass graft surgery. Anesthesiology 1989;71:16–25.
5. van Daele ME, Sutherland GR, Mitchell MM, et al. Do changes in pulmonary capillary wedge pressure adequately reflect myocardial ischemia during anesthesia? A correlative preoperative hemodynamic, electrocardiographic, and transesophageal echocardiographic study. Circulation 1990;81:865–71.

6. Cahalan MK, Lurz FW, Eger Ed, et al. Narcotics decrease heart rate during inhalational anesthesia. Anesth Analg 1987;66: 166–70.
7. Miller RR, Awan NA, DeMaria AN, et al. Importance of maintaining systemic blood pressure during nitroglycerin administration for reducing ischemic injury in patients with coronary disease. Effects on coronary blood flow, myocardial energetics and left ventricular function. Am J Cardiol 1977;40:504–8.
8. Epstein SE, Borer JS, Kent KM, et al. Protection of ischemic myocardium by nitroglycerin: experimental and clinical results. Circulation 1976;53(3 Suppl):I191–8.
9. Borer JS, Redwood DR, Levitt B, et al. Reduction in myocardial ischemia with nitroglycerin or nitroglycerin plus phenylephrine administered during acute myocardial infarction. N Engl J Med 1975;293:1008–12.