Perioperative MI: Data, practice, and questions

Except in emergency or specific high-risk surgery, or for extremely fragile high-risk patients, we anticipate a successful outcome from noncardiac surgery. The skills and tools of our anesthesiology colleagues have advanced to the point that severe intraoperative and immediate postoperative complications are rare.

Preoperative risk assessment and perioperative medical management in large medical centers are now largely done by hospital-based physicians with interest and expertise in this subspecialty, and are integrated into the care of the surgical patient. This has likely contributed to improved patient outcomes. Yet postoperative cardiovascular events still cause significant morbidity (although they generally occur in less than 10% of patients).

The entity of perioperative myocardial infarction (MI) has an interesting history. We have recognized for several decades that its presentation is often different than the usually diagnosed MI: perioperative MI is often painless and may manifest as unexplained sinus tachycardia, subtle changes in mental status, or mild dyspnea. These symptoms, if they occurred while the patient was at home, would often be mild enough that the patient would not seek immediate medical attention. Autopsy studies suggested that many of these MIs result from a different pathophysiology than the garden variety MI; plaque rupture with or without secondary thrombosis may be less common than myocardial injury resulting from an imbalance between cardiac demand and blood flow. Studies initially suggested that postoperative MI occurred many days after the surgery. But as tests to diagnose myocyte injury became more sensitive (electrocardiography, creatine kinase, creatine kinase MB, and now troponin), it was recognized that cardiac injury actually occurred very soon after or even during surgery.

With the advent of highly sensitive and fairly specific troponin assays, it seems that perioperative cardiac injury is extremely common, perhaps occurring in up to 20% of patients (if we include patients at high risk based on traditional criteria). This has led to the newly described entity of "myocardial injury after noncardiac surgery" (MINS). MINS patients, diagnosed by troponin elevations, usually are asymptomatic, and many do not meet criteria for any type of MI. But strikingly, as discussed in this issue of the *Journal* by Horr et al (page 595), simply having a postoperative troponin elevation predicts an increased risk of clinical cardiovascular events and a decreased 30-day survival rate.

Adding postoperative troponin measurement to the usual preoperative screening protocol significantly increases our ability to predict delayed cardiovascular events and mortality. As pointed out by Cohn in his accompanying editorial (page 603), the benefit, if any, of screening low-risk patients remains to be defined. But an even more important issue, as commented upon in both papers, is what to do when an elevated troponin is detected in a postoperative patient who is otherwise doing perfectly well. Given our current knowledge of the pathophysiology of postoperative MI and the still overall low mortality, it seems unreasonable to immediately take all of these patients

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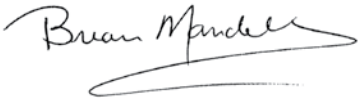
to the catheterization suite. Yet with current knowledge of the prognostic significance of troponin elevation, this can't be ignored. Should all patients receive immediate high-intensity statin therapy, antiplatelet therapy if safe in the specific perioperative setting, and postdischarge physiologic stress studies, or should we "just" take it as a potential high-impact teaching moment and advise patients of their increased cardiovascular risk and offer our usual heart-healthy admonitions?

The confirmed observation that postoperative troponin elevation predicts morbidity and mortality over the subsequent 30 days, and perhaps even longer, has triggered the start of several interventional trials. The results of these will, hopefully, help us to further improve perioperative outcomes.



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