Section Editor: **Richard Prielipp**
SPECIAL ARTICLE

Perioperative Troponin Screening

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Myocardial injury is the most common cause of death during the 30 days after noncardiac surgery. Only 14% of patients who are experiencing a perioperative myocardial infarction will have chest pain, and 65% are entirely clinically silent, which means that they will go undetected without routine troponin screening. Although it is tempting to dismiss asymptomatic troponin elevation, mortality is similar with and without symptoms. Furthermore, mortality at 30 days in patients who have postoperative troponin elevation is a concerning 10%, which represents a 5-fold increase from background risk. Among inpatients ≥45 years of age who are having noncardiac surgery, the number necessary to screen to detect myocardial injury after noncardiac surgery, that would otherwise be missed, is only about 15 patients. Thus, troponin screening seems appropriate for most surgical inpatients who are ≥45 years of age. Potential acute interventions include initiating therapy such as aspirin, statins, and angiotensin-converting enzyme (ACE) inhibitor antihypertensives, along with chronic lifestyle improvements such as smoking cessation, healthful eating, and exercise. (Anesth Analg 2016;123:359–60)

In recent decades, intraoperative mortality has decreased by a factor of 10, even though we now care for much sicker and older patients.[1] Preventable anesthetic-related intraoperative mortality is now so rare that it is hard to quantify.[2] In contrast, postoperative mortality remains substantial. Overall, 30-day postoperative mortality after noncardiac surgery is about 1% in the United States and about 2% among inpatients (outpatients die much less frequently).[3,4] To put this mortality in perspective, if the postoperative period were considered a disease, it would represent the third leading cause of death in the United States.[5] Approximately half of all 30-day postoperative deaths are cardiovascular or consequent to cardiovascular events,—with myocardial ischemia being the most common by far.[6]

Worldwide, 8% of surgical inpatients >45 years of age sustain postoperative myocardial injury as defined by a troponin elevation that is attributable to an ischemic etiology, with only 42% of these events fulfilling the diagnostic criteria of the universal definition of myocardial infarction.[7] Only 14% of patients who are experiencing a perioperative myocardial infarction will have chest pain, and 65% are entirely clinically silent, which means that they will go undetected without routine troponin screening.[6,8,9]

It is tempting to dismiss asymptomatic biomarker elevation as troponitis and assume that it is inconsequential; but this approach would be mistaken because 30-day mortality in patients with elevated postoperative troponin is similar with and without symptoms.[10] The term myocardial injury after noncardiac surgery (MINS) recognizes that troponin elevations without a nonischemic explanation (eg, sepsis and pulmonary embolus) are clinically important, even in patients whose symptoms and signs do not meet the formal definition of a myocardial infarction.[11]

Mortality at 30 days in patients with MINS is a concerning 10%, which represents a 5-fold increase from background risk. Mortality increases exponentially as a function of peak postoperative troponin concentration, ranging from 9% for fourth-generation troponin T (high-sensitive cardiac troponin T) plasma concentrations 0.03 to 0.29 ng/mL to 17% for concentrations ≥0.3 ng/mL (Table). Moreover, it is not just mortality that is increased; a composite of nonfatal cardiac arrest, congestive heart failure, stroke, and death occurred at a rate of 2.4% in patients without MINS and 18.8% among those with MINS, a factor-of-8 increase.[10]

Among inpatients ≥45 years of age who are having noncardiac surgery, the number needed to screen to detect MINS that would otherwise be missed is only about 15 patients, fewer than for tests we conduct routinely for conditions that are far less deadly. Consistent with this logic, per the third universal definition of myocardial infarction guidelines: "routine monitoring of cardiac biomarkers in high-risk patients after major surgery is therefore recommended."[7] In fact, troponin screening should not be restricted to high-risk patients because the incidence of MINS is 8% among a representative cross-section of surgical inpatients selected only for being ≥45 years of age.[11]

Thus, troponin screening seems appropriate for most surgical inpatients ≥45 years of age. Troponin analysis can be included with routine morning blood sampling on the first, second, and third postoperative mornings while patients remain hospitalized. Screening thereafter is probably not necessary because about 75% of postoperative myocardial infarctions occur within 48 hours after surgery[9] and because about 80% of

Table. Relationship Between Peak Postoperative Fourth-Generation Troponin T and 30-Day Mortality and Time to Death

Peak Troponin (ng/mL)	30-Day Mortality (%)	Time to Death (d)
<0.01	1	—
0.02	4	13
0.03–0.29	9	9
≥0.3	17	6

Modified with permission from the Vascular Events In Noncardiac Surgery Patients Cohort Evaluation (VISION) Study Investigators.[10]

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all 30-day mortality occurs during the initial hospitalization.[12] That being said, blood should immediately be sent for troponin analysis in any patients who has cardiovascular symptoms such as chest pain or shortness of breath. Nonischemic causes of troponin elevation include end-stage renal disease, sepsis, and pulmonary embolism[7]; preoperative plasma troponin assays might help clinicians interpret subsequently elevated values.

Postoperative fourth-generation troponin T concentrations ≥0.03 ng/mL in the absence of alternative explanations should prompt a medical or cardiology consult. This recommendation is supported by evidence that suggests that intensification of cardiovascular therapy in patients with elevated postoperative troponin concentrations reduces the risk of subsequent cardiac events by about 40%.[13] Additional work is required to establish the optimal thresholds for non–high-sensitivity troponin I assays and troponin I and T assays.

However, there are no available randomized trial results that suggest specific treatments for MINS. Nonetheless, potential benefits of troponin screening include a cardiology consultation and patients: (1) being informed that they had myocardial injury and are thus at risk for future heart attacks; (2) potentially starting aspirin; (3) being considered for statin and/or ACE inhibitor therapy; (4) having improved hypertension control, as necessary; and (5) taking advantage of a teachable moment[14] to promote lifestyle changes, including smoking cessation, sensible diet, and enhanced exercise.

Too many anesthesiologists still consider our work done when patients arrive safely in the postanesthesia care unit. Although patients rarely die during surgery, postoperative mortality remains high, with myocardial injury being a leading cause. Troponin screening is an opportunity to extend our influence as perioperative physicians into the postoperative period.

DISCLOSURES

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Troponin Is for Diagnosis, Not Screening

To the Editor

In the recent Special Article "Perioperative Troponin Screening,"[1] Sessler and Devereaux opine that "troponin screening seems appropriate for most surgical inpatients 45 years or older." They also suggest that high-sensitive troponin T concentrations 0.03 ng/mL or greater should prompt a medical or cardiology consultation.

Our reading of the current literature suggests that the likely result of screening every postoperative inpatient >45 years of age for high-sensitive troponins would be overtreatment of patients. The 2012 guidelines[2] from the American College of Cardiology state that in cases of elevated troponin in patients with few or no risk factors, acute coronary syndrome is unlikely. Moreover, improvements in outcomes after non-ST–segment elevation myocardial infarction have occurred mostly in patients at moderate-to-high risk, not those without risk factors, and mostly from interventions rather than drug therapy.[3]

Over the past few years, our understanding of perioperative myocardial infarction has evolved from a type I (plaque rupture and thrombosis of coronary artery) to type II (demand ischemia). Now we know that any minor injury or stress to the myocardium may result in release of troponin[4,5] and that low-level postoperative troponin elevations are associated statistically with an increased incidence of noncardiac complications. High-level elevations are associated statistically with an increased incidence of cardiac complications.[6,7] We also recognize that intensification of therapy with angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, statins, β-blockers, nitrates, and/or aspirin reduced complications in high-risk vascular surgery patients with postoperative troponin elevation in an observational study, not a prospective one.[8] Until we have stronger confirmatory evidence, would it not make more sense to limit screening to patients with risk factors for coronary disease undergoing operations that associate with increased risk of major adverse cardiac events?

We also wonder whether screening troponin in the preoperative period would offer the greatest potential to improve outcomes. As has been shown by Maile et al,[9] increased preoperative troponin concentrations are associated with increased morbidity and mortality in high-risk patients; thus, preoperative screening may be reasonable selectively in high-risk patients per the guidelines from the American College of Cardiology. Finally, no discussion of screening can ignore costs. Can we justify the added expense of screening to those who will pay for the testing?

Drs. Sessler and Devereaux regret that too many anesthesiologists consider their work done when patients arrive safely in postanesthesia care unit. We agree; however, we find their recommendations for a "medical or cardiology consultation" equally unacceptable from those who regard themselves as "perioperative physicians." What would we ask the internist or cardiologist to do? Even if we assume that every elevation of troponin T diagnoses a "non ST segment elevation acute coronary syndrome," the standard initial treatment of this condition includes dual antiplatelet therapy and anticoagulation. Such treatment in an immediate postsurgical patient would usually not be feasible.[10] In a postoperative patient with no symptoms or evidence on electrocardiogram, would we not maintain our goal-directed treatment plan whether or not there was troponin-leak?

"Medical overtreatment" commonly has been criticized in the news media.[11] To maximize patient safety and maintain the integrity of our profession, we should base our decisions on evidence and clinical practice guidelines when they are applicable. We would not favor routinely measuring troponin T in all patients who are 45 years or older who are recovering from surgery. The available evidence and published guidelines do not support this practice. We would emphasize that it is time that we sorted out the ambiguity regarding perioperative myocardial infarction versus myocardial injury, and we need to redefine type II myocardial infarction as has been suggested by Nagele.[12]

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