

Postoperative risk stratification for cardiovascular complications

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Introduction

Current evidence suggests that myocardial oxygen supply-demand imbalance predominates in the early postoperative period. It is likely that flow stagnation and thrombus formation is an important pathway in the development of a perioperative myocardial infarction, in addition to the more commonly recognised role of perioperative tachycardia.¹ Perioperative myocardial infarction is most likely to occur in patients with significant coronary artery stenoses. The impetus to continue to risk stratify into the postoperative period is driven by the fact that the independent predictors of cardiovascular risk change from the preoperative to the postoperative period. Adding independent intraoperative risk factors to preoperative risk prediction models reduces the significance of traditional preoperative risk factors. Only a history of stroke²⁻⁴ and renal dysfunction³⁻⁶ appear to remain as independent predictors, once the intraoperative predictors were added to the models.

Intraoperative risk factors which appear to modify perioperative cardiac adverse events include the type of operation,⁷ and markers of operative severity (duration of surgery and blood loss).^{3,7,8} Physiological variables are desirable indicators of perioperative risk, and intraoperative haemodynamics may also be important predictors, although no models have identified physiological variables as independent predictors of adverse cardiac events.

Postoperative risk stratification allows for appropriate intervention in patients who are at increased risk. If it is carried out timeously, it provides a therapeutic window in which outcome can be altered. It has been shown that simple physiological monitoring, including respiratory rate, heart rate, systolic blood pressure, and level of consciousness, are important predictors of mortality in in-hospital patients.⁹ Physiological variables have been used in early warning

scoring systems to identify patients at increased risk of mortality, requiring early intensive care unit admission.¹⁰⁻¹²

Traditionally, when risk stratifying patients, an attempt is made to identify the vulnerable patient. However, in the postoperative period, as cardiovascular complications may present within the first 24 hours following surgery, there is evidence to suggest that the initial approach should be to exclude the occurrence of a cardiovascular complication. Only once this is done, identification of the vulnerable postoperative patient can continue. Troponin release represents the final common pathway of myocardial injury, following non-cardiac surgery. In addition, troponin release has been associated with both short-term and intermediate-term mortality.^{13,14} There is overwhelming evidence to suggest that if patients are at risk of cardiovascular events following non-cardiac surgery, the first investigation that should be considered is postoperative troponin surveillance.

What are the options for postoperative risk stratification if a patient is troponin-negative?

The first approach should be to identify patients whose risk is deemed to have increased during the intraoperative period. Physiological variables may identify patients at increased risk of mortality, and requiring early intensive care unit admission.¹⁰⁻¹² Data suggest that the combination of a physiological score, and the severity of the surgery, would be preferable.^{6,15} Strong modifiers of postoperative risk include postoperative haemoglobin and transfusion requirements,¹⁶ postoperative brain natriuretic peptide (BNP),¹⁷ and medication withdrawal.^{18,19} It is likely that postoperative BNP is a good biomarker for postoperative risk stratification in patients who are troponin-negative.²⁰

References

1. Biccadd BM, Rodseth RN. The pathophysiology of perioperative myocardial infarction. *Anaesthesia*. 2010;65(7):733-741.
2. Kheterpal S, O'Reilly M, Englesbe MJ, et al. Preoperative and intraoperative predictors of cardiac adverse events after general, vascular, and urological surgery. *Anesthesiology*. 2009;110(1):58-66.
3. Davenport DL, Ferraris VA, Hosokawa P, et al. Multivariable predictors of postoperative cardiac adverse events after general and vascular surgery: results from the patient safety in surgery study. *J Am Coll Surg*. 2007;204(6):1199-1210.
4. Devereaux PJ, Xavier D, Pogue J, et al. Characteristics and short-term prognosis of perioperative myocardial infarction in patients undergoing noncardiac surgery: a cohort study. *Ann Intern Med*. 2011;154(8):523-528.
5. D'Ayala M, Huzar T, Briggs W, et al. Blood transfusion and its effect on the clinical outcomes of patients undergoing major lower extremity amputation. *Ann Vasc Surg*. 2010;24(4):468-473.
6. Ryan D, McGreal G. Why routine intensive care unit admission after elective open infrarenal abdominal aortic aneurysm repair is no longer an evidence based practice. *Surgeon*. 2010;8(6):297-302.
7. Boersma E, Kertai MD, Schouten O, et al. Perioperative cardiovascular mortality in noncardiac surgery: validation of the Lee cardiac risk index. *Am J Med*. 2005;118(10):1134-1141.
8. Kheterpal S, O'Reilly M, Englesbe J, et al. Preoperative and intraoperative predictors of cardiac adverse events after general, vascular, and urological surgery. *Anesthesiology*. 2009;110(1):58-66.
9. Goldhill DR, McNarry AF. Physiological abnormalities in early warning scores are related to mortality in adult inpatients. *Br J Anaesth*. 2004;92(6):882-884.
10. Goldhill DR, McNarry AF, Mandersloot G, McGinley A. A physiologically-based early warning score for ward patients: the association between score and outcome. *Anaesthesia*. 2005;60(6):547-53.
11. Cuthbertson BH, Boroujerdi M, McKie L, et al. Can physiological variables and early warning scoring systems allow early recognition of the deteriorating surgical patient? *Crit Care Med*. 2007;35(2):402-409.
12. Duckitt RW, Buxton-Thomas R, Walker J, et al. Worthing physiological scoring system: derivation and validation of a physiological early-warning system for medical admissions. An observational, population-based single-centre study. *Br J Anaesth*. 2007;98(6):769-774.
13. Redfern G, Rodseth RN, Biccadd BM. Outcomes in vascular surgical patients with isolated postoperative troponin leak: a meta-analysis. *Anaesthesia*. 2011;66(7):604-610.
14. Levy M, Heels-Ansdell D, Hiralal R, et al. Prognostic value of troponin and creatine kinase muscle and brain isoenzyme measurement after noncardiac surgery: a systematic review and meta-analysis. *Anesthesiology*. 2011;114(4):796-806.
15. Biccadd BM, Pooran RR. Validation of a model to predict all-cause in-hospital mortality in vascular surgical patients. *Cardiovasc J Afr*. 2008;19(6):303-308.
16. Bursi F, Barbieri A, Politi L, et al. Perioperative red blood cell transfusion and outcome in stable patients after elective major vascular surgery. *Eur J Vasc Endovasc Surg*. 2009;37(3):311-318.
17. Goei D, van Kuijk JP, Flu WJ, et al. Usefulness of repeated N-terminal pro-B-type natriuretic peptide measurements as incremental predictor for long-term cardiovascular outcome after vascular surgery. *Am J Cardiol*. 2011;107(4):609-614.
18. Shammash JB, Trost JC, Gold JM, et al. Perioperative beta-blocker withdrawal and mortality in vascular surgical patients. *Am Heart J*. 2001;141(1):148-153.
19. Schouten O, Hoeks SE, Welten GM, et al. Effect of statin withdrawal on frequency of cardiac events after vascular surgery. *Am J Cardiol*. 2007;100(2):316-320.
20. Mahla E, Baumann A, Rehak P, et al. N-terminal pro-brain natriuretic peptide identifies patients at high risk for adverse cardiac outcome after vascular surgery. *Anesthesiology*. 2007;106(6):1088-1095.