The American College of Cardiology/American Heart Association (ACC/AHA) has incorporated the Revised Cardiac Risk Index (RCRI) into the algorithm for Perioperative cardiovascular evaluation and care for noncardiac surgery (Fleisher et al, 2007). However, it does not successfully discriminate higher from lower risk patients. In comparison, biomarkers can accurately stratify vascular surgical patients.

There are multiple pathways responsible for perioperative major adverse cardiovascular events (MACE) (Devereaux et al, 2005). Brain natriuretic peptide (BNP) elevation identifies a vulnerable ventricle which is at risk due to either excessive pressure, or volume loading, or myocardial ischaemia (Rodseth, 2009; Struthers and Lang, 2007). Troponin elevation only reflects myocyte necrosis.

Preoperative risk stratification

An individual patient data meta-analysis shows that preoperative BNP is a significantly better predictor of postoperative MACE than the RCRI (Rodseth et al, J Am Coll Cardiol submission). This meta-analysis suggests that the RCRI is essentially redundant in vascular surgical patients.

Data from Inkosi Albert Luthuli Central Hospital show that preoperative BNP significantly improves risk reclassification for patients with and without MACE. In comparison, preoperative troponins only significantly improve reclassification of patients who do not sustain MACE, and not those who had MACE (Biccard et al, Anesth Analg submission). Therefore, preoperative risk stratification based on the BNP alone may be clinically viable and preferable to preoperative troponin or RCRI risk stratification.

Postoperative risk stratification

Troponin elevation occurs predominantly postoperatively (Howell et al, 2006). A study of aortic surgical patients showed that postoperative troponin elevation at 48 hours had a positive likelihood ratio of 10, and a negative likelihood ratio of 0.08, for postoperative myocardial infarction (Le Manach et al, 2005), which is statistically very impressive. A further study showed that postoperative BNP was the most powerful independent predictor of postoperative MACE (Mahla et al, 2007).

However, these studies did not compare BNP and postoperative troponins. Further data from our MRC study suggest that troponin elevation (above the upper reference limit) within the first 24 hours postoperatively is a significantly more important predictor of MACE than postoperative BNP elevation (Biccard, Anesth Analg submission).

In conclusion, preoperative BNP is probably the best means of preoperative risk stratification for vascular surgical patients, while postoperative troponin elevation is a significantly better predictor of MACE than BNP. However, postoperative BNP may still risk stratify patients who do sustain early troponin elevation postoperatively.