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Not All Perioperative Myocardial Infarctions Can Be Prevented with Preoperative Revascularization

CURRENT universal definitions classify myocardial infarction (MI) into five types.[1] In general, perioperative MI (PMIs) are of type I (plaque rupture) or type II (prolonged supply-demand imbalance) variety.[2] Increased catecholamines, hemodynamic instability, inflammation, and coronary vasoconstriction during and after surgery can lead to rupture or erosion of a potentially unstable coronary plaque, often referred to as a "vulnerable" plaque, resulting in acute coronary thrombosis and PMI.[3] However, the relative proportion of plaque rupture, demand ischemia, or their combination as the etiology of PMI is unknown. In this issue of ANESTHESIOLOGY, Galal *et al.*[4] demonstrated that although preoperative dobutamine stress echocardiography can predict patients who are at risk for PMI, it could not predict the location of those PMI in 54 consecutive patients undergoing major vascular surgery.

How can we reconcile this surprising finding? Only 10% of patients undergoing vascular surgery were shown to have pristine coronary anatomy.[5] The majority of them have multivessel disease, and they can be identified as at risk for PMI by preoperative dobutamine stress echocardiography. It was previously shown that 66% of MI occur in plaques with less than a 50% luminal stenosis[6] and that the MI-related coronary vascular territory is frequently not related to the territory with the most severe coronary stenosis as seen by coronary angiography.[7] Moreover, statin therapy markedly reduces the risk of MI[8] with minimal effect on the severity of coronary luminal stenosis.[9] This disconnection between the severity of anatomic obstruction and the MI risk is one of the main pieces of evidence that plaque rupture depends on its composition rather than on its size.[10] Although preoperative dobutamine stress echocardiography and other functional tests are good for the diagnosis of significant coronary artery obstruction and the identification of patients at risk for MI,[11] they may fail to identify the myocardial territory at risk from rupture of a nonobstructive coronary artery plaque. If this etiology contributes significantly to PMI, preoperative revascularization approaches based on dobutamine stress echocardiography and coronary angiography will not protect against all PMI. This may in part explain why it has been difficult to show short-term benefit (up to 3 yr) of preoperative revascularization in reduction of PMI or survival[12,13] but may show better long-term survival.[14] These findings are consistent with the current American Heart Association/American College of Cardiology consensus recommendations that preoperative revascularization is warranted if it would benefit patients in the long-term irrespective of the planned surgery.[15]

Galal *et al.* also found that the new wall motion abnormalities (WMA) detected by intraoperative transesophageal echocardiography (TEE) had 100% positive predictive value and better agreement with the location of PMI compared with preoperative dobutamine stress echocardiography. Deterioration of regional WMA correlates better with in-hospital[16] and long-term adverse cardiac outcomes[17] after cardiac surgery. This led to some positive findings when monoplane TEE was used as an ischemia monitor in smaller trials in noncardiac surgery. However, a larger well-designed trial[18] concluded that TEE findings were sensitive, nonspecific, and did not correlate with postoperative MI. Subsequently, the presence of sustained WMA (3 h) after aortic cross clamp was shown to predict PMI.[19] Eisenberg *et al.*[20] concluded that in 332 patients undergoing vascular and abdominal surgery, TEE offered little incremental value compared with two-lead electrocardiogram monitoring, even though the new WMA by intraoperative TEE had a 2.2 relative risk of predicting postoperative outcomes. It is to be noted that the relevant ischemic outcome in all these studies was seen in very few patients, and there was no attempt to correlate PMI location with intraoperative WMA. Thus, unlike in cardiac surgical patients, routine use of TEE as an intraoperative ischemia monitor in high-risk noncardiac surgery did not gain widespread acceptance due to the paucity of literature, the lack of studies with multiplane TEE, nonselective target population studied, and concerns about personnel availability, cost, and safety. This is reflected in the current American Society of Anesthesiologists/Society of Cardiovascular Anesthesiologists recommendation that TEE can be used in acute persistent hemodynamically unstable and life threatening situations during noncardiac surgery (Class IIa, Level C).

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As TEE interpretation has improved with omniplane imaging, three-dimensional TEE, and strain rate imaging, it is now primed to be a cutting-edge technology for early intraoperative ischemia detection. The findings of Galal *et al.* provide a compelling argument for further exploration of intraoperative TEE in high-risk patients such as the ones with preoperative positive dobutamine stress echocardiography (a high pretest probability increases the posttest probability). Galal *et al.* performed protocol-based TEE ischemia and WMA monitoring and analyzed the recordings in the echo-laboratory afterward. They did not study the effect of intraoperative clinical interventions in response to TEE-detected ischemia on PMI. It will be important to study the effect of comprehensive patient management using preoperative risk stratification, early intraoperative detection of ischemia by TEE, and targeted intra- and postoperative interventions and management of PMI with multimodal therapy in an intensive/high-dependency setting.

On the basis of the findings by Galal *et al.*, the authors suggest that preoperative revascularization will often be ineffective because preoperative myocardium "at risk" identified differs from the actual location of PMI. This is an interesting and a provocative finding; however, there are several caveats to be considered. The composite outcome was seen in just 15 patients, and the agreement of location of PMI, a major focus of this study, is based only on six patients. A detailed description of the characteristics of these six patients could give more insights. It would be interesting to see whether the disagreement in location of PMI was in major *versus* minor coronary arterial distribution. For example, the disagreement between inferior and anterior wall is different from the disagreement between anterior and anteroseptal walls. Whether all PMIs were seen in patients with aortic surgery with cross-clamping is unknown. It may very well be that the aortic cross-clamp is a more severe or a "better" stress test than the dobutamine stress echocardiography. It is to be noted that perioperative aspirin and statin therapy that can stabilize the plaques was achieved only in two thirds of this study population. Other limitations include lack of description of the duration and severity of new WMA, relationship to loading conditions, and correlation to intraoperative electrocardiogram evidence of ischemia. Patients with severe valvular disease, decreased ejection fraction, and female gender were not investigated in this study; therefore, the findings may not be generalized to these populations.

PMI remains a significant cause of morbidity, mortality, and increased healthcare costs. The mechanisms of PMI need to be further explored and well understood before an effective intervention strategy can be established. The current strategies of revascularization and optimal medical therapy to reduce the incidence of PMI have met with some success, but PMI is still a common occurrence. Alternative surgical approaches, such as minimally invasive techniques, is an attractive option but it is not suitable for all procedures or patients. The current challenge is to establish a comprehensive strategy with long-term β-adrenergic blockade, statin therapy, and targeted preoperative revascularization, keeping in mind the added risk of coronary stent thrombosis or dual antiplatelet therapy. The study by Galal *et al.* suggests that intraoperative TEE may be a sensitive method for identification of patients at risk for PMI, should these measures fail and allow for early aggressive treatment of these patients.

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