Apostolakis artery bypass grafting
eComment: Perioperative myocardial infarction following coronary myocardial injury may be associated with acute loss of bypass grafts cardiac protection, and intraoperative defibrillation, while postoperative mechanisms have been proposed to explain myocardial injury after CABG. Intra-
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Factors for perioperative MI included age, left main coronary artery disease and three-vessel disease, impaired left ventricular function, unstable angina, recent MI, and emergent operations [3]. According to the PREVENT IV study, both 30-day and two-year clinical outcomes were worse in patients suffering perioperative MI, as they had longer postoperative ventilation times and intensive care unit and hospital stays. Although rates of angiographic vein graft failure were higher in patients with a perioperative MI, one-third of patients with perioperative MI had patent vein grafts at one-year, which suggests that a substantial portion of perioperative MI was not caused by early vein graft failure and that global or regional myocardial ischemia, possibly related to CABG or worse coronary anatomy, may have an important role [3].

Finally, reperfusion injury occurring with restoration of blood flow to ischemic tissue is associated with myocardial cell death and apoptosis, and microvascular injury. Three to 20% of patients experience MI associated with reperfusion injury after CABG [5]. Adenosine, being a powerful inducer of ischemic preconditioning, has been shown to improve postischemic ventric-
ular function, reduce neutrophil accumulation/activation and reduce further myocardial necrosis. Mangano et al. [5], reported that treatment with acadesine, an adenosine regulating agent, significantly reduced mortality by 4.3-fold, with the principal benefit occurring over the first 30 days after MI. Acadesine is the first therapy proven to reduce the severity of acute postreperfusion MI, substantially reducing the risk of dying over the two years after infarction.

References
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This well-designed study [1] concerning perioperative myocardial infarction (MI) and graft patency, offers us the opportunity to add some data regarding this challenging complication. Coronary artery bypass grafting (CABG) is of considerable benefit for those in need of revascularization; however, it may be associated with significant perioperative and postoperative myocardial damage and necrosis, which may occur in varying degrees. Multiple mecha-
nisms have been proposed to explain myocardial injury after CABG. Intra-
operative injury may result from cardiac manipulation, inadequate myo-
cardial protection, and intraoperative defibrillation, while postoperative myocardial injury may be associated with acute loss of bypass grafts [2]. The incidence of perioperative MI varies considerably, from 3% to 30%, because of different diagnostic criteria and variable patient populations [3]. However, troponin values more than five times the 99th percentile of the

normal reference range during the first 72 hours following CABG, when associated with the appearance of new pathological Q-waves or new left bundle-branch block (LBBB), or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium, should be considered as diagnostic of a CABG related MI [4]. The PREVENT IV study identified such intraoperative risk factors for perioperative MI as prolonged cardiopulmonary bypass or aortic cross-clamp times, perioperative myocardial ischemia, and inadequate revascularization. Moreover, patients with perioperative MI also had longer surgery durations, although it was unclear whether this longer duration was a cause of or caused by perioperative myocardial ischemia. Other well-established risk factors for perioperative MI included age, left main coronary artery disease and three-vessel disease, impaired left ventricular function, unstable angina, recent MI, and emergent operations [3]. According to the PREVENT IV study, both 30-day and two-year clinical outcomes were worse in patients suffering perioperative MI, as they had longer postoperative ventilation times and intensive care unit and hospital stays. Although rates of angiographic vein graft failure were higher in patients with a perioperative MI, one-third of patients with perioperative MI had patent vein grafts at one-year, which suggests that a substantial portion of perioperative MI was not caused by early vein graft failure and that global or regional myocardial ischemia, possibly related to CABG or worse coronary anatomy, may have an important role [3].

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