A thorough preoperative evaluation is necessary in those patients who have an increased risk of myocardial ischemia or infarction in the postoperative period. Comorbidities, exercise tolerance, and the type and length of surgery need to be taken into consideration and appropriate measures taken to prevent complications. It also should be noted that even when all necessary preventative measures are taken, some patients will experience clinical evidence of myocardial ischemia or infarction postoperatively.

**Case discussion**

An 83-year-old white male underwent extensive reconstructive surgery for squamous cell carcinoma of the oral mucosa. Postoperatively, he was found to have venous congestion caused by clot formation within the new skin flap. He was brought back to the operating room on the first postoperative day for a second procedure, an excision and drainage of a left facial/neck hematoma. The patient had been in surgery for approximately 10 hours the day before. He had been stable throughout the case and tolerated the previous procedure well. The patient's medical history included hypertension, coronary artery disease (5-vessel coronary artery bypass graft in 1998), chronic pancreatitis, type II diabetes mellitus, and previous tobacco use. Home medications included metoprolol, hydrochlorothiazide/triamterene, rabeprazole, atorvastatin, pancreatic enzymes, furosemide, budesonide, and sublingual nitroglycerin, as needed.

Preoperative vital signs were as follows: blood pressure, 115/66 mm Hg; pulse, 56 beats per minute; and Spo₂, 98% on 0.30 FiO₂ via tracheotomy collar. The patient had no recent incidents of chest pain or cardiac-related shortness of breath. An electrocardiogram (ECG) completed before the patient's first procedure revealed sinus bradycardia with first degree atrioventricular block and occasional premature ventricular contractions, right bundle branch block, and an inferior infarction of undetermined age. Before the second procedure, the patient's hemoglobin was 9.1 g/dL and the hematocrit was 25.6%. The chemistry and coagulation results also were within normal limits. The patient had an 18-gauge peripheral intravenous catheter and a right radial arterial line that had been placed during the first procedure. Before being transported to the operating room, 1 mg of midazolam was given for anxiolysis. Once in the room, the patient was transferred to the operating table and all standard monitors were applied. The patient was preoxygenated with 1.0 FiO₂ via tracheotomy. Once adequately oxygenated, 80 mg of propofol was given intravenously and 0.6% isoflurane was turned on with 50% nitrous oxide. Muscle relaxation throughout the case was maintained with vecuronium.

Vital signs were maintained within 20% of the patient's baseline values throughout the case. One episode of hypotension after induction was treated with 60 µg of phenylephrine. The patient received a total of 800 mL of lactated Ringer's solution and lost approximately 125 mL of blood. At the completion of surgery the patient was fully reversed with 5 µg of neostigmine and 0.8 mg of glycopyrrolate and resumed spontaneous respiration. The patient exhibited some mild tachycardia with normotension during emergence. Because the blood pressure was adequate, the patient was treated promptly with 20 µg of esmolol. Esmolol was chosen over labetolol because of its selectivity toward beta receptors and fast onset of action. The goal was to treat the heart rate without affecting the patient's blood pressure significantly. When the patient exhibited an adequate respiratory effort and pattern, he was transported to the recovery room on a tracheotomy collar with 10 L/min of oxygen.

In the recovery room, humidified oxygen was
administered and all standard monitors were placed. The patient was noted to be tachycardic with a heart rate of 110. This was treated subsequently with two 20-mg boluses of intravenous esmolol. Marked ST-elevation in lead II and T-wave inversion in lead V5 were noted. The patient appeared comfortable and he denied having any chest pain. A 12-lead ECG was obtained immediately, a nitroglycerin drip was started, and laboratory tests were drawn to check for elevated cardiac enzymes and troponin levels. Cardiology was consulted and once stable, the patient was transported to the cardiac intensive care unit for monitoring.

Discussion
An estimated 30 million patients need surgery annually in the United States and approximately 30% of these patients have coronary artery disease or are at high risk of developing it. Each year, 1% to 5% of patients undergoing noncardiac surgery experience cardiovascular complications postoperatively. It has been reported that high-risk populations, such as diabetics, obese patients, and those undergoing peripheral vascular surgery, have a 34% incidence of perioperative myocardial infarction with mortality rates reaching 25% to 40%. Perioperative myocardial ischemia is the most likely cause of postoperative morbidity and mortality. Risk factors for the development of coronary artery disease include male gender, increasing age, hypercholesterolemia, hypertension, smoking, diabetes mellitus, obesity, and family history.

Coronary artery circulation usually supplies enough blood flow to meet the demands of the myocardium; however, complications such as anemia, hypoxemia, hypcapnia, vasospasm, and tachycardia can adversely affect oxygen supply to the heart. An imbalance between oxygen supply to the myocardium and demand can lead to ischemia. When this imbalance becomes severe, dysrhythmias, heart failure, and myocardial infarction can occur. Persistent tachycardia not only increases work of the myocardium, it also decreases the amount of time the heart spends in diastole. This results in less filling of the coronary arteries since coronary blood flow primarily occurs during diastole. The combination of the two can greatly decrease oxygen supply to the heart and cause ischemia. Vasoconstriction secondary to ischemia can further decrease coronary blood flow. Therefore, it is important to avoid extremes in heart rate and blood pressure during induction, as well as perioperatively, because wide fluctuations in hemodynamics can cause a significant increase in myocardial oxygen demand.

Landesberg et al studied stress-induced ischemia and myocardial infarction in the postoperative period. From the 185 study participants, it was noted that an approximate 18 beats per minute increase in baseline heart rates may have resulted in postoperative ischemia or infarction. It also was observed that 68% of the longest ischemic events started 30 minutes before or 60 minutes following the completion of surgery. The blood pressures during the onset of ischemia were noted to be within the patients’ preoperative ranges.

Several medical interventions may decrease cardiac complications following noncardiac surgeries. The most commonly used treatment, the use of beta blocker in the perioperative period, has been studied extensively. Beta blockers are thought to have a cardioprotective effect due to reduction in heart rate, wall tension, and contractility with a subsequent decrease in myocardial ischemia. Lindauer et al concluded that avoiding beta blockers in patients with known coronary artery disease may actually be detrimental. Of the 72 patients examined, all of whom experienced postoperative myocardial infarctions, 81% appeared to be appropriate candidates for beta blocker therapy, but only 30% were actually treated with some type of beta blocker. The patient described in the present case study, however, was receiving metoprolol (chronically) and esmolol, which was continued into the perioperative period. Use of α2 receptor agonists, calcium channel blockers, nitrates, and lipid-lowering agents also has been implicated. However, conclusions from studies investigating those other agents have had somewhat inconsistent results. For example, Stevens et al suggested that α2 agonists, such as clonidine and mivazerol, may be as effective as a beta blocker for perioperative prophylaxis.

Common signs of myocardial ischemia include angina, dyspnea, and ECG changes. The patient did not complain of chest pain or shortness of breath. In fact, the patient appeared to have a regular respiratory pattern and stated that he was comfortable. Diabetic patients and patients who have previously experienced a myocardial infarction have been known to have a greater chance of experiencing silent myocardial ischemia. However, there are other explanations as to why patients may not experience chest pain postoperatively. Surgical pain may be indistinguishable from angina, for instance. Also, patients are almost always on narcotics that can diminish both surgical and chest pain.

The most convenient method in detecting ischemic changes is the use of ECG monitoring. Ischemia is diagnosed primarily by noting changes in the ST segment. Depressions or elevations of at least 1 mm in the ST segment are indicative of ischemia. Other changes such as T-wave inversion or R-wave changes also can indicate...
ischemia, but can be caused by electrolyte imbalances. Once ECG changes are seen, aggressive treatment is necessary to avoid permanent injury to the heart.

The most sensitive indicator for myocardial ischemia, however, is use of transesophageal echocardiography (TEE). The TEE also is used to examine valvular dysfunction. According to Morgan et al, wall motion abnormalities are a more precise indicator of ischemia. Animal studies suggest that when coronary blood flow is diminished, regional wall abnormalities may occur before changes in an ECG. Not all wall abnormalities are consistent with ischemia; however, as these changes can be attributed to changes in heart rate, blood volume, and vascular resistance.

Tachycardia induced by pain should be treated with narcotics if the patient’s respiratory effort is not already compromised. Persistent elevated heart rate that is not caused by pain should be treated with a beta blocker. In this case study, esmolol was used. The patient was already being treated with metoprolol prior to having any surgery. Hypertension can be treated with a direct-acting vasodilator such as nitroprusside or nitroglycerin. Nitroprusside acts on resistance vessels and can therefore cause a dramatic drop in systemic blood pressure. Because the patient was normotensive (blood pressure 100/60 mm Hg) in the recovery room, nitroprusside was avoided and he was given intravenous nitroglycerin. Since nitroglycerin acts on capacitance vessels, it is less likely to cause significant decreases in systemic blood pressure and coronary perfusion pressure. Nitroglycerin also causes decreases in preload that can facilitate subendocardial blood flow. Additionally, the dilation of coronary vessels caused by nitroglycerin improves oxygen supply to the myocardium. Hypotension can be treated with ephedrine or phenylephrine in conjunction with adequate fluid resuscitation. Inotropic drugs, such as dopamine and dobutamine, can be considered in treatment of hypotension; however, increasing contractility causes an increase in myocardial oxygen demand. Tachycardia also is a common side effect of positive inotropic drugs.

On postoperative evaluation, the patient appeared to be in good condition. Vital signs were stable and he appeared to be comfortable. In the cardiac intensive care unit, the patient’s ECG no longer exhibited any ST abnormality or T-wave inversion. Serial cardiac enzymes and troponin levels that had been drawn from the recovery room until 24 hours after the ischemic event were not elevated, indicating that the patient had not suffered an acute myocardial infarction postoperatively. Cardiac enzymes and troponin levels were continued until 72 hours postoperatively, but the levels never became elevated. An echocardiogram completed during the patient’s stay in the unit showed normal left ventricular function and an ejection fraction of 55%. The patient was transferred from intensive care the next day to a monitored floor bed and was subsequently discharged home 1 week later without incident.

REFERENCES

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