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72 y/o is scheduled for right hemicolecotomy for colon cancer. Past history reveals a myocardial infarction (MI) 5 months ago and placement of a right coronary artery stent. He also has diabetes mellitus type 2. He is treated with atenolol, diltiazem, glyburide, losartan and an occasional sublingual nitroglycerin (NTG).

Discussion Questions:

What are the major preoperative predictors for perioperative cardiac morbidity?

Perioperative myocardial infarction (PMI) is one of the most important predictors of short and long-term mortality and morbidity associated with noncardiac surgery. Therefore, the prevention of PMI relates to improvement in the postoperative outcome. Numerous risk indices and predictors for PCM have been published over the last 30 years.

In 1999, Lee et al. revised the Goldman Cardiac Risk Index, utilizing six independent predictors of cardiac risk:
- High-risk type of surgery
- History of ischemic heart disease
- History of congestive heart failure (CHF)
- History of cerebrovascular disease
- Preoperative treatment of diabetes mellitus with insulin
- Preoperative serum creatinine greater than 2.0 mg per dL

They concluded that the rate of major cardiac complications (myocardial infarction [MI], pulmonary edema, ventricular fibrillation, and primary cardiac arrest) with 0, 1, 2, and more than or equal to 3 predictors were 0.5%, 1.3%, 4%, and 9%, respectively.

According to the 2002 American College of Cardiology (ACC) and American Heart Association (AHA) guideline update on perioperative cardiovascular evaluation (ACC/AHA guidelines), clinical predictors of PCM are categorized into major, intermediate, and minor factors as follows:
Major predictors
- Unstable coronary syndromes
- Acute (MI ~7 days before examination) or recent MI (> 7 days but ~ 1 month) with evidence of important ischemic risk by clinical symptoms or noninvasive study. Notice that the traditional 3 and 6 months interval had been avoided,
- Unstable or severe angina (Canadian class III: Marked limitation of ordinary physical activity. Walking one to two blocks on the level and climbing one flight of stairs in normal conditions and at normal pace and class IV: Inability to carry on any physical activity without discomfort-anginal syndrome may be present at rest).
- Decompensated heart failure
- Significant arrhythmias
- High-grade atrioventricular block
- Symptomatic ventricular arrhythmias in the presence of underlying heart disease
- Supraventricular arrhythmias with uncontrolled ventricular rate
- Severe valvular disease

Intermediate predictors
- Mild angina pectoris (Canadian class I: Ordinary physical activity, such as walking and climbing stairs, does not cause angina; angina is with strenuous or rapid or prolonged exertion at work or recreation. Class II: Slight limitations to ordinary activity. Walking or climbing stairs rapidly; walking uphill; walking or stair climbing after meals, or in cold, or in wind, or under emotional stress, or only during the few hours after awakening. Walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace and in normal conditions.)
- Previous MI by history or pathologic Q waves
- Compensated or prior heart failure
- Diabetes mellitus (particularly insulin-dependent)
- Renal insufficiency

Minor predictors
- Advanced age
- Abnormal electrocardiogram (ECG) Oeft ventricular [LV] hypertrophy, left bundle branch block [LBBB], ST-T abnormalities
- Rhythm other than sinus (e.g. atrial fibrillation)
- Low functional capacity (e.g., inability to climb one flight of stairs while
What are the determinants of myocardial oxygen demand? How are they measured clinically? What factors determine myocardial oxygen supply?

The three major determinants are myocardial left ventricular (LV) wall tension (LV preload and afterload), cardiac contractility and heart rate.

The factors for oxygen supply include cardiac output, arterial oxygen content, heart rate, aortic diastolic pressure, left ventricular (LV) end-diastolic pressure, patency of coronary arteries and coronary vascular tone.

What is the incidence of perioperative reinfarction for noncardiac surgery?

Previously, risk assessment for noncardiac surgery of patients with coronary artery disease (CAD) was based upon the time interval between the myocardial infarction (MI) and surgery. However, with improvements in perioperative care, this difference has become blurred. The importance of the timing of the MI in relation to the proposed surgical procedure may no longer be valid in the era of thrombolitics, angioplasty, and risk
stratification. Although many patients with a history of a MI may continue to have myocardium at risk, others may not. If a stress test does not indicate residual myocardium at risk, the likelihood of reinfarction is low. There is no data available to quote a perioperative reinfarction rate related to the age of the MI; despite this, statements contained in the current American Heart Association! American College of Cardiology (AHA/ACC) guidelines for perioperative cardiovascular evaluation confers a higher risk if the MI is at least 1 month old.

Landesberg states that mortality after PMI is 10% to 15%, similar to the in-hospital mortality after nonsurgical non-Q-wave infarction. This is in contrast to older data showing a higher (50%) mortality after PMI.


Based on his MI, would you recommend that the surgery be postponed for a certain period of time?

The arbitrary delaying of a surgical procedure is not supported by American College of Cardiology/American Heart Association (ACC/AHA) guidelines. According to these guidelines an acute MI (> 7 days before the preoperative evaluation) or recent MI (> 7 days but < 1 month) with evidence of important ischemic risk by clinical symptoms or noninvasive study is a major predictor of perioperative cardiac morbidity (PCM). Therefore, the separation of the interval of MI to operation into the traditional 3- and 6-month intervals has been avoided. Current management of MI provides for risk
stratification during convalescence period. If a recent stress test does not indicate residual myocardium at risk, the likelihood of reinfarction after noncardiac surgery is low. Although there are no adequate clinical trials on which to base firm recommendations, it appears reasonable to wait 4 to 6 weeks after MI to perform elective surgery, due to the decrease in incidence of fatal arrhythmias and ventricular rupture after this period of time.


What is the role of the exercise or pharmacologic stress test for this patient?

The aim of functional cardiovascular testing is to elicit evidence of CAD by subjecting the heart to physiologic stress. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines suggest that the best test for preoperative cardiac risk stratification is an exercise ECG. That recommendation is based on all but the highest-risk patients. As with clinical risk assessment, noninvasive diagnostic testing must be used judiciously. Effective testing should be accurate, add useful information to the overall risk assessment, and avoid unnecessary delays in surgery. Noninvasive tests of myocardial perfusion are classified by the type of stress applied to elicit transient and reversible ischemia or by the mode of detecting the ischemic area. Stress can be applied by exercise (e.g., treadmill, sitting or supine bicycle, or handgrip), by a pharmacologic agent that increases chronotropy and inotropy (e.g., dobutamine, atropine), or by a pharmacologic agent that can cause redistribution of coronary blood flow (e.g., dipyridamole, adenosine). A significant fraction of the high-risk population cannot exercise to an adequate level and will require pharmacologic stress testing. During a stress test, an ischemic event might be suggested or detected by the patient’s reporting of symptoms, appropriate ECG changes (horizontal or down-sloping ST-segment depression of 0.1 mV or ST elevation of 0.15 mV in two contiguous leads, reversible wall motion abnormalities on echocardiography, or reversible perfusion defects on radionuclide imaging with thallium or technetium.
The ACC/AHA Task Force reviewed the literature and determined that dipyridamole-thallium and dobutamine stress echocardiography before vascular surgery predict PMI or death, with a positive predictive value of only 12% to 14% and a negative predictive value of 88% and 94% respectively. The low positive predictive value is expected because stress tests are designed to discover fixed coronary artery stenosis that exceed 70%. However, at least 50% of PMIs are due to plaque rupture, and it is often the noncritical, nonischemia producing lesions that rupture.

Selection of the noninvasive stress test should be based primarily on patient characteristics, local availability, and expertise in interpretation. Because of simplicity, lower cost, and widespread familiarity with performance and interpretation, the standard low-level exercise ECG stress test remains the most reasonable test in patients who are able to exercise and have a resting ECG that is interpretable for ST-segment shifts. Patients with an ECG pattern that would interfere with interpretation of the ST segment (left bundle branch block [LBBB]) should have an exercise test with imaging. Patients who are unable to exercise should have a pharmacologic stress test with imaging.

Dobutamine stress echocardiography is the preferred test if there is an additional question regarding valvular function or left ventricular (LV) dysfunction.


Is there a role for alpha-2 agonist in premedication?

Alpha 2 agonists stimulate prejunctional receptors and decrease norepinephrine release from pre-junctional terminals, thereby decreasing noradrenergic central nervous system transmission, producing sedation, anxiolysis, and analgesia. Studies have shown that clonidine as a premedication reduces hypertension, tachycardia, and norepinephrine levels in patients undergoing aortic reconstruction. Clonidine also suppresses the nonnal postoperative increase in fibrinogen levels and antagonizes epinephrine-induced platelet aggregation. It has also been shown to decrease intraoperative myocardial ischemia. The more selective alpha 2 agonists, dexmedetomidine and mivazerol (IV form only available in Europe), may also reduce postoperative myocardial ischemia events in high-risk patients.


Would you use transesophageal echocardiography (TEE) as a monitor?

TEE is a very sensitive diagnostic method of new onset myocardial ischemia. However, its role as a intraoperative monitor of myocardial perfusion is not supported by many investigators.
The echocardiographic diagnosis of myocardial ischemia is based on the development of new regional wall motion abnormalities (RWMAs), decreased systolic wall thickening, and ventricular dilation. Usually, a cross-sectional view (deep transgastric short axis), of the left ventricle (LV) is imaged because this view displays the myocardial perfusion territories of the three major coronary arteries. TEE was found to be twice as predictive as ECG in identifying coronary artery bypass grafting (CABG) patients who have an myocardial infarction (MI). In CABG patients postbypass RWMAs were related to adverse clinical outcome.

However, preintubation events are missed. Real-time intraoperative analysis of the TEE image is associated with a decreased accuracy of interpretation. The examination could divert the anesthesiologist’s attention from more important clinical details. Despite numerous reports extolling the virtues of TEE as an ischemia monitor, Hollenberg et al. demonstrated little incremental value over the 12-lead ECG.

As presented by many authors, myocardial ischemia occurs frequently at the end of the surgery when tachycardia and hypertension appear as part of the emergence of general anesthesia. During this period TEE use will not be feasible.

The American Society of Anesthesiologists and the Society of Cardiovascular Anesthesiologists Task Force in 1996 published a practice guideline for perioperative echocardiography. In these guidelines the use of TEE as a monitor of ischemia has a class II or III indication, meaning there is weak/little scientific evidence or expert support. However, its use has acquired more validity when ECG monitoring cannot provide accurate information, such as conduction abnormalities. Similarly, TEE use loses validity when clinical factors as preexisting ventricular dysfunction limit the accuracy of wall motion interpretations.


Would you induce anesthesia with etomidate?

Although etomidate has a negative inotropic effect on failing and nonfailing human heart muscle in doses that exceed the clinical dose, it appears to have a better hemodynamic profile than other commonly used induction agents, especially in the setting of coronary heart disease; with an induction dose of 0.2 to 0.3 mg per kg there are minimal changes in heart rate, stroke volume or cardiac output, and the blood pressure may decrease up to 15% because of decrease of systemic vascular resistance. Thiopental at equivalent doses of 3 to 5 mg per kg can produce an increase in heart rate 10% to 36% which could be potentially deleterious. Etomidate does
suppress adrenocortical function, cause myoclonus, and prolong duration of seizure when used for electroconvulsive therapy (ECT).


What is the significance of tight control of the heart rate intraoperatively?

Numerous studies have shown that intraoperative tachycardia could cause myocardial ischemia. Therefore, tight control of heart rate intraoperatively could significantly reduce the risk of perioperative myocardial events. Multiple reviews document how the duration of tachycardia is directly proportional associated with troponin elevation in the perioperative period.

Slogoff et al. in the 1980s were able to correlate tachycardia with the occurrence of intraoperative myocardial ischemia.

Raby et al. demonstrated that by identifying the high-risk patients, whose preoperative Holter tracing showed signs of ischemia, and controlling the heart rate 20% below the ischemia threshold or approximately 60 beats per minute with esmolol during vascular surgeries could reduce the risk of perioperative myocardial ischemia.

Recently, Feringa et al. demonstrated that tight heart rate control instead of just the use of b-blockers reduced perioperative myocardial ischemia and
improved long-term outcome in vascular surgery patients.


What are the postoperative predictors of perioperative cardiac morbidity?

The postoperative period appears to present the highest risk for cardiac morbidity for the noncardiac surgical patient. It is during this period where
67% of the ischemic events occur.

This period is characterized by increase in heart rate, blood pressure, sympathetic discharge and hypercoagulability. Heart rate commonly increases postoperatively by 25% to 50% over intraoperative values, and tachycardia occurs in 10% to 25% of patients. Postoperative myocardial ischemia occurs in 27% to 41% of high-risk patients. Most of these events (50% or higher) are silent (without angina). Postoperative myocardial infarction (MI) is usually preceded by prolonged ST-segment depression. This change on ECG is easily missed if not continuously monitored because most of the changes will revert completely to baseline in almost all cases. Traditionally, ischemia monitoring has been of short duration following operation (24 to 72 hours), but data suggests that period should be increased to 7 days.

Perioperative myocardial infarction (PMI) may be associated with tachycardia and higher pain threshold. Studies involving large numbers of patients suggest that postoperative ischemia is the most important marker of immediate and long-term adverse cardiovascular events. It is associated with a 2.8-fold increase in the odds of all adverse cardiac outcomes.

Earlier observations suggested that most MIs would occur in the third postoperative day. However, this belief has changed. Many authors have found the highest incidence of troponins elevation, a biochemical marker of ischemia, frequently 8 to 24 hours after the surgical procedure; even mild elevations are associated with postoperative cardiac ischemia and they could help to categorize the short- and long-term risk of the surgical patient as they are independent predictors of mortality. The discrepancy of studies could be related to the different biochemical markers used in previous studies (CKICK-MB vs. troponins).

Postoperative MI has also been associated with other clinical conditions as postoperative hypothermia, hypercoagulability, postoperative pain, and anemia.


Is postoperative anemia associated with adverse cardiac outcome? Is postoperative hypothermia associated with postoperative myocardial ischemia?

Patients with ischemic heart disease may be adversely affected by anemia. Based on existing literature the evidence is sufficient to state that transfusions are rarely beneficial if the hemoglobin level exceeds 10 g per dL (Hct >30%) in the absence of acute blood loss. It is also reasonable to state that the patient will benefit from a transfusion if the hemoglobin level falls below 7 g per dL. The controversy remains between Hg concentrations of 8.0 to 10.0 g per dL.

Two groups of investigators have now documented adverse clinical consequences of postoperative, iatrogenic acute anemia. Nelson et al. have identified a hematocrit of 28% as being the threshold associated with increased incidence of morbid cardiac events in postoperative vascular surgical patients. Wu et al. also demonstrated the benefit of higher hemoglobin levels in patients with acute myocardial infarction (MI). Since then two controversial publications: The TRICC trial (Transfusion Requirement in Critical Care) was not able to demonstrate a significant difference in 30-day mortality between patients who were “liberally” transfused (average hemoglobin, 10.7 g/dL) versus the restrictive group (no transfusion unless Hb <7g/dL). Rao et al., published the data from the combination of three large randomized trial stating the risk of death is 3.9 times greater in patients who were transfused compared with those who did not. The controversy remains, however, when confronted with high-risk
patients or those who demonstrate myocardial ischemia, we are more likely to transfuse packed cells to raise the hematocrit to 30%.


