2
CHAPTER

Ischemic Heart Disease

J Divatia, J Doctor, A Chatterjee

A 70-year old hypertensive male patient with history of chest pain is posted for a total gastrectomy.

Q. What are the known risk factors for the development of ischemic heart disease (IHD)?

Age, male gender, and a positive family history are risk factors that cannot be modified. Hypercholesterolemia, hypertension, and cigarette smoking are risk factors for which interventions have been proven to lower IHD risk. Sedentary lifestyle, diabetes mellitus, and obesity are the risk factors for which interventions are likely to lower IHD risk.

Q. Describe the normal coronary blood flow (CBF).

The resting CBF averages about 225 mL/min, which is 4–5% of the total cardiac output in normal adults. The CBF increases three to fourfold to supply the extra nutrients needed by the heart at maximum exercise level. The CBF is determined by the pressure gradient between the aorta and the ventricles. There are phasic changes in CBF during systole and diastole in the left ventricle. The blood flow falls to a low value during systole, especially in the subendocardial area, because of compression of left ventricular muscle around the intramuscular vessels and the small pressure gradient between the aorta and the left ventricle (aortic systolic pressure minus the left ventricular systolic pressure). During diastole, the cardiac muscle relaxes and no longer obstructs the blood flow through the left ventricular capillaries. The pressure gradient (coronary perfusion pressure) is high during diastole (aortic diastolic pressure minus the left ventricular end-diastolic pressure). Thus coronary blood flow occurs maximally in diastole. Conditions and interventions that prolong the diastolic time are beneficial, while those that reduce the diastolic filling time are detrimental. The phasic changes of the CBF are much less in the right ventricle because the force of contraction of the right ventricle is much weaker than that of the left ventricle and the pressure gradient is high between the aorta and the right ventricle during the entire cardiac cycle.

Q. Describe the coronary anatomy.

The right coronary artery system is dominant in 80–90% of people and supplies the sinoatrial node, atrioventricular node, and right ventricle. Right-sided coronary artery disease often manifests as heart block and dysrhythmias (Fig. 2.1).

The left main coronary artery gives rise to the circumflex artery and left anterior descending artery, which supply the

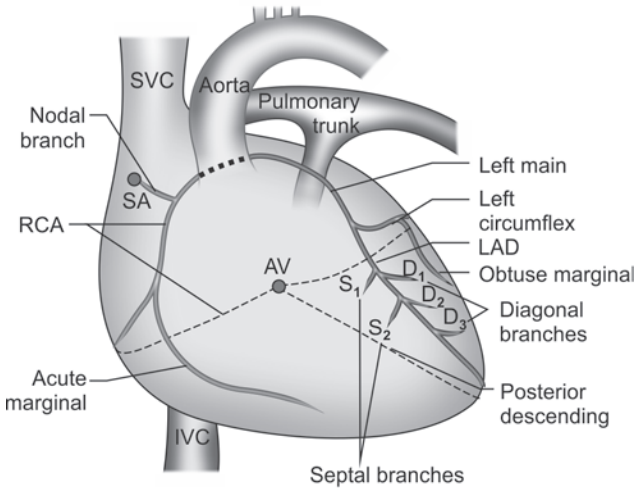


Fig. 2.1: Coronary artery system

Abbreviations: SVC, superior vena cava; SA, sinoatrial; RCA, right coronary artery; IVC, inferior vena cava; AV, atrioventricular; LAD, left anterior descending

majority of the interventricular septum and left ventricular wall. Significant stenosis of the left main coronary artery (left main disease), the proximal circumflex and left anterior descending arteries (left main equivalent) may severely depress myocardial function.

Q. Explain the determinants of myocardial oxygen demand and delivery.

Myocardial oxygen demand is determined by wall tension and contractility. Wall tension (T) is the product of intraventricular pressure (P), radius (r), and the wall thickness (h): ($T = P \times r/2h$) (Laplace's law). Increased ventricular end-diastolic volume (preload) and increased blood pressure (afterload) increases the wall tension and O_2 demand. Increase in contractility in response to sympathetic stimulation or inotropic medications also increase O_2 demand.

Increase in heart rate increases myocardial contractility but decreases the ventricular diameter and the wall tension. Thus, the increase in oxygen demand caused by enhanced contractility is in large part offset by the reduction in oxygen demand that accompanies the reduced wall tension. However, the oxygen demand increases because of more contractions performed per minute.

Myocardial oxygen delivery is determined by oxygen content and coronary blood flow. Oxygen content can be calculated by the following equation:

O_2 content = [1.34 mL O_2/g of hemoglobin × hemoglobin (g/dL) × saturation] + [0.003 × PaO_2]

The oxygen content is decreased in anemia and hypoxemia.

Coronary blood flow is determined by coronary perfusion pressure, the time available for perfusion, and the patency of coronary arteries. Coronary perfusion pressure = Aortic Diastolic pressure – LVEDP. Coronary perfusion pressure is thus reduced by diastolic hypotension, left ventricular hypertrophy, and increased left ventricular end-diastolic pressure (LVEDP). The diastolic time for perfusion is decreased with tachycardia. The patency of the coronary arteries can be influenced by vasospasm or coronary occlusion caused by atherosclerosis.

Q. What is the pathophysiology of myocardial ischemia?

Myocardial ischemia occurs when coronary blood flow is inadequate to meet the needs of the myocardium. The main coronary artery epicardial branches have lumens that are 2–4 mm in diameter. In the absence of collaterals, exertional angina occurs when the lumen area is reduced to 1 mm (50–60% reduction in diameter or 75% reduction in the cross-sectional area) and angina at rest occurs when the luminal area is reduced to 0.65 mm (75% reduction in diameter or 90% reduction in cross-sectional area). Most of the sclerotic lesions are eccentrically located so the remainder of the arterial wall is responsive to vasoactive stimuli and is capable of contraction. Therefore, the severity of the stenosis is dynamic and influenced by the vasomotor activity of the free arterial wall.

Nonstenotic causes of myocardial ischemia include aortic valve disease, left ventricular hypertrophy, ostial occlusion, coronary embolism, coronary arteritis, and vasospasm.

Q. What are the clinical manifestations of myocardial ischemia?

The clinical manifestations of myocardial ischemia are varied. Angina pectoris with or without signs of arrhythmias or heart failure is assumed to be the classic manifestation of myocardial ischemia. However, myocardial ischemia may present as ventricular failure or arrhythmias without angina or may remain clinically silent. The dynamic nature of coronary stenosis accounts for the changes in the caliber of a stenosis that may produce rest pain at one time and angina with varying degrees of exercise at other times.

Q. How can the angina be graded?

The Canadian Cardiovascular Society introduced a grading system for angina:

1. **Class I:** Angina with strenuous or rapid prolonged exertion at work or recreation.
2. **Class II:** Angina with walking or climbing stairs rapidly, walking uphill or walking more than two blocks on the level and climbing more than one flight of ordinary stairs at a normal pace.
3. **Class III:** Angina with walking one to two blocks on the level and climbing one flight of stairs at a normal pace.
4. **Class IV:** Angina may be present at a very low level of physical activity or at rest.

Q. What is unstable angina? Describe the pathogenesis of a perioperative myocardial infarction?

Unstable angina includes a spectrum of syndromes between stable angina and myocardial infarction. The angina attacks may increase in frequency or severity, persist longer, respond less to nitrates and may occur at rest or on minimal exertion.

A myocardial infarction (MI) is usually caused by platelet aggregation, vasoconstriction, and thrombus formation at the site of an atheromatous plaque in a coronary artery. Theoretically, sudden increases in myocardial O_2 demand (e.g. tachycardia, hypertension) or decreases in O_2 supply (e.g. hypotension, hypoxemia, anemia, tachycardia, coronary occlusion) can precipitate MI in patients with IHD. Most perioperative ischemic events are unrelated to hemodynamic perturbations, suggesting that intracoronary

events may be important in the genesis of perioperative ischemia. Complications of MI include dysrhythmias, hypotension, congestive heart failure, acute mitral regurgitation, pericarditis, ventricular thrombus formation, ventricular rupture, and death.

Q. What clinical factors increase the risk of a perioperative major adverse cardiac events (MACE), i.e. death or myocardial infarction following noncardiac surgery?

There are major, intermediate, and minor clinical predictors based on the algorithm for risk stratification and appropriate use of diagnostic testing of the American College of Cardiology/American Heart Association (ACC/AHA) Task Force on Perioperative Evaluation of Cardiac Patients Undergoing Noncardiac Surgery.[1] The guidelines integrate clinical risk factors, exercise capacity, and the surgical procedure in the decision process.

Major factors (markers of unstable coronary artery disease)

- Acute myocardial infarction (<7 days) or recent MI (7–30 days)
- Unstable or severe angina class III and IV
- Decompensated heart failure (NYHA functional class IV worsening or new onset heart failure)
- Significant arrhythmias
  - High grade atrioventricular block (AV block)
  - Mobitz type II AV block
  - Third degree AV block
  - Symptomatic ventricular arrhythmias
  - Supraventricular arrhythmias (including atrial fibrillation) with uncontrolled ventricular rate (heart rate > 100 beats per minute at rest)
  - Symptomatic bradycardia
  - Newly recognized ventricular tachycardia.

Intermediate factors (markers of stable coronary disease)

- History of IHD (excluding revascularization)
- History of congestive cardiac failure (CCF)
- History of a stroke or transient ischemic attack (TIA)
- Preoperative insulin therapy (diabetes)
- Serum creatinine >2 mg% (renal failure).

Minor factors (increased probability of coronary artery disease)

- Familial history of coronary artery disease
- Polyvascular status
- Uncontrolled systemic hypertension
- Hypercholesterolemia
- Smoking
- Electrocardiogram (ECG) abnormalities [arrhythmia, left ventricular hypertrophy (LVH), bundle branch block]
- Postinfarction (>3 months), asymptomatic without treatment
- Post-coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA) >3 months and <6 years, and no symptoms of angina and not on antianginal therapy.

Terminologies/definition

- Emergency procedure
  - Life or limb is threatened without surgery
  - No time, or about <6 hours, minimal clinical evaluation
- Urgent procedure
  - About 6–24 hours time for a limited clinical evaluation
- Time-sensitive procedure
  - Delay of no more than >1 to 6 weeks
    - Most oncologic procedures would fall into this category.
- An elective procedure
  - Procedure could be delayed for up to 1 year.
- A low-risk procedure
  - Combined surgical and patient characteristics predict a risk of a MACE (death or MI) of <1%.

Q. How does the type of surgery influence the risk stratification for perioperative ischemia?

- High-risk procedures [risk of perioperative major adverse cardiac events (MACE) >5%] includes emergent major operations, aortic and major vascular procedures, peripheral vascular surgeries, and anticipated prolonged procedures associated with large fluid shifts and/or blood loss.
- Intermediate-risk procedures (risk of MACE 1–5%) include carotid endarterectomy, head and neck surgery, intraperitoneal and intrathoracic surgery, orthopedic surgery, and prostate surgery.
- Low-risk procedures (risk of perioperative MACE <1%) include endoscopic procedures, superficial procedures, cataract surgery, and breast surgery and ambulatory surgery.

Q. How can cardiac function be evaluated on history and physical examination?

If a patient's exercise capacity is excellent, even in the presence of IHD, the chances are good that the patient will be able to tolerate the stresses of surgery. Poor exercise tolerance in the absence of pulmonary or other systemic disease indicates an inadequate cardiac reserve. All patients should be questioned about their ability to perform daily activities, such as is described in the Canadian classification of angina pectoris or the New York Heart Association classification of exercise tolerance.

Assessment of functional capacity: The Duke Activity Status Index (DASI) and approximate metabolic equivalents (METs – 1 MET represents an oxygen consumption of 3.5 mL/kg/min)

- 1–4 METs
  - Standard light home activities
  - Walk around the house
  - Take care of yourself—eating, dressing, bathing and using the toilet.
- 5–9 METs
  - Climb a flight of stairs, walk up a hill
  - Walk one or two blocks on level ground
  - Run a short distance
  - Moderate activities (golf, dancing, mountain walk)
  - Have sexual relations.
- >10 MET
  - Strenuous sports (swimming, tennis, and bicycle)
  - Heavy professional/domestic work like scrubbing floors, lifting or moving heavy furniture.
  - Signs and symptoms of congestive heart failure, including dyspnea, orthopnea, paroxysmal nocturnal dyspnea, peripheral edema, jugular venous distention, a third heart sound, rales, and hepatomegaly must be recognized preoperatively.

Q. What is the definition of recent and prior MI?

Recent monitoring and therapeutic strategies including aggressive recanalization as well as preoperative optimization of patients for surgery significantly decrease the risk for reinfarction even in patients with recent MI. Based on the decreased risk for perioperative reinfarction; the American College of Cardiology National Database defines an acute MI as one that is ≤7 days old. Recent MI is defined as MI occurring within more than 7 days but less than 30 days. Prior MI is defined as an MI that occurred more than 30 days before the surgery.

Q. What ECG findings support the diagnosis of IHD?

The resting 12-lead ECG remains a low cost effective screening tool in the detection of IHD. It should be evaluated for the presence of ST segment depression or elevation, T wave inversion, old MI as demonstrated by Q waves, disturbances in conduction and rhythm, and left ventricular hypertrophy. Ischemic changes in leads II, III and avF suggest right coronary artery disease, leads I and avL monitor the circumflex artery distribution and leads V3–V5 look at the distribution of the left anterior descending artery.

Q. What is the significance of the presence of IHD?

Patients with unstable or severe stable angina (Class III–IV) or recent MI without adequate revascularization are at high risk for developing perioperative MI and prior to undergoing elective noncardiac surgery should be referred for medical evaluation, coronary angiography and revascularization.

Patients with stable angina (Class I–II) or prior MI need further risk stratification and intervention based on their exercise capacity and the severity of the surgery. Patients with poor exercise capacity (<4 METs) or patients with good exercise capacity (>4 METs), but who are to undergo a high-risk surgery, need noninvasive testing to evaluate reversible ischemic myocardial areas during exercise. Coronary angiography and appropriate revascularization should be done before noncardiac surgery in patients with exercise-induced myocardial ischemia.

Q. What tests can help further evaluate patients with known or suspected IHD?

Further tests to evaluate a patient with known or suspected IHD/coronary artery disease include:

- Resting electrocardiogram
- Stress ECG/stress test
- Resting echocardiogram
- Stress echocardiogram/dobutamine stress echocardiogram
- Cardiopulmonary exercise testing (CPET)
- Dipyridamole-thallium imaging
- Coronary angiography.

Resting Electrocardiogram

According to the AHA Guidelines[1] a resting 12-lead ECG is:

- Reasonable for patients with known coronary artery disease or other structural heart disease, except in low-risk surgery
- May be considered for asymptomatic patients, undergoing moderate to high-risk surgery
- Not useful for aymptomatic patients undergoing low-risk surgical procedures.

Exercise ECG or stress test

Of the noninvasive cardiac tests of the wide variety of tests advocated, the exercise ECG or stress test is the most cost effective means of detecting coronary artery disease (sensitivity ≈ 70%, specificity ≈ 70%). It simulates sympathetic nervous system stimulation that may accompany perioperative events such as laryngoscopy and surgical stimulation.

An exercise ECG:

- Provides an objective estimate of functional capacity
- Can detect myocardial ischemia or arrhythmia
- Can be used to estimate perioperative cardiac risk and long-term prognosis.

Interpretation of the exercise ECG is based on: (1) the duration of exercise that the patient is able to perform, (2) the maximum heart rate that is achieved (Target heart rate achieved), (3) the time of onset of ST segment depression on the ECG, (4) the degree of ST segment depression, and (5) the time until resolution of the ST segment depression during the recovery period.

A normal exercise ECG indicates that the coronary circulation is reasonably adequate. The usefulness of this

technique is, however, limited by the fact that high-risk patients may be unable to exercise to a level required to achieve target rates, and exercise may be contraindicated in others. Also, approximately 10% of the adult population with normal coronary arteries may develop ST segment changes on the exercise ECG that resemble changes observed in the patient with IHD. For this reason, use of the exercise ECG in an asymptomatic patient is of doubtful value.

As per the AHA guidelines:

- A stress test is not necessary in patients with an elevated risk but moderate to excellent functional capacity (>4 METs)
- In patients with <4 METs or unknown functional capacity, it is reasonable to perform a stress test or a cardiac imaging to assess for myocardial ischemia
- In low-risk noncardiac surgeries, routine stress testing is not useful.

Echocardiography:

Used to evaluate left ventricular function and measure the ejection fraction.

As per the AHA Guidelines:[1]

- Routine preoperative evaluation of LV function is not recommended
- In patients with dyspnea of unknown origin or heart failure with worsening dyspnea or other change in clinical status, it is reasonable to evaluate LV function
- Reassessment of LV function in clinically stable patients may be considered.

Pharmacological stress testing:

It reproduces the cardiovascular effects of exercise and is particularly useful in patients who are unable to exercise:

1. Dobutamine stress echocardiography: Stress echocardiography has the advantage of being able to assess both regional wall motion abnormalities resulting from induced myocardial ischemia and left ventricular function (in situations of increased myocardial oxygen demand). It offers the best prediction for perioperative events, with a negative predictive value close to 100% (negative or normal stress ECHO rules out coronary artery disease) and a positive predictive value up to 38% among intermediate or high-risk patients. Areas of wall motion abnormality are considered at risk for ischemia
2. Dipyridamole thallium myocardial imaging, dipyridamole (an adenosine agonist that increases blood flow in normal coronary arteries) is used to simulate the effects of exercise. Images are acquired by the co-administration of a radioisotope, such as thallium, which gets distributed to the myocardium in direct proportion to coronary blood flow. A reversible defect (i.e. a defect seen on the initial stress image that disappears with rest) indicates the presence of coronary artery disease (sensitivity 85–90%, specificity 85–90%).

According to the AHA guidelines:

- Routine screening with noninvasive stress testing is not useful in low-risk noncardiac surgery
- Reasonable to perform these tests in a patient with poor functional capacity and an elevated risk if, it will change the management.

Cardiopulmonary exercise testing:

Cardiopulmonary exercise testing carried out in high-risk surgeries such as abdominal aortic aneurysm surgery, major abdominal surgery (including abdominal aortic aneurysm resection), hepatobiliary surgery, complex hepatic resection, lung resection, colorectal, bladder and renal cancer surgery. A low anerobic threshold was found to be predictive of perioperative cardiovascular complications. An anerobic threshold of approximately 10 mL O_2/kg/min was proposed as the optimal discrimination point, with a range in these studies of 9.9 mL O_2/kg/min to 11 mL O_2/kg/min.

The AHA guidelines currently recommend the use of CPET in patients undergoing elevated risk procedures.

Coronary angiography

Coronary angiography is an invasive procedure, and is indicated only in cases of unstable coronary syndromes, or uncertain stress tests in high-risk patients undergoing major surgery, or when there is a possible indication for coronary revascularization. Cardiac catheterization on its own has been reported to carry a mortality rate of 0.01% to 0.5% and of serious morbidity ranging from 0.03% to 0.25%.[6]

As per the AHA guidelines routine preoperative coronary angiography is not recommended.

Q. How would you stratify risk of anesthesia and surgery for this patient? What are the risk factors in the Lee revised cardiac risk index?

For quantification of risk, three key questions need to be considered:

1. Are there modifiable operative risk factors?
2. If the patient is at high-risk, should the elective operation be modified, delayed or cancelled?
3. Is coronary revascularization and/or valve surgery indicated?

An overall assessment of perioperative cardiac risk requires consideration of the type of surgery planned the presence and type of specific clinical indicators of coronary artery disease and the patient's functional status.

Historically, the Goldman multifactorial cardiac risk index was used to stratify patients according to cardiac risk. The modification of the Goldman index, proposed by Detsky and colleagues, provides improved risk assessment in patients undergoing vascular surgery. In 1999, Lee and colleagues described revised cardiac risk index (CRI) and is reported to be superior to both previous indices (Table 2.1).

Table 2.1: Risk factors and mortality

No. of risk factors	Event rate
0	0.4%
1	0.9%
2	6.6%
>3	11%

Six risk factors were identified in Lee's revised cardiac risk index.[2] This index is now incorporated into the AHA/ACC guidelines for preoperative assessment.

1. High-risk surgical procedure, such as intrathoracic, intraperitoneal or suprainguinal vascular reconstruction.
2. History of IHD (excluding revascularization).
3. History of congestive cardiac failure.
4. History of a stroke or transient ischemic attack (TIA).
5. Preoperative insulin therapy (diabetes).
6. Serum creatinine >2 mg% (renal failure).

The presence of each risk factor contributed one point to the index. The risk of cardiac complications depended on the number of risk factors (Table 2.2).

GUPTA Perioperative cardiac risk calculator: Developed and validated by Gupta et al.[3] for the prediction of cardiac risk after noncardiac surgery. They used the American College of Surgeons National Surgical Quality Improvement Program (NSQIP) Database involving 2,11,410 patients from 180 hospitals from the 2007 data set and validated it with the 2008 data set, which included 2,57,385 patients. The five predictors of perioperative myocardial infarction or cardiac arrest identified included the type of surgery, dependent functional status, abnormal creatinine, American Society of Anaesthesiologists' class, and increasing age. It has a higher accuracy in comparison with the RCRI developed in 1999, and is available as an interactive risk calculator to enable calculating the risk at the bedside or clinics with simplicity and accuracy.

Other risk indices are not commonly used, but are enumerated in Table 2.2.

Table 2.2: Various cardiac risk indices

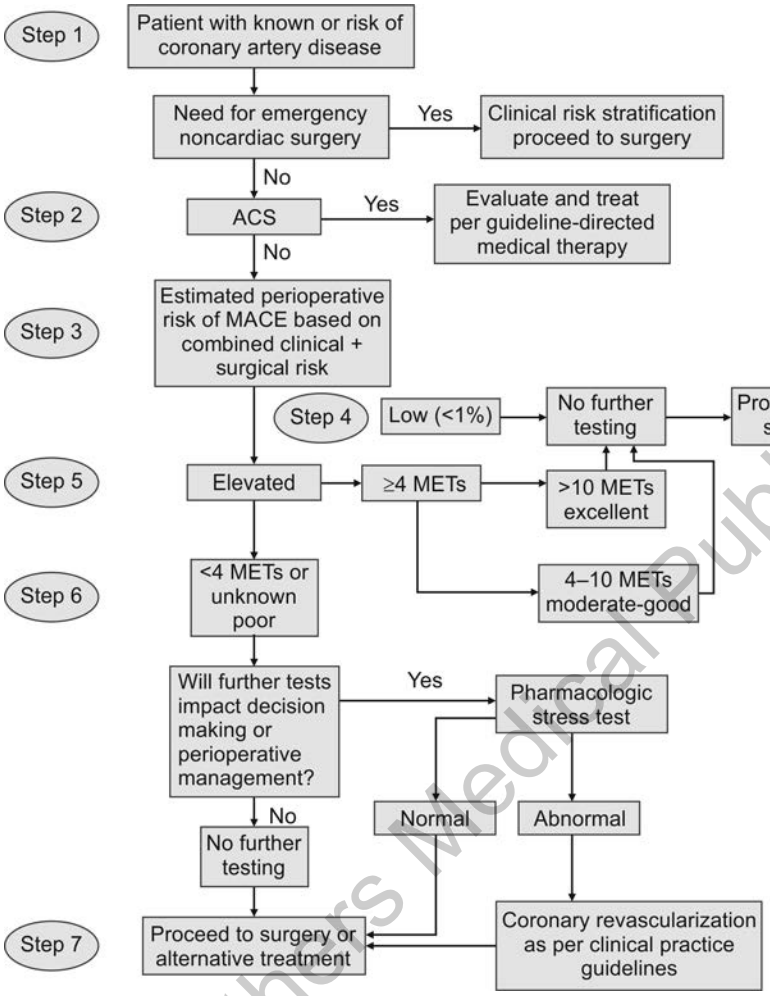
	Goldman 1977	Detsky 1986	Eagle 1989	Lee 1999
Age >70 years	5	5	1	
Emergency surgery	4	10	–	
Intraperitoneal, intrathoracic or aortic surgery	3	–	–	
Canadian Cardiovascular Society Class III angina	–	10	–	
Canadian Cardiovascular Society Class IV angina	–	20	–	
Unstable angina in previous 6 months	–	10	–	
Any angina	–	–	1	
Myocardial infarction in previous 6 months	10	10	–	
Myocardial infarction more than 6 months ago	–	5	–	
Q-wave on ECG	–	–	1	
Third heart sound or jugular vein distension	11	–	–	
Pulmonary edema in previous 7 days	–	10	–	
Any history of pulmonary edema	–	5	–	
Significant aortic stenosis (i.e. gradient >50 mm Hg	3	20	–	
ECG rhythm other than sinus with or without APBs	7	5	–	
>5 PVBs/min documented on ECG before surgery	7	5	–	
History of ventricular ectopy	–	–	1	
Diabetes mellitus	–	–	1	
Poor general medical status	3	5	–	
High-risk surgery				1
History of ischemic heart disease				1
History of congestive cardiac failure				1
History of cerebrovascular disease				1
Preoperative treatment with insulin				1
Preoperative serum creatinine >2 mg%				1
Total possible points	53	120	5	6
Risk Class I	0–5 (1%)	0–15 (0.43)	0 (0–3%)	0 (0.4–0.5%)
Risk Class II	6–12 (6.6%)	20–30 (3.38)	1–2 (6–16%)	1 (0.9–1.3%)
Risk Class III	13–25 (13.8%)	> 30 (10.60)	>3 (25–50%)	2 (4–7%)
Risk Class IV	>26 (78%)	–	–	>2 (9–11%)

Abbreviations: APBs, atrial premature beads; ECG, electrocardiogram

Q. Summarize the plan for evaluation of a patient with IHD undergoing noncardiac surgery.

A stepwise evidence-based approach suggested by the AHA/ACC task force[1] is reproduced in Flowchart 2.1. It takes into account the urgency of surgery, the estimated risk of MACE, and the functional capacity of the patient.

Flowchart 2.1: Evaluation of IHD patient (AHA)



Step 1: Assess risk of coronary artery disease (CAD)
Step 2: Evaluate for acute coronary syndrome (ACS)
Step 3: Calculating risk for major adverse cardiac events (MACE)
Step 4: Low risk of MACE—proceed for surgery
Step 5: Elevated risk of MACE—assess functional status
Step 6: Whether further testing in poor functional capacity (<4 METs) impact decision making/perioperative care
Step 7: Proceed for surgery as per guideline directed medical therapy or alternative strategies

Q. Which patient should be subjected to further non-invasive testing?

A patient in whom any of the following two are present should undergo further noninvasive testing:

- Intermediate clinical predictors
- Poor functional capacity (<4 METs)
- Undergoing a high-risk procedure.

This could include:

a. Tests for resting left ventricular function (LVF)
   - Current or poorly controlled congestive cardiac failure (CCF)
   - Prior LVF and dyspnea of unknown origin

b. Exercise or pharmacologic stress test
   - Active cardiac conditions
   - Patients with elevated risk and poor or unknown functional capacity (<4 METS).

Testing may be omitted in patients with elevated risk but with moderate or good functional capacity.

Noninvasive testing is not indicated for:

- Low-risk procedures
- No risk factors and undergoing intermediate surgery.

Q. What does one understand from the terms level of evidence (LOE) and class of recommendation (COR)?

Both help to understand the certainty of treatment effect.

Level of evidence:

- Level A: Data from multiple RCTs or meta-analyses
- Level B: Data from single RCT or nonrandomized studies

- Level C: Consensus opinion of experts, case studies or standard of care.

Class of recommendation

- Class I: Benefit >>> Risk (Procedure/treatment **should** be performed)
- Class II
  - IIa: Benefit >> Risk (Procedure/treatment **reasonable** to be performed)
  - IIb: Benefit ≥ Risk (Procedure/treatment **may be considered**)
- Class III: No benefit/HARM (Procedure/treatment should not be performed).

Q. Is it necessary to subject all patients with known IHD to coronary revascularization prior to noncardiac surgery in order to reduce the risk of MACE?

According to the AHA 2014 guidelines,[1] revascularization before noncardiac surgery is recommended only when indicated by clinical practice guideline (I C). Routine revascularization for noncardiac surgery is not recommended (III B).

Coronary artery bypass grafting

Older studies had shown a significant improvement in survival of revascularized patients, when they undergo subsequent noncardiac vascular surgery. However, the coronary artery revascularization procedure (CARP) trial by Mc Falls and colleagues failed to show any benefit of coronary artery revascularization before elective vascular surgery among patients with stable cardiac symptoms.

Preoperative CABG or revascularization should be reserved for patients with:

- Left main disease
- a LVEF <20%
- Severe aortic stenosis (AS).

For major noncardiac surgery following recent CABG, one should keep a gap of at least 4–6 weeks possibly for even up to 6 months.

Percutaneous coronary interventions (PCI): PCI before noncardiac surgery has not been shown to be of value.

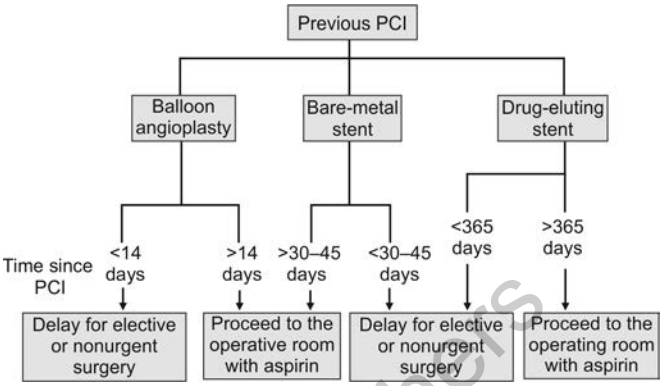
PCI before noncardiac surgery should be limited to those with:

- Left main disease
- Unstable angina, MI
- Life-threatening arrhythmias due to active ischemia and could be managed with PCI.

Patients who require urgent noncardiac surgery but need prior revascularization should undergo a balloon angioplasty or bare-metal stent (BMS) implantation (Flowchart 2.2).

If PCI is necessary, then the urgency of the noncardiac surgery with the risk of bleeding and events such as stent thrombosis, in a patient on dual antiplatelet therapy must be considered carefully.

Flowchart 2.2: Plan for patient with previous PCI/revascularization



Abbreviation: PCI, percutaneous coronary intervention

Q. When can a patient who has undergone a recent coronary revascularization procedure be subjected to a noncardiac surgery?

Unscheduled noncardiac surgery in a patient who has undergone a prior PCI presents special challenges. All patients with stents require dual antiplatelet therapy with aspirin and clopidogrel for a variable period of time. Any surgery performed within 4–6 weeks of a PTCA presents an increased risk of stent thrombosis and reinfarction if the antiplatelet medication is stopped, or of major bleeding if the antiplatelet treatment is continued through the perioperative period. The ACC/AHA guidelines[1] now recommend a delay of at least two weeks and ideally 4–6 weeks between implantation of a bare metal stent and noncardiac surgery. This allows 4 weeks of antiplatelet therapy during stent re-endothelialization and also a safe option of withholding antiplatelet drugs for 2 weeks prior to the surgery without the risk of stent blockage.

Noncardiac surgery should be delayed after PCI:

- Noncardiac surgery should be delayed for 365 days after a drug-eluting stent (DES) implantation
- Elective noncardiac surgery after DES implantation may be considered after 180 days in those patients where the risk of delay in the surgery far outweighs the risk of ischemia and stent thrombosis
- Elective noncardiac surgery should not be performed in patients in whom dual antiplatelet therapy will need to be discontinued perioperatively within 30 days after a bare metal stent or within 12 months of a DES implantation
- Elective noncardiac surgery should not be performed within 14 days of a balloon angioplasty in whom aspirin will need to be discontinued perioperatively.

Approach as suggested by the AHA/ACC Task Force,[1] for the management of a patient who has undergone

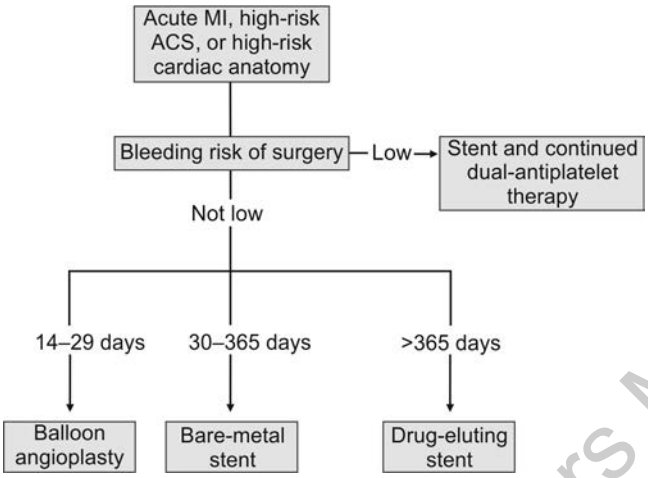
revascularization and would require urgent noncardiac surgery.

Q. Depending on the time frame available to optimize a patient prior to noncardiac surgery, which revascularization procedure would you subject the patient to?

The AHA 2014 guidelines[1] do not recommend revascularization prior to noncardiac surgery solely to reduce the incidence of MACE.

Management of patients requiring revascularization (as per CPGs) prior to noncardiac surgery is as shown in Flowchart 2.3.

Flowchart 2.3: Plan for revascularization prior to urgent noncardiac surgery



Abbreviations: MI, myocardial infarction; ACS, acute coronary syndrome

Q. Discuss the use of beta-blockers to reduce perioperative cardiovascular complications along with evidence for the same.

Rationale for the use of perioperative β-blockade therapy: Perioperative period is a period of physiological stress associated with high levels of catecholamines in blood. Catecholamines increase each of the four determinants of myocardial oxygen consumption (i.e. heart rate, preload, afterload and contractility). β-blockers have the potential of reducing myocardial O_2 consumption (thus improving the myocardial O_2 supply/demand balance) by decreasing sympathetic tone and myocardial contractility, which in turn, decreases the heart rate and arterial pressure. Mangano and colleagues performed a randomized, double-blind, placebo-controlled trial with atenolol in 200 patients with known coronary artery disease or risk factors for atherosclerosis who underwent noncardiac surgery. No perioperative deaths occurred in patients given atenolol, and only one death was reported in patients receiving the placebo. However, by 6 months, there were 8 deaths in the group that received the placebo and none in the group given atenolol ($p < 0.001$). This difference was sustained for the 2-year follow-up period.

However, several other recent trials have not shown any benefit using perioperative beta blockade. The POISE (Perioperative Ischemia Study Evaluation) trial found that while the incidence of cardiac complications was reduced by metoprolol, the overall mortality and the incidence of stroke was significantly higher in patients receiving metoprolol. This may have been due to a higher incidence of hypotension in this set of patients. In the POISE trial, a higher dose of a long-acting beta-blocker was started immediately before the noncardiac surgery. Subsequently, three studies[4-6] where a lower dose of beta-blocker was used, showed no harm or benefit.

Evidence supports the fact that the use of beta-blockers can reduce perioperative cardiac events. Benefit however may be offset by a higher risk of perioperative strokes and uncertain mortality benefit.

The 2014 ACC/AHA guideline summarizes the recommendations on perioperative beta blockade[7] (Level of evidence) as follows:

Class I: Patients on beta blockers (β-blockers) should continue their use in the perioperative period (I B)

Class II:

- Beta-blockers can be started preoperatively in patients with:
  - Intermediate or high-risk preoperative tests (IIb C)
  - Those with ≥3 RCRI factors (IIb B)
- The usefulness of β-blockers is uncertain in patients
- With long-term indication for beta-blockers but no other RCRI risk factor (IIb B)
- Preoperative beta-blockers where indicated should be started well ahead preferably > 1 day prior to the surgery to assess for safety and tolerability (IIb B)
- Restarting/ modifying dose of beta-blockers after surgery to be guided by clinical circumstances, independent of when the beta-blocker was started (IIa B).

Class III: Beta-blockers are not to be started on day of surgery (III B).

Starting beta-blockers on day of surgery may in fact be harmful. It is preferred to start the therapy at least 2–7days prior to the surgery.

Q. Which beta-blockers should be used and in what doses? What should be the targeted effect?

Drug of choice: Atenolol or metoprolol can be used. If the patient is on another β-blocker, there is no need to change to a cardioselective β-blocker. However, the dose should be adjusted to maintain a heart rate (HR) <70 beats/minute. Avoid bradycardia and hypotension.

- Beta-blocker initiation: Target heart rate between 50–70 beats/minute

- Preoperatively if HR >60 beats/minute and blood pressure (BP) >100 mm Hg: Start metoprolol 25–50 mg twice daily (BID) dose or atenolol 50–100 mg daily for at least a week prior to surgery
- Immediately preoperative if the HR >70 beats/minute and the BP >100 mm Hg: Give metoprolol 2.5–5 mg IV every 10 minutes to get the HR <70 beats/minute and BP >100 mm Hg
- Intraoperatively if the HR >70 beats/minute and the BP >100 mm Hg: Give metoprolol 2.5–5 mg IV every 10 minutes to get the HR <70 beats/minute or an esmolol infusion (50–200 μg/kg/min IV) (can be started to maintain HR <70 beats/minute.

Q. What is the role of α_2-adrenergic agonists?

Not everyone with an increased perioperative cardiac risk can tolerate a β-blocker. Clonidine, a centrally acting α_2-adrenergic agonist may prove to be an effective alternative. Perioperatively, it is used as a sedative, anxiolytic and an analgesic. It reduces hypertension, tachycardia and norepinephrine release associated with surgical stress.

The effect of α_2- adrenergic agonists has also been studied in the perioperative period. A meta-analysis[8] of 31 trials involving 4,578 patients demonstrated that α_2-adrenergic agonists reduced death and myocardial ischemia overall but sudden discontinuation was found to be associated with hypertension, headache, agitation and tremor. However, the POISE -2 trial which enrolled patients with or at a risk for atherosclerotic disease undergoing noncardiac surgery, had a different finding. Here, clonidine was not found to reduce the rate of death or nonfatal MI, but it did increase rate of nonfatal cardiac arrest and clinically important hypotension.

At present AHA guidelines do not recommend the use of α_2-adrenergic agonists for prevention of cardiac events (III B).

Q. What about the use of statins in the perioperative period?

Statins are lipid lowering drugs. In addition to plaque stabilization, they have been found to have anti-inflammatory and antioxidative properties, improve endothelial function and increased nitric oxide bioavailability.

They are found to be very effective in primary and secondary prevention of cardiac events in high-risk patients.[9]

The AHA guidelines recommend:

- Continuation of statins in the perioperative period in those who are already on them (I B)
- Reasonable to start preoperatively in those undergoing vascular surgery (IIa B)
- May be started preoperatively in patients with a clinical risk factor undergoing high-risk procedures (IIb C).

Q. Should all cardiac medications be continued throughout the perioperative period?

Patients with a history of IHD are usually on-medications intended to decrease myocardial oxygen demand by decreasing the heart rate, preload, afterload, or contractile state (e.g. β-blockers, calcium channel antagonists, nitrates) and to increase the oxygen supply by causing coronary vasodilatation (nitrates). These drugs are generally continued throughout the perioperative period. Abrupt withdrawal of β-blockers can cause rebound increases in heart rate and blood pressure.

Antagonists of the renin-angiotensin-aldosterone system may occasionally cause refractory hypotension that responds only to vasopressin or terlipressin. Based on evidence from randomized controlled trials.[10, 11] previous recommendations have suggested that angiotensin receptor blockers (ARBs) or ACE inhibitors (ACEI) be discontinued on the morning of surgery.

However the recent AHA guidelines (2014)[1] suggest:

- Continuation of ACEI and ARBs in the perioperative period (IIa B)
- If withheld prior to surgery, restart as soon as clinically feasible postoperatively (IIa C).

Q. How would you manage a patient on dual antiplatelet therapy if he /she presents for a noncardiac surgery?

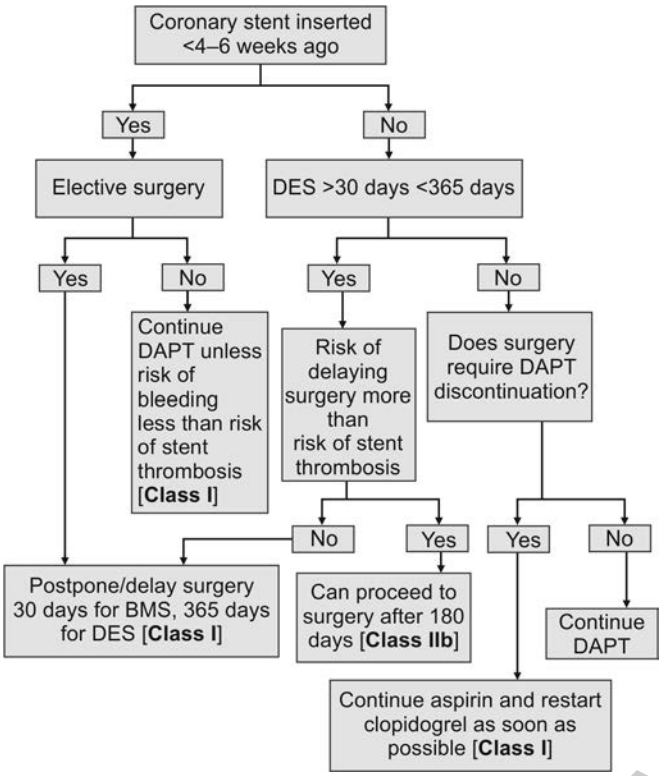
All patients with stents require dual antiplatelet therapy (DAPT) with aspirin and clopidogrel for a variable period of time. These patients present a challenge to the anesthetist. The risk of bleeding exists with the continuation of DAPT while discontinuing the antiplatelet therapy runs the risk of stent thrombosis and reinfarction. The current AHA Guidelines (2014) for management of a patient on DAPT are as shown in Flowchart 2.4.

Q. How long should a patient with a recent MI wait before undergoing elective noncardiac surgery?

The risk of reinfarction during surgery after a prior MI has traditionally depended on the time interval between the MI and the procedure. Postoperative MI and 30-day mortality rates are known to decrease substantially as the length of time from the MI to the operation is increased. The risk modified further by coronary interventions at the time of the MI. In the past ACC/AHA task force suggested that elective surgery is associated with prohibitive risk only during the initial 4–6 weeks. However, the AHA 2014 guidelines suggest that noncardiac surgery in the absence of any coronary intervention could be performed ≥60 days after a MI.

A recent MI, defined as having occurred within 6 months of noncardiac surgery, is considered as an independent risk factor for perioperative stroke, with an 8-fold increase in the perioperative mortality rate.[12]

Flowchart 2.4: Management of a patient on DAPT (AHA Guidelines 2014)



Abbreviations: DAPT, dual antiplatelet therapy; DES, drug-eluting stent

Q. How is premedication useful in the setting of IHD and surgery?

Patient anxiety can lead to catecholamine secretion and increased oxygen demand. In this regard, the goal of premedication is to produce sedation and amnesia without causing deleterious myocardial depression, hypotension, or hypoxemia. Morphine, scopolamine, and benzodiazepines, alone or in combination, are popular choices to achieve these goals. All premedicated patients should receive supplemental oxygen.

Q. Outline the hemodynamic goals of induction and maintenance of general anesthesia in patients with IHD.

The anesthesiologist's goal must be to maintain the balance between myocardial O_2 demand and supply throughout the perioperative period. During induction, wide swings in heart rate and blood pressure should be avoided. Ketamine should be avoided because of the resultant tachycardia and hypertension. Prolonged laryngoscopy should be avoided, and the anesthesiologist may wish to blunt the stimulation of laryngoscopy and intubation by the addition of opiates, β-blockers, or laryngotracheal or intravenous lignocaine.

Maintenance drugs are chosen with knowledge of the patient's ventricular function. In patients with good left ventricular function, the cardiac depressant and vasodilatory effects of inhaled anesthetics may reduce myocardial O_2 demand. An opioid-based technique may be chosen to avoid undue myocardial depression in patients with poor left ventricular function. Muscle relaxants with minimal cardiovascular effects are usually preferred.

Blood pressure and heart rate should be maintained near baseline values. This can be accomplished by blunting sympathetic stimulation with adequate analgesia and aggressively treating hypertension (e.g. anesthetics, nitroglycerin, nitroprusside, β-blockers), hypotension (e.g. fluids, sympathomimetics, inotropic drugs), and tachycardia (e.g. fluids, anesthetics, β-blockers).

Q. Which monitors are useful for detecting ischemia intraoperatively?

The following monitors are useful for detecting perioperative ischemia:[13]

Electrocardiogram (ECG): In the perioperative period, ECG monitoring most commonly helps to identify stress induced ST depression type of ischemia. Most current monitors do not simultaneously allow monitoring of 12 ECG leads. Selecting which chest leads to monitor is important particularly in noncardiac surgery. The combination of various leads with their sensitivity for detecting perioperative MI is as given below:

- The V5 (Sensitivity: 75%) is the most sensitive single ECG lead for detecting ischemia and should be monitored routinely.
- Lead V4 (61%)
- Leads V4 & V5 (90%)
- Lead II & V5 (80%)
- Leads II, V4 & V5 (98%).

Transesophageal echocardiography (TEE): It can provide continuous intraoperative monitoring of left ventricular function. Detection of regional wall motion abnormalities with this technique is the most sensitive monitor for myocardial ischemia. However, according to the AHA 2014 guidelines:

- Routine use of intraoperative TEE in noncardiac surgery is not recommended (III C)
- Emergency use of TEE in patients with hemodynamic instability is reasonable only if experts are readily available (IIa C).

Pulmonary artery catheters: Pulmonary artery occlusion (wedge) pressure gives an indirect measurement of left ventricular volume and is a useful guide to optimizing intravascular fluid therapy. Sudden increases in the wedge pressure may indicate acute left ventricular dysfunction due to ischemia. As per AHA 2014 guidelines:

- The routine use of pulmonary artery catheters in patients with IHD has not been shown to improve outcome and hence is not recommended (III A)
- May be considered in patients in whom the underlying medical conditions that significantly affect hemodynamics cannot be corrected preoperatively (IIb C).

Q. What are the goals of anesthetic management in this patient?

The primary goal of anesthetic management in a patient with IHD for noncardiac surgery is the avoidance of myocardial ischemia and myocardial infarction (MI). This is accomplished by preventing ischemia through measures that improve the myocardial oxygen supply-demand balance, primarily by controlling the patient's hemodynamics, and by detecting and treating myocardial ischemia when and if it does occur. The pathophysiology of perioperative myocardial infarction differs somewhat from that of myocardial infarction occurring in the nonoperated patient, where rupture of a coronary arterial atherosclerotic plaque leads to platelet aggregation and thrombus formation (Type I MI). In contrast, plaque rupture is not always the cause of perioperative myocardial infarctions; most MI's which occur perioperatively are due to a prolonged imbalance between myocardial oxygen supply and demand in the setting of coronary artery disease (Type II MI).[13]

Myocardial oxygen supply-demand balance

The myocardium extracts most of the oxygen from the blood flowing through the coronary arteries in a normal individual at rest. Increased cardiac work for any reason (e.g. physical exertion, emotional stress, and hypertension), increases myocardial oxygen demand, which is compensated in a healthy person by an increased coronary blood flow. With IHD, the arteries are narrowed and the coronary blood flow is already maximal. When oxygen demand by the myocardium exceeds its oxygen supply, myocardial ischemia develops, which, if prolonged, results in myocardial infarction.

Table 2.3: Causes of imbalance in myocardial oxygen supply and demand

Decreased oxygen supply	Increased oxygen demand
Decreased coronary blood flow • Tachycardia (diastolic perfusion time) • Hypotension (especially diastolic) • Increased preload (perfusion pressure) • Hypocapnia (coronary vasoconstriction) • Coronary artery spasm	*Tachycardia* • Increased wall tension • Increased preload • Increased afterload (hypertension) • Increased myocardial contractility
Decreased oxygen content and availability • Anemia • Hypoxemia • Reduced oxygen release from hemoglobin (e.g. pH, 2,3-DPG, temperature)	

Hence, the cornerstone of prevention and treatment of myocardial ischemia is to improve oxygen supply and reduce oxygen demand. It is important to know the conditions detrimental to myocardial oxygen balance because they are encountered frequently in the perioperative period, and can be caused and/or manipulated to some degree by the anesthesiologist (Table 2.3).

Q. Describe perioperative anesthetic management for the above patient?

All anesthetic techniques must aim to keep myocardial oxygen supply greater than demand, and therefore avoid ischemia. The essential requirements of general anesthesia for a patient with IHD are avoiding tachycardia and extremes of blood pressure, both of which adversely affect the balance between oxygen supply and demand.

Premedication: An anxious patient may be tachycardic and require an anxiolytic premedication. The patient may already be on medication for angina or hypertension. These drugs include β-blockers, nitrates, and calcium antagonists. These protect against the hemodynamic stresses of surgery and should be continued through the perioperative period. However, general anesthesia may exaggerate the hypotensive actions of such drugs.

Induction: All intravenous anesthetic agents have a direct depressant action on the myocardium, and may also reduce vascular tone. This causes hypotension (especially in the hypovolemic patient), often with a compensatory tachycardia, which may cause myocardial ischemia. To avoid hypotension at the time of induction all agents should be given slowly and in small increments. However, since ketamine causes indirect stimulation of the sympathetic nervous system, leading to both hypertension (increased afterload) and tachycardia, it should be avoided.

Intubation: Laryngoscopy is a powerful stressor, causing hypertension and tachycardia. This can be avoided with a supplemental dose of intravenous induction agent or liberal dose of an opioid like fentanyl prior to laryngoscopy. Also minimize duration of laryngoscopy (15 seconds or less) which may be useful in reducing the magnitude of sympathetic stimulation.

Maintenance: Volatile agents have minimal effects on cardiac output, although they do reduce myocardial contractility, especially halothane. They cause vasodilatation, and isoflurane has been implicated in the 'coronary steal' syndrome. The theory is that prestenotic vasodilatation diverts blood away from already ischemic areas of the myocardium. However, there is doubt as to the clinical significance of this phenomenon. Vagal stimulation due to halothane can cause bradycardias and nodal rhythms. Bradycardias can be beneficial by allowing greater coronary diastolic filling, provided blood pressure is maintained.

Iatrogenic hyperventilation, which greatly decreases $PaCO_2$, should be avoided as hypocapnia may evoke coronary artery vasoconstriction. It is important to avoid persistent and excessive changes in heart rate and systemic BP. A common recommendation is to strive to maintain patient's heart rate and BP within 20% of normal awake value. Maintain good pain relief and adequate depth of anesthesia to minimize sympathoadrenal response. Pain relief should be provided with liberal use of opioids. Theoretically, nonsteroidal anti-inflammatory drugs (NSAIDs) may have both a useful postoperative analgesic action and an antiplatelet effect which may reduce coronary thrombosis.

Maintain normothermia during intraoperative period as hypothermia will lead to peripheral vasoconstriction thus leading to increased afterload. Also these patients will develop shivering in postoperative period thus increasing myocardial work and oxygen demand.

Anemia is well-tolerated in the general population, but can cause a critical reduction in myocardial oxygen supply in those with IHD—a hematocrit of 30% or more is recommended. The American Association of Blood Banks 2012 Clinical Practice Guidelines[14] recommends:

- Transfusion in patients with CAD if:
  - Hemoglobin is less than 8 g/dL
  - Patient is symptomatic for chest pain, orthostasis and congestive heart failure
- In postoperative patients, it is recommended to maintain hemoglobin level of ≥8 g/dL unless patient exhibits symptoms mentioned above.

As per the AHA 2014 guidelines:

- It is reasonable to use either a volatile anesthetic or total intravenous anesthesia (TIVA) in noncardiac surgery (IIa A)
- Maintenance of normothermia may be reasonable to prevent perioperative cardiac events (IIb B)
- Use of intravenous nitroglycerine (NTG) prophylactically is not effective in reducing the incidence of perioerative myocardial ischemia (III B).

Reversal and recovery: Reversal of muscle relaxation with a combined anticholinesterase/antimuscarinic causes tachycardia, and extubation in itself is a stressor. Problems in the recovery phase which can cause ischemia include: tachycardia, pain, hypothermia, shivering, hypoxia, and anemia. These should be treated not just in the immediate postoperative period, but throughout the hospital admission. The use of supplemental oxygen in the postoperative period is one of the simplest, yet most effective measures in preventing myocardial ischemia.

Neuraxial anesthesia and perioperative pain management: The use of regional anesthetic techniques has theoretical advantages—epidural anesthesia reduces preload and afterload, coagulation responses and in the case of thoracic epidurals, causes coronary vasodilatation. These effects should reduce perioperative myocardial ischemia, but this is not supported by any evidence. However, good epidural analgesia may reduce the incidence of tachycardia arising due to postoperative pain. However, care should be taken to optimally fill these patients before giving a central neuraxial blockade. Despite decrease in myocardial O_2 requirements produced by peripheral sympathetic nervous system blockade it is important to realize that flow in the coronaries narrowed by atherosclerosis is pressure dependent. Therefore, decreases in systemic BP with epidural or spinal anesthesia should not be permitted to persist. In a patient with IHD, local anesthetic techniques whenever feasible such as brachial plexus block should be encouraged in order that the hemodynamic responses to general anesthesia are avoided. However, even under local anesthesia, the patient will be subject to the stresses of the surgical procedure itself as well as anxiety related to it, which can have marked hemodynamic effects.

Recent evidence[15,16] have shown that intraoperative neuraxial anesthetic does not provide any cardioprotective benefit over general anesthesia. However, the current AHA 2014 guidelines suggest the use of:

- Neuraxial anesthesia for postoperative pain relief to decrease the incidence of perioperative MI in patients undergoing abdominal aortic surgery (IIa B)
- Preoperative epidural analgesia may be considered to decrease the incidence of preoperative cardiac events in patients with hip fractures (IIb B).

Q. Describe your strategies to prevent perioperative MI (PMI) in this patient.

The risk of perioperative MI peaks within first 72 hours after surgery with most events occurring on day of surgery or next 24 hours. PMI is due to either acute plaque disruption (Type I MI) or due to a myocardial oxygen supply/demand mismatch (Type II MI). Increases in sympathetic discharge with accompanying elevation of heart rate and BP, pro-coagulant postoperative environment and mobilization of interstitial fluid administered during perioperative period may promote plaque rupture and subsequent cardiac event.

Strategies to prevent perioperative MI would therefore include:

- Optimization as per the current AHA guidelines as mentioned above
- Continuation of appropriate pharmacotherapy perioperatively
- Maintain a supply-demand balance.

Q. What postoperative care would you advice for this patient?

- Continuous ECG monitoring and surveillance for post-operative myocardial ischemia and infarction

- Continuation or institution of beta-blockade guided by clinical parameters
- Temperature control: Decreases in body temperature that occur intraoperatively may predispose to shivering on awakening leading to abrupt excessive increases in myocardial O_2 requirement
- Provision of supplemental O_2
- Adequate postoperative pain relief
- Maintenance of hemodynamic parameters with IV fluids
- Deep venous thrombosis prophylaxis.

Q. How would you diagnose and manage perioperative MI in this patient?

Mechanisms of perioperative myocardial infarction (PMI):
The risk of PMI peaks within first 72 hours after surgery with most events occurring on day of surgery or next 24 hours. Most of these events are silent and of non-ST-elevation myocardial infarction (NSTEMI) variety, as shown by various studies. In PMI, acute plaque disruption and hemorrhage in the infarct-related coronary artery seems to be common. On the other hand, a myocardial oxygen supply/demand mismatch is often believed to be the main trigger of myocardial injury. However, myocardial oxygen supply/demand mismatch and plaque rupture are not mutually exclusive mechanisms, and MI's may develop by different mechanisms at different locations in the same patient. In cases of fatal PMI's autopsy findings have shown plaque rupture as cause of PMI in 45–55% cases whereas half of the patients had no evidence of plaque rupture in their coronaries in spite of having extensive CAD. Increases in sympathetic discharge with accompanying elevation of heart rate and BP, procoagulant postoperative environment and mobilization of interstitial fluid administered during perioperative period may promote plaque rupture and subsequent cardiac event.

Most PMI's (>80%) which occur early after surgery, are asymptomatic, are of the non-Q-wave type and are most commonly preceded by ST-segment depression rather than ST-segment elevation. The majority of PMI's are silent showing no signs or symptoms and may be completely overlooked if continuous ECG monitoring is not performed. Long duration (single duration >20–30 min or cumulative duration >1–2 h) rather than merely the presence of postoperative ST-segment depression, seems to be the important factor associated with adverse cardiac outcome. The frequent combination of increases in heart rate preceding the ischemic episodes, ST-segment depression rather than elevation during all ischemic episodes; non-Q-wave rather than Q-wave MIs in almost all cases; the lack of angiographically visible thrombus or ruptured plaques in some patients who underwent coronary angiography following PMI; and complete reversal of ECG changes to baseline are highly suggestive that prolonged stress-induced myocardial ischemia is the likely primary cause of PMI. Repeated brief ischemic episodes may well have a cumulative effect and ultimately cause myocardial necrosis.

In summary, it can be said that both plaque rupture and prolonged perioperative ischemia in presence of stable CAD contribute to PMI. Probably, this could explain the benefit of beta-blockade therapy.

Diagnosis:
According to the definition of the World Health Organization (WHO), at least two of three criteria must be fulfilled to diagnose MI: (i) typical ischemic chest pain; (ii) increased serum concentration of creatine kinase (CK)-MB isoenzyme and troponin; and (iii) typical electrocardiographic findings, including development of pathological Q-waves.

Perioperative MI is mostly silent. In the past, studies have shown that ST segment changes postoperatively were associated with an increased incidence in MACE. However, recent evidence has suggested that an *electrocardiogram* may not provide sufficient information in this setting. Even in nonsurgical patients ECG is diagnostic only in 50% of cases, some abnormality but not classic MI changes in 40% and normal in 10% patients. Also pain if present is often masked by analgesia and residual anesthetics in the immediate perioperative period. Therefore, if the diagnosis of MI is based solely on the classical triad considerable under-reporting could happen.

Routine ICU monitoring with two lead ECG and ST segment trending detects ischemia only in 3% of high-risk postoperative patients when compared to 12 lead ECG.[5] The majority of ischemic events occurred in leads V1, V2, and V4 and not in commonly monitored leads II and V5. Also preexisting bundle branch blocks, digitalis effects or paced beats make interpretation of ECG difficult.

All these limitations put heavy emphasis on biochemical markers as a diagnostic test in perioperative period. *CK-MB* is the most routinely available and done marker and has sensitivity of 60–75% and specificity of 80–95% in perioperative period.

The *cardiac troponins* (troponin T and I) are cardiac injury markers that are rapidly released into the circulation after myocyte injury. Troponins have nearly absolute myocardial tissue specificity and a high sensitivity. Several large studies have demonstrated the superiority of troponin testing to ECG in identifying patients with Type I and Type II MI, and suggest that it may be superior initial test in the diagnosis of MI. Additional testing with *ECHO* to assess LV function may be required to establish definite diagnosis with the detection of a new regional wall motion abnormality (RWMA).

The recent AHA guidelines recommend that:

- Troponin levels should be measured in the setting of signs and symptoms suggestive of MI (I A)
- ECG should be recorded in the setting of signs and symptoms suggestive of MI (I B)
- Usefulness of postoperative screening with either troponins or ECG in patients with high risk for perioperative MI but without signs and symptoms is uncertain (IIb B)
- Routine postoperative screening in unselected patients without any signs and symptoms of MI or ischemia is not useful for guiding perioperative management (III B).

Management of PMI:

The major difference between perioperative patients and nonsurgical patients is the risk of life-threatening bleeding with thrombolytic therapy in the former. Thus thrombolysis is almost always contraindicated. The bleeding risk is high even with aggressive use of antiplatelet agents and anticoagulants. Hence, in the perioperative setting, a more conservative approach is recommended. Urgent angiography and percutaneous coronary interventions are reserved for patients with STEMI or those with NSTEMI who are hemodynamically unstable. The main stay of management is medical line of treatment consisting of good pain control, beta blockade, antiplatelet agents and anticoagulants.

a. *Oxygen:* It should be supplemented in patients with an arterial saturation less than 90%, patients in respiratory distress, or those with other high-risk features for hypoxemia.[17]
b. *Pain relief:* Pain if present should be treated with opioids, most commonly used being morphine, as it decreases preload, thus decreasing myocardial oxygen consumption.
c. *Nitrates:* As certain studies did not show survival benefit for nitrates, use of nitrates is optional for patients without evidence of ongoing ischemia, but for patients with symptomatic ischemia IV nitroglycerine is effective.
d. *Antiplatelet agents:* Aspirin should be administered as soon as MI is suspected in the dose of 375 mg orally/through NGT. Other antiplatelet agents are available, clopidogrel being the widely used one. Patients with allergy to aspirin, clopidogrel can be used. Aspirin being a weak antiplatelet agent it is advisable to combine both; however, studies have shown that combination increases the relative risk for major perioperative bleeding by approximately 50%.
e. *Anticoagulants:* Acute MI is often associated with increased thrombin activity. Unfractionated heparin is associated with indirect thrombin inhibition and is frequently used to treat acute MI. Low molecular weight heparins act through their anti-Xa activity and have predictable kinetics but suffer from the longer half life. In the perioperative setting IV UFH is indicated if suspicion of plaque rupture is high and bleeding risk is low.
f. β-*blockers:* β-blockers should be used for all the patients with acute coronary event unless there is significant bradycardia, decompensated CHF, or severe COPD. Calcium channel blockers have no survival benefit in such situations and should not be used.
g. *Statins*: Are currently recommended for all patients who develop a perioperative MI.

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