

## **WHEN TO RAISE THE ALARM TO STOP SURGERY**

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**Abstract:** *Perioperative myocardial ischaemia and infarction (PMI) is a major cause of short and long term morbidity and mortality in the surgical population. It is estimated that more than one half of postoperative deaths are caused by cardiac events, most of which are ischaemic in origin.*

### **Introduction**

Perioperative myocardial ischaemia and infarction (PMI) is a major cause of short and long term morbidity and mortality in the surgical population.

It is estimated that more than one half of postoperative deaths are caused by cardiac events, most of which are ischaemic in origin.

Over 50,000 patients each year sustain a perioperative MI at an average additional cost to the health infrastructure of \$12,000 per patient.

Thus prevention of a PMI is important to improve overall postoperative outcome.

Perioperative myocardial infarction is a significant issue in patients undergoing not only high risk surgery neurosurgery but also those with clinical predictors that put them at a higher risk even with minor surgical interventions.

Though the understanding of the pathophysiology and management of ischaemia in the preoperative period has increased tremendously over the last decade but lot of questions still remain unanswered.

In view of the poor positive predictive value of non-invasive cardiac stress tests, the emphasis is on a combination of selective non-invasive testing aggressive preoperative pharmacological therapy or cancellation of surgery.

### **Myocardial Ischemia –Definition**

Myocardial ischemia is a dual state composed of inadequate myocardial oxygenation and accumulation of anaerobic metabolites and occurs when myocardial oxygen demand exceeds the supply.

Myocardial ischemia is characterized by an imbalance between myocardial oxygen supply and demand. The Two most common conditions that predispose to myocardial ischemia are CAD and left ventricular hypertrophy (LVH).

Increases in coronary blood flow secondary to an increase in myocardial oxygen demand and sympathetic nervous system activation (e.g. during exercise, mental stress or increase in heart rate) induce vasodilatation in normal coronary arteries but lead on to paradoxical vasoconstriction in the atherosclerotic vessels.

Such limitation of coronary flow and the inability of vessels to dilate near the site of an atherosclerotic plaque may result in regional myocardial supply- or low-flow ischemia.

### **Myocardial Ischemia-Clinical Manifestations**

The clinical manifestations of myocardial ischemia range from –

- Asymptomatic or “silent” episodes
- Angina
- Arrhythmia
- Conduction blocks
- Wall motion abnormalities
- Pulmonary congestion
- Infarction and
- Sudden cardiac death

Systolic (contractile) and diastolic (ventricular filling) dysfunction occur first; followed by electrocardiographic changes, and finally by chest pain.

### **Myocardial Ischaemia**

Preoperatively, the single most common abnormality that is often associated with ischemia is **Tachycardia**. Preoperative tachycardia can result from many causes, including-

- Light plane of anesthesia
- Endotracheal intubation and extubation
- Hypovolemia
- Fever,
- Anaemia
- Congestive heart failure(CHF), and
- Postoperative pain

### **Other causes-**

- Hypertension -can cause increased demand and
- Hypotension -can lead to decreased supply in the perioperative period.

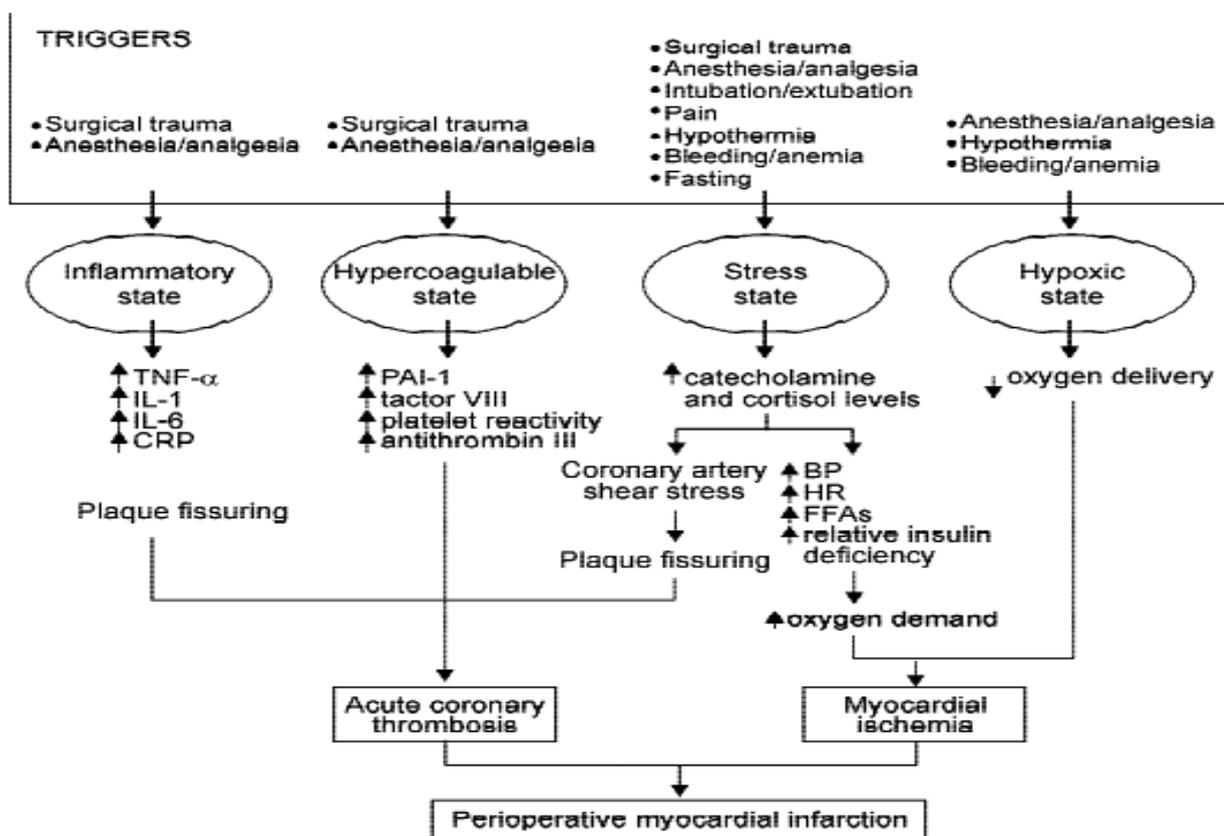
### **Timing of surgery**

Most ischemic episodes tend to start at the end of surgery and during emergence from anesthesia. This period is characterized by increases in-

- Heart rate (HR)
- Arterial blood pressure (BP)
- Sympathetic tone, and
- Procoagulant activity.

### **Mechanisms and triggers of preoperative myocardial injury**

Surgery, with its associated trauma, anesthesia and analgesia, intubation and extubation, pain, hypothermia, bleeding and anemia, and fasting, is analogous to an extreme stress test.



### Diagnosis of perioperative myocardial ischaemia

**Chest pain:** The onset of new cardiac pain can be extremely important when one is diagnosing myocardial ischaemia preoperatively, during surgery under local or regional anaesthesia and in the recovery room. Typically cardiac pain is a sense of chest constriction and may be referred to arm, neck, jaw, teeth or even post scapular area. However diabetics may have silent ischaemia because pain pathways are impaired by diabetic neuropathy.

**Electrocardiography (ECG):** Preoperative myocardial ischaemia has predominantly been detected and defined by ECG. The reported incidence of perioperative myocardial ischaemia greatly depends on choice and number of precordial leads, on the definition of ischaemic ST segment change (extent and duration of ST-segment change), and on the mode of data acquisition (continuous vs intermittent).

The standard ECG consists of 12 leads, however, during anaesthesia, monitoring is usually limited to five or seven leads.

Blackburn et al demonstrated that 89% of significant ST depression was found in precordial lead V5.

They also demonstrated that 100% of ST segment changes can be detected by recording leads V3 -V6 and leads II and aVF.

In routine practice, leads II and V5 are continuously monitored, giving views of inferior and lateral myocardium. .

Horizontal or down sloping ST segment depression of 1mm or more indicates significant subendocardial ischaemia while ST segment elevation greater than 1mm indicates severe transmural ischemia.

Although ST-segment depression usually reflects subendocardial ischaemia and is often regarded as reversible injury, it is not inconsistent with an MI. During the early evolution of an MI, significant ST-segment elevation may belacking.

For that reason, in current clinical practice, acute MI is divided into ST-segment and non-ST-segment elevation MI (which ultimately develop with little cross-over into Q-wave and non-Q-wave MI, respectively).

In most studies on perioperative cardiac ischaemic events, the populations consisted largely of elderly patients. Thus, prolonged ST-segment depression may reflect ongoing myocardial ischaemia (ultimately leading to MI), or it may reflect the beginning of an evolving MI.

### **Limitations of ECG**

Patients with left ventricular hypertrophy, left bundle branch block (LBBB), digitalis effect, ventricular pacing and those not in sinus rhythm are not suitable for ECG-derived diagnosis of myocardial ischaemia.

In addition, perioperative changes in acid-base balance and electrolytes can affect the ECG in a way that interferes with ischaemia detection.

### **Biochemical markers:**

**CPK-MB**- Increased concentration of CPK-MB is not useful intraoperatively because the leakage of these enzymes into the circulation can occur 8-24 hours after an MI.

**Cardiac Troponins**-Because of the inherent limitations of CPK-MB concentration and lack of specificity, MI may be best detected with cardiac TnT concentrations.

While CPK-MB concentrations may rise only 10-20 times of normal during infarction and return to normal within 72 hrs, TnT and TnI levels may rise more than 20 times above the reference range within 3 hrs after onset of chest pain and may persist for up to 10-14 days. This may assist in late diagnosis of infarction.

### **Pulmonary artery pressure:**

The quantitative increase in pulmonary capillary wedge pressure and characteristic changes in its waveform have been suggested as an ischaemia monitor, but it is believed that the pulmonary artery catheter is an insensitive monitor of myocardial ischaemia and should not be inserted with this as a primary indication.

In addition, the use of pulmonary artery catheters in the perioperative period may actually contribute to increased morbidity.

### **Transoesophageal echocardiography (TEE):**

New or worsening of baseline SWMA, akinesia dyskinesia, reduction in ejection fraction, ischemic mitral regurgitation, change from prior TTE.

### **Limitations:**

- Small Q wave MI
- non Q wave MI
- prior MI with baseline SWMAs
- reversible ischemia
- Stunning hibernating myocardium

### **Preoperative risk assessment**

### Non cardiac surgery

The fundamental purpose of ascertaining the presence of coronary artery disease, myocardial ischaemia, or both preoperatively is

To determine which patients are at risk and whether any further preoperative treatment is necessary,

To design an intraoperative management plan to reduce the incidence and consequences of ischaemia in patients at risk, and

In these at risk patients attempt to reduce the risk of adverse outcome by implementing aggressive preventive and treatment modalities Interventions based on this assessment may include preoperative medical optimization, coronary revascularization or both.

American Heart Association/American College of Cardiology (AHA/ACC) published a guideline for perioperative cardiovascular evaluation for noncardiac surgery in 1996. This guideline has been recently updated in 2002 and offers the most comprehensive approach to preoperative cardiac evaluation for noncardiac surgeries.

It focuses clinicians' attention on three major areas:

Clinical risk predictors, surgery-specific risks, and functional capacity

#### Clinical predictors of perioperative cardiac risk

Major	Intermediate	Minor
Acute or recent MI Unstable angina Decompensated CHF Significant arrhythmias Severe valvular disease	Mild angina Prior MI Compensated CHF Diabetes mellitus Renal insufficiency	Advanced age Abnormal ECG Rhythm other than sinus History of stroke Uncontrolled hypertension

#### Surgery-specific cardiac risks

High (5%)	Intermediate (<5%)	Low (1%)
Emergent surgery Aortic or major vascular surgery Peripheral vascular surgery Large fluid shifts and blood loss	Carotid endarterectomy Head and neck surgery Intraperitoneal intrathoracic procedures Orthopaedic surgery	Endoscopic procedures Superficial procedures Cataract surgery Breast surgery

#### Preoperative interventions for prevention of Perioperative MI

Two principal strategies used in an attempt to reduce the incidence of PMIs and other cardiac events are:

A. Preoperative coronary revascularization- PCI or CABG

B. Pharmacological Two principal strategies used in an attempt to reduce

**β-blockers**

Antiplatelet therapy (APA)-aspirin, clopidogrel and glycoprotein

Inhibitors

Alpha 2-adrenoceptor agonists

Statins

Nitroglycerin

Miscellaneous (ACE inhibitors, Antithrombin therapy, Adenosin modulators, Nicotidil)

**Intraoperative management-**

The basic challenge during the perioperative period is to prevent myocardial ischaemia, this goal is logically achieved by maintaining the balance between myocardial oxygen delivery and demand.

Intraoperative events that influence the balance between myocardial oxygen delivery and myocardial oxygen requirements

**Decreased oxygen delivery**

Decreased coronary blood flow

Tachycardia

Diastolic hypotension

Hypocapnia ( coronary artery vasoconstriction)

Coronary artery spasm

**Decreased oxygen content**

Anaemia

Arterial hypoxaemia

Shift of the oxyhaemoglobin dissociation curve to the left

**Increased oxygen requirements**

Increased preload (wall tension)

Sympathetic nervous system stimulation

Tachycardia

Systemic hypertension

Increased myocardial contractility

Increased afterload

**Choice of anaesthetic technique**-Induction of anaesthesia in patient with IHD should be smooth and attempts should be made to minimize pressor response to laryngoscopy and intubation. Various drugs like lidocaine, nitroprusside, esmolol, fentanyl, nitroglycerine etc. have been used for this purpose.

Choice of drugs for maintenance depends on left ventricular function as determined by preoperative evaluation. In patients with normal LV function, a combination of N<sub>2</sub>O-opioid with addition of volatile agent (isoflurane, desflurane, sevoflurane) is acceptable. In patients with severely impaired LV function, a high dose opioid (fentanyl 50-100 µg/kg-1IV) may be utilized as sole anaesthetic.

Opioid-based anaesthetics have become popular because of the cardiovascular stability associated with their use.

**Neuraxial anaesthetic techniques** - can result in sympathetic blockade, resulting in decreases in both preload and afterload. The decision to use neuraxial anaesthesia for the high-risk cardiac patient may be influenced by the dermatomal level of the surgical procedure.

Infrainguinal procedures can be performed under spinal or epidural anaesthesia with minimal haemodynamic changes if neuraxial blockade is limited to those dermatomes.

Abdominal procedures can also be performed using neuraxial techniques; however, high dermatomal levels of anaesthesia may be required and may be associated with significant haemodynamic effects.

High dermatomal levels can potentially result in hypotension and reflex tachycardia if preload becomes compromised or blockade of the cardioaccelerators occurs.

### **Treatment of perioperative myocardial ischaemia**

#### **Prevention of myocardial ischaemia:**

Careful attention to prevention of tachycardia during anaesthesia is extremely important. Maintenance of adequate depth of anaesthesia along with judicious use of ultra-short acting blockers can be very useful in preventing tachycardia.

Apart from this, one should take adequate measures to attenuate pressor responses to laryngoscopy and endotracheal intubation.

**Treatment of myocardial ischaemia without accompanying haemodynamic alterations:** In susceptible patients, myocardial ischaemia often occurs without attendant haemodynamic alterations. In these patients nitroglycerine (sublingual or intranasal) can be useful. Nitroglycerine decreases preload and wall tension, dilates epicardial coronary arteries, and increases subendocardial blood flow.

**Treatment of myocardial ischaemia accompanied by tachycardia and hypertension:** A combination of tachycardia and hypertension can precipitate myocardial ischaemia by disturbing the myocardial oxygen demand and supply balance. After ensuring adequate ventilation, oxygenation and anaesthetic depth, blockers (esmolol or metoprolol) may be administered in a titrated manner provided there is no evidence of CHF or bronchospasm.

**Treatment of myocardial ischaemia accompanied by tachycardia and hypotension:** A combination of tachycardia and hypotension (mostly due to hypovolemia) can precipitate myocardial ischaemia because both can drastically reduce myocardial oxygen supply.

Apart from volume replacement, it is important to restore coronary perfusion pressure and slow the rate.

**Severe resistant myocardial ischaemia:** Occasionally one may come across severe myocardial ischaemia, which is resistant to all antianginal drugs. Here intraaortic balloon pump (IABP) can be useful; it acutely

Decreases myocardial oxygen requirements and may increase myocardial oxygen supply.

### **Conclusions**

There is still some way to go before we can have formal strategies and guidelines for preventing, identifying and managing perioperative subclinical myocardial injury and further research in this area is the need of the hour.