

**PERIOPERATIVE MYOCARDIAL INFRACTION**Ravindra S. Giri<sup>1</sup>, Ajay G. M<sup>2</sup>, Thyagaraju N<sup>3</sup>**HOW TO CITE THIS ARTICLE:**

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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. Over 50,000 patients each year sustain a perioperative MI. Thus prevention of a PMI is important to improve overall postoperative outcome. Myocardial ischaemia is a dual state composed of inadequate myocardial oxygenation and accumulation of anaerobic metabolites and occurs when myocardial oxygen demand exceeds the supply. Myocardial infarction is defined as the death of myocardial myocytes due to prolonged ischaemia. In patients with, or at risk of coronary artery disease (CAD), the reported incidence of perioperative myocardial ischaemia is 20-63%. Various studies have shown that postoperative myocardial ischaemia was consistently found to occur considerably more often than preoperative and intraoperative ischaemia (Ratio approximately 3:1 and 5:1 respectively). As more and more patients coming for non-cardiac surgeries who have already undergone coronary intervention such as balloon angioplasty, stenting or CABG, we as anaesthesiologists should have thorough knowledge of the perioperative implications of the same in a day to day practice. Secondly, as the geriatric population is increasing there are more chances of encountering patients with known or unknown ischaemic heart disease both on an emergency and elective basis.

**KEYWORDS:** Myocardial Ischaemia, Myocardial Infarction, Myocardial-Oxygen Supply Demand, Metabolic Equivalents, AHA-Guidelines 2014.

**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. The incidence of perioperative cardiac injury is a cumulative result of the patient's preoperative medical condition, the specific surgical procedure, the expertise of the surgeon, the diagnostic criteria used to define MI, and the overall medical care at a particular institution. The risk of perioperative death due to cardiac causes is less than 1% in patients who do not have ischemic heart disease. The incidence of perioperative MI in patients who undergo elective high risk vascular surgery is between 5 to 15%.

The risk is even higher for emergency surgery. Perioperative MI are associated with a 20% mortality.<sup>(1,2)</sup>

**PATHOPHYSIOLOGY OF MYOCARDIAL ISCHEMIA AND MYOCARDIAL INFARCTION:** Myocardial ischemia is defined as a dual state arising due to an imbalance between the increased oxygen demand and inability of coronary blood vessels to increase the oxygen delivery superimposed on a lesion that causes coronary arterial obstruction. Myocardial infarction is defined as the death of cardiac myocytes due to prolonged ischaemia.<sup>(1,3)</sup> The 2 conditions which are usually associated with ischemia is coronary artery disease and left ventricular hypertrophy.

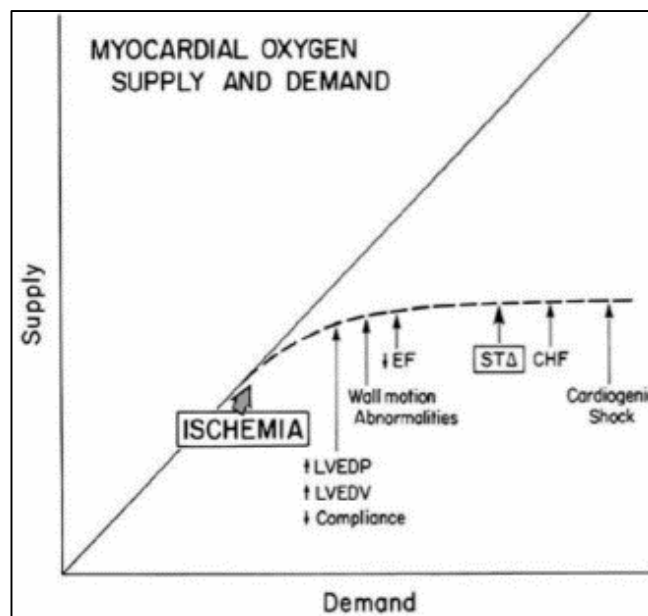
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Decreased oxygen supply or low-flow ischaemia due to coronary vasoconstriction, intracoronary platelet aggregation or thrombus formation is largely responsible for myocardial infarction and unstable angina.

In myocardial ischemia there is also impairment in endothelial function of the coronary arteries predisposing to coronary thrombosis. In a normal coronary vasculature, as myocardial oxygen demand increases there is subsequent coronary vasodilation whereas in atherosclerotic vessels there is paradoxical vasoconstriction.

Most of the perioperative myocardial ischemia/infarction occurs first 24 to 48 hrs. The clinical manifestations of myocardial ischaemia may vary from asymptomatic or "Silent" episodes to 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. All these events often occur in a short time course of less than 1 minute. An 80% reduction in coronary blood flow causes akinesis, whereas a 95% decrease causes dyskinesis. As the ischemia worsens there is increase in left ventricular end diastolic pressure leading to pulmonary edema.<sup>(1,2)</sup>

**Figure 1: Sequence of events in myocardial ischaemia**



**Fig. 1**

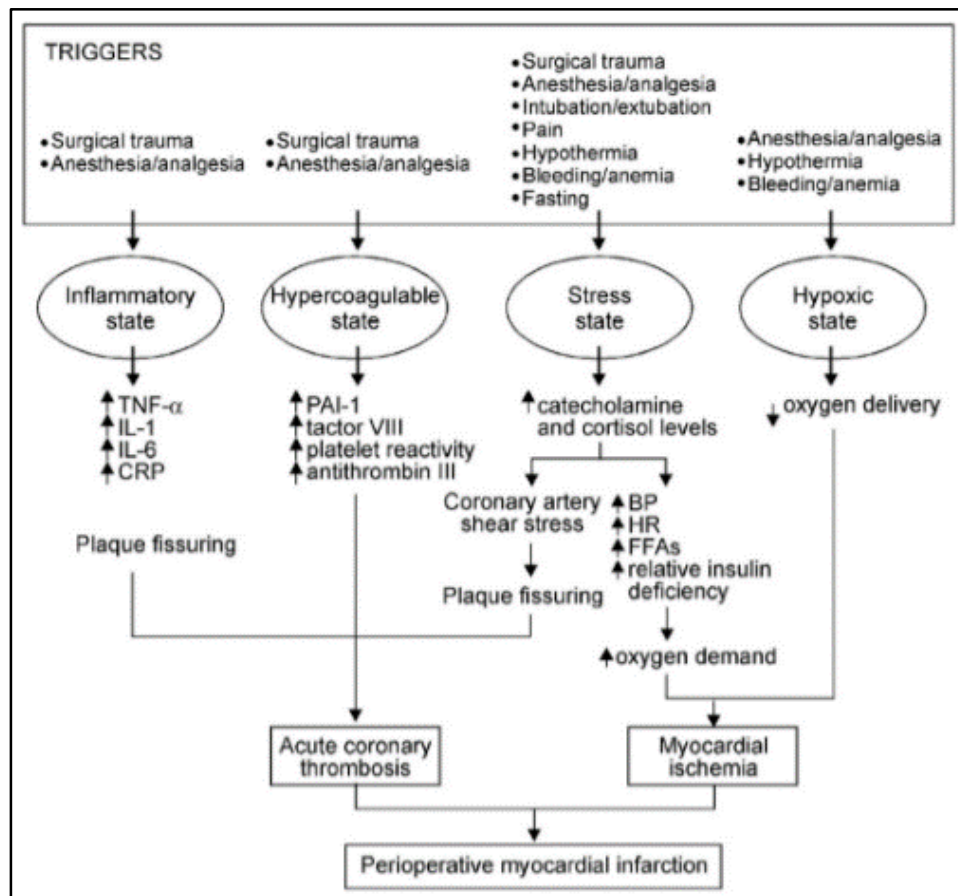
Another theory describes the occurrence of perioperative myocardial infarction is due to the sudden development of thrombotic process associated with vulnerable plaque rupture. A vulnerable plaque is an inflamed fibroatheroma with a lipid-rich core containing cholesterol crystals and necrotic debris, a thin fibrous cap with an infiltration of macrophages and lymphocytes, and low smooth muscle content. Endothelial injury at the site of a plaque rupture triggers the cascade of platelet aggregation and release of mediators. Aggregation of platelets and activation of other inflammatory and non-inflammatory mediators potentiates thrombus formation and leads to dynamic and physical blood vessel narrowing cause ischemia or infarction.<sup>(1,2)</sup>

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In the post-operative period, changes in blood viscosity, catecholamine concentrations, and plasminogen activator inhibitor levels create a prothrombotic state.

Changes in the heart rate and blood pressure as a result of the stress response can increase the propensity for a plaque to fissure and develop endothelial damage. In combination, these factors can precipitate thrombus formation in an atherosclerotic coronary artery and lead to the development of an STEMI. Hence there are two different pathophysiologic process which lead to perioperative MI, one due to the acute coronary thrombus formation and second due to the imbalance between the oxygen supply and demand.<sup>(2)</sup>

**Figure 2: Potential triggers of states associated with perioperative elevations in troponin levels, arterial thrombosis and fatal myocardial infarction.<sup>(1)</sup>**



**Fig. 2**

**DIAGNOSIS OF MYOCARDIAL ISCHEMIA OR INFARCTION:** There is no accepted gold standard for the diagnosis of myocardial ischaemia. The diagnosis can usually be based on clinical, Haemodynamic (Pulmonary artery capillary wedge and/or left atrial pressure wave), electrocardiographic (ECG), functional (Echocardiogram), metabolic (Coronary lactate production), Biochemical (release of creatine kinase-MB isoenzyme and/or troponin) or Regional perfusion (Scintigram) parameters.

Diagnosis of acute MI requires the typical rise and subsequent fall in plasma levels of biochemical markers of myocardial necrosis in combination with atleast one of the following:

1. Ischemic symptoms.
2. Development of pathologic Q waves on the ECG.
3. ECG changes indicative of ischemia as mentioned earlier (ST segment elevation or depression).
4. Imaging evidence of a new loss of viable myocardium or new regional wall motion abnormality.<sup>(1,2)</sup>

**Chest pain:** Cardiac pain is a sense of chest constriction and may be referred to arm, neck, jaw, teeth or even post scapular area. The new onset of chest pain may be appreciated by the patient preoperatively, post operatively or even intra-operatively if the patient has received regional or local anaesthesia. There is high propensity of diabetic patients presenting as silent MI due to the neuropathy associated with diabetes.<sup>(1,3,2)</sup>

**Electrocardiography:** Perioperatively, most of the time myocardial ischemia or infarction has been detected by ECG. The incidence of perioperative myocardial ischaemia is determined by the choice and number of precordial leads, on the definition of ischaemic ST segment change (Extent and duration of ST-segment change). During anaesthesia, electrocardiographic monitoring is limited to 5–7 leads. Among the ECG leads, lead V5 is considered most sensitive for the detection of ischemia or infarction, which can detect upto 80 – 85 %of the ischaemic events. Almost 100 %of the ST segment changes can be detected by monitoring with Lead V3 – V6, lead II and aVF. It is advised to calibrate ECG to deflection of 10mm for 1mV potential so as to avoid missing of minimal but significant ST segment depressions.<sup>(1,3,2,4)</sup>

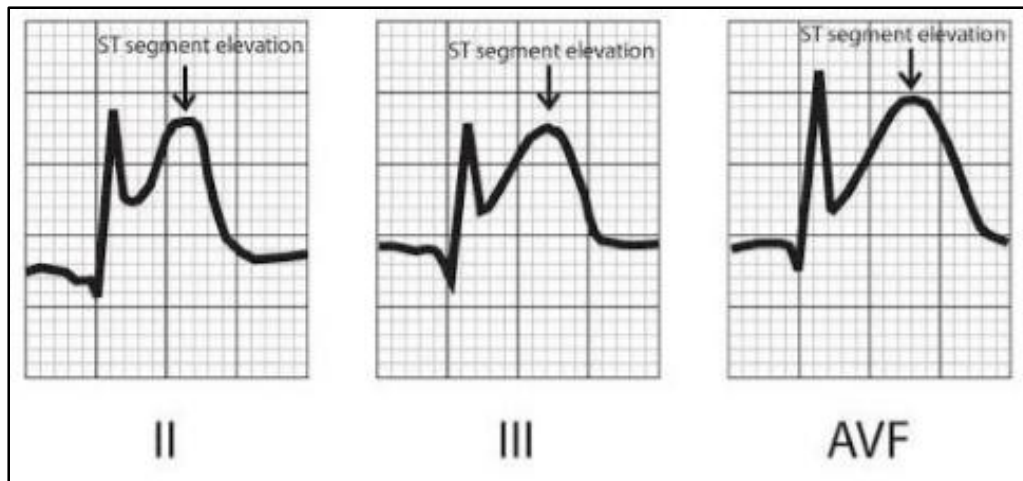
Horizontal or down sloping ST Segment depression of 1mm or more that coincides in time with anginal chest pain indicates significant subendocardial ischaemia. This may be accompanied by transient symmetrical T wave inversion. Patients with old h/o MI or IHD with chronic T-wave inversion may present as normalisation of T wave labelled as pseudo-normalisation of T wave indicating myocardial ischaemia

In a c/o infarction, ECG may show changes of subendocardial (ST segment depression) or transmural ischaemia (ST elevation >1mm). The vast majority of peri-operative myocardial infarctions are of the non-Q-wave type and preceded by episodes of ST-segment depression and T wave inversion. Long duration of ST segment change – single duration >20–30min or cumulative duration >1–2 h is associated with adverse cardiac outcome. Along with the ECG changes a very important finding includes elevation of cardiac biomarkers which forms one of the diagnostic criteria for the diagnosis of MI.<sup>(1,3,4)</sup>

**Figure: ECG strip showing ST Segment depression >2mm indicating ischaemia**



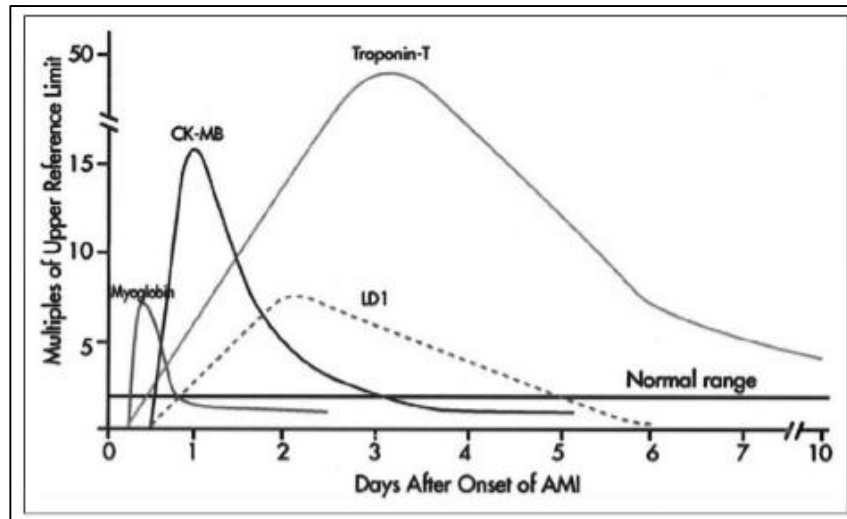
**Fig. 3**

**Figure: ECG strip showing acute myocardial infarction changes****Fig. 4**

**Cardiac biomarkers:** Troponin is a cardiac specific protein and biochemical marker for acute MI. An acute increase in the circulating concentration of troponin occurs early after myocardial injury. Cardiac troponin (T or I) increase within 3 hours after myocardial injury and remain elevated for 7 – 10 days. Elevated troponin levels and the ECG are powerful predictors of adverse cardiac events in patients with anginal pain. Troponin is more specific than CK-MB for determining myocardial injury. The currently accepted definition of MI recommends assessing the magnitude of the infarction by measuring quantitative elevation of cardiac enzymes above the normal reference range. <sup>(3)</sup>

<b>Biomarker</b>	<b>Range of time to initial elevation</b>	<b>Mean time to peak elevation</b>	<b>Time to return to normal</b>
CK-MB	3-12 hr.	24 hr.	48-72 hr.
Troponin I	3-12 hr.	24 hr.	5-10 day
Troponin T	3-12 hr.	12 hr. – 2 days	5-14 day
Myoglobin	1-4 hr.	6-7 hr.	24 hr.
CK-MB tissue isoform	2-6 hr.	18 hr.	Unknown
CK-MM tissue isoform	1-6 hr.	12 hr.	38 hr.

**Table 1: cardiac enzyme levels during Myocardial infarction and its time correlation.<sup>(2)</sup>**

**Figure: cardiac enzyme levels during MI.<sup>(1)</sup>****Fig. 5****Echo Cardiography:****Trans oesophageal echocardiography:**

TEE is a highly sensitive ischaemia monitor and demonstrates development of new regional wall motion abnormalities, decreased systolic wall thickening, and ventricular dilation. Usually, a trans gastric cross-sectional view of the left ventricle is imaged because this view displays the myocardial perfusion territories of the three major coronary arteries. The use of TEE has become increasingly common in the operating room for cardiac surgery but is less frequently used in non-cardiac surgery. The sudden appearance of severe wall motion abnormalities in patients being monitored by TEE may also indicate MI. It may be difficult to distinguish evolving infarction, stunned myocardium and hibernating myocardium using TEE.<sup>(1,2)</sup>

Limitations of TEE are; pre-intubation events are missed. The image plane may miss events in other areas of the myocardium. It has been shown that if the anaesthesiologist has the training, experience and equipment to perform TEE, it can be valuable in the early detection of myocardial ischaemia.<sup>(1)</sup>

**Definitions of Urgency and Risk:** An emergency procedure is one in which life or limb is threatened if not in the operating room where there is time for no or very limited or minimal clinical evaluation, typically within <6 hours.

An urgent procedure is one in which there may be time for a limited clinical evaluation, usually when life or limb is threatened if not in the operating room, typically between 6 and 24 hours.

A time-sensitive procedure is one in which a delay of >1 to 6 weeks to allow for an evaluation and significant changes in management will negatively affect outcome. Most oncologic procedures would fall into this category.

An elective procedure is one in which the procedure could be delayed for up to 1 year.<sup>(5,6)</sup>

**Risk Stratification:** In the process stratifying the risk preoperatively the guidelines integrate.

1. Clinical risk factors.
2. The type of surgical procedure the patient is undergoing.
3. Exercise capacity or the effort tolerance (metabolic equivalents).<sup>(2)</sup>

### **Clinical risk factors:**

#### **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 I.
- 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 >2mg% (renal failure).

#### **Minor Factors (Increased probability of coronary artery disease).**

- Familial history of coronary artery disease.
- Uncontrolled systemic hypertension.
- Hypercholesterolemia.
- Smoking.
- ECG abnormalities (arrhythmia, LVH, bundle branch block).
- Post infarction (>3 months), asymptomatic without treatment.<sup>(2)</sup>

#### **Risk Stratification Depending Upon Type of Surgery:**

1. High-risk procedures (Risk of perioperative adverse cardiac events >5%) includes Emergent major operations,  
Aortic and major vascular procedures.  
Anticipated prolonged procedures associated with large fluid shifts and/or blood loss.
2. Intermediate - risk procedures (risk of perioperative adverse cardiac events 1–5%) include.
  - Intermediate - risk procedures (risk of perioperative adverse cardiac events 1–5%) include.
  - Carotid endarterectomy,
  - Head and neck surgery,
  - Intraoperative and intrathoracic surgery,

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- Orthopedic surgery.
- Prostate surgery.

Low-risk procedures (risk of perioperative adverse cardiac events < 1%) include.

- Endoscopic procedures
- Superficial procedures
- Cataract surgery
- Breast surgery
- Ambulatory surgery.<sup>(2)</sup>

Risk stratification depending on functional capacity – DASI (Duke activity status index) and Metabolic equivalents.

### 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).

### > 10 METs

- Strenuous sports (Swimming, tennis, and bicycle).
- Heavy professional/domestic work like scrubbing floors, lifting or moving heavy furniture.

Metabolic equivalents	Functional levels of exercise
1	Eating, working at a computer, dressing
2	Walking downstairs or in your house, cooking
3	Walking 1-2 blocks
4	Raking leaves, gardening
5	Climbing 1 flight of Stairs
6	Playing golf, carrying clubs
7	Playing singles tennis
8	Rapidly climbing stairs, jogging slowly
9	Jumping rope slowly
10	Swimming quickly, running or jogging briskly
11	Skiing cross country, playing full-court basketball
12	Running rapidly for moderate to long distances

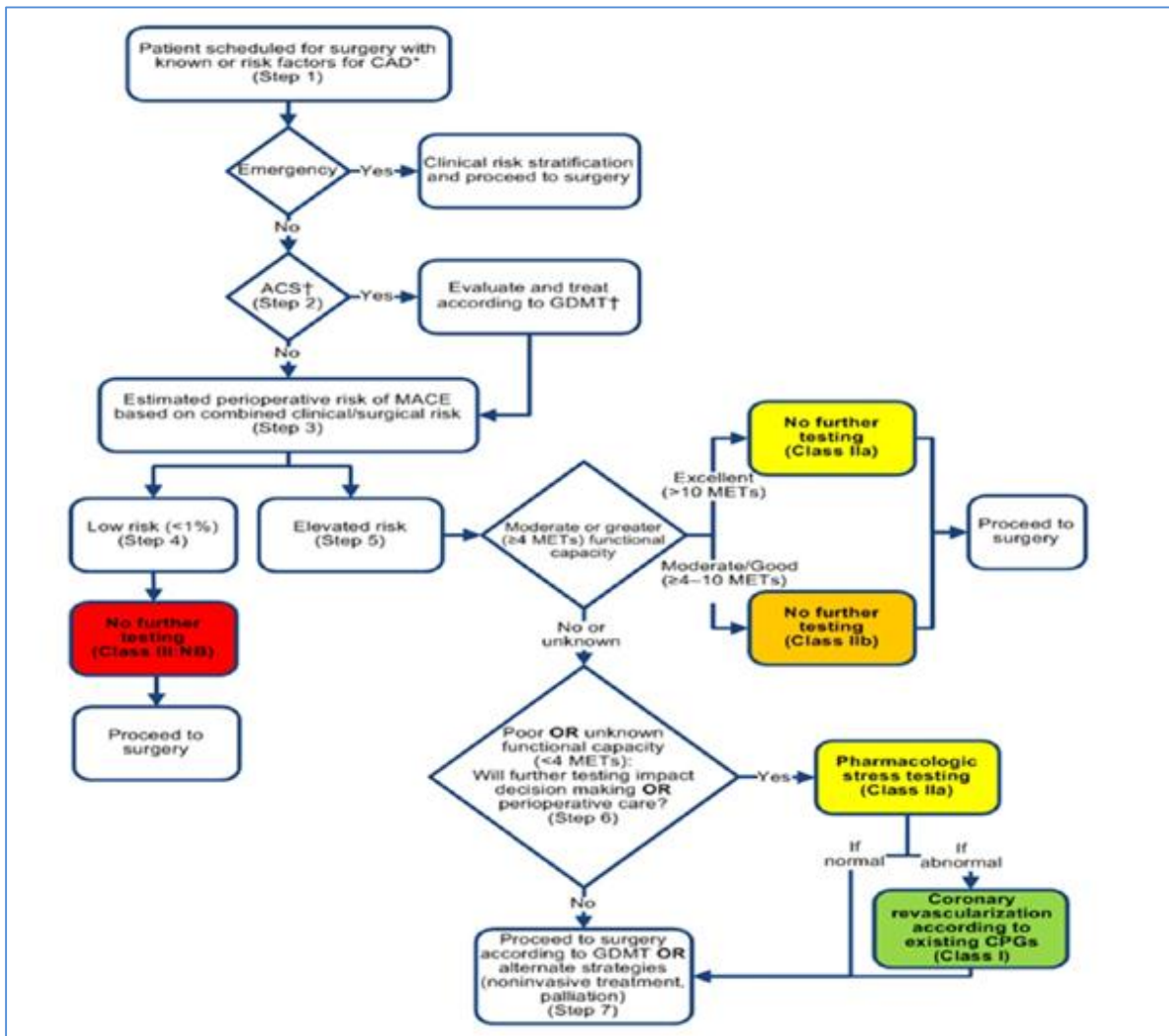
Table 2: Metabolic equivalents of functional capacity.<sup>(7)</sup>

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.



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**AHA Guidelines 2014:** A low-risk procedure is one in which the combined surgical and patient characteristics predict a risk of a major adverse cardiac event (MACE) of death or myocardial infarction (MI) of <1%. Selected examples of low-risk procedures include cataract and plastic surgery. Procedures with a risk of MACE of  $\geq 1\%$  are considered elevated risk. Many previous risk-stratification schema have included intermediate- and high-risk classifications. Because recommendations for intermediate- and high-risk procedures are similar, classification into 2 categories simplifies the recommendations without loss of fidelity.<sup>(6)</sup>



**Fig. 6**

### Indications for further non-invasive testing:

- Major clinical predictors.
- Poor functional capacity (< 4 METs).
- High risk procedure.

Resting LV function is not predictive of perioperative ischemic events, so in patients in whom further testing is indicated a stress test should be performed.

**The tests include:**

- Exercise stress testing.
- Non-exercise stress testing.
- Dobutamine stress Echo.
- Dipyridamole/adenosine thallium testing.
- Ambulatory ECG.<sup>(1,3,2)</sup>

**Exercise stress test/stress ECHO**

- It is the initial test of choice.
- Cost effective means of detecting coronary artery disease (Sensitivity 70 %, specificity 70 %).
- Provides an objective estimate of functional capacity.
- Detects Myocardial ischaemia or arrhythmia.
- Estimates long term prognosis.
- Simulates sympathetic nervous system stimulation that may accompany perioperative events – laryngoscopy/surgical stimulation.
- Interpretation is based on.
- Duration of exercise the patient is able to perform.
- The maximum heart rate that is achieved.
- Time of onset of ST segment depression on the ECG.
- The degree of ST segment depression.
- The time until resolution of the ST segment depression during the recovery period.
- **Limitations:** often exercise is contraindicated in high risk patients and cannot be performed by patients with poor exercise tolerance.<sup>(1,2)</sup>

**Pharmacologic stress testing:****Indications:**

- Elderly patients with decreased functional capacity and possible CAD.
- Patients with chronic debilitation and possible CAD.
- Younger patients with functional impairment due to injury, peripheral neuropathy, myopathies, or peripheral vascular disease, in which a maximal heart rate is not easily achieved with routine exercise stress testing because of an early onset of fatigue due to musculoskeletal, neurologic, or vascular problems rather than cardiac ischemia.
- Patients on beta-blockers or other negative chronotropic agents that would inhibit the ability to achieve an adequate heart response to exercise.

**Dipyridamole thallium myocardial imaging:**

- Dipyridamole an adenosine agonist is administered which Increases blood flow in normal coronary arteries.
- Images acquired by the co administration of a radioisotope, such as thallium - distributes to 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% and specificity 85–90%.<sup>(1,3,2)</sup>

## **Dobutamine stress testing:**

- Dobutamine is a synthetic catecholamine which directly stimulates both beta-1 and beta-2 receptors.
- A dose-related increase in heart rate, blood pressure, and myocardial contractility occurs just as similar to physical exertion, dobutamine increases regional myocardial blood flow based on physiological principles of coronary flow reserve.
- A similar dose-related increase in subepicardial and subendocardial blood flow occurs within vascular beds supplied by significantly stenosed arteries, with most of the increase occurring within the subepicardium rather than the subendocardium. Thus, perfusion abnormalities are induced by the development of regional myocardial ischemia.
- Dobutamine is infused in incremental doses starting at 5 mcg/kg/min for 3 minutes.
- Then, 10, 20, 30, and 40 mcg/kg/min are administered until the stress end point is reached.
- The end points are similar to those of exercise stress testing (e.g., target heart rate, chest pain with ECG changes, hypotension).
- The indications for early termination of dobutamine stress testing are similar to those of exercise stress testing.
- ST elevation and ventricular tachycardia are more likely with dobutamine stress testing than any other type of stress testing.
- Adverse effects requiring early termination subside within 5-10 minutes of discontinuation of the infusion as the half-life of dobutamine is 2 min.
- The effect of dobutamine can be reversed with beta-blockers.

**Coronary Angiography:** It is a invasive procedure indicated in cases of unstable coronary syndromes, evidence for high risk of adverse outcome based on noninvasive test results and if equivocal non-invasive test results in patients at high clinical risk undergoing high risk surgery.<sup>(1,3,2)</sup>

## **Management of Risk Stratification:**

Three therapeutic options are available before elective non-cardiac surgery

1. Revascularisation by surgery (CABG)
2. Revascularisation by PCI
3. Optimal medical Management <sup>(2)</sup>

## **Coronary artery bypass grafting**

### **Preoperative CABG should be reserved for patients with:**

- Acceptable coronary revascularization risk and suitable viable myocardium with left main stenosis.
- Three vessel CAD in conjunction with LV dysfunction.
- Two vessel disease involving severe proximal LAD obstruction.
- Intractable coronary ischemia despite maximal medical therapy.
- CABG is indicated only if anatomy or symptoms mandate CABG independent of the planned noncardiac surgery.
- For major noncardiac surgery following recent CABG, a gap of at least 4–6 weeks possibly for even up to 6 months is necessary.

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**Percutaneous coronary intervention:** Percutaneous coronary angioplasty was introduced as an alternative to CABG to mechanically open stenosed coronary arteries. It was effective, but restenosis of the angioplasty site occurred in 15 – 60 % of patients. To solve the problem of abrupt coronary closure after angioplasty, bare metal stents were introduced. However, coronary restenosis due to neointimal hyperplasia was observed in around 30% of the patients with BMS. Drug eluting stents were introduced to reduce the neointimal hyperplasia and subsequent stenosis.

Mechanically opening a blood vessel by angiography causes vessel injury, especially destruction of the endothelium. This makes the area prone to thrombosis. It takes about 2 to 3 weeks for the vessel to reendothelialize after balloon angioplasty. After BMS placement, reendothelialization can take up to 12 weeks, and a DES may not be completely endothelialized even after 1 year. Thus thrombosis after angioplasty and stent placement is a major concern.

Stent thrombosis is categorized by the time interval between its occurrence and the PCI:

- Acute (within 24 hours),
- Subacute (between 2 and 30 days),
- Late (between 30 days and a year), and
- Very late (after a year).

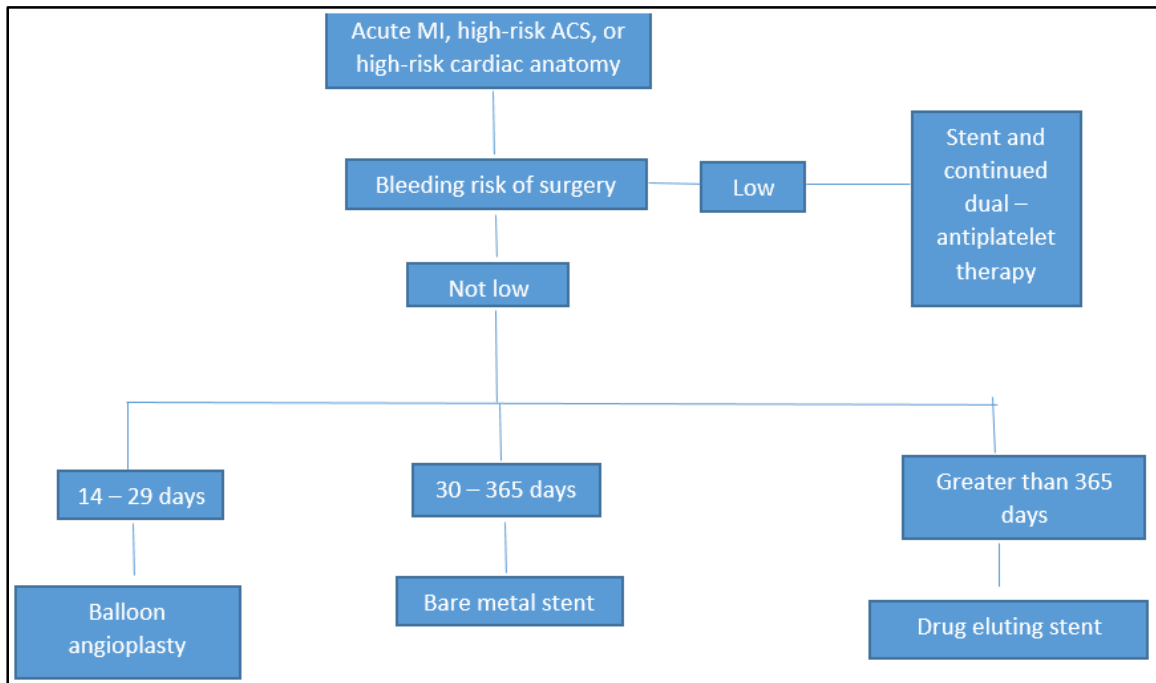
Early stent thrombosis is usually mechanical in origin and due to coronary artery dissection or under expansion of the stent. In contrast, late stent thrombosis is typically related to stent malposition, abnormal reendothelialization, or hypersensitivity. Platelets play an important role in the pathophysiology of stent thrombosis, and use of antiplatelet drugs is critical in these patients until the stent becomes less prone to thrombosis.

Five factors should be considered when caring for a patient with a coronary stent.

1. Timing of the operation after PCI – PCI to surgery interval.
2. Continuation of dual antiplatelet therapy.
3. Perioperative monitoring strategies.
4. Anaesthetic technique.
5. Immediate availability of an interventional cardiologist.<sup>(13,2,8)</sup>

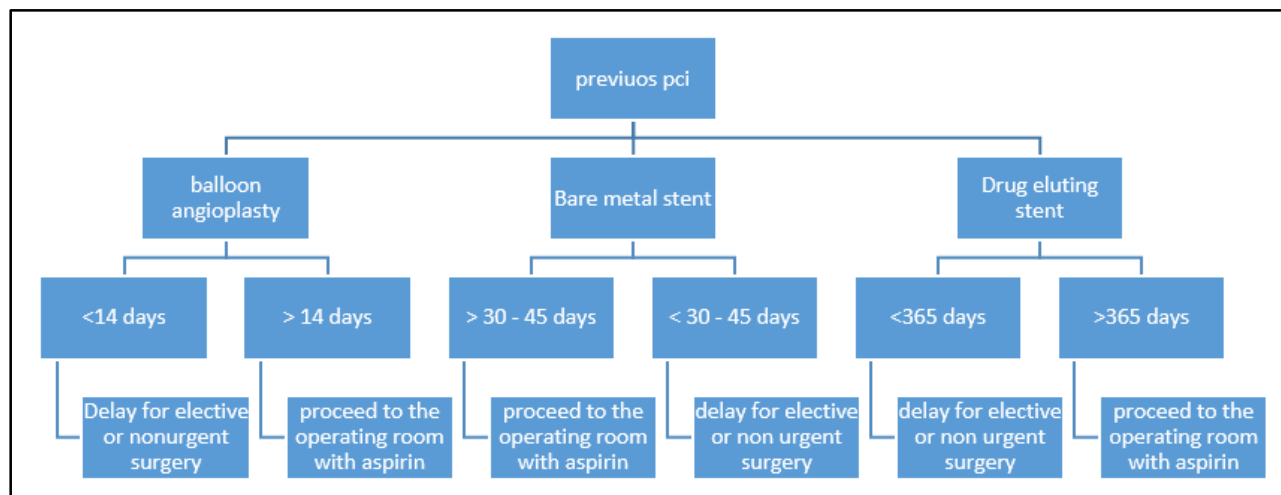
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**Figure: Proposed treatment for patients requiring percutaneous coronary intervention (PCI) who need subsequent surgery. <sup>(5)</sup>**



**Fig. 7**

**Figure: proposed approach to the management of patients with previous percutaneous coronary Intervention. <sup>(5)</sup>**



**Fig. 8**

### Pharmacological optimization:

**Beta blockers:** It forms the mainstay of treatment both for cardiac prophylaxis and treatment following an ACS. If patient is already on beta blockers, it has to be continued perioperatively, if

withdrawn suddenly there is again propensity for rebound hypertension. Helps in maintaining balance between demand – supply ratio by negative chronotropic and inotropic effect. If beta blockers are used for prophylactic purposes in the perioperative period, they should be initiated at least a week before elective surgery and acute administration of high dose beta blockers in high risk populations is not recommended. For ease of dosing and consistency of effect, longer acting beta blockers such as atenolol is more efficacious in the perioperative period.

The AHA guidelines 2014 regarding the perioperative use of beta blockers for non-cardiac surgery concludes that perioperative “beta blockade started within 1 day or less before non-cardiac surgery helps prevent nonfatal MI but at the cost of increased risks of stroke, death, hypotension, and bradycardia.”<sup>(1,2,9)</sup>

**Anti platelet therapy:** Aspirin forms an integral part of the acute therapy not only after an ACS but also in patients not known to have a pre-existing CAD. It eliminates the diurnal variation in plaque rupture. Apart from reducing platelet aggregation, its anti-inflammatory effect may be additive to its antithrombotic effect in patients with plaque instability.

For those patients who are on combination of two APA, the anaesthesiologist needs to discuss the risk benefit ratio with the surgical team and preferably put the patient on monotherapy at least 5 days preoperatively.

### **AHA recommendations post – PCI:**

- In patients receiving a stent (BMS or DES) during PCI for ACS, P2Y12 inhibitor therapy should be given for at least 12 months. Options include clopidogrel 75 mg daily, prasugrel 10 mg daily and ticagrelor 90mg twice daily.
- In patients receiving DES for a non-ACS indication, clopidogrel 75 mg daily should be given for at least 12 months if patients are not at high risk of bleeding.
- In patients receiving BMS for a non-ACS indication, clopidogrel should be given for a minimum of 1 month and ideally upto 12 months (unless the patient is at increased risk of bleeding; then it should be given for a minimum of 2 weeks.<sup>(5)</sup>

### **1. Statin therapy:**

- Hydroxy methyl glutaryl coenzyme A (HMGCoA) reductase inhibitors or statin therapy is associated with major benefits in patients with vascular disease.
- Pleiotropic effects of statins independent of their lipid lowering action have been proposed as the mechanism of their beneficial effects.
- Statins cause stabilization of the atherosclerotic plaque surface and cap, making plaque rupture less likely and enhancing plaque remodelling
- Statins should be continued perioperatively.

### **2. ACE inhibitors:**

- ACE inhibitors have a proven benefit in patients with a recent ACS and also in patients with vascular disease and normal left ventricular function.
- Reduction in incidences of progression to microalbuminuria in diabetics.
- ACE inhibitors have anti-ischaemic actions with a 20% relative reduction for myocardial infarction.

### 3. Alpha 2 adrenergic receptor agonists:

- These drugs attenuate perioperative haemodynamic instability, inhibit central sympathetic discharge, reduce peripheral norepinephrine release, and dilate post-stenotic coronary vessels.<sup>(1,2,6,7)</sup>

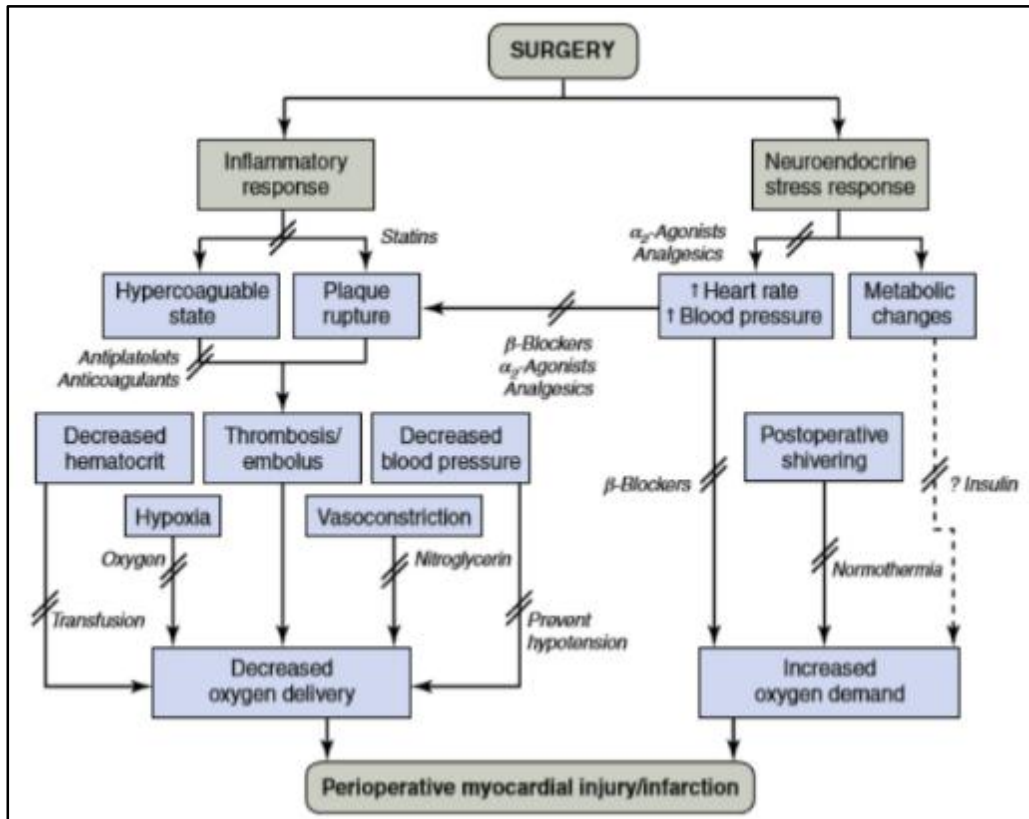


Fig. 9

Figure: interventions that can modulate the triggers of perioperative myocardial infarction.<sup>(2)</sup>

#### INTRAOPERATIVE MANAGEMENT:

##### Induction of anaesthesia:

- Intravenous anesthetic agents have a direct depressant action on the myocardium and also reduce vascular tone leading to hypotension and compensatory tachycardia which leads to myocardial ischemia.
- Small incremental doses preferred.
- Ketamine avoided: sympathetic stimulation causing tachycardia and hypertension.
- Opioid induction preferred: fentanyl 50 – 150ug/kg
- Intubation: minimise duration of laryngoscopy to < 15sec.<sup>(2)</sup>

##### MAINTAINANCE OF ANAESTHESIA:

- Volatile inhalational agents: decreases myocardial contractility and cardiac output.
- Prestenotic vasodilation – especially isoflurane diverts blood away from ischaemic areas precipitating coronary steal phenomenon.
- Vagal stimulation causing bradycardia and nodal rhythm, mainly seen with halothane.

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- Iatrogenic hyperventilation causing reduction in paco<sub>2</sub> which produces coronary vasoconstriction.(2)
- Adequate pain relief by the use of optimal use of opioids.
- NSAIDS: antiplatelet and anti inflammatory activity.
- Muscle relaxants: muscle relaxants with minimal or no effect on heart rate and systemic blood pressure are preferred. Ex: Vecuronium, rocuronium or cisatracurium.
- Reversal: Anticholinesterase/anticholinergic drug combination safely used.
- Glycopyrolate: less chronotropic.
- Maintain normothermia: because hypothermia causes vasoconstriction and increase in afterload.

### **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.
- Adequate ventilation and oxygenation.
- Adequate anaesthetic depth.
- Beta blockers (Esmolol or metoprolol) - administered in a titrated manner.

### **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.
- Volume replacement.
- Restore coronary perfusion pressure and.
- Slow the heart rate.

### **OTHER ASPECTS OF TREATMENT:**

- Morphine - venodilator that reduces ventricular preload and oxygen requirements.
- It is the analgesic of choice for continuing pain unresponsive to nitrates, and it is also effective in patients with pulmonary vascular congestion complicating ACS.
- Aspirin 325 mg is administered orally (through ryle's tube if unable to take orally) and is continued thereafter.
- Hypotension should be rapidly treated in order to restore coronary perfusion pressure (CPP). Moderate hypotension often responds to volume expansion with 300 – 500 ml of crystalloid.
- If severe hypotension (60-80mmHg systolic) persists despite volume expansion, vasoactive or inotropic drugs may be given to elevate CPP above critical value.(2)

### **TREATMENT OF CARDIAC DYSRHYTHMIAS:**

- Correct metabolic acidosis (Due to low cardiac output),
- Correct electrolyte imbalance (Especially hypokalemia),
- Ensure good oxygenation.
- Treat arrhythmias appropriately.



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- Sinus bradycardia - Common following acute MI especially inferior wall infarctions reflecting acute ischaemia of SA/ AV node. Treatment with atropine and a temporary pacemaker is needed when there is haemodynamic compromise. Patients presenting with severe bradycardia requires emergency cardiac pacing.
- Atrial fibrillation - Occurs in > 10% of patients following acute MI. Result from an acute increase in left atrial pressure caused by LV dysfunction. When AF is haemodynamically significant – cardio version should be promptly done.
- Ventricular tachycardia (VT) - common following acute MI. Sustained or haemodynamically significant VT is treated with electrical defibrillation. Asymptomatic VT is treated with lidocaine, procainamide, or amiodarone.
- Ventricular fibrillation (VF) - occurs in 3-5% of patients with acute MI. Prophylactic lidocaine is no longer recommended because of an increased incidence of bradydysrhythmias and associated asystole. When VF occurs, rapid defibrillation with 120-200 Joules biphasic or 360 Joules monophasic current should be administered.

### THROMBOLYTIC OR REPERFUSION THERAPY:

- Thromboplastin activator (t-PA) or streptokinase is recommended to minimize the damage caused by intraoperative infarct.<sup>(1,3,2)</sup>
- To be effective, they should be preferably given within 4 hrs. (Maximum up to 12 hrs.).
- The major limitation is a predisposition for bleeding. So it is contraindicated in patients with fresh surgical wounds. However it may be useful in patients who have suffered PMI after procedures like cystoscopy or direct laryngoscopy.

### INTRA AORTIC COUNTER PULSATION DEVICE:

- It acutely decreases myocardial oxygen requirements and may increase myocardial oxygen supply.<sup>(1,2)</sup>

**CONCLUSION:** As more and more patients coming for non-cardiac surgeries who have already undergone coronary intervention such as balloon angioplasty, stenting or CABG, we as anaesthesiologists should have thorough knowledge of the perioperative implications of the same in a day to day practice. Secondly, as the geriatric population is increasing there is more chances of encountering patients with known or unknown ischaemic heart disease both on an emergency and elective basis.

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