Review Article

Indian Journal of Anesthesia and Analgesia
2017, 4 (4, Part-II), 1272-1275
DOI: http://dx.doi.org/10.21088/ijaa.2349.8471.4417.56

Myocardial Oxygenation during Preoperative Period

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Abstract

Adequate myocardial oxygenation during the perioperative period is crucial for a good postoperative outcome. This requires the maintenance of myocardial oxygen supply demand ratio, which can be disrupted by various perioperative events. Normally there is a significant reserve in the myocardial oxygen supply, but it can be reduced in pathologic conditions like coronary artery disease, anemia and hypoxemia, which can result in myocardial ischemia. Though myocardial oxygenation is autoregulated over a wide range of perfusion pressures, perioperative events that affect the heart rate, myocardial contractility and coronary perfusion pressure can precipitate myocardial ischemia. Optimal outcome of these patients require good anaesthetic planning and prompt manipulation of any adverse hemodynamic events during the perioperative period.

Keywords: Myocardial Oxygenation; Perioperative Period.

Amongst the human organs, heart has the highest oxygen consumption per tissue mass. The resting coronary blood flow is about 250 ml min[1] (5% of cardiac output). The myocardial arterial oxygen extraction is 70–80%, as against 25% for the rest of the body tissues [1]. Hence increase in oxygen consumption must be met by an increase in coronary blood flow.

Myocardial Oxygen Supply and Demand

Myocardial oxygen supply is determined by the coronary blood flow and its oxygen carrying capacity. Under normal conditions, oxygen demand is met by its supply with a significant reserve. In pathologic conditions like coronary artery disease, anemia and hypoxemia, the supply may be reduced causing an imbalance resulting in ischemia [2]. Perioperative factors, such as changes in blood rheology, hematocrit, and coronary collateral blood flow also influence the myocardial oxygen supply demand ratio.

When the myocardial oxygen demand exceeds its supply, the resulting imbalance can cause myocardial ischemia. Myocardial ischemia may result in reversible (myocardial stunning) or irreversible damage (infarction), ventricular arrhythmias, or cardiogenic shock.

Determinants of Coronary Blood Flow

Heart Rate: The single most common abnormality associated with myocardial ischaemia is tachycardia, which causes both an increase in demand and reduction in oxygen supply [3]. Coronary blood flow occurs mainly during the diastolic phase, as during this phase the compressive forces are minimum and coronary resistance is at its nadir. Any factor that shortens this phase such as increase in the heart rate or

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Received on 06.12.2017, **Accepted on** 20.12.2017

prolonged ventricular systole will impair the blood supply. This is most pronounced in the subendocardium and hence this region is most susceptible to ischemia. Changes in diastolic phase have minimal impact in blood flow to the epicardial layers which are perfused throughout the cardiac cycle.

Increase in heart rate also leads to increased oxygen consumption and compromise the supply by an increase in the demand. This is most affected in the left ventricular subendocardium.

Coronary Perfusion Pressure

Coronary perfusion pressure is the difference between the aortic diastolic pressure and left ventricular end-diastolic pressure (LVEDP). During systole, intramyocardial blood vessels get compressed and twisted, decreasing the coronary blood flow to the left ventricle to its lowest level. The force is greatest in the subendocardium, where it is almost equal to the intramyocardial pressure and decrease from endocardium to epicardium. Blood flow resumes during diastole when the myocardium relaxes. Thus, the duration of diastolic phase is crucial to the subendocardial perfusion. Alteration in coronary flow to the right ventricle is less pronounced because of the lesser amount of myocardial mass and force of contraction.

Myocardial Contractility

Myocardial contractility is influenced by the autonomic nervous system, heart rate, blood calcium level, temperature, and other factors. Left ventricular preload and afterload also affect myocardial oxygen demand by altering end-diastolic and end-systolic wall tension. Greater the contractility, the more oxygen the myocardium consumes.

Systolic Wall Tension

Myocardial wall tension is proportional to ventricular systolic pressure, ventricular radius, and inversely proportional to ventricular wall thickness (Laplace law). Preload influences the ventricular radius and afterload the magnitude of systolic pressure. Systolic wall tension decreases with increasing ventricular wall thickness.

Autoregulation

Under normal conditions, myocardial blood flow is autoregulated over a range of perfusion pressures of 60-160 mm Hg to maintain perfusion. When perfusion pressure falls below this level, coronary arteries dilate and flow becomes pressure dependant. In pathologic conditions (e.g. coronary artery disease) the perfusion pressure may fall below this critical threshold, thereby rendering the distal vascular bed maximally dilated and dependent on the perfusion pressure. In severe coronary stenosis, ability of the distal vascular bed to dilate will become exhausted. In this situation, the autoregulation fails to maintain coronary blood flow, and perfusion becomes dependent on coronary perfusion pressure.

Metabolic Control

The metabolic rate of myocardium is related to its blood flow, and the metabolite act as a potent coronary vasodilator. When the myocardial metabolism exceeds production of this substrate, it induces coronary vasodilation. Myocardial metabolism releases adenosine which accumulates with increased metabolic rates or decreased blood flow causing vasodilation and increase in blood flow. Diminished oxygen tension also has vasodilatory effect on smooth muscle cells and on tone of pre-capillary sphincters of vascular beds.

Perioperative Period

Perioperative myocardial infarction can occur due to mismatch between the myocardial oxygen supply and demand, due to various factors which increase the sympathetic activity. This supply demand relationship can be favourably altered to reduce the myocardial oxygen demand while increasing its supply using pharmacologic and anesthetic interventions. Prevention of myocardial ischemia is more important than the type of anesthetic or the drugs chosen.

The morbidity associated with ischemia during anesthesia is difficult to predict. Increase in heart rate may be tolerated in a patient with mild coronary artery disease, but may result in global deterioration of left ventricular function due to profound ischemia in a patient with severe multi-vessel disease.

Pharmacologic Prophylaxis

β-blockers: β- blockers cause a decrease in sympathetic tone and myocardial contractility, preventing myocardial ischemia by maintaining the myocardial oxygen supply demand balance, thus reducing perioperative morbidity and mortality [4].

The anti ischemic mechanism of β-blockers is due to reduction in heart rate and myocardial contractility. A target heart rate of 50-70 beats per minute is commonly recommended. Decrease in heart rate increases the duration of diastole, enhancing the coronary perfusion time, increases subendocardial blood flow, and reduces myocardial oxygen consumption. β antagonists also cause "reverse" coronary steal by increasing coronary vascular tone in normal regions by reducing the oxygen demand. These drugs attenuate the adverse effects of sympathetic nervous system activation, including increase in heart rate and myocardial contractility, decreases in coronary blood flow secondary to constriction of large epicardial coronary vessels. β-blockers also have antiarrhythmic properties. The reduction in heart rate is compensated by an increase in stroke volume *via* Frank-Starling mechanism, maintaining the cardiac output.

β-blockers should be started at least 24 hours before elective procedures and titrated to attain a heart rate of 50-60 beats per minute. Atenolol has been shown to be anti-ischemic, protect against myocardial reinfarction, and reduce the mortality during the perioperative period [5]. Esmolol administered to maintain a heart rate lower than the ischemic threshold has been found to reduce the incidence of postoperative ischemic events.

α^2-Adrenergic agonists (Clonidine, Dexmedetomidine): These drugs has sedative, anxiolytic and analgesic properties and reduce the central sympathetic nervous system activity by preventing tachycardia, hypertension, and increased sympathetic tone. They have advantage over β-blockers because of their ability to attenuate the adverse effects of sympathetic nervous stimulation mediated by peripheral á- as well as â-adrenoceptors.

Nitrates

Nitroglycerin reduces myocardial oxygen demand by decreasing left ventricular preload and end-diastolic wall tension. It also increases the coronary collateral perfusion by dilating large epicardial coronary arteries and collateral conduit vessels. It is also a donor of nitric oxide, which may have direct cardioprotective properties.

Nitrates can be deleterious if a decrease in coronary perfusion pressure occurs, especially in patients with hypovolemia. Increasing intravascular volume and temporary use of phenylephrine may be useful.

Calcium channel antagonists: Nifedipine and nicardipine have action on peripheral arterial tone and may produce baroreflex-mediated increases in heart rate that can result in an increase in myocardial oxygen consumption. Diltiazem reduces heart rate and may offer some benefit in the treatment of intraoperative ischemia.

Factors Affecting Myocardial Oxygen Supply-Demand Balance

Prevention of myocardial ischemia by optimizing myocardial oxygen supply and reducing myocardial oxygen demand (maintainance of the myocardial oxygen balance) is more important than choosing a specific technique or drug for anaesthesia. A common recommendation is to keep the heart rate and blood pressure within 20% of the normal awake value.

Anaesthetic Goals

The primary aim is to avoid factors which impair the perioperative myocardial oxygen supply-demand balance, thereby preventing myocardial ischemia rather than choosing a particular anaesthetic drug or technique. Anaesthetic management should focus upon to keep the myocardial oxygen supply greater than the demand to avoid ischemic events.

Pre-Operative Preparation

- Patients stabilized on beta blockers should continue it throughout the perioperative period.
- Anxiolysis (Psychological and pharmacological if indicated) to prevent tachycardia.
- Avoid tachycardia inducing drugs in premedication (e.g. atropine)

Induction of Anaesthesia

Most intravenous inducing agents are myocardial depressants and cause decrease in systemic vascular resistance.

- Etomidate is usually preferred due to its minimal cardiovascular effects.
- Avoid drugs causing significant hypotension (Thiopentone sodium) or hypertension (Ketamine).
- Maintainance of stable hemodynamics is more important than selection of the drug.
- A quick gentle laryngoscopy (within 15 sec.) is more important than using drugs to blunt the

tracheal intubation response (lidocaine, esmolol, fentanyl).

Maintatinance of Anaesthesia

- Prevent undesirable fluctuations in heart rate and blood pressure (proper depth of anaesthesia, adequate analgesia, gentle surgical manipulations).
- Adequate neuromuscular blockade using cardiostable drugs (rocuronium, vecuronium, cisatracurium)

Reversal of Anaesthesia

- Reverse after adequate recovery
- Glycopyrrolate is preferred to atropine
- Extubation should be smooth, avoiding sympathetic stimulation (opioids or â-blockers used).

Postoperative Management

- Elective ventilation if indicated
- Prevent tachycardia, hypercarbia
- Adequate pain relief (epidural, opiates)
- Prevent hypothermia, shivering
- Continue monitoring till stable hemodynamics
- Monitor for fresh ischemic insults

Regional Anaesthesia causes reduction of intraoperative stress responses, which is a great advantage in maintaining the myocardial oxygen balance. However, central neuraxial blockade can cause undesirable systemic hypotension, which can be prevented by optimal preload and use of vasopressors like phenylephrine. It avoids intubation responses and the adverse effects of positive pressure ventilation. Epidural or intrathecal local anesthetics or opioids may be beneficial for effective postoperative analgesia patients with coronary artery disease.

Regional anaesthesia alone or in combination with general anaesthesia is often better choice in selected cases, provided the myocardial oxygen balance can be maintained (oxygen supplementation, prevention of blood loss, adequate hydration, maintaining acceptable heart rate and blood pressure).

Conclusion

Stress associated with anaesthesia and surgery along with unfavorable perioperative events can be detrimental in patients prone for myocardial ischemia[6]. Anaesthetic management should focus on maintaining the perioperative myocardial supply-demand balance, rather than choosing individual drugs or techniques. Vigilant monitoring should be adopted for the early detection and treatment of ischemic events for a good outcome in high risk patients.

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