Cardiovascular system: critical incidents

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Abstract

Cardiovascular events are an important group of critical incidents in anaesthesia. They include hypotension, hypertension, myocardial ischaemia/infarction, arrhythmias and cardiac arrest. These incidents require prompt recognition to stabilise the patient.

Keywords Arrest; arrhythmia; hypertension; hypotension; infarction; ischaemia

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Learning objectives

After reading this article, you should be able to:

- understand the meaning of the term 'critical incident' in the anaesthetic literature
- appreciate the generic presentations of acute cardiovascular disturbance
- be able to outline the management of critical cardiovascular incidents.

Introduction

A critical incident is an occurrence that leads to an undesirable outcome, or which could have done if not corrected in time.[1] Cardiovascular events represent a particularly important group of anaesthesia critical incidents with the intraoperative incidence reported at 5%–15%.[2] Perioperative cardiac morbidity is a leading cause of death following surgery and those with pre-existing cardiac disease are most at risk.[3] This article discusses hypotension, hypertension, myocardial ischaemia/infarction, bradycardia, tachycardia and cardiac arrest. The ability to manage these generic events is an essential skill for all anaesthetists. In addition, it includes consideration of other rarer situations that may manifest under anaesthesia with life-threatening cardiac symptoms.

Hypotension

Perioperatively hypotension can be defined as a drop of more than 20% from the baseline systolic blood pressure (BP). It is a common problem whose potential causes can be considered as:

- inadequate preload (e.g. hypovolaemia, tension pneumothorax, pericardial tamponade, pulmonary embolism)
- impaired cardiac function (e.g. arrhythmias, ventricular failure, negative inotropes)
- decreased systemic vascular resistance (e.g. central neuraxial block, vasodilating drugs, sepsis, anaphylaxis).

In addition to BP monitoring, other manifestations of significant hypotension may reflect the changes in cardiac output (e.g. reduced end-tidal CO_2) or in systemic vascular resistance (e.g. pathological vasodilation or compensatory vasoconstriction).

Immediate management is directed towards confirmation of the condition and rapid treatment:

- ensure adequate oxygenation and ventilation
- stop or review any vasodilating or negatively inotropic drugs
- increase preload with intravenous fluids and consider lifting the patient's legs
- administer an intravenous bolus of a vasopressor (e.g. metaraminol or ephedrine).

Subsequent management will be guided by the likely cause and the response to treatment. If hypotension persists, further measures may include additional monitoring (e.g. invasive pressures, urinary output, and haemoglobin concentration) and inotrope and/or vasopressors infusions.

Hypertension

Like hypotension, hypertension is common in anaesthesia and usually regarded as significant when more than 20% from baseline. 'Severe hypertension' is defined in absolute terms as a blood pressure of ≥180/≥110 mmHg, while 'hypertensive emergencies' are characterised by acute damage to target organs (e.g. myocardial ischaemia).[4]

The causes of perioperative hypertension may be classified as follows:

- anaesthetic factors (e.g. airway instrumentation, hypoxia, hypercapnia, inadequate analgesia/anaesthesia, sympathomimetic drugs)
- surgical factors (e.g. tourniquet pain, pneumoperitoneum, aortic cross-clamping)
- cardiac disorders (e.g. essential hypertension)
- extra-cardiac disorders (e.g. pre-eclampsia, raised intracranial pressure, thyroid storm, phaeochromocytoma).

Immediate management depends upon aetiology and severity:

- ensure adequate oxygenation and ventilation
- stop the surgery if necessary
- check drug administration, increase the depth of anaesthesia, and give analgesia
- consider other potential causes, giving consideration to the clinical context
- in the absence of a correctable cause, severe hypertension warrants symptomatic treatment to treat or prevent acute target organ damage. If there is an associated tachycardia, β-blockade (e.g. esmolol) is helpful. Otherwise, administer a vasodilator either as a bolus (e.g. phentolamine) or an infusion (e.g. glyceryl trinitrate).

Further management involves monitoring of arterial pressure, detection of target organ damage (e.g. 12-lead ECG), and treatment of the underlying cause.

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Myocardial ischaemia/infarction

Ischaemia occurs when there is an imbalance between myocardial oxygen supply and demand with the perioperative incidence, in at-risk patients, quoted between 20 and 60%.[5] The greatest risk occurs in the early postoperative period, with ischaemia during emergence from anaesthesia.[6]

The clinical expressions of myocardial ischaemia include symptoms, ECG abnormalities, and haemodynamic disturbance. Unfortunately, the sensitivity and specificity of these modalities is limited. For example, ischaemia is often silent and ECG changes may be subtle or shortlived.[6,7] However, intraoperative ischaemia is most commonly detected by ECG, with ST-segment deviations of at least 1 mm from baseline considered significant.[8] Multi-lead monitoring is recommended, and the combination of leads II and V5 will detect 80% of all ischaemic changes.[7,8] In the perioperative period, the risk of bleeding complicates the otherwise routine use of antithrombotic therapies and coronary intervention. Immediate management must therefore be directed towards the haemodynamic and oxygen-carriage factors that shape the equilibrium between myocardial oxygen supply and demand:

- correct the factors outlined in Table 1. The pathogenic importance of tachycardia should be emphasised, because it disturbs both the supply and demand aspects of oxygen balance
- consider invasive monitoring to help guide haemodynamic therapy
- discuss the problem with the surgeon and consider abbreviating the surgical plan

Postoperative management may include cardiology referral and admission to a high dependency environment. Further investigations aim to stratify risk (e.g. serial ECGs and troponins) and assess myocardial function (e.g. echocardiography to assess new regional wall motion abnormalities).

Myocardial infarction: most perioperative myocardial infarctions occur within 24–48 hours after surgery and not on days 3–5 as previously reported.[7]

Bradycardia[9]

Bradycardia is usually defined as an absolute heart rate of less than 60 beats/min, but is better described as any rate inappropriately slow for the haemodynamic state. In the normal heart, cardiac output is not significantly altered until the heart rate drops below about 40 beats/min.

Factors that impair myocardial oxygen supply–demand balance

Reduced supply	Increased demand
Reduced coronary blood flow	Tachycardia
• Tachycardia	Increased wall tension
• Hypotension	• Hypertension
Reduced arterial oxygen content	• Hypervolaemia
• Anaemia	Increased contractility
• Hypoxaemia	

Table 1

The causes of bradycardia include:

- cardiac disorders (e.g. sick sinus syndrome, atrioventricular [AV] block)
- extra-cardiac disorders (e.g. raised intracranial pressure)
- anaesthetic factors (e.g. hypoxia, tracheobronchial suction, remifentanil, high central neuraxial block)
- surgical factors (e.g. ocular stimulation, cervical dilatation)

Immediate management involves identifying the cause and assessment of the rhythm/haemodynamic status:

- ensure adequate oxygenation and ventilation. Bradycardia in children is commonly due to hypoxia
- stop surgical causes
- define the rhythm by 12-lead ECG; if this is not immediately feasible ensure that lead II is displayed on the monitor (since the axis of lead II parallels the cardiac axis)
- haemodynamic instability is suggested by shock, syncope, myocardial ischaemia, or heart failure. Promptly treat the unstable patient with atropine. Other pharmacologic options for symptomatic bradycardia include glycopyrrolate, adrenaline, and glucagon.

Cardiac pacing will be required if the patient fails to respond to atropine or if there is a risk of asystole (which is suggested by recent asystole, Möbitz type II block, complete heart block, or ventricular standstill >3 seconds). Transcutaneous (external) pacing may be employed until the transvenous approach is established; set the rate to 80/min and adjust the current to achieve capture. Seek expert help.

Tachycardia[9]

Tachycardias are characterised by at least three consecutive complexes occurring at a rate exceeding 100 beats/min. In the normal heart, haemodynamic instability is uncommon with ventricular rates below 150 beats/min (because the elevated rate maintains cardiac output despite a reduction in stroke volume). Patients with impaired cardiac function will demonstrate instability at lower rates.

Causes of the tachycardias include:

- cardiac disorders (e.g. myocardial ischaemia, long QT)
- extra-cardiac disorders (e.g. electrolyte abnormalities, pyrexia, thyrotoxicosis, phaeochromocytoma)
- anaesthetic factors (e.g. hypoxia, hypercapnia, inadequate analgesia/anaesthesia, sympathomimetic drugs)
- surgical factors (e.g. tourniquet pain).

The immediate management of tachycardia follows the same general principles as for bradycardia. With tachycardia, haemodynamically unstable patients require synchronised cardioversion.

Stable patients allow for precise rhythm diagnosis and pharmacological therapies. Treatment algorithms for stable tachycardia are based primarily upon the QRS duration and the regularity of the complexes.

- **Regular narrow-complex tachycardia:** causes include sinus tachycardia, paroxysmal supraventricular tachycardia, and atrial flutter. Sinus tachycardia represents a normal response to an underlying process (e.g. sepsis) and treatment should be directed at the cause. Atrial flutter frequently produces a tachycardia of 150 beats/min, which corresponds to an atrial rate of 300/min with 2:1 conduction. Flutter may require rate control with β-blockade.