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 and treatment.

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

Royal College of Anaesthetists CPD Matrix: 2A06

Learning objectives

After reading this article, you should:

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

Introduction

A critical incident is an occurrence that either leads to an undesirable outcome or could have done if not corrected.[1] Cardiovascular events represent a particularly important group of anaesthesic 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. The ability to manage these events is an essential skill for all anaesthetists. This article also considers some rarer situations which 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. 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 blood pressure measurement, other monitors reflect the changes in cardiac output including loss of the pulse oximeter trace and a fall in the measured end tidal CO_2. 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 such as 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. urine output, haemoglobin concentration, invasive pressures, cardiac output monitoring) plus vasopressors and/or inotrope infusions.

Hypertension

Hypertension is also common under 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 or higher, while 'hypertensive emergencies' are characterized by acute damage to target organs (e.g. myocardial ischaemia).[3] 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 cause 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 prevent or treat acute target organ damage. If there is an associated tachycardia, β-blockade may be helpful. Otherwise, administer a vasodilator either as a bolus or an infusion. Further management involves monitoring of arterial pressure, detection of target organ damage (e.g. 12-lead ECG) and treatment of the underlying cause.

Myocardial ischaemia/infarction

Ischaemia occurs when there is an imbalance between myocardial oxygen supply and demand. The perioperative incidence is

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high in at-risk patients, with the greatest risk of ischaemia during emergence from anaesthesia or during the early postoperative period. Most perioperative myocardial infarctions occur within 24–48 hours after surgery and not on days 3–5 as previously reported.[4]

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 short-lived.[4,5] However, intraoperative ischaemia is most commonly detected by ECG, with ST-segment deviations of at least 1 mm from baseline considered significant.[6] Multi-lead monitoring is recommended, and the combination of leads II and V5 will detect 80% of all ischaemic changes.[7] In the perioperative period, the risk of bleeding complicates the otherwise routine use of antithrombotic therapies and coronary intervention, and 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 emphasized, 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 seeks to confirm the diagnosis (serial ECGs, raised cardiac biomarkers) and stratify risk. Imaging can also assess myocardial function (e.g. echocardiography to assess new regional wall motion abnormalities). Referral to a cardiologist and admission to a high-dependency environment are necessary.

Bradycardia

Bradycardia is usually defined as an absolute heart rate of less than 60 beats/minute, 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/minute.

Factors that impair myocardial oxygen supply–demand balance

Reduced supply

Reduced coronary blood flow

- Tachycardia
- Hypotension

Reduced arterial oxygen content

- Anaemia
- Hypoxaemia

Increased demand

- Tachycardia

Increased wall tension

- Hypertension
- Hypervolaemia
- Increased contractility

Table 1

The causes of bradycardia include:

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

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

- Ensure adequate oxygenation and ventilation. Bradycardia in children is commonly due to hypoxia.
- Stop surgical cause.
- 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 and adrenaline.

Cardiac pacing will be required if the patient fails to respond to atropine or if there is a risk of asystole (suggested by 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/minute, adjust the current to achieve capture and seek expert help.

Tachycardia

Tachycardias are characterized by at least three consecutive complexes occurring at a rate exceeding 100 beats/minute. In the normal heart, haemodynamic instability is uncommon with ventricular rates below 150 beats/minute (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 tachycardia include:

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

The immediate management of tachycardia follows the same general principles as for bradycardia. With tachycardia, haemodynamically unstable patients require synchronized cardioversion. If the patient is stable, however, there is time for rhythm diagnosis and consideration of pharmacological therapies. Treatment algorithms for stable tachycardia are based primarily upon the QRS duration and the regularity of the complexes:[4]

- **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/minute, which corresponds to an atrial rate of 300/minute with 2:1

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