Anaesthetic implications of congenital heart disease for children undergoing non-cardiac surgery

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Learning objectives

After reading this article, you should be able to:

- categorize congenital heart disease and describe the anatomy and physiology
- carry out a preoperative assessment pertinent to the CHD patient
- have a framework to aid decisions regarding stratification of risk and triggers for referral to specialist centres
- explain the perioperative management specific to congenital heart disease

Abstract

A diagnosis of congenital heart disease (CHD) increases perioperative risk for children having surgical procedures. Some will require anaesthesia at a specialist cardiac centre, while for others it is safe and appropriate to have their procedure in a local district general hospital (DGH). Children with complex CHD and poor physiological status carry the highest risk of cardiac arrest and mortality. Clinical features of cardiomyopathy, cyanosis, pulmonary hypertension, arrhythmia and cardiac failure are most likely to require specialist input and tertiary referral, whereas those with a preoperative stay of less than 10 days undergoing elective, minor surgery, who are older than 2 years of age and physiologically well may be safely anaesthetized in a DGH. In order to maximize safety, the anaesthetist must carry out a thorough preoperative assessment, have an understanding of the patient's individual physiology and be prepared for the potential effects of general anaesthesia. Systemic vascular resistance is reduced by most induction agents and volatile anaesthetics, pulmonary vascular resistance is affected by changes in ventilation and gas exchange. The anaesthetist should be alert to particular potential complications, such as arrhythmias, hypoxia, bleeding and cardiac arrest.

Keywords Anaesthesia; cardiac; congenital heart disease; non-cardiac surgery; paediatric; peri-operative

Royal College of Anaesthetists CPD Matrix: 1A01, 2A03, 2D01, 2D02, 2D07, 3D00, 3G00

Introduction

Advances in prenatal diagnosis, surgical intervention and critical care of patients with congenital heart disease (CHD) has led to improved survival of patients,[1] and an increased prevalence in the general population. With up to 1% of all live births having CHD,[2] there is a likelihood of presentation for non-cardiac surgery at non-specialist centres. The increased risk of perioperative complications with this population are well documented,[3] and include increased morbidity, incidence of perioperative cardiac arrest and 30-day mortality. However, CHD includes a spectrum of conditions, and the complication rate carried by physiologically well patients is much less than complex patients with signs of cardiac failure or pulmonary hypertension, for example. There is a broad range of presentations and variation in physiological compensation, and 30% of CHD patients have non-cardiac congenital anomalies,[4] which require consideration. The diversity of the CHD population deems it impossible to recommend an anaesthetic 'recipe' for these patients; instead each case must have an individually tailored plan.

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Pathophysiology and anatomy

The anaesthetist must have a fundamental understanding of their patient's physiology to inform their anaesthetic plan. Congenital cardiac lesions can be grouped as normal ('in series'), balanced ('in parallel'), or completed single ventricular circulation; or categorized physiologically by the shunt and direction of flow.

Normal ('in series') circulation

This circulation is the simplest to understand, since the predominant path of blood flow follows that of a structurally normal heart. This group includes small septal defects, with simple shunts, and many patients with a surgically corrected CHD. The degree of shunt depends on both size of the defect and the pressure gradient (Figure 1).

Balanced ('in parallel') circulation

Large cardiac defects lead to mixing of deoxygenated and oxygenated blood, and consequently a parallel or 'balanced' circulation (Figure 2). Flow to the systemic and pulmonary vasculature depends upon the relative resistances (i.e. the balance) of the pulmonary vascular resistance (PVR) and systemic vascular resistance (SVR). Excessive blood flow to one area compromises blood flow to the other. High pulmonary blood flow can result in pulmonary oedema and pulmonary hypertension, the resultant decreased systemic perfusion increases the risk of coronary and splanchnic ischaemia. Conversely,

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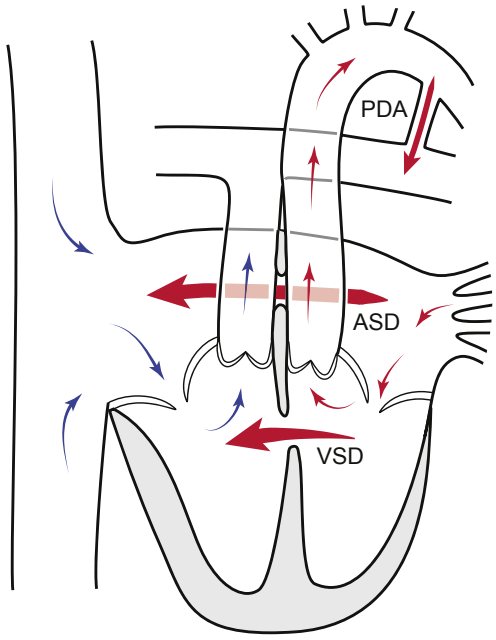

Figure 1 Schematic diagram illustrating an 'in series' circulation with examples of shunts. VSD, ventricular septal defect (VSD); ASD, atrial septal defect; PDA, patent ductus arteriosus.

insufficient pulmonary blood flow can cause cyanosis. This balance of SVR and PVR must be managed perioperatively. Factors affecting pulmonary blood flow are summarized in Table 1. Chronic hypoxia can lead to abnormal clotting and hyperviscosity.

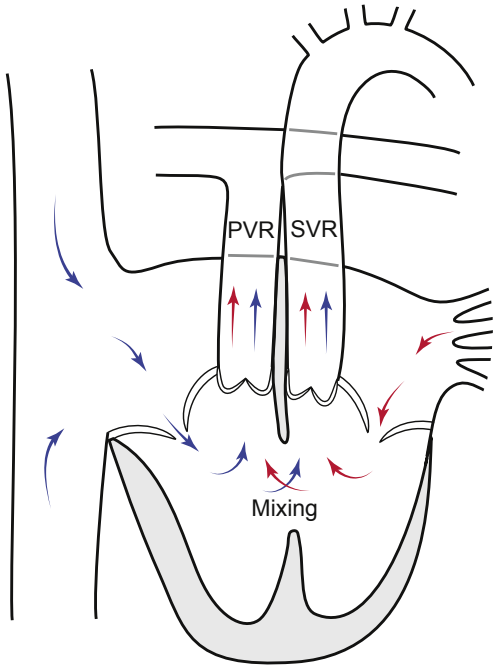

Figure 2 In parallel 'balanced' circulation with mixing. Example of a balanced circuit, such as a large ventricular septal defect (VSD). The proportion of blood going into the pulmonary artery and aorta is dependent on the relative systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR).

Factors affecting pulmonary blood flow

Factors reducing pulmonary blood Flow	Factors increasing pulmonary blood flow
Increased PVR	Reduced PVR
• Hypoxia	• High FiO_2
• Hypercarbia	• Hypocarbia
• Acidosis	• Alkalosis
• Hyperinflation	• Normal function residual capacity
• ↑Haematocrit	• ↓ Haematocrit
Decreased SVR	Increased SVR
• Pyrexia	• Hypothermia
• General anaesthetic agents (excluding Ketamine)	• Vasoconstrictor drugs
• Sympathetic block	• Sympathetic stimulation

Table 1

Single ventricle circulation

When patients are born with a hypoplastic ventricle (Figure 3a), surgery is palliative and leads to a single ventricular circulation. This is usually a three stage process, illustrated in Figure 3b–d:

Stage 1: **Norwood Procedure** (Figure 3b); The hypotrophic anatomical aorta is anastomosed to the pulmonary artery, which becomes the 'neo-aorta'. Pulmonary blood flow is supplied by a modified Blalock-Taussig shunt (mBTS) or an RV-PA conduit ('Sano modification'). Atrial septectomy is performed to ensure blood flow.

Stage 2: **Glenn shunt/bidirectional Glenn/hemi-Fontan** (Figure 3c). Aged 3–5 months. Superior vena cava connected to right pulmonary artery and the mBTS taken down. Postoperatively, the patient remains cyanosed with oxygen saturations between 75 and 85%.

Stage 3: **Total cavopulmonary circulation (TCPC)/Fontan procedure** (Figure 3d). Aged 3–5 years. Inferior vena cava connected to the right pulmonary artery, therefore separating the systemic and pulmonary circulations, pumped by a single ventricle. A fenestration can be created to decompress high pulmonary venous pressure in the setting of increased postoperative PVR.

Blood flow to the systemic circulation is pumped by the single ventricle, while pulmonary blood flows passively from the venae-cavae to the pulmonary artery. Pulmonary flow depends upon the pressure gradient which is affected by the PVR and intrathoracic pressure. Avoiding rises in PVR and optimizing ventilation strategies is therefore important. Factors influencing PVR are shown in Table 2. Although spontaneous ventilation increases blood flow through the lungs, positive pressure ventilation may allow better control of oxygenation and minute volume. If controlled ventilation is necessary, pulmonary blood flow should be optimized by minimizing peak inspiratory time, peak inspiratory pressure, and positive end expiratory pressure.

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