Postoperative care of adult cardiac surgery patients

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Abstract

Over 35,000 cardiac operations are performed in the UK each year, with the majority requiring cardiopulmonary bypass (CPB). CPB replaces the heart and lung function temporarily by providing non-pulsatile oxygenated blood flow in order to facilitate arrested heart surgery. The use of an extracorporeal circuit and cardioplegia causes the release of pro-inflammatory cytokines inducing a systemic inflammatory response, coagulation cascade activation, haemodilution and transient myocardial depression among other effects. These manifest as a series of typical pathophysiological derangements, which require the adoption of a standard management strategy. The aim of this article is to provide an overview of the key issues including cardiac, respiratory, neurological, renal and haemostatic complications, which may arise while managing the postoperative cardiac surgical patient.

Keywords Bleeding; cardiac surgery; complications

Introduction

The immediate management of the postoperative cardiac surgical patient is different from that of other surgical patients due to the physiological and pathological changes associated with cardiac surgery. This may present a challenge for surgical trainees. The purpose of this article is to outline the approach to the cardiac surgical patient and to discuss the most common scenarios that could emerge when participating in their postoperative care.

Patient handover

When the patient returns to the intensive care unit from theatre, a detailed handover from the surgical team should be given to the ITU staff. This should include, but not be limited to, the following information: the nature of the operation that the patient had and any surgical concerns, the findings on intraoperative transoesophageal echocardiography (TOE), whether the patient came off cardiopulmonary bypass smoothly or significant inotropic or vasopressor support was required, cross clamp time, the patient's rhythm and if they required epicardial pacing, any bleeding issues and, finally, any other relevant data from the patient's past medical history that would impact the management plan.

Patients with significant carotid artery disease, long-standing arterial hypertension or chronic renal dysfunction may benefit from higher blood pressure targets to provide adequate organ perfusion. In patients with preoperative left ventricular dysfunction, there will be a lower threshold to start inotropic support post operatively. Also, careful fluid balance is imperative to avoid precipitating congestive heart failure. In patients with significant left ventricular hypertrophy, for example, patients with preoperative severe aortic stenosis, adequate ventricular filling and preserved atrial contractility are essential to guarantee maximum cardiac output. Lastly, the on-duty doctor should be aware of the location of the emergency sternotomy trolley in the ITU and be familiar with the instruments it contains and their use.

How to identify the sick patient

Adequate cardiac output is usually associated with good blood pressure (without or on minimal inotropic support), good urine output (ideally more than 1 ml/kg/hour), good peripheral warming (no discrepancy between core temperature and skin temperature), and absence of metabolic acidosis on blood gases. Low cardiac output will result in derangement of some or all of these parameters. A crucial aspect however in managing a sick cardiac surgical patient is identifying a problem that may be rectified surgically. Examples include: massive surgical bleeding, cardiac tamponade with subsequent peripheral organ hypoperfusion, graft dysfunction with myocardial ischaemia and acute right ventricular failure refractory to medical management. Proactive management of these acute surgical problems by early return to theatre may be the best option to minimize the damage and to save the patient's life. Communication with senior member staff is essential for timely identification of these problems and correct patient management.

Cardiovascular

Low cardiac output state (LCOS)

Cardiac output, expressed in litres per minute, is the amount of blood the heart pumps in one minute. It is the product of stroke volume and heart rate ($CO = SV \times HR$). Stroke volume is affected by ventricular contractility, preload and afterload. Understanding the practical relevance of these components is important when diagnosing and treating low cardiac output states. When managing a low cardiac output state (LCOS) all of the following four areas must be optimal.

Heart rate: within the physiological range, increasing heart rate will directly increase CO. However, with severe tachycardia, the diastolic filling time will be significantly reduced, this will result in exponential reduction in stroke volume and significant drop in CO. Normal sinus rhythm will boost the cardiac output in patients with reduced ventricular compliance. This is discussed further in cardiac rhythm and pacing section.

Contractility: contractility is defined as the intrinsic strength of the ventricle independent of loading conditions. It is affected intrinsically by circulating catecholamines and extrinsically by

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exogenous inotropes. The most common inotropes used in the ITU setting are described in Table 1.

Preload: preload refers to the amount of filling of the ventricle just prior to contraction. This depends on heart rate, rhythm and venous return. Normal sinus rhythm or atrial pacing will ensure that the atrium is contributing to the CO (see pacing section). Adequate heart rate will ensure adequate diastolic filling time. High positive pressure ventilation will impede venous return (by increasing the intrathoracic pressure) and will negatively affect the preload. Also, adequate filling will increase the venous return. It is important to optimize the filling status before escalating the dose of inotropic support to treat LCOS.

Afterload: afterload is the force against which the ventricle must contract in order to eject blood. This is largely due to peripheral vascular resistance (and therefore blood pressure). In patients with severe systemic hypertension, vasodilator agents will result in better blood pressure control and a more optimized CO. Similarly, treating acute right ventricular failure by giving selective pulmonary vasodilators (e.g. milrinone or nitric oxide) will reduce the right ventricular afterload and will result in improved right ventricular contractility and CO.

Vasoactive support

In a low cardiac output state, vasoactive agents may be required to increase cardiac output, maintain adequate mean arterial pressures and therefore maintain tissue perfusion. The main receptor that affects rate and contraction within the heart is beta 1. Beta 1 agonists bind to the receptor, resulting in increased calcium availability, increasing the binding with troponin-C and hence enhance myocardial contractility.

Dobutamine acts primarily on the beta 1 receptors. It has an effect additionally on beta 2 receptors, leading to vasodilatation, decreasing afterload and improving coronary and renal perfusion. A drop in systolic pressure may require a concomitant vasopressor (such as noradrenaline). Side effects include tachycardia, dysrhythmias and raised myocardial oxygen demand.

Vasoactive agents

	HR	MAP	CO	PCWP	SVR
Dopamine	↑↑	↑	↑	↑	↓/↑
Dobutamine	↑↑	↓/↑	↑	↓	↓
Epinephrine	↑↑	↑	↑	↑	↓/↑
Norepinephrine	↑	↑↑	↑	↑↑	↑↑
Milrinone	NA	↓	↑	↓	↓↓

Dopamine and dobutamine are first-line agents for low cardiac output (CO). Dobutamine is a vasodilator, increasing coronary flow, but may reduce systemic blood pressure initially (though this is minimal due to its inotropic action). Tachycardia may limit the use of dopamine and dobutamine. Epinephrine is a potent inotrope, with an initial vasodilating effect. The primary action of norepinephrine is vasoconstriction, raising systemic blood pressure. Milrinone usually requires concurrent administration of a vasopressor due to its inodilatory action, resulting in a decreased systemic vascular resistance (SVR) and mean arterial pressure (MAP). HR, heart rate; PCWP, pulmonary capillary wedge pressure.

Table 1

Isoprenaline is similar to dobutamine; however, it has a greater effect on heart rate. Isoprenaline can be used as a continuous infusion for severely bradycardic patients (in the absence of pacing wires).

Dopamine in low doses (2–5 μg/kg/minute) has mainly dopaminergic effects, as this is increased (5–10 μg/kg/minute) the beta adrenergic action predominates. Higher doses of dopamine have an increasingly alpha receptor effect, leading to vasoconstriction and increased myocardial oxygen demand.

Noradrenaline is primarily a vasopressor, useful for treating hypotension due to reduced systemic vascular resistance. It acts mainly on alpha one receptors. The alpha agonist action increases blood pressure by increasing vascular resistance. In high doses, renal artery blood flow may become compromised and myocardial oxygen demand can increase. Phenylephrine is another commonly used vasopressor in critical care. It can be given as a bolus in order to temporarily increase blood pressure for an unstable patient on the wards prior to transfer to a critical care environment.

Adrenaline acts on both alpha and beta adrenergic receptors. At low doses it is more a beta agonist, and acts predominantly as an alpha agonist at higher doses. It is not used typically for vasoactive support as its action is non-specific and side effects include raised lactate and glucose.

Milrinone, an inodilator, is a phosphodiesterase-3 (PDE3) inhibitor. PDE3 is an enzyme present on cardiac and smooth muscle cells. Inhibition of PDE3 increases intracellular calcium (via cAMP and PKA), resulting in increased myocardial contractility, and a reduction in pulmonary vascular resistance (and a reduction in right ventricular afterload). It is useful in heart failure, where beta 1 receptors may be downregulated, as it acts independently from the adrenergic receptors.

Cardiac tamponade

Cardiac tamponade is a syndrome caused by fluid collecting in the pericardial space resulting in reduced ventricular filling and low CO with haemodynamic compromise. Small volumes of fluid without any compromise are usually present around the heart after surgery but the incidence of cardiac tamponade is relatively infrequent (1–6%).[1] There should be a high index of suspicion for cardiac tamponade as it is a potentially fatal condition if not recognized and managed promptly. The classical signs are arterial hypotension (rise in vasopressor/inotropic supports), raised central venous pressures and pulsus paradoxus. This is invariably associated with evidence of organ hypoperfusion such as low urinary output and worsening metabolic acidosis. CXR may show a globular heart or a widening mediastinum in comparison to the preoperative one. Trans-thoracic echocardiogram (TTE) may show compression of the right-sided chambers adjacent to the collection and distension of the inferior vena cava. Unfortunately, neither of these investigations are totally reliable to exclude cardiac tamponade, therefore in the setting of a convincing clinical picture, the diagnosis of cardiac tamponade should still be suspected. Early return to theatre to confirm the diagnosis and evacuate the collection must take place without any delay.

Heart rate and rhythm

Arrhythmias are one of the most common complications after cardiac surgery. Causes include surgical disturbance of the

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