The intra-aortic balloon pump and other methods of mechanical circulatory support

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Abstract

Mechanical circulatory support plays an increasing role in the management of patients with cardiac failure. Of the several forms to be discussed, intra-aortic balloon pump (IABP) counterpulsation is the most widely utilized. This involves percutaneous insertion of a balloon catheter through the femoral artery that inflates during diastole and deflates prior to systole, improving ventricular performance by simultaneously increasing myocardial oxygen supply during diastole and reducing myocardial oxygen demand by reducing afterload. Other forms of more advanced mechanical circulatory support that will be discussed include extracorporeal membrane oxygenation, ventricular assist devices and the total artificial heart. These technologies can replace rather than simply support cardiac function, as in the case of the IABP, and as such patient selection is critical. The use of these technologies must be balanced against potential complications which can be significant.

Keywords Counterpulsation; diastolic augmentation; extracorporeal membrane oxygenator; intra-aortic balloon pump; mechanical circulatory support; total artificial heart; ventricular assist device

Intra-aortic balloon pump (IABP) counterpulsation is the most widely available and utilized method of mechanical circulatory support. This and other techniques, such as extracorporeal membrane oxygenator pumps (ECMO) and ventricular assist devices (VADs) are used to support the circulation mechanically in a range of clinical scenarios, from prophylactic use in high-risk coronary artery procedures, to use in the management of advanced cardiogenic shock or even cardiopulmonary arrest.

Intra-aortic balloon pump counterpulsation

The IABP was first used clinically in the 1960s by Kantrowitz in patients with cardiogenic shock. The concept for this form of mechanical circulatory support arose following appreciation that myocardial oxygen supply occurs during diastole and that coronary arterial perfusion depends on the difference between pressure in the aorta and the coronary arteries. This resulted in animal model work investigating how manipulation of the arterial pressure pulse could augment coronary arterial flow with 'diastolic augmentation'.[1]

Principles of IABP counterpulsation

The IABP system comprises two components: the balloon catheter and a drive console. The balloon catheter is a flexible catheter with two lumens: one which permits pressure monitoring and the other through which helium gas is delivered to and removed from a closed balloon. Helium is the gas of choice due to its low density facilitating rapid transfer, and it has high solubility in blood in case of balloon rupture. The drive console has a computer that controls helium transfer and determines the inflation and deflation cycle.

Insertion and positioning: the balloon catheter is most commonly inserted percutaneously into the femoral artery using the Seldinger technique and advanced retrogradely up the descending thoracic aorta. The balloon catheter can also rarely be inserted surgically, antegradely into the ascending aorta (Figure 1).

The tip of the catheter should lie just distal to the origin of the left subclavian artery (approximately at the level of the carina on a chest radiograph). If the catheter is placed proximal to the left subclavian artery there is a risk of impeding cerebral blood flow, whereas if it is positioned too low mesenteric and renal perfusion can be compromised during balloon inflation. The catheters are often inserted under fluoroscopic guidance to aid positioning. Intraoperative placement may be confirmed instead by transoesophageal echocardiography. The position should be confirmed following insertion on a chest radiograph.

Following insertion, in the absence of any contraindication, patients with an IABP must be therapeutically anticoagulated to reduce the risk of thrombosis and subsequent embolic events.

Physiological effect of counterpulsation: inflation and deflation of the balloon is timed to occur at precise points in the cardiac cycle (Figure 2), with the aim of improving ventricular performance by increasing myocardial oxygen supply whilst simultaneously reducing myocardial oxygen demand:

- ***Balloon inflation*** occurs at the end of systole after aortic valve closure which causes augmentation of the diastolic pressure and a second peak in the arterial pressure waveform. This 'diastolic augmentation' displaces blood within the aorta resulting in a peak diastolic pressure that can exceed systolic pressure. The resultant effect is to increase the difference between aortic and left ventricular pressure, which leads to increased coronary blood flow and an increased myocardial oxygen supply. Distal displacement of blood within the aorta can additionally improve systemic perfusion.
- ***Balloon deflation*** occurs immediately before opening of the aortic valve during systole. The rapid deflation creates a vacuum effect by reducing aortic volume and therefore impedance for forward flow, leading to reduced left ventricular afterload. This results in reduced myocardial oxygen consumption. The reduced tension in the left ventricular wall and improved oxygen availability results in improved contractility and increased cardiac output.

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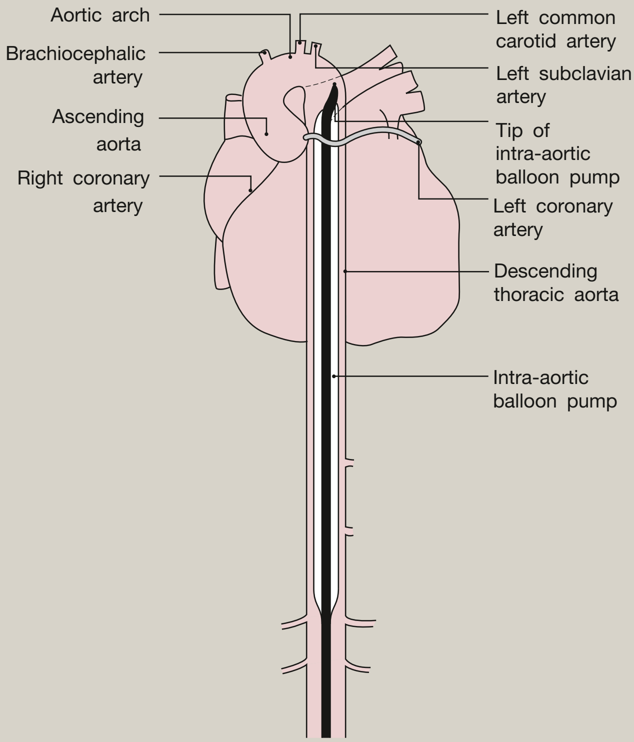
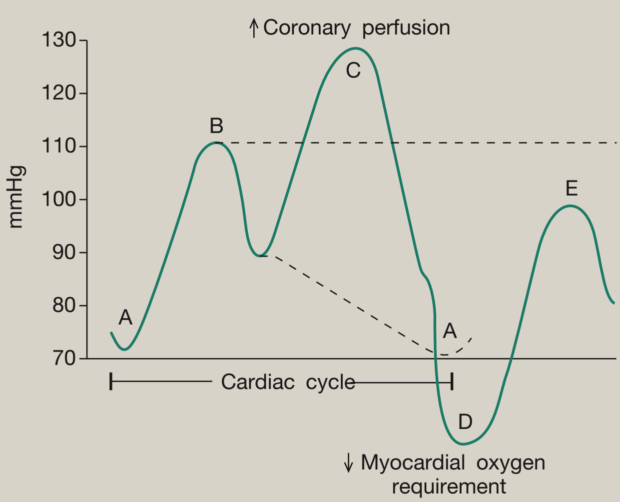
Positioning of the intra-aortic balloon pump



The correct position of the tip of the inflated intra-aortic balloon pump is 1–2 cm distal to the left subclavian artery.

Figure 1

Pressure changes as a result of intra-aortic balloon pump therapy



A Unassisted aortic end-diastolic pressure
B Unassisted systolic pressure
C Diastolic augmentation
D Assisted (↓) aortic end-diastolic pressure
E Assisted (↓) systolic pressure

Figure 2

The magnitude of the haemodynamic effect depends on several factors:

i) Balloon volume – the volume of blood displaced is directly proportional to the inflation volume.
ii) Heart rate – the higher the heart rate, the shorter the period of diastole and therefore opportunity for augmentation.
iii) Aortic compliance – the lower the compliance of the aorta, the greater the diastolic augmentation achieved.

Data obtained from studies with invasive monitoring have suggested the impact on haemodynamic parameters to be only modest, with an absolute increase in cardiac output of approximately 0.5–1 l/minute. These findings may relate to coronary autoregulation, whereby coronary arterial blood flow is not dependent upon perfusion pressure. In the majority of clinical situations in which IABP counterpulsation is utilized though, it is predicted that coronary autoregulation may not be effective and therefore the reported data likely underestimate the clinical impact of IABP counterpulsation in these patients.[2]

Control of counterpulsation: the drive console can be programmed to trigger balloon inflation or deflation in response to electrical or pressure signals. The console receives electrical signals in the form of the ECG waveform. The balloon is inflated at the onset of diastole (middle of the T-wave), and deflates just prior to systole (peak of the R-wave). Electrical triggering can be problematic in the presence of poor ECG quality, cardiac arrhythmias and with electrical interference (e.g. in the operating theatre with diathermy use). In these situations, and others, the console can be programmed to trigger from the systemic arterial pressure waveform instead.

Other parameters can also be controlled by the console, usually utilized to facilitate weaning. The proportion of augmented beats can be altered, for example, from every beat (1:1) to alternate (1:2) or fewer beats. Additionally, the inflation volume can be reduced. Both of these strategies reduce the haemodynamic support offered by the IABP. Importantly, the IABP must never be switched off due to the high risk of developing a thrombus on the stationary balloon.

Indications

There are several indications for the use of IABP counterpulsation, summarized in Box 1.[2]

Cardiogenic shock: IABP therapy is effective in improving haemodynamics of patients suffering cardiogenic shock as a complication of an acute myocardial infarction, characterized by low cardiac output and hypotension refractory to pharmacological management, and can be utilized as a bridge to definitive therapy.

Unstable angina: IABP therapy is utilized in patients with symptomatic myocardial ischaemia refractory to pharmacological therapy, usually as a bridge to revascularization. Improving coronary perfusion and haemodynamics results in symptomatic relief and can reverse ischaemic ECG changes.

Low cardiac output following cardiopulmonary bypass: IABP therapy can be used to facilitate weaning from cardiopulmonary

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