# Principles of intra-aortic balloon pump counterpulsation

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**Key points**

The primary goal of intra-aortic balloon pump (IABP) treatment is to increase myocardial oxygen supply and decrease myocardial oxygen demand.

Decreased urine output after the insertion of IABP can occur because of juxta-renal balloon positioning.

Haemolysis from mechanical damage to red blood cells can reduce the haematocrit by up to 5%.

Suboptimal timing of inflation and deflation of the balloon produces haemodynamic instability.

An IABP is thrombogenic; always anticoagulate the patient.

Never switch the balloon off while *in situ*.

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Intra-aortic balloon pump (IABP) remains the most widely used circulatory assist device in critically ill patients with cardiac disease. The National Centre of Health Statistics estimated that IABP was used in 42 000 patients in the USA in 2002. Advances in technology, including percutaneous insertion, smaller diameter catheters, sheathless insertion techniques, and enhanced automation, have permitted the use of counterpulsation in a variety of settings, with greater efficacy and safety.

## History

Kantrowitz[1] described augmentation of coronary blood flow by retardation of the arterial pressure pulse in animal models in 1952. In 1958, Harken[2] suggested the removal of some of the blood volume via the femoral artery during systole and replacing it rapidly in diastole as a treatment for left ventricular (LV) failure, so called diastolic augmentation. Four years later, Moulopoulos and colleagues[3] developed an experimental prototype of an IABP whose inflation and deflation were timed to the cardiac cycle. In 1968, Kantrowitz[1] reported improved systemic arterial pressure and urine output with the use of an IABP in two subjects with cardiogenic shock, one of who survived to hospital discharge. Percutaneous IABs in sizes 8.5–9.5 French (rather than 15 French used earlier) were introduced in 1979, and shortly after this, Bergman and colleagues[4] described the first percutaneous insertion of IABP. The first prefolded IAB was developed in 1986.

## Basic principles of counterpulsation

Counterpulsation is a term that describes balloon inflation in diastole and deflation in early systole. Balloon inflation causes 'volume displacement' of blood within the aorta, both proximally and distally. This leads to a potential increase in coronary blood flow and potential improvements in systemic perfusion by augmentation of the intrinsic 'Windkessel effect', whereby potential energy stored in the aortic root during systole is converted to kinetic energy with the elastic recoil of the aortic root.

## Physiological effects of IABP therapy

The primary goal of IABP treatment is to improve the ventricular performance of the failing heart by facilitating an increase in myocardial oxygen supply and a decrease in myocardial oxygen demand. The overall haemodynamic effects of IABP therapy are summarized in Table 1. Although these effects are predominately associated with enhancement of LV performance, IABP may also have favourable effects on right ventricular (RV) function by complex mechanisms including accentuation of RV myocardial blood flow, unloading the left ventricle causing reduction in left atrial and pulmonary vascular pressures and RV afterload.[5] IABP inflates at the onset of diastole, thereby increasing diastolic pressure and deflates just before systole, thus reducing LV afterload. The magnitude of these effects depends upon:

(i) *Balloon volume*: the amount of blood displaced is proportional to the volume of the balloon.
(ii) *Heart rate*: LV and aortic diastolic filling times are inversely proportional to heart rate; shorter diastolic time produces lesser balloon augmentation per unit time.
(iii) *Aortic compliance*: as aortic compliance increases (or SVR decreases), the magnitude of diastolic augmentation decreases.

### Myocardial oxygen supply and demand

Inflation of IAB during diastole increases the pressure difference between aorta and left

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**Table 1** Summary of haemodynamic effects of IABP therapy

| | |
|---|---|
| Aorta | ↓systolic pressure, ↑diastolic pressure |
| Left ventricle | ↓systolic pressure, ↓end-diastolic pressure, ↓volume, ↓wall tension |
| Heart | ↓afterload, ↓preload, ↑cardiac output |
| Blood flow | ↑→ coronary blood flow |

ventricle, the so-called diastolic pressure time index (DPTI). The haemodynamic consequence of this is an increase in coronary blood flow and, therefore, myocardial oxygen supply. Myocardial oxygen demand is directly related to the area under the LV systolic pressure curve, termed as tension time index (TTI). Balloon deflation during systole causes a reduction in the LV afterload, thereby decreasing TTI. Thus, the ratio of oxygen supply (DPTI) to oxygen demand (TTI), known as the endocardial viability ratio (EVR), should increase if the IABP is working optimally. This can be evidenced by a decrease in coronary sinus lactate.

### Coronary perfusion

According to the Hagen Poiseuille principle, flow through a tube is directly proportional to the pressure difference across it and the fourth power of the radius while being inversely proportional to the length of the tube and the viscosity of fluid flowing through it. Hence, in patients with severe coronary artery disease in whom autoregulation is perceived to be absent, coronary blood flow is directly related to diastolic perfusion pressure. Therefore, IABP should theoretically improve coronary flow in these patients.

### Renal function

Renal blood flow can increase up to 25%, secondary to increase in cardiac output. Decrease in urine output after insertion of IABP should raise the suspicion of juxta-renal balloon positioning.

### Haematological effects

The haemoglobin levels and the haematocrit often decrease by up to 5% because of haemolysis from mechanical damage to the red blood cells. Thrombocytopenia can result from mechanical damage to the platelets, heparin administration, or both.[6]

## Indications

Over the years, indications for the use of IABP have developed in clinical practice and are summarized along with contraindications in Table 2.

### Acute myocardial infarction

IABP is aimed at achieving haemodynamic stability until a definitive course of treatment or recovery occurs. By decreasing myocardial work and SVR, intracardiac shunting, mitral regurgitation, or both (if present) are reduced while coronary perfusion is enhanced.

**Table 2** Indications and contraindications for the use of IABP therapy

| | |
|---|---|
| *Indications* | |
| Acute myocardial infarction | Refractory LV failure |
| Cardiogenic shock | Refractory ventricular arrhythmias |
| Acute MR and VSD | Cardiomyopathies |
| Catheterization and angioplasty | Sepsis[9] |
| Refractory unstable angina | Infants and children with complex cardiac anomalies[10] |
| Cardiac surgery | |
| Weaning from cardiopulmonary bypass | |
| *Contraindications* | |
| **Absolute** | **Relative** |
| Aortic regurgitation | Uncontrolled sepsis |
| Aortic dissection | Abdominal aortic aneurysm |
| Chronic end-stage heart disease with no anticipation of recovery | Tachyarrhythmias |
| Aortic stents | Severe peripheral vascular disease |
| | Major arterial reconstruction surgery |

Severe mitral regurgitation secondary to papillary muscle dysfunction or rupture after myocardial infarction can lead to significant haemodynamic instability. This can initially be managed by IABP, pending definitive surgery.

### Ventricular arrhythmias

IABP is also effective in stabilizing patients with refractory ventricular ectopy after myocardial infarction by increasing the coronary perfusion pressure, reducing ischaemia and trans-myocardial wall stress, and maintaining adequate systemic perfusion.

### Cardiogenic shock

This is life-threatening complication of acute myocardial infarction, is characterized by low cardiac output, hypotension unresponsive to fluid administration, elevated filling pressures and tissue hypoperfusion leading to oliguria, hyperlactaemia, and altered mental status. IABP therapy is considered to be a class I indication (ACC/AHA guidelines) for the management of cardiogenic shock not rapidly reversed by pharmacological therapy.[7]

### Unstable angina

Unstable angina refractory to drug treatment is an indication for IABP. These patients are at increased risk of developing acute myocardial infarction and death. By improving the haemodynamic condition of these patients, IABP can facilitate further percutaneous interventions or bridge the patient to surgery.

### Refractory ventricular failure

IABP has a role in managing patients with refractory ventricular failure outside the setting of acute myocardial infarction, such as those with cardiomyopathy or severe myocardial damage associated with viral myocarditis. This can aid the progression to more definitive treatments such as ventricular assist device or cardiac transplantation.

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## Cardiac surgery

IABP is used for stabilization of patients with acute myocardial infarction referred for urgent cardiac surgery. IABP support is often initiated in the cardiac catheterization laboratory and continued through the perioperative period. Elective placement is considered in high-risk patients such as those with significant left main stem disease, severe LV dysfunction (ejection fraction <30%), congestive heart failure, cardiomyopathy, chronic renal failure, or cerebrovascular disease. Weaning from cardiopulmonary bypass may be difficult in cases where aortic cross-clamping is prolonged, revascularization is only partially achieved, or pre-existing myocardial dysfunction is present. Separation from cardiopulmonary bypass may be marked by hypotension and a low cardiac index despite the administration of inotropic drugs. The use of IABP in this setting decreases LV resistance, increases cardiac output, and increases coronary and systemic perfusion, facilitating the patient's weaning from cardiopulmonary bypass.

## Contraindications

The contraindications to IABP are summarized in Table 2. It is contraindicated in patients with aortic regurgitation because it worsens the magnitude of regurgitation. IABP insertion should not be attempted in case of suspected or known aortic dissection because inadvertent balloon placement in the false lumen may result in extension of the dissection or even aortic rupture. Similarly, aortic rupture can occur if IABP is inserted in patients with sizable abdominal aortic aneurysms. Patients with end-stage cardiac disease should not be considered for IABP unless as a bridge to ventricular assist device or cardiac transplantation.

IABP device placement should be avoided in patients with severe peripheral vascular disease. Percutaneous femoral IABP device insertion is contraindicated in the presence of bilateral femoral–popliteal bypass grafts. Uncontrolled sepsis and bleeding diathesis are relative contraindications to the placement of IABP device.

## Technique of insertion and operation

The IABP device has two major components: (i) a double-lumen 8.0–9.5 French catheter with a 25–50 ml balloon attached at its distal end; and (ii) a console with a pump to drive the balloon. The balloon is made of polyethylene and is inflated with gas driven by the pump. Helium is often used because its low density facilitates rapid transfer of gas from console to the balloon. It is also easily absorbed into the blood stream in case of rupture of the balloon.

Before insertion, the appropriate balloon size is selected on the basis of the patient's height (as supplied by Datascope, for a patient <152 cm in height, a balloon volume of 25 cc is appropriate; for height between 152 and 163 cm, balloon volume 34 cc; for height 164–183 cm, balloon volume 40 cc, and for height >183 cm, balloon volume 50 cc). Smaller balloons are available for paediatric use. The diameter of the balloon, when fully expanded, should not exceed 80–90% of the diameter of the patient's descending thoracic aorta.

**Table 3** Complications associated with IABP

| |
|---|
| Transient loss of peripheral pulse |
| Limb ischaemia |
| Thromboembolism |
| Compartment syndrome[11] |
| Aortic dissection |
| Local vascular injury—false aneurysm, haematoma, bleeding from the wound |
| Infection |
| Balloon rupture (can cause gas embolus) |
| Balloon entrapment |
| Haematological changes, for example thrombocytopenia, haemolysis |
| Malpositioning causing cerebral or renal compromise |
| Cardiac tamponade |

The IABP catheter is inserted percutaneously into the femoral artery through an introducer sheath using the modified Seldinger technique. Alternative routes of access include subclavian, axillary, brachial, or iliac arteries. The catheter can also be inserted surgically using a transthoracic or translumbar approach, but this is associated with an increased periprocedural mortality.[8] Once vascular access is obtained, the balloon catheter is inserted and advanced, usually under fluoroscopic guidance, into the descending thoracic aorta, with its tip ~2 to 3 cm distal to the origin of the left subclavian artery (at the level of the carina). Intraoperatively, balloon placement can be ascertained using transoesophageal echocardiography.

The outer lumen of the catheter is used for delivery of gas to the balloon and the inner lumen can be used for monitoring systemic arterial pressure. Complications associated with IABP are summarized in Table 3.

The console is programmed to identify a trigger for balloon inflation and deflation. The most commonly used triggers are the ECG waveform and the systemic arterial pressure waveform. The balloon inflates with the onset of diastole, which corresponds with the middle of the T-wave. The balloon deflates at the onset of LV systole and this corresponds to the peak of the R-wave. Poor ECG quality, electrical interference, and cardiac arrhythmias can result in erratic balloon inflation.

The balloon is set to inflate after the aortic valve closure (which corresponds to the dicrotic notch on the arterial waveform) and deflate immediately before the opening of the aortic valve (which corresponds to the point just before the upstroke on the arterial pressure waveform). IABP timing refers to inflation and deflation of the IAB in relation to the cardiac cycle. The cardiac cycle is monitored by continuous display of the arterial pressure waveform. As the balloon inflates at the onset of diastole, a sharp and deep 'V' is observed at the dicrotic notch (Fig. 1). Balloon inflation causes augmentation of diastolic pressure and a second peak is observed. This peak is referred to as diastolic augmentation. Diastolic augmentation is ideally higher than the patient's systolic pressure except when reduced stroke volume causes a relative decrease in augmentation. Depending upon the patient's

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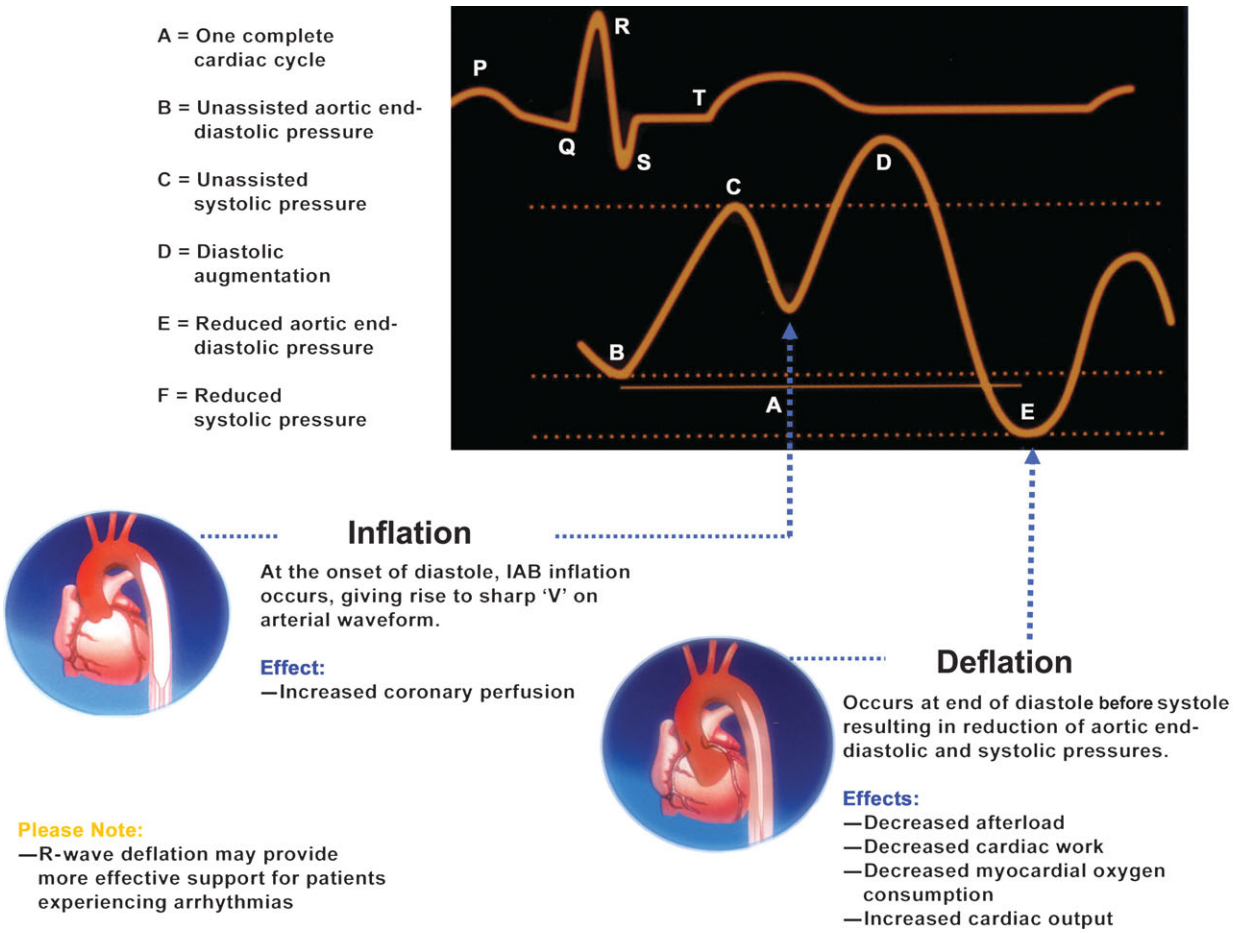


**Fig 1** One complete cardiac cycle and the corresponding waveform of the IABP during inflation and deflation. Reproduced with permission from Datascope®.

haemodynamic status, the balloon is programmed to assist every beat (1:1) or less often (1:2, 1:4, or 1:8). With haemodynamic improvement, the device can be 'weaned' to less frequent cycling before complete removal. However, the device should never be left unused *in situ* to prevent thrombosis.

Suboptimal timing of inflation and deflation of the balloon will result in haemodynamic instability (Fig. 2A–D): Examples of this include:

(i) *Early inflation*: inflation of the IAB before aortic valve closure (Fig. 2A).
(ii) *Late inflation*: inflation of the IAB markedly after closure of the aortic valve (Fig. 2B).
(iii) *Early deflation*: premature deflation of the IAB during the diastolic phase (Fig. 2C).
(iv) *Late deflation*: deflation of the IAB after the onset of systole (Fig. 2D).

Weaning from IABP should be considered when the inotropic requirements are minimal, thus allowing increased inotropic support if needed. Weaning is achieved gradually (over 6–12 h) reducing the ratio of augmented to non-augmented beats from 1:1 to 1:2 or less and/or decreasing the balloon volume. The balloon should never be turned off *in situ* except when the patient is anticoagulated because of the risk of thrombus formation on the balloon.

Patient care should be carried out with three primary goals in mind:

(i) evaluation in terms of haemodynamic status, systemic perfusion, and relief of cardiac symptoms;
(ii) observation for early signs of complications including limb ischaemia, balloon malpositioning, thrombus formation, bleeding, and infection;
(iii) ensuring proper functioning of IABP, including correct timing, consistent triggering, and troubleshooting of alarms.

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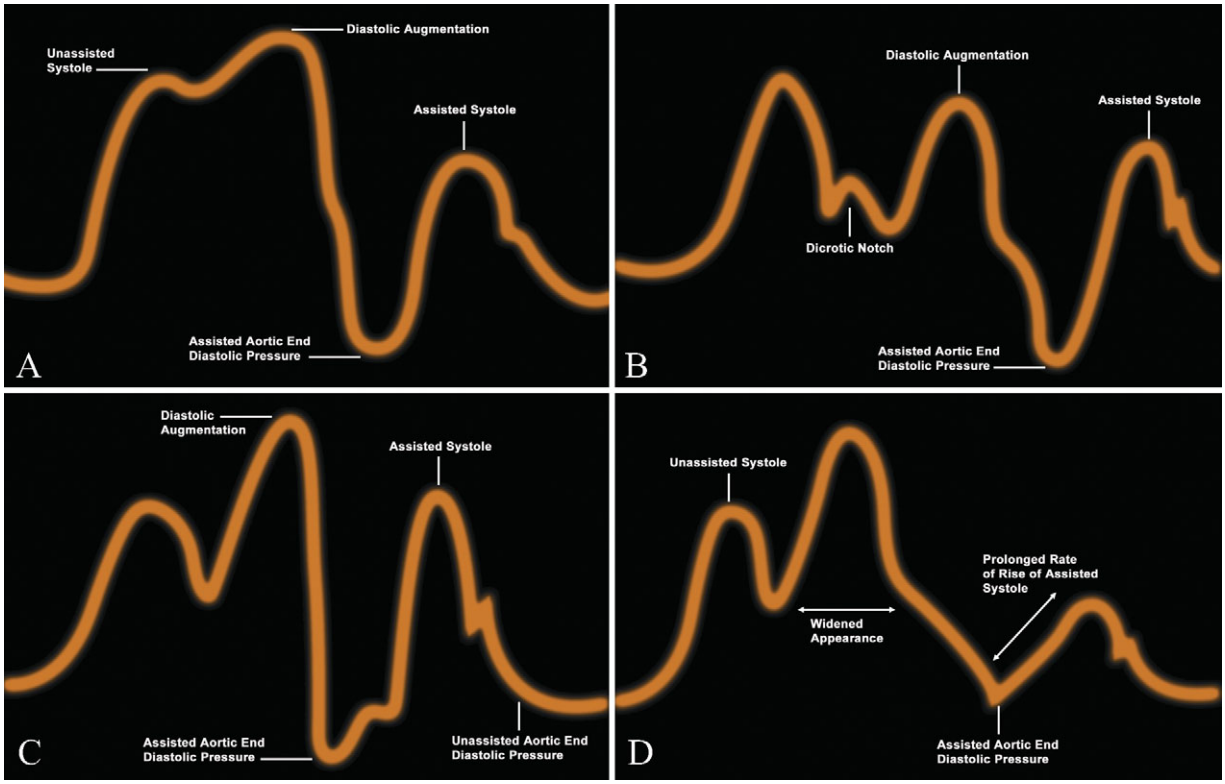


**Fig 2** (A) *Waveform characteristics*: inflation of IAB before dicrotic notch; diastolic augmentation encroaches onto systole, may be unable to distinguish. *Physiological effects*: potential premature closure of the aortic valve; potential increase in LVEDV and LVEDP or PCWP; increased LV wall stress or afterload; aortic regurgitation; increased $MVO_2$ demand. (B) *Waveform characteristics*: inflation of IAB after the dicrotic notch; absence of sharp 'V'. *Physiological effects*: suboptimal coronary artery perfusion. (C) *Waveform characteristics*: deflation of IAB is seen as a sharp decrease after diastolic augmentation; suboptimal diastolic augmentation; assisted aortic end-diastolic pressure may be equal to or less than the unassisted aortic end-diastolic pressure; assisted systolic pressure may increase. *Physiological effects*: suboptimal coronary perfusion; potential for retrograde coronary and carotid blood flow; suboptimal afterload reduction; increased $MVO_2$ demand. (D) *Waveform characteristics*: assisted aortic end-diastolic pressure may be equal to the unassisted aortic end-diastolic pressure; rate of increase of assisted systole is prolonged; diastolic augmentation may appear widened. *Physiological effects*: afterload reduction is essentially absent; increased $MVO_2$ consumption because of the left ventricle ejecting against a greater resistance and a prolonged isovolumetric contraction phase; IAB may impede LV ejection and increase the afterload. Reproduced with permission from Datascope®.

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**Please see multiple choice questions 21–25**

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