

INTRA-AORTIC BALLOON PUMP COUNTERPULSATION

ANAESTHESIA TUTORIAL OF THE WEEK 220

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INTRODUCTION

The intra-aortic balloon pump (IABP) is a temporary coronary and systemic perfusion assist device. Since introduction into clinical practice in the 1960s it has become widely used in critically ill patients with coronary disease and cardiac pump failure. This article will review the basic principles underlying IABP counterpulsation, the device's indications, contraindications, techniques for insertion and operating instructions. Understanding the basic principles, physiological effects, appropriate set-up and potential complications of the IABP are essential for improving patient outcomes.

BASIC PRINCIPLES AND PHYSIOLOGICAL EFFECTS

Synchronized counterpulsation is the core principle of IABP therapy. This describes inflation in diastole and deflation in systole of a balloon situated in the descending aorta. The overall aim is to improve myocardial function by increasing myocardial oxygen supply and decreasing myocardial oxygen demand. The method by which this is achieved is by displacement of blood in the aorta, both proximally and distally during balloon inflation. Figure 1 illustrates schematically how this is achieved.

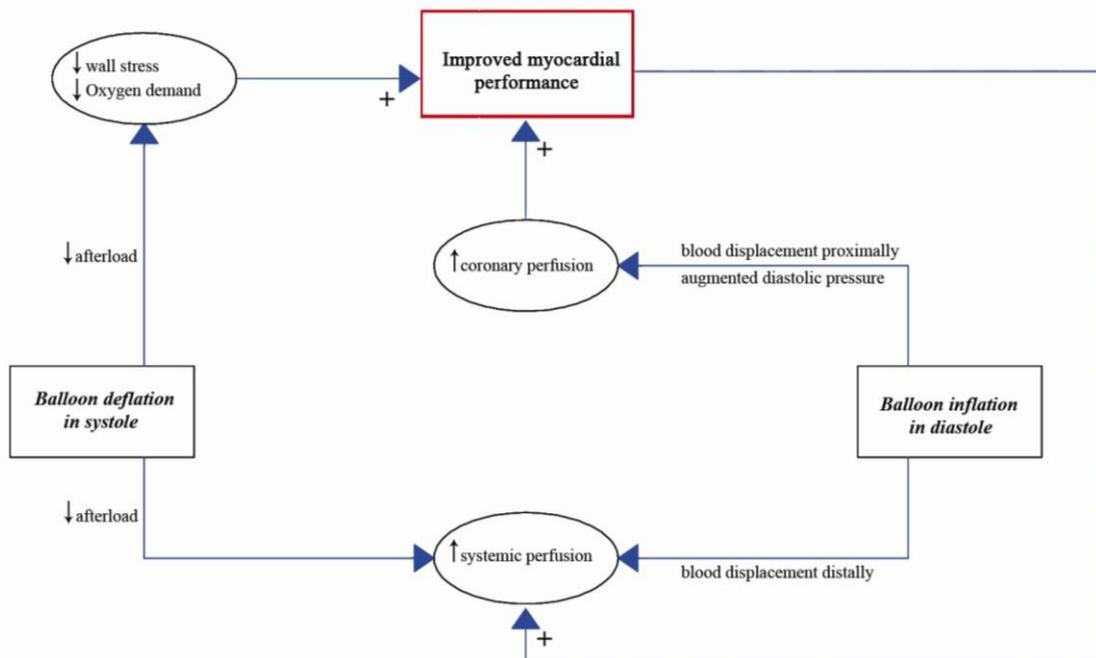


Figure1. Physiological effects of IABP

Balloon inflation occurs at the start of diastole. This displaces blood proximally and provides increased coronary blood flow by increasing aortic root diastolic pressure. This results in forcible “active” filling of the coronary arteries. This is in contrast to coronary artery filling in normal circumstances that is an autoregulated, passive process. Blood in the descending aorta is also displaced distally during balloon inflation. This may provide improved blood flow in the coeliac, renal and mesenteric vessels. However the significant improvement in systemic perfusion that is seen with successful use of an IABP is predominantly due to the reduction of afterload that occurs with rapid *balloon deflation* at the start of systole. This reduction in afterload results in a decrease in the workload of the left ventricle and a decrease in myocardial oxygen consumption. The overall effect of an improved ratio of myocardial oxygen supply to myocardial oxygen demand is improved myocardial performance.

The magnitude of the effects of IABP counterpulsation on systemic and coronary haemodynamics depends on a number of factors. These include the relationship of balloon volume to aortic size, aortic compliance, heart rate and heart rhythm.¹ Overall, IABP counterpulsation is expected to increase diastolic pressure by 30% (ideally to a value greater than systolic pressure), decrease the systolic pressure by 20% and improve cardiac output by 20%.² However the most appropriate way of assessing the adequacy of IABP therapy is by repeated assessment of the adequacy of systemic and coronary circulation through regular patient examination and assessment of clinical and laboratory parameters.

INDICATIONS

Since its introduction in the 1960s there has been a considerable increase in the indications for IABP use. Current indications for IABP are shown below. Indications have been divided into those with clear evidence of benefit, those with probable benefit and those with no evidence to suggest benefit.

Indications with proven benefit

- Cardiogenic shock secondary to AMI refractory to medical therapy³
- Mechanical complications of AMI: acute MR and VSD
- Refractory ventricular arrhythmias⁴
- Refractory unstable angina⁵
- Decompensated systolic heart failure (as a bridge to definitive treatment)

Indications with probable benefit

- Peri-operative support for high risk coronary artery bypass surgery⁶
- Peri-operative support for high risk cardiac patients undergoing non-cardiac surgery
- Decompensated aortic stenosis¹

Indications with no evidence to suggest benefit

- Sepsis
- Routine use in high-risk patients undergoing PCI⁷

AMI – acute myocardial infarction, MR – mitral regurgitation, VSD – ventricular septal defect, PCI – percutaneous coronary intervention

Contrary to the results and recommendations of several small observational studies, one large randomised controlled trial found that elective IABP insertion before high risk PCI adds no significant benefit in reducing the incidence of major adverse cardiovascular and cerebral events at 4 weeks⁷. Routine IABP therapy in high risk patients undergoing PCI is not recommended and should be preserved to patients who are unstable or likely to become unstable during PCI.

CONTRAINDICATIONS

The contraindications to IABP are summarised in below.

Absolute

- Significant aortic regurgitation and aortic dissection
- Aortic stent
- End-stage cardiac disease with no viable other treatment options
- Bilateral femoral-popliteal bypass grafts (femoral route only contraindicated)

Relative

- Uncontrolled sepsis
- Abdominal aortic aneurysm
- Severe bilateral peripheral vascular disease
- Uncontrolled bleeding disorder
- Prosthetic ileo-femoral grafts/iliac artery stents

COMPLICATIONS

The Benchmark Counterpulsation Outcomes Registry published data on nearly 1,700 patients who underwent IABP therapy between 1996 and 2000⁵. In this series 2.8% of patients experienced one or more major complications (defined as death, major limb ischaemia, severe bleeding or balloon leak). The incidence of minor complications was 4.2%.

The most common complications of IABP therapy are related to the presence of the IABP catheter itself and the technique used for insertion. Rapid inflation and deflation of the balloon causes trauma to red blood cells and platelets, commonly resulting in anaemia and/or thrombocytopenia. Device related thrombus formation and subsequent embolization are also significant risks. Because of these factors, patients with an IABP in situ are usually systemically anticoagulated, resulting in an increased risk of bleeding at the insertion site.

The most common vascular complication is limb ischaemia, noting that many patients in whom IABP therapy is indicated in will have either established peripheral arterial disease or multiple risk factors. All patients must therefore have their peripheral pulses, capillary return and skin temperature monitored on at least an hourly basis whilst an IABP catheter is in situ. Unresolved limb ischaemia may require removal of the device and urgent vascular surgery review if ischaemia persists.

Balloon rupture is a rare complication. It can be indicated by a sudden loss of inflation pressure or the presence of blood in the balloon line and is associated with a risk of intra-balloon thrombus and helium embolisation. If balloon rupture occurs the console will alarm and withdraw helium from the balloon before shutting down. Surgical removal of the balloon may be required to ensure complete removal.

The complications of IABP are shown below.

Vascular

- Limb ischaemia
- Vascular laceration and local vascular injury at time of insertion
- Aortic dissection
- Spinal cord and visceral ischaemia
- Peripheral thrombotic embolization
- False aneurysm and AV fistula formation

Balloon related

- Misplacement or migration of the balloon (may lead to occlusion of renal or subclavian arteries or perforation of the aortic arch)
- Balloon perforation or rupture leading to gas embolization
- Thrombocytopenia
- Anaemia

Miscellaneous

- Infection

TECHNIQUES OF INSERTION AND OPERATION

Device insertion

IABP catheters are supplied with an instruction booklet that should be reviewed before use. The size of the balloon is selected based on the patient's height. The usual range for an adult patient is between 25cc (for patients under 5 feet tall) to 50cc (for patients over 6 feet tall) but individual manufacturer's guidance should be followed. The IABP is usually inserted percutaneously using the Seldinger technique and strict aseptic technique. The most common site of access is the femoral artery. Alternative access sites include brachial and axillary artery percutaneous techniques and open surgical aortic approach. After successful puncture of the chosen artery a J-shaped guide wire is inserted to the level of the aortic arch. A combined dilator and sheath combination is then inserted, allowing the balloon to then be fed over the guide wire into the descending aorta, usually under fluoroscopic guidance. The tip of the IABP catheter should lie 1 – 2 centimetres distal to the origin of the left subclavian artery. This corresponds to the level of the second rib. Alternative methods for guiding IABP catheter placement include transoesophageal echocardiography or open sheathless insertion during cardiac surgery. Percutaneous sheathless insertion can be used in patients with peripheral vascular disease but this method is associated with an increase in minor bleeding and infection.

Initial set-up (triggering and timing)

Following insertion of an appropriately sized device the balloon catheter is connected to the console and the system is purged with helium. The central lumen of the catheter is then connected to a pressure transducer. An aortic pressure waveform should be visible after this is done. Blood samples must not be taken from this line. Systemic anticoagulation is usually now given, aiming for a target activated partial thromboplastin ratio of 2.

Correct timing of balloon inflation and deflation during the cardiac cycle is vital to ensure optimal effects of counterpulsation whilst minimizing potentially harmful effects related to mistiming. Most commonly the ECG waveform is used to trigger balloon inflation and deflation. The arterial pressure waveform is an alternative technique that may be useful if either the ECG trace is poor or there are cardiac arrhythmias. Modern machines allow either method to be easily selected. The balloon starts to inflate at the onset of diastole. This corresponds to the middle of T wave on the ECG waveform and the dicrotic notch of the arterial pressure trace. As aortic valve closure has occurred balloon inflation caus-

es a sharp upstroke on the arterial pressure waveform followed by a tall peak which represents the assisted diastolic pressure. Deflation occurs at the onset of systole immediately before opening of the aortic valve. This corresponds with the peak of the R wave on the ECG trace and the point just before the upstroke of systole on the arterial pressure trace. As the balloon deflates, the assisted aortic end-diastolic pressure dips down to create the second deep wave, usually U shaped on the arterial pressure waveform. IABP timing in relation to the cardiac cycle is monitored by display of arterial pressure waveform, the ECG trace and an intra-balloon pressure trace as shown in **Figures 2 and 3**.

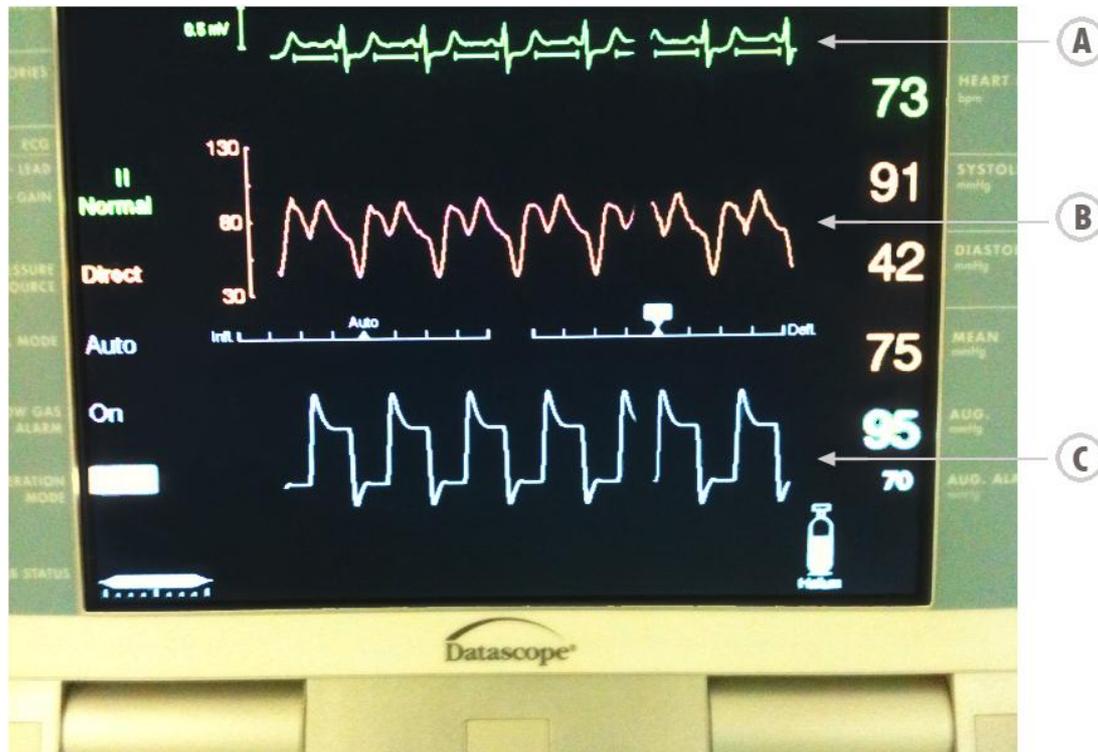


Figure 2. Datascope® IABP display unit during ECG triggered 1:1 augmentation.

Waveforms A ECG trace with horizontal line displaying duration of balloon inflation
 B Aortic pressure waveform during augmentation
 C Balloon pressure waveform

Heart rate 73 bpm
Systolic pressure 91 mmHg
End diastolic pressure 42 mmHg
Mean pressure 75 mmHg
Augmented diastolic pressure 95 mmHg

The augmentation frequency ratio refers to the number of augmented beats per cardiac cycle. The ratio is selected depending on the haemodynamic status of the patient and the presence of myocardial ischaemia. In practice an augmentation ratio of 1:1 is initially used. This may easily be changed using the console controls. As already referred to, inaccurate timing of inflation and deflation will worsen haemodynamic instability and further compromise coronary perfusion.

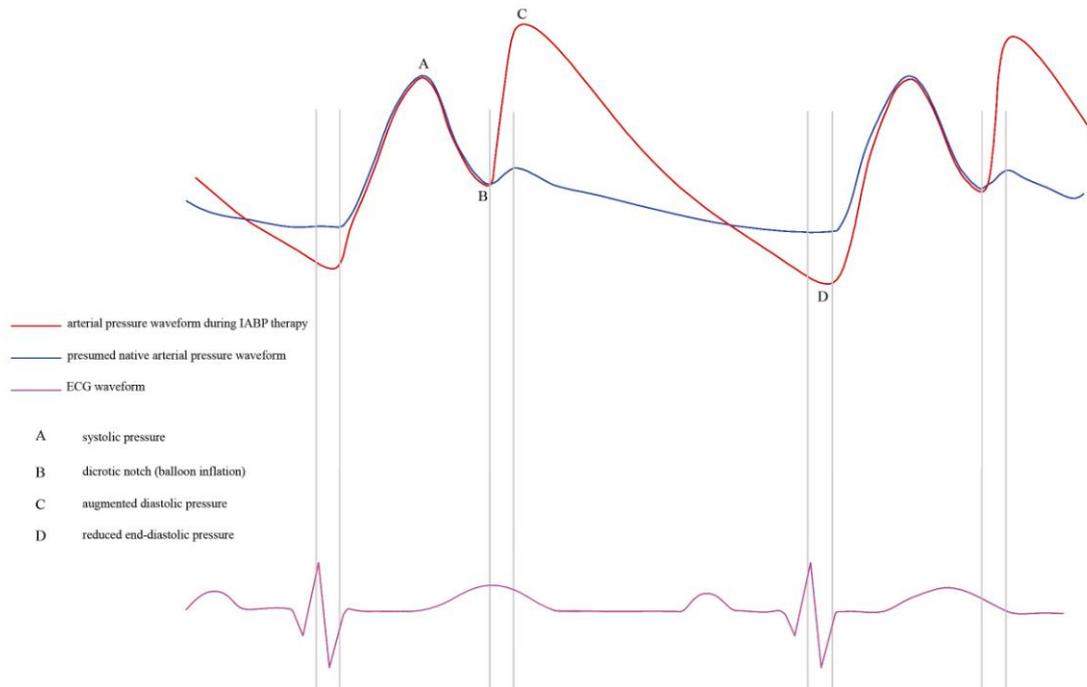


Figure 3. IABP arterial waveform in relation to cardiac cycle (1:1 augmentation)

WEANING FROM IABP COUNTERPULSATION

Successful weaning from IABP requires the patient to not be in cardiogenic shock and to have an adequate blood pressure whilst on little or no inotropic support. Reasonable target values to aim for prior to weaning are a mean arterial pressure of 65 mmHg and, if monitored, a cardiac index of ≥ 2 l/min/m². IABP counterpulsation is usually weaned by reducing the ratio of augmented to non-augmented beats. This can be done by reducing the augmentation frequency every 1 – 6 hours, from ratios of 1:1 to 1:2 to 1:3. If a ratio of 1:3 is tolerated for 6 hours then the device should be put into standby and removed. An alternative weaning method is to decrease the balloon filling volume by 10ml every 1 - 6 hours until a filling volume of 20ml is reached. The balloon pump should not be left in situ once switched off as this is associated with a high chance of thrombus formation on the balloon and distal embolization. A ratio of 1:3 should also not be used for prolonged periods as this is also associated with a significant increase in thrombosis risk.

MAIN POINTS OF CARE

- Most hospitals will have care protocols for patients with an IABP in situ. These should be closely adhered to. The majority of protocols will include the following:
- Patients should be managed in an appropriate area by staff familiar with IABP management.
- Ensure consistent triggering and correct timing.
- Regularly monitor vital signs and the need for augmentation.
- Record patient's blood pressure from IABP console even if another arterial line is in situ.
- Patients should be anticoagulated as per unit protocol unless there is a contraindication.
- Do not take blood samples from the balloon arterial pressure line.
- Perform hourly assessment for signs of peripheral hypo-perfusion or limb ischaemia in the limb distal to the catheter insertion site. This should include assessment of colour, capillary return, sensation and presence of pulses by palpation or Doppler studies. Presence of limb ischaemia should prompt consideration of removal of the device and sheath and urgent vascular surgery review.

- Perform hourly perfusion assessment of the left upper limb, which may become compromised in the event of proximal migration of the catheter compromising left subclavian artery blood flow.
- The patient should be log rolled and the end of the bed should not be elevated to more than 30 degree to minimize proximal catheter migration.
- Regularly assess insertion site for oozing, bleeding, swelling and signs of infection.
- Regularly check haemoglobin (risk of bleeding or haemolysis)
- Regularly check platelet count (risk of thrombocytopenia)
- Regularly check renal function (risk of acute kidney injury secondary to distal migration of IABP catheter)
- The balloon pump must not be left in standby by mode for any longer than necessary and never longer than 20 minutes in view of high risk of device related thrombus formation.

SPECIAL SITUATIONS

Cardiac arrest

In the event of a cardiac arrest pressure triggering should be selected on the IABP console. Reducing the pressure threshold may be necessary. If there is no arterial pressure trace then the console should be switched to “internal mode,” however this increases the chance of “asynchronous counterpulsation”

Defibrillation

IABP counterpulsation does not need to be discontinued during defibrillation, but staff must remain clear of the IABP console and connections when a shock is delivered.

SUMMARY

Intra-aortic balloon pump counterpulsation is the most commonly employed coronary and systemic circulation assist device. The overall aim of this therapy is to improve myocardial function by better matching of myocardial oxygen supply and demand. This is achieved by synchronized counterpulsation, which describes inflation in diastole and deflation in systole of a balloon situated in the descending aorta. There are a number of evidence based indications for use of IABP therapy. These include cardiogenic shock secondary to AMI refractory to medical therapy, some mechanical complications of AMI, refractory ventricular arrhythmias, refractory unstable angina and decompensated systolic heart failure (as a bridge to definitive treatment). Proper use of the IABP includes setting an appropriate augmentation frequency ratio and trigger and ensuring correct timing of balloon inflation and deflation. Major complications occur in fewer than 3% of cases but adequate knowledge of potential complications and their management is necessary for all users.

REFERENCES

- 1- Weber KT, Janicki JS.
Intraaortic balloon counterpulsation. A review of physiological principles, clinical results, and device safety.
Ann Thorac Surg. 1974;17(6):602-36.
- 2- Scheidt S, Wilner G, Mueller H, Summers D, Lesch M, Wolff G, et al.
Intra-aortic balloon counterpulsation in cardiogenic shock. Report of a co-operative clinical trial.
N Engl J Med. 1973;288(19):979-84.
- 3- Ryan TJ, Antman EM, Brooks NH, Califf RM, Hillis D, Hiratzka LF, et al
1999 Update: ACC/AHA Guidelines for the Management of Patients With Acute Myocardial Infarction: Executive Summary and Recommendations A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction)
Circulation. 1999;100:1016-30
- 4- Fotopoulos GD, Mason MJ, Walker S, Jepson NS, Patel DJ, Mitchell AG, et al.
Stabilisation of medically refractory ventricular arrhythmia by intra-aortic balloon counterpulsation.
Heart. 1999;82(1):96-100.
- 5- Ferguson JJ 3rd, Cohen M, Freedman RJ Jr, Stone GW, Miller MF, Joseph DL, et al.
The current practice of intra-aortic balloon counterpulsation: results from the Benchmark Registry.
J Am Coll Cardiol. 2001;38(5):1456-62.
- 6- Rubino AS, Onorati F, Santarpino G, Abdalla K, Caroleo S, Santangelo E, et al
Early IABP following perioperative myocardial injury improves hospital and mid-term prognosis.
ICVTS 2009;8:310-315
- 7- Perera D, Stables R, Thomas M, Booth J, Pitt M, Blackman D, et al. BCIS-1 Investigators.
Elective intra-aortic balloon counterpulsation during high-risk percutaneous coronary intervention: a randomized controlled trial.
JAMA. 2010 Aug;304(8):867-74.