International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2019): 7.583

A Review of the Intra Aortic Balloon Pump Counter Pulsation Device

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Abstract: ***Intra-aortic balloon pump (IABP) counterpulsation has emerged as one of the most effective and most frequently employed methods of mechanical circulatory support. Specifically, it relies on the twin concept of diastolic augmentation and afterload reduction to facilitate the functioning of an ischemic and failing myocardium. 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. (1)***

Keywords: IABP, circulation, support

1. History

Dr Kantrowitz (1918 –2008) was an American cardiac surgeon whose team performed the world's first pediatric heart transplant at Maimonides Medical Center in Brooklyn on December 6, 1967. The actual surgery was performed in the middle of the night by his resident. It was only the second time that a human heart had been transplanted into another human being, taking place just three days after Christiaan Barnard's seminal attempt in South. Kantrowitz invented the intra-aortic balloon pump (IABP), a left ventricular assist device (L-VAD), and an early version of the implantable pacemaker. He described augmentation of coronary blood flow by retardation of the arterial pressure pulse in animal models in 1952.

In 1958, Harken 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 developed an experimental prototype of an IABP whose inflation and deflation were timed to the cardiac cycle.

In 1968, Kantrowitz 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 described the first percutaneous insertion of IABP. (2)

The first prefolded IAB was developed in 1986.

2. Introduction

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 an 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.

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. 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 after load. The IABP inflates at the onset of diastole, thereby increasing diastolic pressure and deflates just before systole, thus reducing LV after load. The magnitude of these effects depends upon:

a) *Balloon volume*: the amount of blood displaced is proportional to the volume of the balloon.
b) *Heart rate*: LV and aortic diastolic filling times are inversely proportional to heart rate; shorter diastolic time produces lesser balloon augmentation per unit time.
c) *Aortic compliance*: as aortic compliance increases (or SVR decreases), the magnitude of diastolic augmentation decreases. (3,4)

Myocardial Oxygen Supply and Demand

Inflation of IAB during diastole increases the pressure difference between aorta and left 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), will increase if the IABP is working optimally. This can be evidenced by a decrease in coronary sinus lactate. (5)

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 will 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. (6,7)

Indications for IABP

Over the years, a variety of indications for the use of IABP have developed in clinical practice. The hemodynamic criteria for mechanical circulatory support is when, despite adequate preload and max pharmacological support, cardiac index < 1.8lt/min, systolic arterial pressure < 90 mmHg, LA or RA pressure > 20 mmHg, urine output < 20 ml/h, SVR > 2100 or there is persistent metabolic acidosis.

Clinical indications are left ventricular failure, cardiogenic shock, refractory unstable angina, failure to wean from cardiopulmonary bypass, high risk/failed PTCA, as a bridge to heart transplantation, stunned myocardium, any low cardiac output state due to myocardial contusion, myocarditis or cardiomyopathy.(8)

Variables that influence diastolic pressure augmentation during balloon inflation are,

a) Balloon position: the closer to the aortic valve the greater the diastolic pressure elevation.
b) Balloon Volume: When the balloon volume is equal to the stroke volume the diastolic augmentation is maximized.
c) Balloon diameter and occlusivity: The greatest augmentation occurs with complete aortic occlusion.
d) Balloon Configuration/Driving gas & timing.
e) Stroke volume: If stroke volume is less than 25 ml little diastolic augmentation can be expected.
f) Arterial pressure: Aortic elasticity is important as aortic volume doubles between a mean arterial pressure of 30 mmHg and a normal mean pressure of 90 mmHg.

Steps are involved during insertion of an IABP:

1) An initial physical examination focusing on peripheral vasculature should be conducted including palpation and demarcation of the femoral, popliteal, dorsalis pedis, and posterior tibial pulses and auscultation for femoral and abdominal bruits.
2) The side with the better arterial pulsations should be selected for insertion.
3) The inguinal region should be inspected for landmarks and the femoral artery should be identified.
4) The inguinal region should be prepared and draped in a sterile fashion.
5) Following administration of a local anesthetic agent, a skin incision is made 2 to 3 cm below the inguinal ligament.
6) Using a modified Seldinger technique, the femoral artery is cannulated with a needle and a J-tipped guide wire is then advanced through the needle after brisk flow of arterial blood is confirmed.
7) The guide wire should be advanced to the level of the descending aorta under fluoroscopic guidance.
8) A dilator is inserted and removed until an arterial sheath can be safely placed.
9) The intra-aortic balloon is passed over the guide wire to a position just distal to the origin of the left subclavian artery.
10) The guide wire is subsequently removed and the catheter lumen is aspirated to remove any residual air or thrombus.
11) The intra-aortic balloon is connected to the drive system console and counterpulsation can subsequently begin.
12) The hemodynamic tracing should be inspected for proper timing.
13) A chest radiograph should be obtained to document correct positioning.
14) The intra-aortic balloon catheter and femoral sheath should be secured with sutures.

The IABP Device

The IABP device has two major components:

a) A double-lumen 8.0-9.5 French catheter with a 25-50 ml balloon attached at its distal end and
b) A console with a pump to drive the balloon. The balloon is made of polyethylene and is inflated with gas driven by the pump. Both helium and carbon dioxide have been used as driving gases. Helium has the advantages of speed of gas entry and retrieval as well as maintenance of a larger volume of gas within the balloon for a longer period of time; due to lower viscosity of helium. 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 (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.

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 peri procedural mortality. 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.

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 dicrotic notch on the central arterial pressure tracing/middle of the T-wave. The balloon deflates at the onset of LV systole and this corresponds to the peak of the R-wave. As the balloon inflates at the onset of diastole, a sharp and deep 'V' is observed at the dicrotic notch. Poor ECG quality, electrical interference, and cardiac arrhythmias can result in erratic balloon inflation.

Depending upon the patient's 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.

Arterial pulse timing: For situations when the patient is not paced, nor is the ECG any good. It is a poor second to ECG timing because of a noticeable delay to the balloon inflation. Ideally you'd expect the balloon to start inflating about 40 msec before the dicrotic notch (to compensate for the fact that even helium doesn't flow instantly). By using pressure trigger, one relies on the propagation of the pressure wave, which - though brisk, ~100m/sec - is not as fast as the electrical signals. Delays ranging from 60-119 msec were seen. l This has the effect of decreasing diastolic augmentation and increasing afterload, which could be disastrous.

Asynchronous timing: This also an option. The pump defaults to a regular rate of 80 bpm, irrespective of what the myocardium is doing. The inflations aren't timed to the cardiac cycle in any sense and only useful if there is no cardiac cycle, i.e. the patient is asystolic

IABP Waveforms

In normal inflation-deflation timing, balloon inflation occurs at the onset of diastole, after aortic valve closure; deflation occurs during isovolumetric contraction, just before the aortic valve opens. In a properly timed waveform, as shown, the inflation point lies at or slightly above the dicrotic notch. Both inflation and deflation cause a sharp V shape. Peak diastolic pressure exceeds peak systolic pressure; peak systolic pressure exceeds assisted peak systolic pressure. (9)

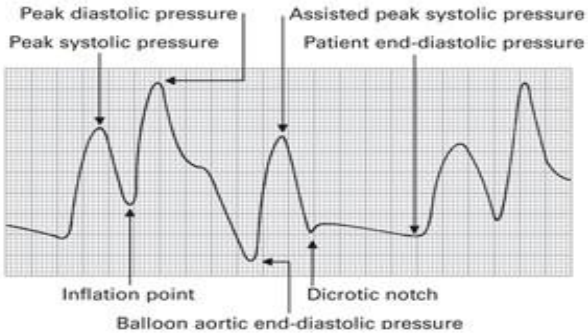

Figure 1: Normal IABP Inflation Deflation Timings

Early inflation: The inflation point lies before the dicrotic notch, during systole, before the aortic valve is closed, which dangerously increases myocardial stress and decreases CO. (Fig 2)

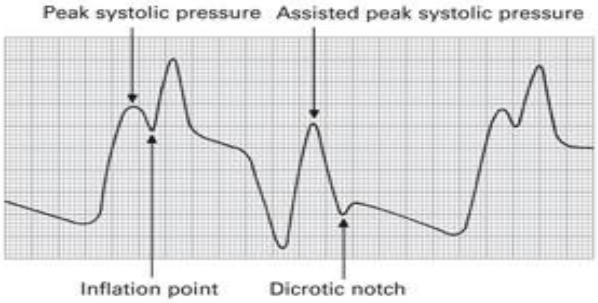

Figure 2: Early IABP inflation

Late inflation: The balloon inflates after the aortic valve closes. The dicrotic notch precedes the inflation point, and the notch and the inflation point create a W shape. This can lead to a reduction in peak systolic pressure and coronary perfusion pressure. (Fig 3)

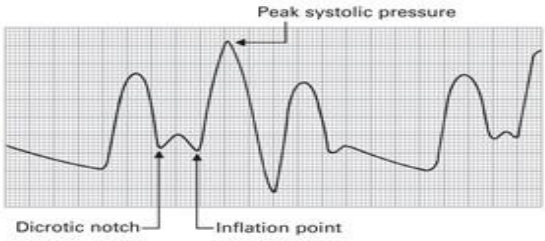

Figure 3: Late IABP inflation

Early deflation: A 'U' shape appears and peak systolic pressure is less than or equal to assisted peak systolic pressure. This won't decrease afterload or myocardial oxygen consumption. (Fig 4)

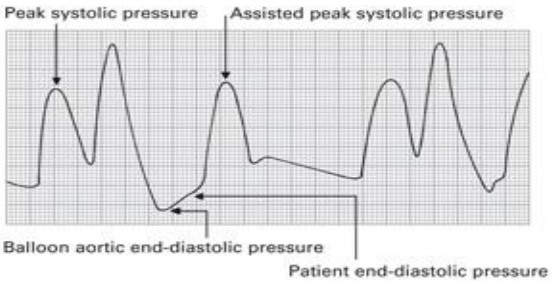

Figure 4: Early IABP deflation:

Late deflation: The peak systolic pressure exceeds assisted peak systolic pressure. This threatens the patient by increasing after load, myocardial oxygen consumption, cardiac workload, and preload. It occurs when the balloon has been inflated too long or inflates at the beginning of ventricular ejection. As a result, the left ventricle has to eject blood against the resistance of the inflated balloon. (Fig 5)

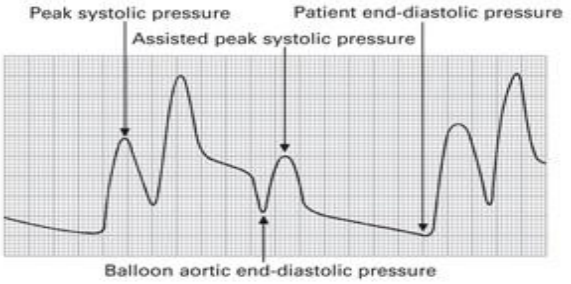

Figure 5: Late IABP deflation

Details during different clinical scenarios and IABP

1) **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. Severe mitral regurgitation secondary to papillary muscle dysfunction or rupture after myocardial infarction can lead to significant haemodynamic instability. With an IABP, there appears to be a 20% to 30% increase in cardiac output in patients with low-output syndromes and a significant amount of afterload reduction as demonstrated in reduction of mitral regurgitation. Direct measurement of coronary blood flow during IABP function has demonstrated augmentation in non diseased and post-angioplasty vessels, but no increase in vessels distal to significant stenosis. This can initially be managed by IABP, pending definitive surgery.
2) **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.
3) **Cardiogenic Shock**
This is a life-threatening complication of acute myocardial infarction and 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.
4) **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.
5) **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.
6) **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 for IABP

IABP 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, bleeding diathesis and irreversible brain death are relative contraindications to the placement of IABP device. (10)

Weaning from IABP

This 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.

It is possible to slave the pump to the temporary pacemaker pulse generator, to time the deflation of the balloon according to the pacemaker pulse instead of the R

wave. Clever modern pumps have "atrial" and "ventricular" pacing trigger settings, with appropriate timing offsets.(11)

Steps for removal of an IABP

1) Anticoagulation should be stopped; confirm that the activated clotting time (ACT) is less than 180 seconds or the activated partial thromboplastin time (aPTT) is less than 40 seconds.
2) Conscious patients should receive a low dose narcotic and/ or analgesic agent.
3) The securing sutures are cut.
4) The drive system console is turned off.
5) The intra-aortic balloon is completely deflated by aspiration with a 20-mL syringe attached to the balloon inflation port.
6) The sheath and intra-aortic balloon catheter are pulled as one unit.
7) Blood is allowed to flow from the arterial access site for a few seconds to remove any thrombi.
8) Manual pressure is applied above the puncture site for 30 minutes or longer if hemostasis is not obtained; a mechanical compression device can also be used to help apply pressure to promote hemostasis.
9) Distal arterial pulsations are palpated.
10) The patient should remain recumbent for a minimum of 6 hours to prevent any subsequent hemorrhage or vascular complications at the arterial access site

3. Conclusion

Although IABP provides less hemodynamic support when compared with newer mechanical circulatory support devices like Cardiopulmonary Bypass Pumps/ ECMOs, internal or external. Counterpulsation can still be the mechanical support device of choice in appropriate situations because of its relative simplicity of insertion and removal, need for smaller size vascular access and better safety profile.

The efficacy of the IABP is reflected by the positive outcomes of the high number of patients who are weaned from the device. The success rate is higher for patients, in whom the device is deployed early, reflecting the reversible nature of ischaemic pathology and the positive contribution of the IABP to preventing the cascade of complications of heart disease.

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