

# Intra-Aortic Balloon Counterpulsation Therapy and Its Role in Optimizing Outcomes in Cardiac Surgery

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## 1. Introduction

Several discoveries and inventions in medicine have revolutionized its practice. Examples would include the discovery of Insulin by Dr William Banting in 1920. The discovery of Heparin by Dr Jay McLean and its first clinical use in Toronto in 1933-36, the advent of the membrane oxygenator, heart lung machines with progressively smaller footprints, intra aortic balloon (IAB) pumps and VAD's (ventricular assist device) would be some of the devices which significantly impacted outcomes in cardiac surgery.

The fundamentals of IAB technology were first tested by Harken in 1958, who is credited with the first use of diastolic augmentation. The pump for Harken's system was a failure due to massive hemolysis. Mouloupoulos (in the 1960's) from the Cleveland Clinic developed the first successful prototype of an Intra-aortic balloon pump (IABP) which could be timed to the cardiac cycle.

The IABP device as we know it was reported by Dr Adrian Kantrowitz (Fig 1) and his team from Grace Sinai hospital in Detroit. The first clinical implant was performed at Maimonides Medical Centre, Brooklyn, NY in Oct 1967 for a 48 yr old woman in cardiogenic shock unresponsive to traditional therapy. The IAB was inserted through a cut down of the left femoral artery (LFA) and pumping performed for 6 hrs. The shock was reversed and the patient discharged. The device was further developed for cardiac surgery by Dr David Bregman at New York Presbyterian Hospital in 1976.

Studying the history of counterpulsation elucidates the great strides in IAB technology and its clinical applications. The size of the balloons initially inserted were as large as 15 Fr. Two operations were required for balloon usage, one to insert the balloon by cut down in a femoral artery, and a second operation to remove the balloon. Advances in technology afforded progressively smaller IAB catheter sizes and eventually 8 and 9Fr. balloons were developed. Current IAB catheter sizes are 7 and 7.5Fr.

In 1968 -Kantrowitz and his group began to use the IABP regularly in clinical practice.

Since 1979 balloon placement utilizes the Seldinger (percutaneous) technique.

The wrapped IAB was developed in 1985. Advances in technology facilitated graduating from cut down insertions to percutaneous and sheathless insertions going from cut down insertions to percutaneous and finally sheathless insertions. Smaller diameter catheters permitted this along with user friendly consoles with automated and real time timing algorithms.



Fig. 1. Datascope System 80

## 2. Fundamentals of cardiac physiology

Understanding of counterpulsation, requires a knowledge of relevant cardiovascular physiology. The heart works as a series circuit of pumps, the left system and the right system.

The right atrium (RA) receives blood from the inferior and superior vena cava and coronary sinus, most of which flows passively into the right ventricle (RV) through the tricuspid valve. An additional 20% of ventricular filling occurs through the atrial kick corresponding to the “p” wave of the Electrocardiogram (ECG). (Quaal 1993)

The right system feeds the low pressure pulmonary vasculature which offers little resistance to the blood ejected from the right ventricle (RV). Consequently the right ventricular musculature is one third the girth of the Left Ventricle. The left atrium receives blood from four pulmonary veins and passively empties the blood into the left ventricle through the mitral valve. Since there are no valves in the pulmonary veins, elevation of left atrial pressure results in an increase in pulmonary vascular resistance (PVR).

Generation of high pressure is required by the left ventricle, most of the pressure being generated occurs during isovolumetric contraction (Fig2-phases of contraction-electrical and mechanical) in order to open the aortic valve, and overcome SVR and aortic end diastolic pressure (AEDP), which is a function of the systemic vascular resistance.

The intraventricular septum also contributes to the left ventricular ejection along with the thick circular posterior and lateral walls.

Some of the energy imparted to the blood through ventricular ejection is stored in the proximal aorta and large arteries as potential energy during their peak expansion. This is known as the Windkessel effect. In diastole, this energy is transformed into kinetic energy by the aorta and large arteries causing a recoil, which maintains a pressure head in the aorta. This in turn maintains a runoff during diastole into the peripheral arteries. It's important to note that coronary arteries fill during diastole.

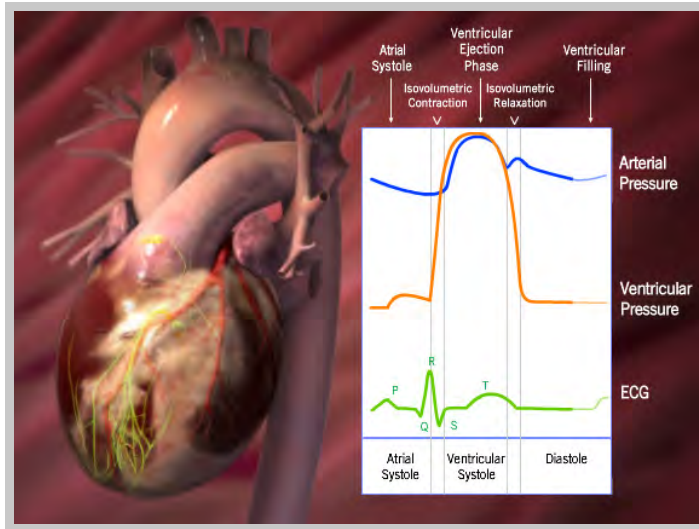


Fig. 2. Phases of electrical and mechanical contraction

### 3. Cardiac output

Cardiac output is the product of stroke volume and heart rate. Stroke volume is the amount of blood ejected by the Left Ventricle with every beat.

Stroke volume is dependent on

1. Preload
2. Afterload
3. Inotropy or (ventricular contractility) and
4. Heart rate

### 4. Preload

This concept suggests that the length of ventricular muscle fiber determines the magnitude of contraction. The length of the LV muscle fiber in turn is dependent upon the left ventricular end diastolic volume (LVEDP). In other words increase in left atrial filling would increase the magnitude of LV contraction. The ability of the LV to vary the strength of its contraction as a function of the LVEDP and end diastolic muscle fiber length is defined as Frank Starling law (fig 3). This gain in contractility is impaired when the stretch goes beyond physiological limits. In clinical practice the length of the muscle fiber is proportional to the LVEDP 'which is measured indirectly as the pulmonary artery wedge pressure (PAWP) by means of a Swan Ganz catheter. In a normal LV, very slight changes in PAWP or LVEDP, produces significant increases in stroke volume.

### 5. Afterload

Afterload is defined as the resistance to LV ejection. Major components are SVR and AEDP. AEDP is the resistance the LV has to overcome in order to open the aortic valve. Ninety percent of myocardial oxygen consumption takes place during the isovolumetric

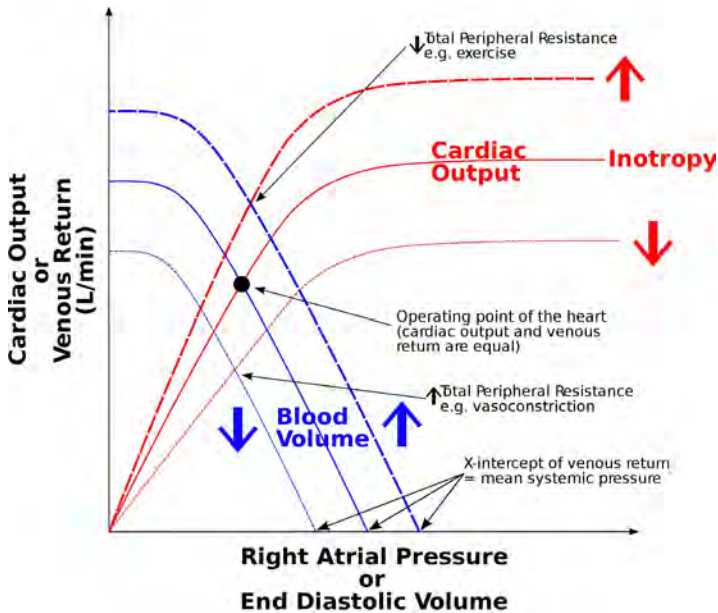


Fig. 3. Starling Law

contraction phase when the LV is trying to overcome the resistance caused by the AEDP in order to open the Aortic valve. The aim of counterpulsation is to lower AEDP, thereby reducing afterload and myocardial oxygen consumption during isovolumetric contraction (fig 4).

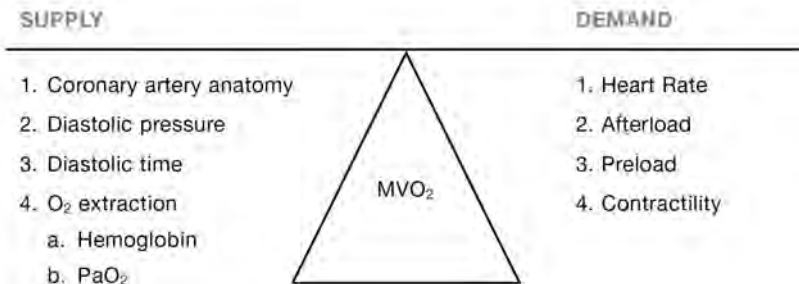


Fig. 4. Myocardial Oxygen Supply and Demand

### 6. Contractility

Contractility is defined as the change in the force of contraction, independent of myocardial fiber length. Contractility inotropic performance can be increased by endogenous production of catecholamines or exogenous administration of vasopressors like norepinephrine (NE), dopamine, calcium etc. Myocardial contractility decreases with hypoxemia and drugs like barbiturates, procainamide, lidocaine, propranolol etc.

## 7. Ventricular function

In order for us to appreciate the need for the IAB, we need to understand ventricular function, end diastolic volume and the Fick equation. Ventricular function in critical care is assessed by the Ejection Fraction (EF). EF is defined as the volume of blood ejected per beat (stroke volume or SV) divided by the volume in the LV prior to ejection, end diastolic volume (EDV). Stroke vol equals EDV minus end systolic volume (ESV). The equation for EF would then be:

$$EF = \frac{EDV - ESV}{EDV} = \frac{SV}{EDV}$$

Ventricular function is graded into four groups by two dimensional echocardiography according to ejection fraction as shown in fig 5 (Conolly HM & Kohl J 2012).

LV Grade	Ejection Fraction
Grade 1 LV	≥ 55%
Grade 2 LV	45 to 54%
Grade 3 LV	30 to 44%
Grade 4 LV	≤ 30%

Fig. 5. LV Grade and Corresponding Ejection Fraction

**Diastolic volume index:** EDV indexed to the patients body surface area (BSA) is another measure of ventricular performance. A normal index is considered less than 100ml/m<sup>2</sup>. In patients with regurgitant valves and volume overload, the end diastolic volume index (EDVI) may be high despite preserved LV function.

**The Fick Equation:** Defines the relationship between C.O and oxygen extraction.

$$O_2 \text{ consumption} = C.O \times O_2 \text{ content difference (sao}_2\text{-svo}_2\text{)}$$

$$C.O = O_2 \text{ consumption} / O_2 \text{ content difference (sao}_2\text{-svo}_2\text{)}$$

Therefore CO is inversely proportional to O<sub>2</sub> content difference, assuming that O<sub>2</sub> consumption remains constant. A normal value for arterio-venous oxygen difference would be less than 5 ml/dl or 50ml/L. A low cardiac output state would encourage increased oxygen extraction, resulting in widening of arterio-venous o<sub>2</sub> content of greater than 5ml/dl. (Hensley et al.,1995 )

## 8. Heart rate

Increase in the frequency of contraction increases C.O at a given filling pressure. This is called the staircase (Bowditch) effect. Reduction in diastolic time can result in reduction of ventricular filling, thus limiting C.O increase with tachycardia. An important corollary to remember is that the filling of coronary arteries takes place in diastole and tachycardia can compromise diastolic coronary filling.

## 9. Coronary circulation and anatomy

The myocardium is perfused during diastole through the coronary system (fig 6).

The major vessels of the coronary circulation are the left main coronary that divides into left anterior descending and circumflex branches, and the right main coronary artery. The left and right coronary arteries originate at the base of the aorta from openings called the coronary ostia located just distal to the aortic valve leaflets.

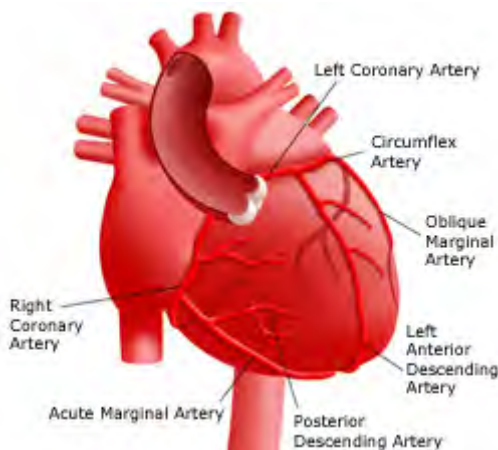


Fig. 6. The Coronary System

The left and right coronary arteries and their branches lie on the surface of the heart, and therefore are sometimes referred to as the epicardial coronary vessels. These vessels distribute blood flow to different regions of the heart muscle. When the vessels are not diseased, they have a low vascular resistance relative to their more distal and smaller branches that comprise the microvascular network. As in all vascular beds, it is the small arteries and arterioles in the microcirculation that are the primary sites of vascular resistance, and therefore the primary site for regulation of blood flow. The arterioles branch into numerous capillaries that lie adjacent to the cardiac myocytes. A high capillary-to-cardiomyocyte ratio and short diffusion distances ensure adequate oxygen delivery to the myocytes and removal of metabolic waste products from the cells (e.g.,  $\text{CO}_2$  and  $\text{H}^+$ ). Capillary blood flow enters venules that join together to form cardiac veins that drain into the coronary sinus located on the posterior side of the heart, which in turn drain into the right atrium. There are also anterior cardiac veins and thebesian veins that drain directly into the cardiac chambers. The LAD supplies blood to the front (anterior-septal) portion of the heart and the LCX supplies the side (anterio-lateral) and back (posterior) of the left ventricle. The right coronary artery supplies blood to the ventricles, the right atrium (RA), the inferior portion of the myocardium and the sino-atrial node. The LAD give rise to various branches called the diagonals and marginal's while the RCA bifurcates into the posterior descending artery (PDA) and the acute marginal artery. The RCA supplies blood to the inferior portion of the myocardium.

## 10. Heart failure

Forward heart failure or congestive heart failure is defined as the inability of the heart to keep up with its demand. Forward heart failure can lead to backward heart failure. Heart failure can be caused by structural heart disease, coronary artery disease, Cardiomyopathy or conduction disorders, more often than not requiring surgical correction. Conduction disorder can impair C.O by causing a too slow/fast heart rate, loss of atrial kick or loss of conduction. Left ventricular failure is directly related to the need for an IAB. Heart failure can be manifested as angina, crescendo or unstable angina or exercise induced angina. Heart failure causes ischemia and myocardial ischemia can be silent. Ninety seven percent of peri-operative

ischemia has been found to be silent (Hensley 1995). Silent ischemia can translate into peri-operative infarct or ischemia both of which could cause heart failure. Left Ventricular failure can be divided into three stages. Stage 1 (fig 7) is manifested as vasoconstriction leading to decreased pumping efficiency, increased LV volume and pressure. These physiological events activate baroreceptors leading to increased heart rate, increased afterload (SVR), increased myocardial oxygen demand and ultimately an increase in preload (LVEDP).

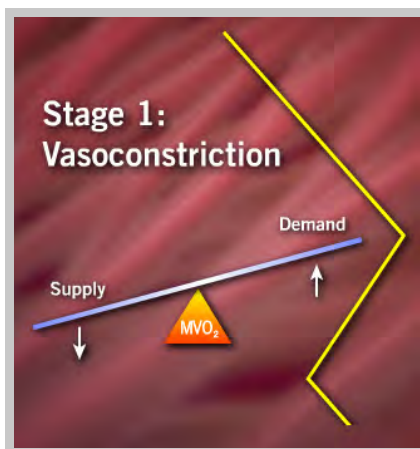


Fig. 7. Stage 1 Vasoconstriction

Stage two (Fig 8) leads to hypervolemia as the heart tries to compensate with the additional afterload. There is a decrease in cardiac output and glomerular filtration pressure, further activating the Renin-Angiotensin system. There is an increase in sodium and water reabsorption leading to an increase in preload and afterload. There is a decrease in cardiac

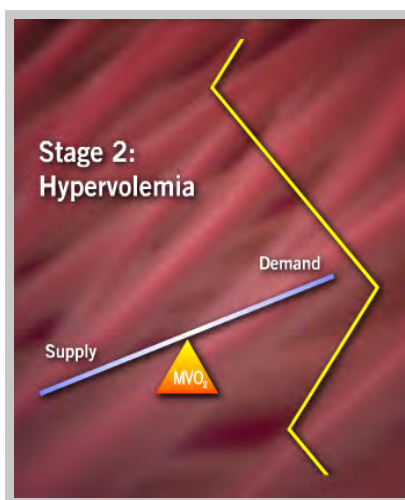


Fig. 8. Stage 2 - Hypervolemia

output, decrease in oxygen supply, increase in heart rate and increase in oxygen demand. There is an increase in pulmonary artery wedge pressures.

The final stage of LV failure (Fig 9) is the manifestation of tissue hypoxia. Decreased cardiac output, decreased MAP, decreased oxygenation coupled with pulmonary edema causes acceleration of anaerobic metabolism, lactic acid production, Tissue anoxia and finally tissue death. Its ideal to insert the IAB in stage one or by stage two, so that we prevent the final stage of heart failure.

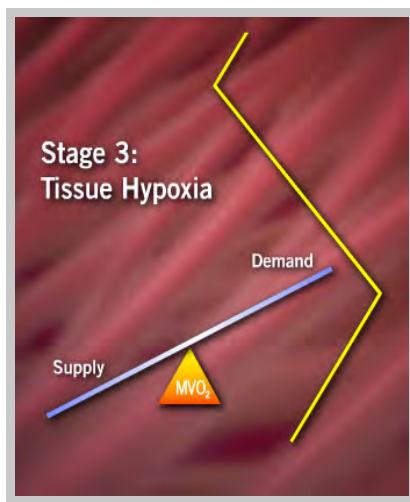


Fig. 9. Stage 3 - Tissue Hypoxia and Apoptosis

## 11. Indications for mechanical circulatory support

Before we cite specific indications for IAB insertion, we should discuss indications for mechanical circulatory support. In other words, we should be able to delineate or define ventricular failure. LV failure would be defined as cardiac index (CI) of less than 1.8 L/min/m<sup>2</sup> with a systolic blood pressure of less than 90 mmhg (ref: Hensley Martin) despite maximized preload (mean atrial pressure > 20 mmhg), optimized heart rate (> 80) and normalized ionized calcium. This could be extrapolated to RV failure except for the systolic blood pressure. RV work is the function of the difference between the RA and PA mean pressure. As the difference between the two approaches zero, pulmonary blood flow is passive and RV failure is present. RV failure can occur with or without pulmonary hypertension.

Mechanical support is suggested when the above criteria are present despite maximum inotropic support. Maximum inotropic support can be defined as any two or more (*High et al., 1995*) of the following combinations:

1. > 10 µg/kg/min of Dopamine
2. > 10 µg/kg/min of Dobutamine
3. > 0.2 µg/kg/min of Epinephrine
4. > 0.75 µg/kg/min of Milrinone after loading dose
5. > 10 µg/kg/min of Nor-epinephrine

For patients with severe forms of LV failure, ventricular assist devices (VAD) are indicated. The initial indicated mechanical support in these scenarios is the IABP. Large and prolonged



inotrope infusion will only tend to increase the workload of the ventricle. The IABP does the exact opposite, decreases the workload of the heart.

## 12. Indications for IAB insertion

In cardiac surgery the IAB is indicated for the following situations:

- A. Pre-op predictors:
  - 1. Grade 3 to 4 LV dysfunction or ejection fraction (EF) of less than 0.30 (*Dietl CA et al.,1996*)
  - 2. Severe and/or multiple valvular disease with end stage myocardial impairment not including aortic regurgitation
  - 3. Anticipated prolonged CPB time
  - 4. Coronary artery disease (CAD) only partially correctible by grafting with concomitant LV dysfunction
  - 5. Persistent ST Changes before, during or after induction of general anesthesia
  - 6. Coronary obstruction via clot or otherwise
- B. Intra/post-op predictors:
  - 1. Pre/post CPB ischemia
  - 2. Incomplete repair or bypass
  - 3. Prolonged CPB time
  - 4. Large ventriculotomy or LV resection for LV aneurysm repair.
  - 5. Particulate or air embolus in coronary arteries.
  - 6. Persistent ST changes post CPB.

Clinical indications for the IAB are listed below (fig 10 showcases benchmarks in 2005 for IAB use)

- 1. Ventricular failure after myocardial infarction (MI) – (*Barron et al., 2001*) or acute myocardial infarction (AMI). As in all other cases the IAB will would decrease afterload and increase coronary perfusion. Early insertion is recommended to ameliorate the threatening extension of MI.
- 2. Angina- chest pain is usually the initial stages of an MI and here again early insertion is recommended
- 3. Unstable angina with or without ST segment elevation
- 4. Cardiomyopathy- In the majority of cases when cardiomyopathy occurs the patient suffers from dilated cardiomyopathy and the IAB assist in raising mean blood pressure and reducing afterload. In a very few cases of initial stages of hypertrophic cardiomyopathy, where the ventricle tends to be a small volume ventricle (extremely thick myocardial wall), a sudden decrease in AEDP would result in insufficient volume left to fill the increased capacitance . This would actually result in a drop in MAP and systolic blood pressure, especially at an augmentation of 1:1
- 5. Acute mitral valve regurgitation (*Abid et al.,2002*) and/or stenosis with LV rupture
- 6. Aortic stenosis without aortic insufficiency (AI) or accompanied by mild AI
- 7. Congestive heart failure
- 8. Intractable ventricular arrhythmias secondary to ischemia or otherwise. In these cases IAB's with fibre optic technology can be used to track rapid or irregular heart rhythms
- 9. Ventricular irritability
- 10. Bridge to transplant or destination therapy
- 11. Support in the catheterization laboratory (*Stone GW et al.,1997*) for stenting or PTCA (Percutaneous Transluminal Coronary Angioplasty)

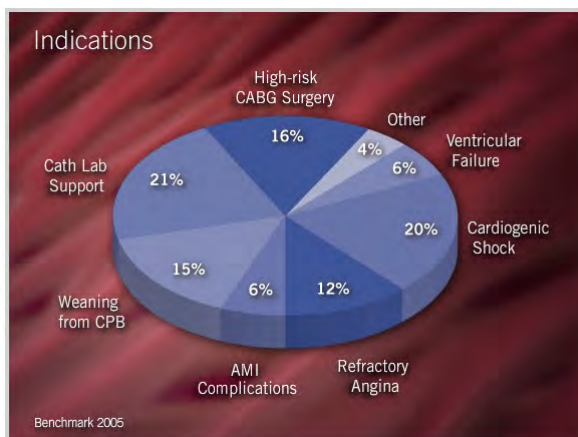


Fig. 10. BENCHMARK for IAB use (2005)

12. Myocardial ischemia or stunning of the heart- In this scenario much of the myocardium has suffered reversible damage and scarring has occurred only in a portion of the heart.
13. VSD (ventricular septal defect) - particularly post MI. This may be accompanied by papillary muscle rupture and acute mitral regurgitation
14. Support in the General OR for patients with ongoing heart disease or a history of heart disease
15. Transport (*Sinclair TD & Verman HA,2009*)for unstable patients
16. Left main disease
17. Cardiac contusion and/or trauma
18. Septic shock or pre-shock syndrome
19. Pulsatile flow (*Onorati F et al.,2009*)during CPB

Freedman coined the term "Myoconservation" (*Quaal 1993*). It is defined as the hemodynamic support provided within the crucial window of opportunity, ensuring that enough myocardium remains viable to permit normal function of the heart following definitive coronary therapy-whether it be CABG or coronary stenting/PTCA. The IAB is one of the very important modalities available to the cardiac surgeon/cardiologist which supports myoconservation by supporting the coronary circulation, supporting the systemic circulation, reduction in LV stress and reduction in LV workload.

Miller et al in 1986 summarize "The result of our clinical experience suggest that more aggressive use of IAB is likely to save lives of coronary disease patients who develop severe complications of their disease. There have been no deaths reported among our patients with refractory unstable angina who had an IAB inserted and all evidence suggests that short of actually opening the vessel to obtain relief from angina, IAB insertion is the most effective method to treat these patients"

More recent studies (*Christenson JT 1999*) have confirmed the efficacy of pre-operative IAB use in high-risk coronary patients.

### 13. Absolute contraindications for IAB insertion

1. Aortic regurgitation or insufficiency. In this physiology, raising AEDP would result in increase of regurgitant factor thereby increasing workload of the heart. In instances of mild AI a decision can be made if the benefits outweigh the risks.

2. Aortic dissection. Precludes IAB insertion. Attempting to Place an IAB in this situation may lead to placement in the false lumen or at the very least increase circulation into the false lumen.

#### **14. Relative contraindications for IAB insertion**

1. Severe peripheral vascular disease (PVD). Although this sometimes precludes insertion, we have had some experience with this. A discussion on PVD patients will be conducted a little later.
2. Unresected thoracic, abdominal or thoraco-abdominal aneurysm. An insertion of a mechanical device like the IAB and the counterpulsation of such a device against a diseased aortic wall may result in aortic dissection
3. Sepsis or infection
4. Severe thrombocytopenia
5. Coagulopathy or coagulopathic disorder
6. End stage terminal disease
7. End stage cardiomyopathy unless bridge to transplant/destination therapy
8. Severe atherosclerosis

#### **15. Pre-insertion predictors of risks associated with IAB insertion**

1. Age- Increased age appears to be an increased risk. It is probably because of increased atherosclerosis associated with increased age (*Goldberger 1986*). There are other studies reporting inconsistent results.
2. Gender- women tend to have a greater risk of complication due to their smaller stature. This is probably due to the smaller lumen size of the femoral artery in women. The IAB catheter was expected to occupy greater lumen space in women thereby increasing the likelihood of ischemia and/or thrombus formation. Women are 1.6 to 1.8 more likely to experience limb ischemia/vascular complications than men (*Skillman JJ et al, 1988*).
3. Peripheral vascular disease (PVD) - These patients have higher likelihood for IAB complications due to insertion difficulties. Gottlieb suggested a three times likelihood for complications (*Gottlieb SO et al.,1984*), others have suggested less (*Skillman JJ et al.,1988*).
4. Type-II diabetes- Due to severe and diffuse atherosclerotic disease, higher incidence of hypertension and dampened resistance to bacterial contamination, diabetics tend to have a higher risk of complication post IAB. Some investigators found that diabetics (*Wasfie T et al.,1988*) had a 22% incidence of complications post IAB insertion as compared to 14% for non-diabetics. For insulin dependent diabetes a higher complication rate of 34% (*Alderman JD et al., 1987*) was suggested.
5. Duration of IAB therapy- Findings for the duration of safe use of IAB therapy remain inconclusive (*Quaal 1993*)

#### **16. Complications of IAB insertion**

1. Loss of pedal pulses- Occurs in 15 – 25% of patients (*funk M et al.,1989*). Asymptomatic loss occurs transiently without resulting in limb ischemia and usually returns spontaneously or after IAB removal.

2. Limb ischemia- Occurs in 12 - 47% patients and is the most frequently reported complication (*Curtis JJ et al., 1988*). This may result from decreased cardiac output subsequent to heart failure, elevated SVR, low output syndrome, intimal injury or dissection, vessel catheter discrepancy, catheter occlusion or distal thrombo-embolism. Limb ischemia usually preceded by pain in the affected limb, change in pallor, cyanotic color changes, mottling, decreases in sensation, motor function loss, decrease in temperatures in extremity and loss of pedal pulses. Treatment is usually done by removal of IAB, thrombectomy, femoro-popiteal grafting and/or papaverine administration.
3. Thromboembolism- Percutaneous insertion/removal of IAB may result in dislodging of plaques or clots into the renal, splanchnic, hepatic or peripheral arteries. Clots can be seen while removing IAB catheters. It is our institutional policy to have some sort of anti-coagulation during IAB therapy. Although Low molecular weight Dextran has been used, heparin is preferable as it can be reversed with protamine. Generally maintaining the aPTT 1.5 - 2 times normal is sufficient to prevent formation of thrombin, thereby protecting from subsequent embolism. It is also recommended to flush the IAB catheter with heparinized saline prior to insertion. The only incidence where this may not be necessary is when the patient is fully heparinized and on CPB. It is always a good practice even in this situation to flush with heparinized saline. At Southlake we use pre-mixed heparinized saline bags with 5000 units of heparin in 500ml of saline.
4. Compartment syndrome- The legs and thighs of humans are made up of compartments containing bone, muscle, nerve tissue and blood vessels, surrounded by a fibrous membrane or fascia. Compartment syndrome is caused by an increased pressure within the non-distensible fascial space reducing capillary blood flow which in turn compromises enclosed fascial tissue. A fasciotomy can be performed to relieve the pressure.
5. Aortic Dissection- Is the most serious of complications from IAB insertion. Often unrecognized until removal or until IAB therapy is discontinued resulting in hemodynamic instability and death. Diagnosis is confirmed upon autopsy. Isner in 1980 found an incidence of aortic dissection in 36% of IAB patients (*Isner JM et al., 1980*). With the advent of smaller French sizes in IAB catheters and better techniques and training, incidence of aortic dissection as a direct consequence of IAB insertion has drastically reduced. Symptoms of aortic dissection include severe back and/or abdominal pain, falling hematocrit and mediastinal enlargement. Aortic Dissection has to be treated aggressively and immediately by attempting to repair the dissection in the operating room.
6. Local injury- False aneurysm formation, hematoma, lymphedema, lymph fistula, wound hemorrhage, laceration of the femoral/iliac artery or the aorta are common complications and can be treated by evacuation and/or arterial repair.
7. Infective complication- Local wound infection is possible necessitating debridement, drainage, systemic antibiotics and rare cases removal. Sterility is very important while inserting the IAB whether in the peri-operative setting or otherwise. Generally the thigh is prepared by applying betadine prior to insertion and is covered by sterile dressing after insertion and anchorage of the catheter by two sutures.

8. IAB rupture/entrapment- IAB rupture is rare but immediate removal/replacement is required. Rupture is more likely in women and patients with smaller size and usually occurs because of the constant contact between IAB membrane and atherosclerotic plaque on the femoral arterial/Aorta walls. The most common sign of IAB leak is blood in the drive line (fig 11). IAB leak can cause either a helium embolus (in instances of patient hypotension where helium pressure within the IAB exceeds blood pressure in the aorta) or entrapment of the catheter. The leak can cause a clot to form within the catheter resulting in prevention of removal of the catheter at the time of IAB removal. This may necessitate a cut down removal of the IAB catheter.

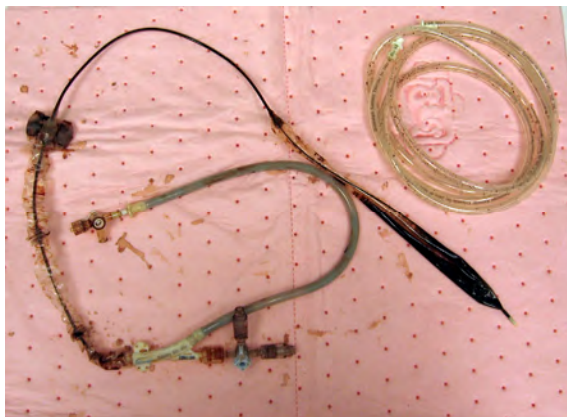


Fig. 11. IAB Catheter with Blood in Drive Line and Balloon

9. Hematologic effects- Long term use of IAB has been associated with increased destruction of red cells and thrombocytopenia. The degree of decrease appears to be related to the duration of therapy (*McCabe 1978*)
10. Malposition of the IAB catheter- If positioned too high, the IAB may occlude blood flowing to the head vessels causing cerebral ischemia and or embolism. If positioned too low it may occlude the renal arteries thereby causing renal ischemia/perfusion and non-optimal coronary perfusion/afterload reduction.

## 17. Insertion techniques

1. Cut down technique- By 1978 the IAB was a 12fr catheter and prior to that year, cut down insertion was common. Initially, the common femoral artery is dissected and the vascular fascia is opened. With a retractor the femoral artery can be dissected up to a length of 5 cms and umbilical tapes are placed around and behind the femoral artery. Heparin 5000u is administered and the IAB catheter is introduced at an angle to the femoral artery. Placement is confirmed and augmentation initiated. This technique can also be used in patients where femoral pulse is not palpable or where difficulty of insertion is anticipated as in patients suffering from PVD.
2. Percutaneous (Seldinger) technique: An 18 gauge cannula is introduced into the common femoral artery at a 45 degree angle or less. The guide wire is advanced through the needle and the needle removed. The tissue tract around the arterial

puncture is then dilated by the dilator provided in the IAB catheter set. The introducer-dilator set is advanced over the guidewire. Finally the dilator is removed and the IAB catheter advanced over the guidewire. The IAB is introduced over the guide wire and placed in the second or third intercostal space. It is recommended to maintain a continuous flush of the IAB catheter with heparinized saline (1000u in 500ml) after placement. It is also recommended to periodically flush the arterial line coming from the IAB in order to prevent clotting of the arterial line coming from the IAB catheter. The manufacturer recommends the IAB catheter to be anchored with two sutures to the subcutaneous tissue of the thigh (Fig 12) at the anchors provided on the side of the base of the catheter or by the sutureless securement device provided.



Fig. 12. Proper Anchoring of the IAB Catheter

3. Sheathless insertion- Initially IAB catheters were enveloped by sheaths with bigger French sizes. E.g. The 8Fr IAB catheter has a sheath whose outer diameter is 10Fr. By not using the sheath, the practitioner is reducing trauma to the groin and femoral artery. Current IAB catheters of 7 and 7.5Fr are inserted in our institution without the sheath. Sheathless insertion also reduces the length of catheter indwelling in the tissue, thereby reducing limb ischemia.
4. Subclavian insertion- Currently the manufacturer and FDA (Food and Drug Administration) recommends (in their IFU-instruction for use manual) femoral insertion of all IAB catheters. IAB catheters have regulatory clearance for femoral insertion and are labeled as such. There are situations where the subclavian artery has been used in order to avoid an aorto-iliac stenosis. A subclavicular incision is done and the subclavian artery isolated via cut down. An IAB catheter is placed antegrade down the descending thoracic aorta.
5. Brachial insertion- The brachial artery can be used as a point of insertion where the patients have bilateral obstructive femoral and iliac disease and/or the patient has bilateral femoro-popliteal graft. Due to discomfort associated with positioning, the authors would recommend only in patients who are intubated and on a ventilator. Care

should be taken to insert the IAB on the left rather than the right side. As right sided insertion would not give the IAB enough length to cross the aortic arch and lie in the descending Aorta. We came across a 74 years female diagnosed with coronary artery disease (CAD) and triple vessel disease (TVD) who had aorto-bifemoral grafts inserted in 2004. Her risk factors included hypertension, active smoking history, type 2 Diabetes and previous MI. Her EF was only 28%, she had moderate MR, moderate PAH and left carotid bruit. She was turned down for surgery. She presented in emergency with angina. Had a syncopal episode with rapid atrial fibrillation. She was cardioverted twice and transported to the cath lab for IAB insertion. An IAB (Fig 13) was initially inserted through the right brachial artery and was observed via fluoroscopy to lie in the aortic arch. The IAB was then removed and re-inserted via the left brachial artery and inserted antegrade down the thoracic aorta. Augmentation was initiated, the patient survived the hypotensive episode, was placed in the CCU and discharged on the 9<sup>th</sup> post-procedure day.



Fig. 13. Transbrachial Insertion

6. Transthoracic Insertion- In instances where the IAB insertion is precluded due to severe PVD or has been tried and failed, The IAB catheter can be introduced down the aorta in operative room situations. We had one such patient- a 65 year old male with active smoking history, no hypertension/daibetic history and a history of pericarditis in 2003. He had a history of severe PVD with bilateral femoral claudication and severe pain in his legs. Angiogram was facilitated through the right radial approach. He underwent CPB and uneventful CABG to his LAD and PIV (posterior interventricular artery). The patient had a hypotensive episode during sternal closure, CPB was re-initiated, mammary spasm suspected and a vein graft anastomosed distal to the previous mammary artery anastomosis. We were unable to separate from CPB, at this time a decision was made to insert an IAB transfemorally. Transfemoral insertion failed and an IAB catheter (Fig 14) was placed antegrade down the thoracic aorta. At this point the patient was able to separate from CPB. Due to suspicion of myocardial edema, late sternal closure was decided upon, the skin closed, dressing placed and patient transported to the CVICU. The patient was taken back to the OR post-op day 3, the IAB

removed and the chest closed. The patient was extubated post-op day 9 and discharged after 25 days. No post-operative morbidity noted other than associate with his history of peripheral vascular disease (*Datt B 2007*)



Fig. 14. Trans-thoracic Insertion of IAB Catheter

### 18. Mechanics of IAB functioning

IAB therapy is often referred to as counterpulsation therapy as the balloon inflates in diastole or counter to the hearts contraction (systole). The IAB is a polyethylene balloon catheter placed percutaneously in the thoracic aorta through the groin (Fig 15).

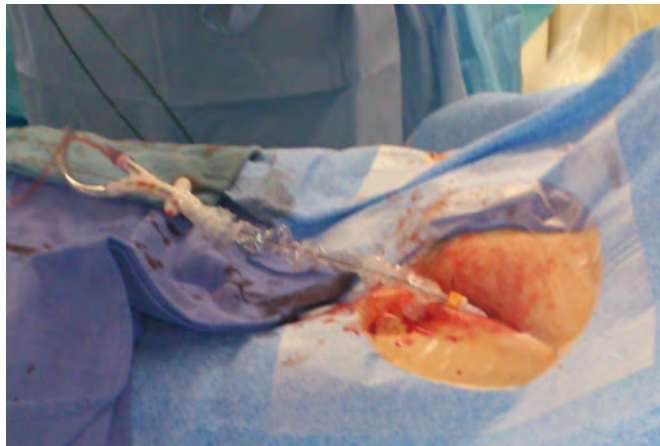


Fig. 15. Femoral Insertion of IAB Catheter

Balloon inflation actively pushes blood into the coronary arteries, increasing myocardial oxygen supply. Balloon deflation decrease AEDP thereby directly reducing myocardial work load and myocardial oxygen consumption by reducing the time period for isovolumetric contraction. This gives the heart time to rest and gives a chance for hibernating myocardium to recover.



The IAB is connected by a driveline to a helium chamber or pressurized gas reservoir which is connected to the IAB catheter via a solenoid valve. The inflation or helium supply is linked to a trigger for the balloon which is usually a synchronized ECG (Electrocardiogram) or the patient's blood pressure. Earlier on in IAB development CO<sub>2</sub> was used due to its high solubility and safety in case of balloon leak/rupture and gas embolization. Helium started being used due to its smaller Reynold's number (lower density), thereby allowing a smaller drive line/catheter. Smaller balloon catheters improved gas shuttle speeds, reduced trauma to the groin and resulted in fewer complications post-insertion.

The balloon is usually placed 2 cms below the subclavian artery in the second to third intercostals space. This optimizes coronary perfusion and decreases the chances for renal artery occlusion. Balloon placement is verified in the OR by TEE or post-operatively by CXR (Fig 16).

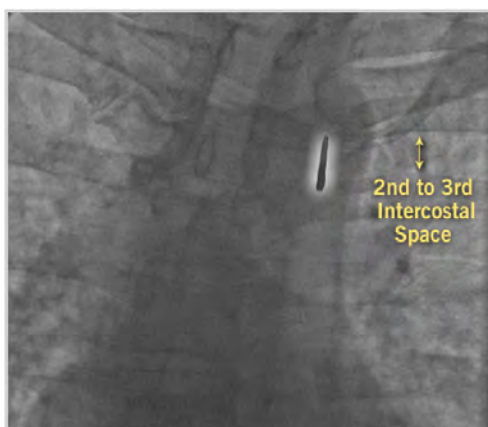


Fig. 16. Chest X-ray Confirmation of IAB Placement

Synchronization of the IAB is achieved usually by using the R wave of the QRS complex to deflate the IAB catheter. In the operating room some practitioners tend to use pressure trigger to circumvent electrical interference from the cautery or other devices. If the patient is pacer dependent, the pacer spike can also be use to trigger IAB deflation. Correct timing is verified by observing the dicrotic notch on the arterial pressure waveform on the balloon console and making sure that balloon inflation takes place just after the dicrotic notch (aortic valve closure) and deflates prior to LV ejection (R wave on QRS complex-Fig 17 & Fig 18)

While using the pressure trigger, it's important to use the dicrotic notch as the marker for inflation. This will prevent the balloon from impinging on LV ejection and adding to the hearts afterload.

At Southlake we use the Maquet IAB console (fig 19) CS 100 which has an auto feature for timing. Another IAB console which the reader may come across would be the ACAT II WAVE console (fig 20) marketed by Arrow international.

The console uses a unique algorithm to establish the initial timing using the ECG or arterial pressure waveforms. It will also automatically readjust the inflation and deflation timing for changes in heart rate or rhythm. This allows for ease of nursing care post-operatively and decreases the necessity of the perfusionist except for troubleshooting. The IAB has three choices for augmentation. 1:1, 1:2 and 1:3. The IAB is generally initiated at 1:2 in order to be

able to check the timing of the IAB catheter. The timing can be verified by using the inflation interval option on the console. Generally at a higher heart rate (>100), a better augmentation pressure is achieved by lowering the ratio to 1:2 or 1:3. The (stroke) volume of the balloon used is dependent on the size of the balloon and is chosen based on the height of the target patient (fig 21).

Recently, due to the reduction in french size of the IAB catheters to 7Fr, it is possible to insert 40cc balloons in most patients, except those very small or very large.



Fig. 17. Inflation (1:1) After timing IAB (pacing trigger)

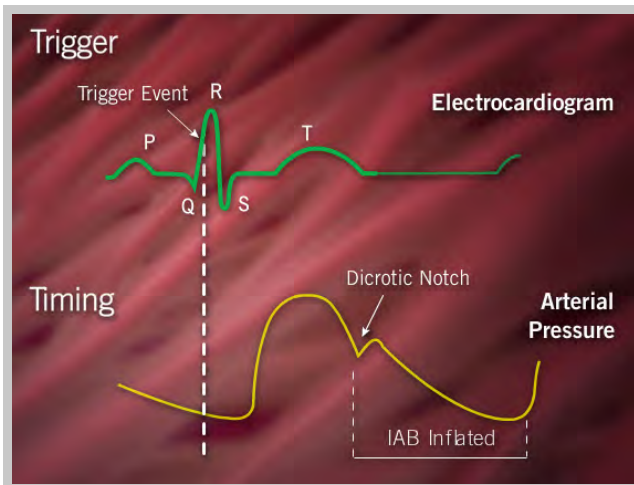


Fig. 18. Proper IAB Timing



Fig. 19. CS100 MAQUET IABP



Fig. 20. ACAT II WAVE Arrow IABP

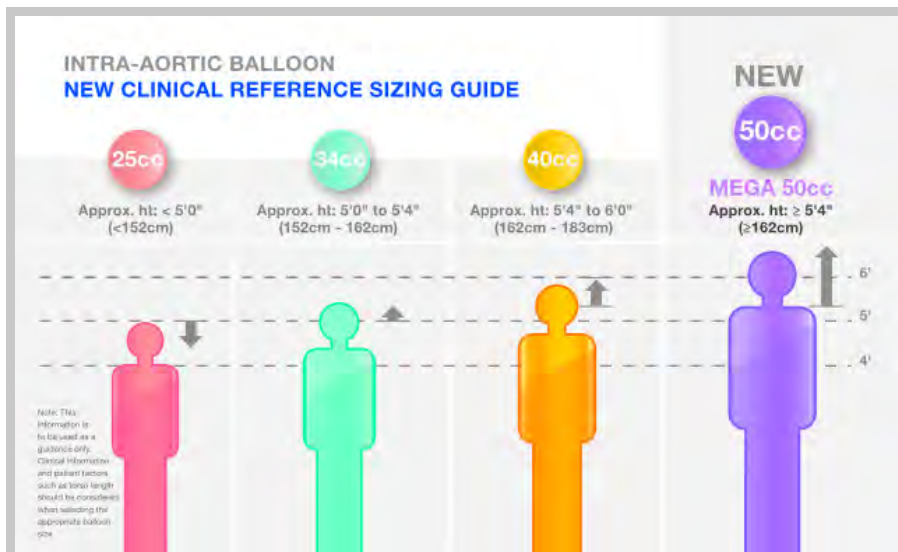


Fig. 21. Size Selection Criteria for IAB Catheter

## 19. Timing errors

There are four major timing errors with the IAB. The IAB is usually timed at a 1:2 ratio (one inflation every second heart beat) and the deflation is triggered off the R wave in the QRS complex. If the IAB is timed improperly, it can actually add to the afterload of the heart by inflating prior to the closure of the aortic valve. In this situation the cardio-vascular hemodynamics can deteriorate quickly and it is important for the practitioner to understand and avoid these errors.

1. *Early inflation:* The waveform shows inflation of IAB prior to diastolic augmentation, diastolic augmentation encroaches into systole (fig 22) and is not hard to miss. The physiological effects would be potential premature closure of aortic valve, increase in LVEDP, LVEDV (Left Ventricular End Diastolic Volume) and PCWP (Pulmonary Capillary Wedge Pressure). The net effect would increase afterload, myocardial oxygen consumption and possibility of adding to Aortic regurgitation.
2. *Late inflation:* Inflation of IAB after the diastolic notch, indicated by the lack of a sharp "V" on the waveform (fig 23). The physiological effects include sub-optimal coronary perfusion and decreased diastolic augmentation.
3. *Early deflation:* Viewed as a sharp drop in the waveform after diastolic augmentation. Assisted AEDP can be equal to the unassisted AEDP (fig 24). There may be little or no decrease in assisted systolic pressure. There is an absence of a sharp "v" or pre-systolic dip. This error will also lead to sub-optimal coronary perfusion, mvo<sub>2</sub> and afterload reduction. There is also a potential for causing retrograde coronary and carotid blood flow with the latter causing an increase in angina.
4. *Late deflation:* Possibly the most dangerous of all timing errors. Rate of rise of assisted systolic pressure may be prolonged. Assisted AEDP may be equal to unassisted AEDP. Diastolic augmentation waveform may be dampened (fig 25), depending on how late

the deflation is. Physiologically, afterload reduction is absent. IAB is actually impeding LV ejection and increasing afterload. Isovolumetric contraction phase increases along with myocardial oxygen consumption. Hemodynamics deteriorate rapidly.

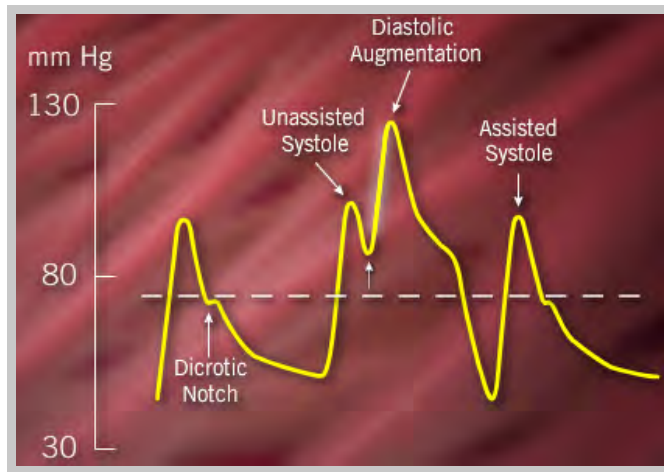


Fig. 22. Early Inflation

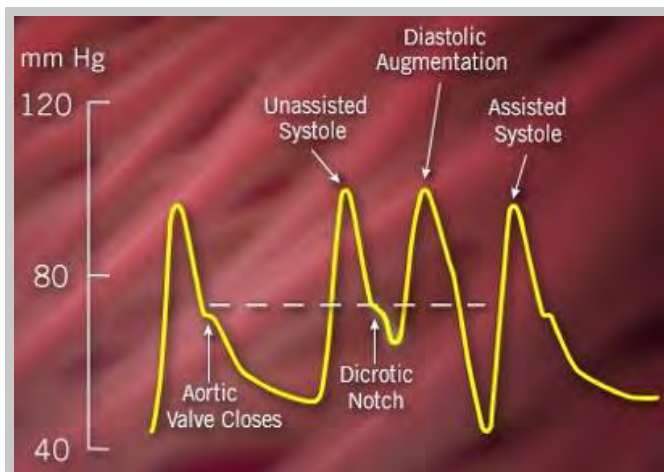


Fig. 23. Late Inflation

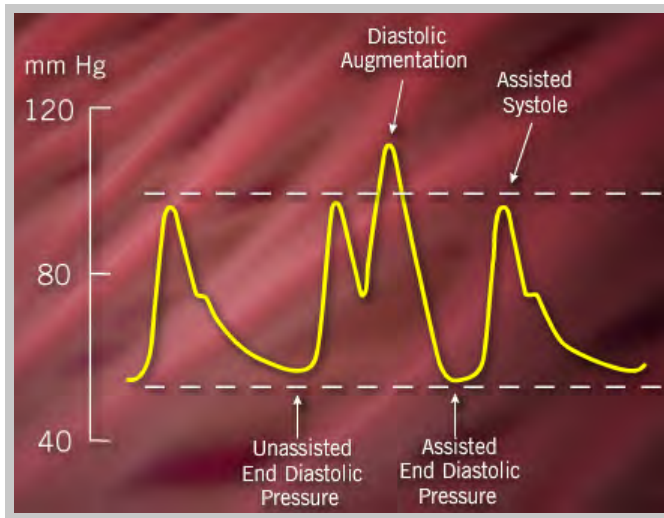


Fig. 24. Early Deflation

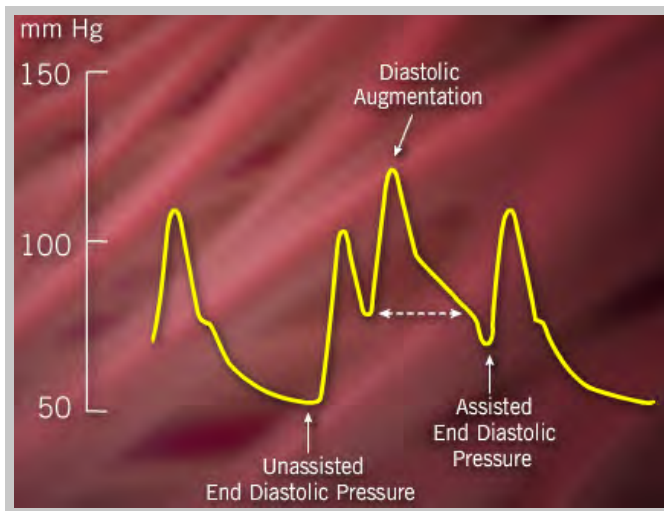


Fig. 25. Late Deflation

## 20. IAB weaning

IAB weaning is usually done when the following physiological parameters are observed in target patients.

1. No afterload reduction as seen on balloon waveform
2. Increased urine output
3. Improved LV function and hemodynamics

4. Cessation and/or improvement of ventricular and/or atrial arrhythmias
5. Cessation or reduction of angina
6. When transportation or operative procedure (revascularization of left main or aortic/mitral valve repair/replacement) is completed and the patient hemodynamics appear to be stable
7. When the shock phase has been successfully tided over

The IAB is generally weaned by reducing the ratio from 1:1 to 1:3, after which the augmentation volume of the balloon catheter can be reduced. If the IAB volume is being decreased as a part of the weaning process, ensure adequate movement of the IAB catheter in order to minimize clotting or thrombus formation. The IAB is never turned off when the IAB catheter lies in the aorta due to the risk of thrombus formation. Afterload reduction by the IAB can be explained via numbers. The stroke volume of an average sized patients in normal health is usually 70 to 80 ml. In a patient in compromised cardio-vascular state, this stroke volume is reduced to 40 or 50ml. What a 40cc IAB catheter does is to complement the native stroke volume of 30-40ml until hibernating myocardium recovery and myocardial rest result in an increased stroke volume of that patient.

## **21. Advancements in IAB catheter and IAB console technology**

Considerable improvements have been made in IAB catheter and console technology in the more than 40 years since counterpulsation was introduced to clinical practice. Early IABP consoles required intensive user intervention, as all aspects of console operation were manually controlled. Early IAB catheters were 12 Fr. and required surgical insertion via a cutdown to the femoral artery. Surgical insertion could take 1-2 hours and complication rates were high. In 1979, IAB catheter insertion improved significantly with the development of percutaneous IAB insertion. Insertion time was reduced, typically to 15 minutes.

Additional improvements to the catheter in the early years included the addition of an inner lumen to facilitate wire-guided insertion and the lumen could then be used to monitor the arterial blood pressure. Prefolding of the membrane during manufacturing eliminated the need for the clinician to wrap the membrane prior to insertion through the introducer sheath. Catheters were also made smaller, from 12 Fr. to 10.5 Fr. and then to 9.5 Fr. Catheters are now 7 and 7.5 Fr. and this has helped reduce the incidence of limb ischemia to less than 3%. Reducing the catheter shaft diameter required a significant change to the way catheters were designed. With early dual lumen catheters, the inner lumen used for wire guiding the insertion and monitoring the arterial pressure was a separate catheter within the balloon catheter gas lumen. With the development of a co-lumen design where the two lumens of the catheter were extruded together with the inner and outer lumens sharing a common wall, the catheter diameter was reduced while maintaining the gas shuttle lumen and maximizing the inner lumen to accommodate the largest guide wire possible. The range of balloon membrane sizes was also expanded over the years from 40 cc in the early days of counterpulsation to 25 cc, 34 cc, 40 cc, and 50 cc IAB membranes. The size of balloon used is determined by patient height. The most recent improvement in IAB catheter technology has been the addition of a fiber optic pressure sensor to acquire the arterial pressure signal. This technology is immune to artifact, resulting in a high fidelity arterial pressure waveform for accurate IAB timing.

While innovations in IAB catheter technology were being developed, improvements in IAB console technology were also occurring. Major improvements to the console over the years

have been aimed at making the algorithms smarter, the pneumatics faster, and the consoles smaller and lighter. Early consoles required constant operator intervention to maintain optimal triggering and timing. Improvements to the algorithms in the console resulted in the automation of timing so that once the operator established the initial timing, the console could adjust the timing for changes in patient heart rate and rhythm. Console pneumatic improvements reduced IAB inflation and deflation time, which was important for tracking tachycardic rhythms. Consoles were made smaller and lighter to facilitate transport by aircraft or ambulance.

The most significant improvement to console design occurred with the development of fully automated operation. With this capability, the console can automatically select the most reliable trigger and establish initial timing. It will then automatically readjust timing for changes in heart rate and rhythm, using advanced algorithms to track predictable (regular) as well as unpredictable (irregular) rhythms. The algorithms will also select an alternate trigger if the current trigger is lost. This automation, along with smaller fiber optic catheters offers the clinician the most advanced IAB counterpulsation system for optimal patient benefit.

## **22. Intra-aortic balloon pump use during percutaneous coronary angioplasty**

The Intra-aortic balloon pump (IABP) can also provide important hemodynamic support during complex percutaneous coronary intervention (PCI), both in the elective setting, and for acute coronary syndromes, including ST segment elevation myocardial infarction. Modern coronary interventions can generally be performed to a wide array of patient subsets with low risk of procedural complications. However, optimal patient outcomes in PCI require identification of higher risk patients, and attempts to modify those risks. One of the main predictors of outcomes in PCI is Left Ventricular dysfunction, i.e. Ejection fraction <30%. The availability of mechanical support helps provide "backup" or reserve during the procedure and decreases the risk of hemodynamic compromise. Typically, mechanical support is initiated prior to commencing PCI, and removed shortly after successful PCI. Intra-aortic balloon pump use results in the dual benefits of increased peak diastolic pressure, while lowering the end-systolic pressure. There is a resultant reduction in after-load (Perera D *et al*,2010) and improved coronary perfusion, which leads to improved myocardial oxygen supply, with decreased myocardial energy demands.

The common scenarios for IABP use in PCI include:

1. Acute Coronary Syndromes (ACS) / ST elevation Myocardial Infarction (STEMI), complicated by cardiogenic Shock, due to LV systolic dysfunction or a mechanical complications such as acute mitral regurgitation or ventricular septal defect.
2. PCI in a coronary artery supplying a large territory of myocardium, typically with underlying Left ventricular dysfunction (practically speaking, Grade 3 or worse - EF < 30%). Specific scenarios include i) coronary intervention on a diseased vessel that also supplies flow to another occluded vessel (via channels, called collateral arteries, that fill that territory in retrograde fashion), ii) unprotected left main stenting, iii) PCI to the only patent artery iv) PCI with concomitant valvular disease, if anticipated deterioration in the valvular disease (ex. patients with at least moderate ischemic mitral regurgitation, and planned PCI that could worsen degree of regurgitation).
3. If patients have clinical signs of abnormal resting hemodynamics, prior to PCI: low systolic blood pressure (SBP) <100, with objective or clinical suspicion of elevated left heart filling pressures - pulmonary capillary wedge pressure >20mmHg.



4. Survival Dependent vessel: Any one artery supplying a sufficient amount of myocardium such that (in the opinion of the cardiologist) closure of the vessel would be fatal.

It should be noted that there is considerable practice variation with Intra-aortic balloon support during high risk coronary intervention. Recognizing the higher risk of access related and other vascular complications with its insertion, some clinicians prefer to have the IABP readily available and use it only if hemodynamic compromise develops. . One trial conducted in centers in the United Kingdom, The Balloon Pump-Assisted Coronary Intervention Study, assessed whether routine intra-aortic balloon pump insertion, prior to PCI, reduced the risk of major adverse cardiac and cardiovascular events in patients with severe left ventricular dysfunction and advanced coronary disease. The results of this trial involving just over 300 randomized patients did not support routine use, given no difference between the groups. Clinical judgment is certainly exercised in situations (such as those described above) where the IABP is inserted "pre-emptively", with anticipated hemodynamic stress during PCI.

### **23. Counterpulsation – An anesthesiologists perspective**

As an anaesthetist, working in the perioperative care of cardiac surgery patients for the last 20 years, I have observed a significant change in the attitudes and uses of the intra aortic balloon pump. I have watched the use of the IABP change from a last resort salvage technique for the patient on maximal inotropes with low output syndrome and a dismal prognosis, to a first line tool aiming for preservation of myocardial tissue in unstable cardiac surgery patients. The presumed IABP's favorable myocardial oxygen supply/demand profile and the ease of percutaneous insertion have been instrumental in this change (*Trost JC 2006*).

In our institution IABPs are often inserted in preoperative cardiac surgery patients: who are experiencing unstable angina, especially post myocardial infarction, not responsive to standard non-invasive therapies; that have low output syndrome or congestive heart failure secondary to severe left ventricular dysfunction or mechanical complications such as a ventricular septal defect or mitral regurgitation induced by a myocardial infarction ; and rarely who have acute myocardial ischemia causing intractable arrhythmias. It appears that the earlier the balloon is placed in a hemodynamically struggling patient, the more stable the patient is in the post-operative period. This practice is consistent with the Task Force on Practice Guidelines of the American Heart Association and American College of Cardiology (*Ryan TJ 1999*). Frequently, IABPs are inserted for preoperative cardiac surgery patients with symptomatic left main disease. The intention of the IABP is the preservation of viable ischemic myocardial tissue. The mechanism of the preservation may relate more to decreased myocardial demand and collateral flow as Kimura showed that in the presence of a coronary artery stenosis of greater than 90% the IABP diastolic augmentation did not result in an increased post-stenotic pressure (*Kimura A 1996*). The insertion of the IABP into the aforementioned patients facilitates the anaesthetist's ability to maintain cardiovascular stability and a favourable myocardial oxygen supply/delivery ratio at induction, maintenance and release from bypass. Replace with" The importance and validity of the IABP in maintaining a favorable myocardial oxygen supply/demand ratio is realized in the post-operative period (*Christenson 1997*).

Intraoperatively, IABPs are inserted into our patients when difficulty to wean from the bypass machine is anticipated due to instability in the pre-bypass or bypass period or for failure to wean from the bypass machine, once reversible causes for the failure are ruled out. There may be a survival advantage to placing the IABP prior to coming off bypass based on one review. Delaying insertion of the IABP in the unstable perioperative patient who is unresponsive to reasonable doses of inotropes or who has rapidly accelerating inotropic requirements may have deleterious effects. Theoretically this is supported by the following facts. High inotropic doses increase myocardial inotropy and tension resulting in increased myocardial oxygen demands. Impaired diastolic relaxation and increased ectopy and afterload, as seen with certain inotropes, may further tip the myocardial supply/demand balance in favour of demand (*Katz AM 1990*). Myocardial injury may be further exacerbated by the inotropes increased delivery of calcium to the cell, overcoming the ischemic myocardium's presumed protective attempt at limiting available calcium. Early insertion of the IABP in the unstable post-bypass patient appears to theoretically and anecdotally improve outcome (*Poole Wilson PA 1990*).

Postoperative insertion of the IABP in the majority of our cases is for myocardial failure or signs of ischemia. Many times there is a dramatic improvement post balloon insertion. The mechanism producing this improvement is often not clear. Possibilities include a heightened diastolic pressure overcoming decreased coronary flow secondary to low mean arterial pressure, left internal mammary spasm or air within a coronary artery, improved collateral flow (*Kern MJ 1991*), or decreased demands secondary to decreased afterload and preload resulting in decreased myocardial wall tension. The IABP often assists in stabilization of the patient, although more definitive therapy may be required. The location of the ischemia, as visualized by the electrocardiogram and often echocardiogram, the magnitude of the failure, the surgical knowledge of the coronary disease and surgery and the response to the IABP determine the need for further management.

We have come to use IABP's earlier, and the indications have broadened to include all stages of the perioperative care. The theoretical protective effect of the IABP with respect to the myocardial supply/demand ratio, the ease of insertion of the IABP by the percutaneous route clinical observations and limited studies support this practice.

## 24. Conclusion

The IABP since its advent in 1960 has progressively become an indispensable technology available to the healthcare team in cardiology and cardiac surgery. Improvements in membrane technology have reduced the french sizes required to be used for femoral percutaneous insertion. This has minimized trauma caused by femoral insertion of the IAB catheter.

Earlier on in my training as a perfusionist, old school cardiac surgeons always inserted IAB's later rather than sooner. Counterpulsation technology was always thought to be a last ditch effort and mortality was thought to be higher once the IAB was inserted. This perception has changed radically over the last twenty years.

The ease of insertion (compared to VAD's and artificial lungs), ease of removal, low risk of complications and availability in the peri/post and pre-operative environment in a hospital setting has made counterpulsation an ideal choice to treat acute or chronic heart failure. There is no doubt at this point of the positive effects of counterpulsation on early mortality and post-operative morbidity in cardiac surgery. In instances where IAB insertion is

precluded or insertion has failed, transthoracic (in a peri-operative setting), transbrachial or subclavian insertion can also be performed with a positive impact on survival. Morbidity and mortality however are higher in these kinds of cases and may have to do with the pre-operative risk factors these patients come into surgery for. The cardiac surgeon has to tread the fine line between deciding the optimal timing for IAB insertion by weighing the benefits of counterpulsation vs the low risk of complications. It is increasingly being understood that for a poor LV and /or a patient with a risk of peri-operative infarct, it is optimal to insert an IAB sooner rather than later. The catheter can easily be removed first or second post-operative day after the surgery has performed and the risk for infarct been mitigated.

At Southlake, a 380 bed community hospital, we perform approximately 1000 heart surgeries and 3000 PTCA's annually. In support we insert 250 IAB catheters annually and are the largest IAB user in Ontario-Canada. We are a comparatively new heart centre, having started our cardiac surgery program in 2004. Aggressive use of counterpulsation reflects the new belief in early IAB insertion in cardiac patients at risk of pre, peri or post-operative infarct. Transfusion rates at SRHC for CABG in the first half of 2009 approximate 26% (Fig 26).

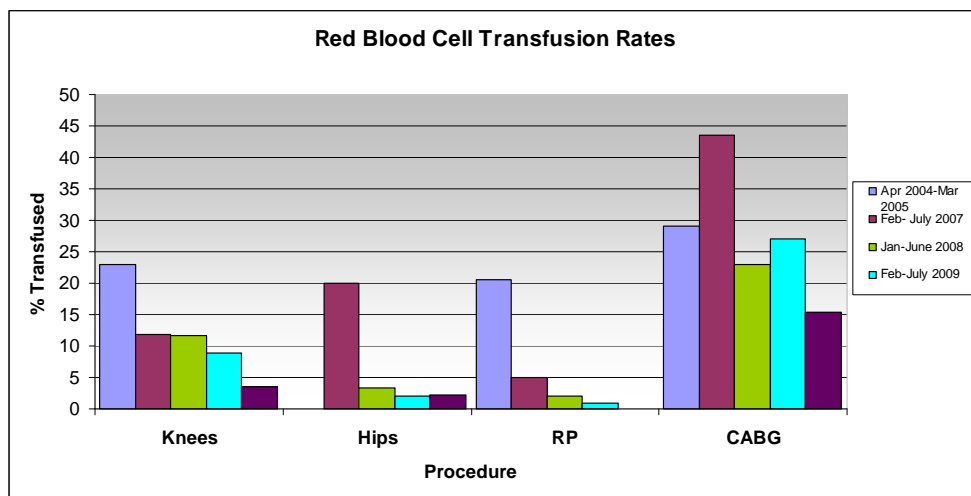


Fig. 26. Red Blood Cell Transfusion Rate for 2009 at SRHC.

Mortality at SRHC for late 2009 (July to Dec 2009) was based on Toronto Risk Score (TRS). The observed mortality ranged from 0.5% (TRS 2-4) to 8.5% (TRS >8) as shown in fig 27.

	TRS 0-2 (n=286)	TRS 3-4 (n=420)	TRS 5-7 (n=432)	TRS >8 (n=236)
Incidence of Observed Mortality	0	0.5%	2.1%	8.5%
Incidence of Observed Mortality by Count	0	2	9	20
Calculated Expected Mortality	1.52%	2.17%	3.95%	10.16%

Fig. 27. Observed and Expected Mortality at SRHC by Toronto Risk Score

It is the opinion of the authors that the above average results in morbidity and mortality for our cardiac patients may partly be due to the aggressive use of IAB in cardiac procedures. Aggressive use of counterpulsation therapy preempts infarction for the cardiac patient and in turn improves outcomes in cardiac surgery.

## 25. Acknowledgement

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