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Pre-Reading Package:

Introduction to IABP

MAYOHEALTHCARE EDUCATION SERVICE

Introduction to IABP

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Important note:

Every effort has been made by the author to ensure the information presented in this package is accurate and up to date. New research and experience in the medical field however, results in necessary changes to treatments and therapies. It is the responsibility of the professional, relying on experience and knowledge of the patient, to determine the best treatment for each individual patient. The author accepts no liability for any consequences from the application of the information in this package.

Table of Contents

Acknowledgements

This package has been based on the Mayo Healthcare Intra-Aortic Balloon Pump self-directed learning package, created by Alanna McInerney in August 2003. Her contribution has been invaluable in the completion of this package. Thanks also to the Neapean Hospital Intensive Care Unit, whose IABP self-directed learning package was used as a basis for the original Mayo Healthcare package.

Introduction

This package provides an introduction to the concepts and principles of intraaortic balloon pumping. It has been designed to be used as a pre-learning package, prior to attendance at a Mayo Healthcare IABP workshop.

ICON KEY	
ℰ	Application note
✎	Test your knowledge

There are practical application notes and review questions located throughout the package to help you apply the information presented. They can be identified by an icon from this key. A glossary of common cardiac terminology has also been included to assist you in your learning.

During the workshop it will be assumed that all participants have read and have a good understanding of the content included in the pre-learning package. If you have any questions whilst completing the package, please bring your questions along to the training.

The questions at the end of each section should be completed and the completed package __________________ brought to the workshop.

Regards,

Mayo Healthcare Education Team

Chapter 1

Cardiac Anatomy & Physiology

Intraaortic balloon pumps are designed to provide temporary support of heart function. An understanding of normal heart function is therefore vital in understanding when an intra-aortic balloon pump is required and how it works.

Heart Structure and Function

The primary function of the heart is to pump blood to the body tissues, supplying them with oxygen and other nutrients and allowing removal of metabolic waste products such as carbon dioxide at a rate to meet the body's needs.

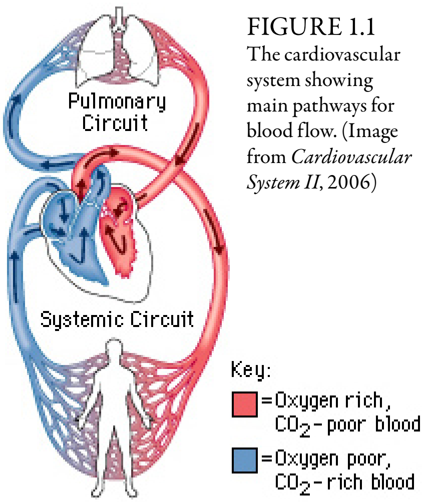


FIGURE 1.1 The cardiovascular system showing main pathways for blood flow. (Image from *Cardiovascular System II*, 2006)

- The right side of the heart receives blood from the body and pumps the blood through the pulmonary arteries to the lungs where it is replenished with oxygen (oxygenated), and carbon dioxide is removed.
- The left side of the heart receives the oxygenated blood from the lungs and pumps it to the body tissues and organs.

Each of the pumps contains two chambers, an upper chamber called an *atrium*, and a lower chamber, called a *ventricle*.

Each chamber has a specific role in moving blood efficiently through the body.

- The atrium acts as a reservior for blood coming to the heart, and then actively assists in filling the ventricle.
- The ventricle pumps the blood into the circulatory system, with the right ventricle pumping blood to the lungs, and the left ventricle pumping blood to the body tissues.

The process of pumping blood through the heart is referred to as the *cardiac cycle*.

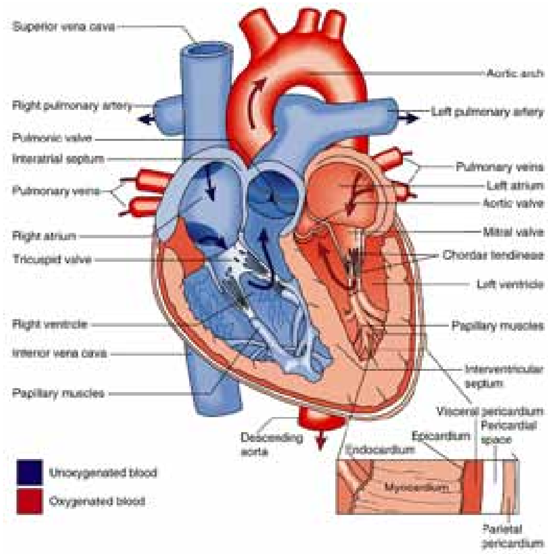


FIGURE 1.2 Structure of the heart. Arrows show course of blood through the heart chambers. (Image from Bare & O'Connell, 2004)

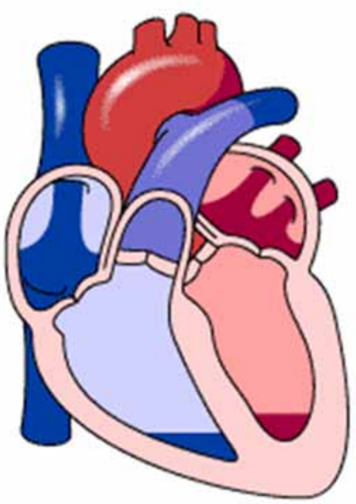
Cardiac Cycle

There are two main phases of the cardiac cycle each of which can be divided into 3 further phases.

- Diastole -the resting and filling phase
- Systole - the working and ejection phase.

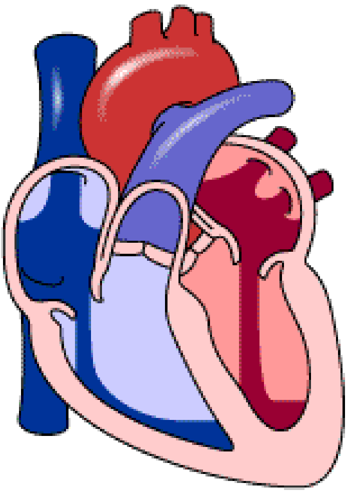
Diastole

Isovolumentric Relaxation



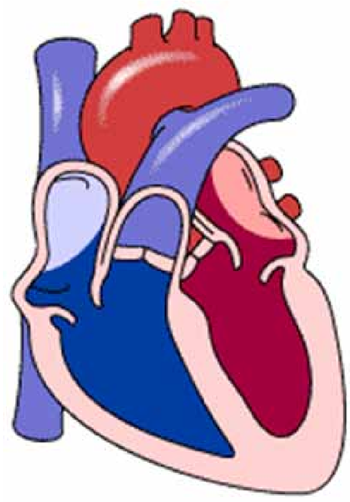
At the start of diastole all the valves are closed and the ventricle is relaxing. There is no movement of blood into or out of the ventricle. This is known as *isovolumetric relaxation* (*IVR*).

Rapid Ventricular Filling



The valves between the atria and ventricles (*AV valves*) then open, allowing blood to rapidly move move from the atria, where it had collected, into the ventricles.

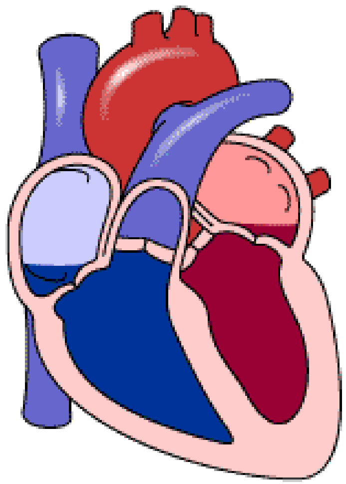
Atrial Contraction



The atria then contract, pushing the remaining blood into the ventricles. This contraction contributes 10-30% of the total volume of blood in the ventricles at the end of diastole (Darovic, 2002).

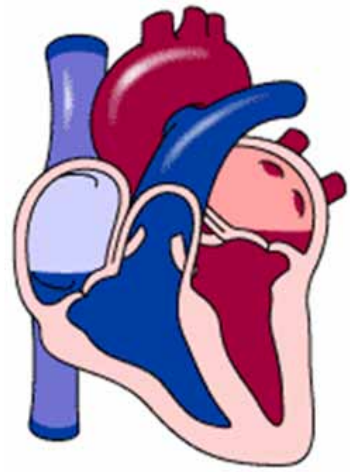
Systole

Isovolumentric Contraction



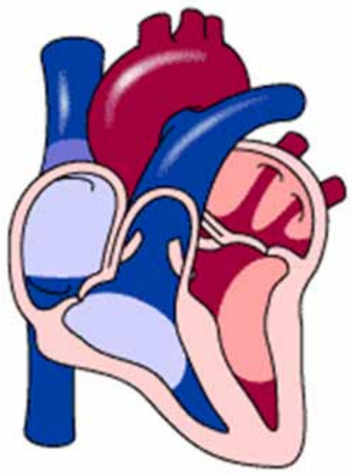
Following the atrial contraction the AV valves close and the ventricle begins to contract. As all the valves are closed there is no movement of blood into or out of the ventricle.

Rapid Ventricular Ejection



When the pressure inside the ventricle exceeds the pressure in the pulmonary artery and aorta, the valves leading to the vessels are pushed open and blood is rapidly ejected into the circulation.

Reduced Ventricular Ejection



As the pressure difference begins to equalise between the ventricle and great vessels, the rate of ejection slows. When the pressure in the vessel exceeds that in the ventricle the valves will close.

Cardiac Output

The heart needs to regulate adequate blood flow in order to meet the changing needs of the body. During exercise and illness there is an increase in cellular metabolism and therefore an increased need for blood flow to provide nutrients to meet the metabolic demand and remove the metabolic wastes produced. The amount of blood ejected from the heart per minute is referred to as the *cardiac output (C.O.)*. On average the normal cardiac output is 4-8 litres per minute. The cardiac output is affected by:

- the volume of blood ejected with every beat, or *stroke volume* (SV)
- the number of beats per minute, or *heart rate (HR)*
- Can be calculated *C.O. = SV x HR*

Stroke volume is affected by the cardiac preload, afterload and contractility

The body attempts to compensate for changes in one factor by increasing the other; for example a decrease in stroke volume will stimulate the heart to increase the heart rate in order to maintain the cardiac output at the same level and continue to meet metabolic demand.

Stroke volume

The stroke volume, or amount of blood ejected from the heart with each beat, is determined by three main factors; preload, afterload and contractility.

Preload

Preload refers to the amount of stretch of the myocardial muscle fibres caused by the volume of blood in the ventricle at the end of diastole prior to contraction. The greater the volume in the ventricle, the greater the stretch and therefore the greater the volume of blood ejected from the heart (known as Starling's Law).

Afterload

Afterload refers to the force that the heart must contract against to eject blood into the arterial circulation. The most important factor that affects the afterload is the vascular resistance. If there is increased resistance to blood flow in the circulatory system, the heart has to work harder to eject the blood, and the volume able to be ejected may fall.

High arterial diastolic pressure will increase afterload and reduce cardiac output

When systemic and pulmonary blood vessels constrict the diameter of the vessels decreases and causes a significant increase in the resistance to blood flow. The pressure within the blood vessels increases and the heart must generate an even greater pressure in order to open the valve and eject the blood. This increases the work of the ventricle and makes it harder to eject all the volume, thus potentially reducing stroke volume.

Other factors that affect the impedence, or force against which the ventricle must contract, are the:

- Mass of blood - the greater the mass the more effort required to start the blood moving.
- Viscosity, or thickness, of the blood - the more viscous, the harder it is to push it through the circulation.
- Compliance of the arterial walls - the less stretchy the arterial walls, the harder it is puch the blood into the vessels.

Contractility

Contractility refers to the intrinsic ability of the myocardial muscles fibres to contract, regardless of the preload and afterload. Contractility cannot be directly measured at the bedside, however once preload and afterload have been optimised it can be judged by assessing:

- the percentage of the total ventricular volume that is ejected, known as the *ejection fraction* (EF). Normally 55-65% of the total ventricular volume is ejected (Bond & Halpenny, 1995).
- chamber wall movement during echocardiography.

- peripheral blood supply, or perfusion.

Application note

Echocardiography is a diagnostic test which uses ultrasound waves to make images of the heart chambers, valves and surrounding structures. The movement of the heart chamber walls, as well as the movement of blood through the heart, can been seen to help identify any areas of poor contractility.

Contractility may be affected by:

- Positive stimuli that increase contractility (known as *positive inotropes*) including naturally occuring or administered substances such as adrenaline and dopamine.
- Negative stimuli that decrease contractility (known as negative inoptropes) including some drugs (e.g. beta-blockers a common form of blood pressure medication), electrolyte imbalances, acidemia or hypoxemia which directly affect the function of the myocardial cells.

Myocardial Oxygen Balance

The heart pumps blood into the pulmonary and systemic circulation to help provide nutrients to organs and body tissue. The heart also requires it's own circulation to provide the myocardial cells with nutrients and remove waste products from metabolism. In fact the heart requires large volumes of oxygen to maintain normal function. Even at rest the heart extracts approximately 70% of the oxygen from the coronary blood, as opposed to the systemic circulation where only 25-35% is used by body tissues at rest (Darovic, 2002a). Due to the high energy expenditure and therefore oxygen consumption of the heart there is minimal oxygen reserve for times of increased myocardial oxygen need. The myocardium therefore requires high blood flow to ensure that supply meets the demand. The harder the heart works, the more blood flow it requires.

90% of myocardial work & oxygen consumption occurs during isovolumetric contraction

Application note

When myocardial oxygen supply does not meet demand, the cells will be low in oxgyen, or ***hypoxic****, which will affect their ability to carry out their function (for e.g. a muscle cell to contract) and result in* ***heart failure****. Prolonged periods of hypoxia can lead to cell damage and cell death, or* ***infarction****, which may cause permanent heart dysfunction.*

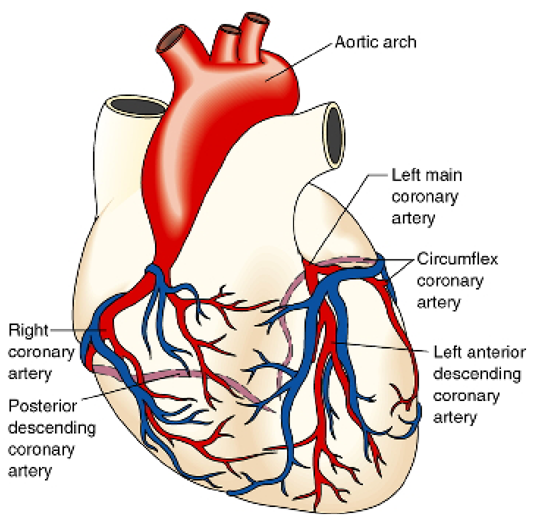


FIGURE 1.3 The major coronary arteries (red vessels) and coronary veins (blue vessels). (Image from Bare & O'Connell, 2004)

Myocardial Oxgyen Supply

The coronary circulation is a network of arteries and veins covering the surface and permeating through the heart muscle to provide oxygen and other nutrients to the myocardial cells. The coronary arteries come off the aorta in the aortic root near the aortic valve, where blood is ejected from the left ventricle (see Figure 1.3). During systole the coronary arteries are compressed by the contraction of the ventricular muscle, raising the pressure in the arteries and impeding blood flow into them. At the start of diastole as the ventricles relax the pressure drops within the coronary arteries, falling below the pressure in the aorta, causing blood to move in to the coronary arteries. It is in this isovolumetric relaxation phase of diastole that 90% of coronary artery filling, or *perfusion*, occurs.

In the healthy heart the coronary arteries are able to constrict

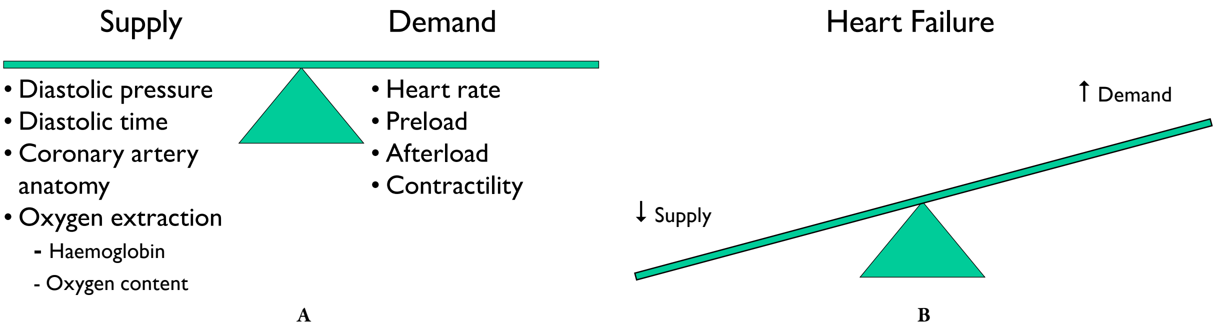


FIGURE 1.4 Myocardial oxygen balance. (**A**) Factors affecting the balance of myocardial oxygen supply and demand. (**B**) Imbalance during heart failure.

and dilate in order to regulate the blood flow to meet the myocardial demand, known as *autoregulation*. Coronary artery disease however impairs this autoregulation with coronary blood flow, and therefore myocardial oxygen supply, heavily reliant on the pressure gradient between the aortic root and the coronary arteries (this is referred to as the *perfusion pressure*). Increasing pressure in the aorta during diastole, when the coronary arteries are filling, will increase coronary perfusion, whereas low pressures will reduce coronary perfusion and therefore myocardial oxygen supply.

Other factors that may affect myocardial oxygen supply are listed in Figure 1.4.

Myocardial Oxygen Demand

As previously discussed, the heart needs to be able to respond to the body's changing needs, increasing and decreasing the cardiac output to meet the body's metabolic demand. Increasing the cardiac output, by increasing the heart rate or stroke volume, will require more work for the heart muscle and the myocardial cells will consume more oxygen to achieve it. The myocardial oxygen demand will therefore be increased by an increase in heart rate, preload, afterload or contractility (see Figure 1.4).

Application note

The release of adrenaline by the body into the blood stream in response to stress or illness will cause an increase in heart rate, constriction of the blood vessels (increasing the afterload and preload) and an increase in the force of contraction (contractility). All these factors will increase myocardial oxygen demand, requiring a subsequent increase in supply.

Myocardial Oxygen Imbalance

When myocardial oxygen demand outweighs supply, the myocardial cells do not have the nutrients needed to function effectively. An imbalance may occur from a restricted supply, where there is not enough blood flow to meet normal demands, or from an increase in demand, beyond the supply capactiy. The healthy heart is usually able to compensate for changes in one factor, however in the presence of heart disease, or multiple factors, the heart is unable to compensate fully and heart function will be affected. Common conditions that result in an imbalance of myocardial oxygen supply and demand include:

- *Coronary heart disease* is where there is myocardial impairment due to an imbalance between coronary blood flow and myocardial oxygen requirements caused by changes in the coronary circulation. This can be acute (short-term)or chronic (long-term) due to atheroscerosis, blood clots, or spasm of the coronary arteries blocking the blood flow. The heart is therefore unable to significantly increase the supply when there is increased demand and may result in injury to the myocardium with myocardial ischaemia (cell hypoxia), which can manifest as chest pain or angina, or infarction (death of cell), commonly known as a heart attack. This injury starts a series of physiological changes which results in a further imbalance between myocardial oxygen supply and demand.

An increase in aortic pressure at the beginning of diastole increases coronary artery perfusion pressure

- In *cardiac failure*, cardiac output falls resulting in a reduction in myocardial oxygen supply. In an attempt to compensate, preload, afterload and heart rate increase. This results in an increase in myocardial oxygen demand. As the failure progresses a cycle develops whereby a greater imbalance between myocardial oxygen supply and demand occurs, resulting in worsening pump failure. Myocardial oxygen supply will continue to decrease while oxygen demand will continue to rise.

- *Shock* refers to a complex state where there is insufficient circulation to adequately perfuse body tissues, resulting in impaired cellular metabolism and organ failure. This circulatory insufficiency may be caused by heart failure from reduced contractility (*cardiogenic shock*), insufficient blood volume reducing preload (*hypovolaemic shock*), or massive vascular dilation (*distributive shock*) reducing preload when the increased vascular capacity reduces the blood volume returning to the heart. In *septic shock* there is not only dilation, but toxins are also released which depress myocardial contractility, reducing cardiac output even further.

Test Your Knowledge

Try to answer the questions below without initially referring to the chapter. The short answer questions should be completed in your own words.

Matching Exercises

a. Stroke volume	1. ___ Working and ejection phase of the cardiac cycle.
b. Diastole	2. ___ When most coronary perfusion occurs.
c. Right ventricle	3. ___ Contributes 10-30% of total ventricular volume.
d. Atrial contraction	4. ___ Pumps oxygenated blood to the body tissues.
e. Ejection fraction	5. ___ Pumps deoxygenated blood to the lungs.
f. Left ventricle	6. ___ Volume of blood ejected with each beat.
g. Systole	7. ___ Gives an indication of myocardial contractility.

True/False

Please indicate whether the following statements are true or false.

1. ___ Isovolumetric relaxation is the beginning of diastole.

2. ___ A decrease in aoritc pressure will increase blood flow through the coronary arteries.

3. ___ The pulmonic and aortic valves open during ventricular systole.

Multiple Choice

Please circle the correct answer

1. The phase of the cardiac cycle where the majority of myocardial oxygen is consumed is:
   a. The ventricular ejection
   b. Isovolumetric contraction
   c. Atrial contraction
   d. Ventricular filling

2. Preload is the:
   a. Impedance against which the left ventricle must pump
   b. Pressure of volume in the ventricle at the end of diastole
   c. Aortic root pressure
   d. Peripheral vascular resistance

3. Afterload is the:
   a. Impedance against which the left ventricle must pump
   b. Pressure or volume in the ventricle at the end of diastole

c. Ability of the myocardial fibers to stretch

d. Affected by the amount of circulating blood volume

4. Coronary artery perfusion occurs predominantly during:

a. Ventricular systole

b. Isovolumetric contraction

c. Reduced ventricular ejection

d. Isovolumetric relaxation

6. During isovolumetric relaxation:

a. The AV valves are open

b. Coronary artery perfusion occurs

c. 90% of myocardial oxygen consumption occurs

d. Aortic valve is open

Short answer/Fill-in Questions

1. What is the primary function of the heart?

2. Describe three factors that affect stroke volume, providing an example of what may affect each of them.

a.

b.

c.

3. Briefly describe the role of the:

a. Atria

b. Ventricles

3. Which blood vessels supply the heart muscle with oxygen and other nutrients?

4. Name three things that affect myocardial oxygen supply.

a.

b.

c.

5. Name three things that affect myocardial oxygen demand.

a.

b.

c.

6. Describe what may happen if there is an imbalance of myocardial oxygen supply and demand.

Chapter 2

Counterpulsation

The intra-aortic balloon pump (IABP) was introduced into clinical practice in the late 1960's as a way of providing temporary mechanical support for patients with cardiogenic shock after myocardial infarction (Darovic, 2002b; Laurent-Bopp & Shinn, 1995). Since then many published studies have documented the efficacy of this mechanical assist device in an expanding number of clinical situations for patient's with actual or potential life-threatening circulatory problems (Darovic, 2002b). See Figure 2.6 for a list of common indications for IABP therapy.

The IABP decreases myocardial oxygen demand & increases myocardial oxygen supply

The IABP is a left ventricular assist device used to improve blood supply to the heart and reduce the work of the heart during pumping. The IABP acts to help rebalance myocardial oxygen supply and demand in order to arrest any deteriotation in patient condition and stabilise their circulation until the myocardial function recovers after an acute injury or mechanical problems are surgically repaired. It assists cardiac function by:

- Decreasing left ventricular workload
- Enhancing myocardial oxygen supply
- Supporting the systemic circulation.

Principles of Intraaortic Balloon Counterpulsation

Intraaortic balloon counterpulsation involves placing a balloon inside the aorta that can be inflated and deflated in time with the cardiac cycle. The intraaortic balloon pump system comprises:

- A pump to quickly inflate and deflate the balloon with a micropressor to control when it should inflate and deflate.
- An intraaortic balloon catheter which has two separate lumens, a balloon or gas lumen, and a central lumen to monitor the aortic pressure.
- Gas drive line tubing to connect the catheter to the pump.

A sausage-shaped balloon is located at the distal end of the catheter which will fill the decending aorta (with ideally 85-90% occulsion) when inflated. The balloon size must therefore be chosen prior to insertion to ensure a correct fit, with either a 30ml, 40ml, or 50ml balloon, according to the patient's height and body surface area. The catheter is inserted into the aorta, usually via the femoral artery in the groin, until the catheter tip lies 1-2cm below the origin of the left subclavian artery (see Figure 2.2). The position of the catheter tip should be checked via fluroscopy or chest x-ray to ensure that the balloon is not occluding the left subclavian or carotid arteries (if placed too high), or the renal arteries (if placed too low). The catheter is usually inserted in the cardiac catheter laboratory or operating theatre but may be inserted at the bedside if necessary.

FIGURE 2.1 Intraaortic balloon pump system

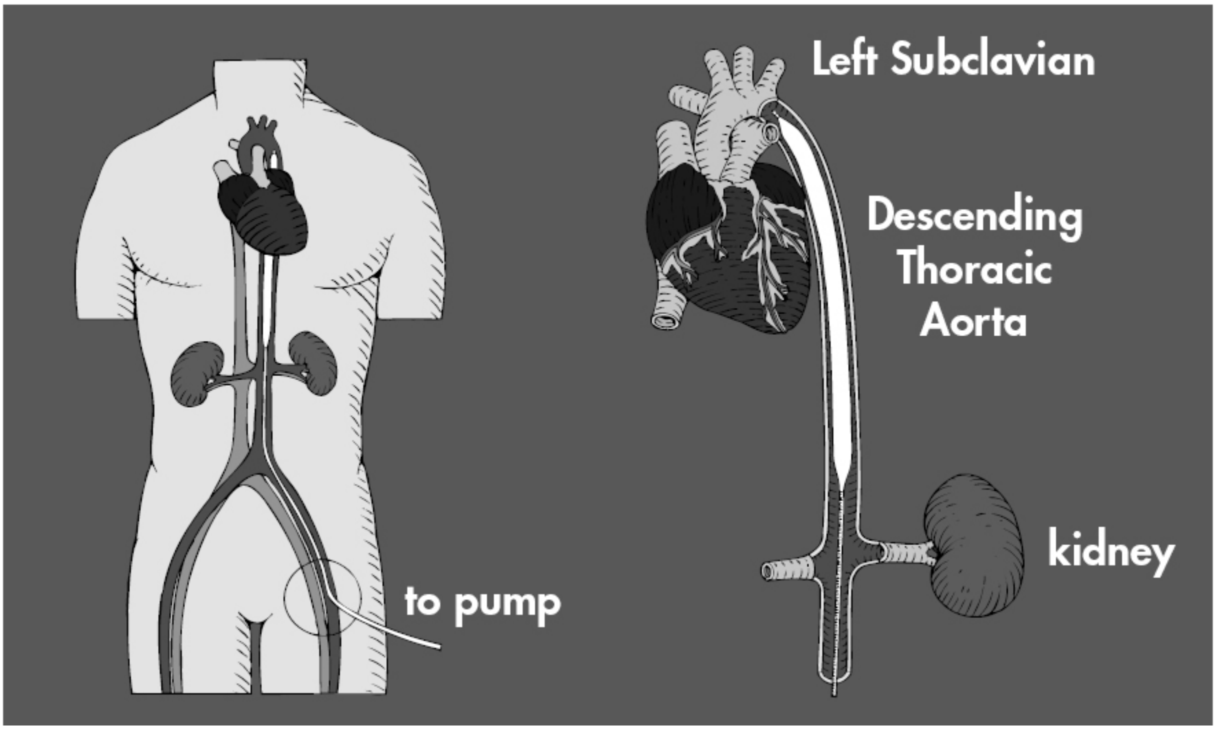


FIGURE 2.2 Correct placement of the Intraaortic Balloon. (Image from *Counterpulsation Applied*, 2006)

Once the balloon is in place the pump can shuttle gas into and out of the balloon via the gas drive line. Helium is used to inflate the balloon as it can move very quickly back and forward The pump however needs to be able to coordinate the inflation and deflation with the patient's cardiac cycle. The pump uses the electrocardiograph (ECG) signal, received through electrodes (or leads) connected to the patient, to determine when the cardiac cycle is occuring and time it's pumping.

Application note

Helium is used to inflate and deflate the balloon as it has a low density, and can thereofre move back and forward through the catheter at high speeds. It is also a safe gas as it is inert and will dissipate quickly if a small amount escapes into the blood stream.

The balloon assists the heart by increasing myocardial oxygen supply and decreasing demand. It increases aortic pressure during diastole (resting phase of the heart) to improve coronary artery perfusion (blood supply), and it decreases aortic pressure just prior to systole (contraction) to lessen the work load on the left ventricle. This is accomplished through inflation and deflation of the balloon. The balloon will be inflated during diastole and deflated during systole.

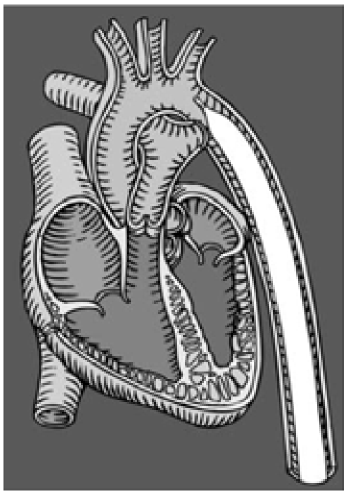


FIGURE 2.3 Intraaortic Balloon Inflation. (Image from *Counterpulsation Applied*, 2006)

Balloon Inflation

The balloon inflates at the beginning of diastole, during isovoumetric relaxation, when the ventricles are relaxing and 90% of coronary filling normally occurs. The inflation of the balloon in the aorta increases the aortic diastolic pressure, which:

- Increases coronary blood flow and therefore myocardial oxygen supply as oxygenated blood is pushed into the coronary circulation.
- Increases systemic perfusion.
- Potentially increases coronary collateral circulation.
- Is referred to as diastolic augmentation, or peak diastolic pressure as the inflation of the balloon augments, or increases, the diastolic pressure.

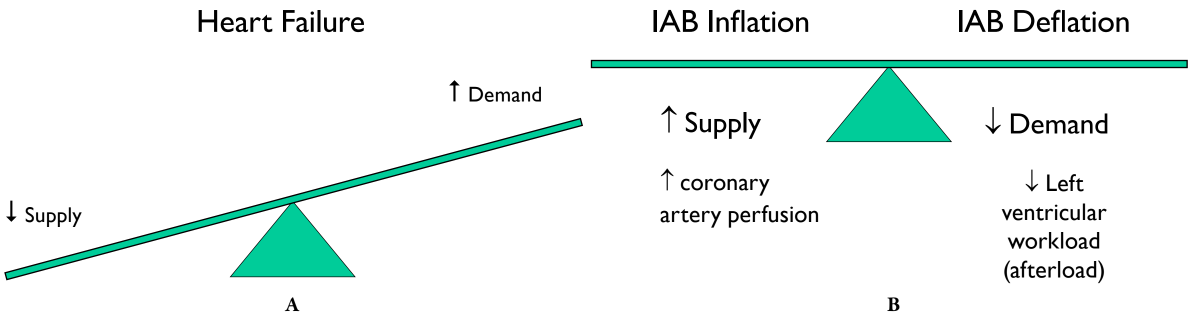


FIGURE 2.4 Affect of intraaortic balloon pump therapy on myocardial oxygen balance. (**A**) Imbalance during heart failure. (**B**) Affect of balloon inflation and deflation on myocardial oxygen supply and demand, with inflation increasing supply and deflation decreasing demand.

Balloon Deflation

The balloon deflates immediately before the left ventricle contracts, during isovolumetric contraction when 90% of myocardial workload occurs. The rapid deflation of the balloon drops the pressure in the aorta, which:

- Decreases the afterload, or the pressure in the aorta against which the heart has to work.
- Decreases the systolic wall tension of the ventricle, or left ventriclular workload, reducing the myocardial oxygen demand.
- Increases stroke volume as it is easier for the heart to emtpy, allowing cardiac output to increase.
- Is reflected by a reduction in end-diastolic pressure and the systolic pressure of the following cycle.

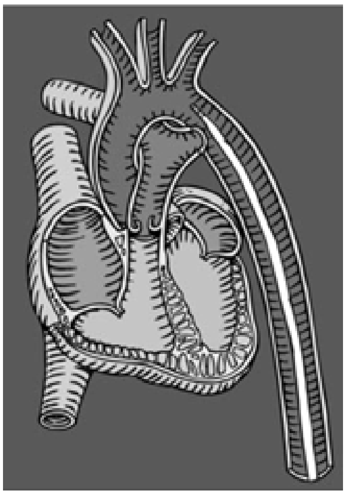


FIGURE 2.5 Intraaortic Balloon Deflation. (Image from *Counterpulsation Applied*, 2006)

Use In The Clincial Setting

The use and management priorities of IABP therapy differs amongst units within a hospital dependent on the patient population and unit focus.

Cardiac Catheter Laboratory (CCL)

Historically, the majority of IAB catheters have been inserted in the operating theatres (OT). With the trend away from invasive surgical treatments for CHD, the majority of intra-aortic balloon catheters are now inserted in the CCL to assist patients actuely compromised by their coronary heart disease. They may be placed:

- Electively to increase myocardial blood supply during coronary interventions, such as angioplasty, where necessary occlusion of vessels during the procedure may cause ischaemia due to a lack of alternative blood vessels to supply the area of myocardium.
- In an emergency when there has been myocardial injury during the procedure causing heart failure. IABP therapy will assist by increasing myocardial oxygen supply and decreasing workload and demand until the injury can be repaired surgically or the myocardium recovers.

IAB insertion within the cardiac catheter laboratory is often urgent, with priorities being fast insertion and pump set-up in order to assist the patient as soon as possible. The laboratories are equipped with fluroscopy, allowing easy assessment of correct balloon catheter placement. Once the IABP is inserted and supporting the patient, the patients is then transferred either to the operating theatre, coronary care or intensive care unit for ongoing management.

Indications for Intraaortic Balloon Pump Therapy

Cardiac catheter laboratory
- Support during elective percutaneous coronary angiography or angioplasty for high-risk patients (multiple-vessel disease, poor left ventricular function)
- Emergency support after injury during percutaneous coronary angiography or angioplasty

Coronary Care & Intensive Care Units
- Severe unstable angina
- Cardiogenic shock after myocardial infarction
- Acute cardiac defects awaiting emergency surgery or post infarction resulting in shock (ventricular septal defects, mitral regurgitation)
- Recurrent postinfarction angina
- Ventricular arrythmias unresponsive to treatment after acute myocardial infarction
- Haemodynamic deterioration in patients awaiting cardiac transplant

Cardiac Operating Theatre
- Perioperative support of high-risk cardiac and general surgical patients
- Weaning from cardiopulmonary bypass

FIGURE 2.6 Common indications for intraaortic balloon therapy. (Adapted from Darovic, 2002b; Laurent-Bopp & Shinn, 1995)

Operating Theatre (OT)

The insertion of IAB pumps in OT are now restricted primarily to the cardiac theatres with insertion prior to or during cardiac surgery. After cardiopulmonary bypass there can be some stunning of the myocardium, causing a reduction in the heart's ability to relax and fill with blood and to contract efficiently. Systemic vasoconstriction (contraction of arterial vessels) also results in an increase in the heart's workload (afterload). These factors result in low cardiac outputs and an inability to wean from the total mechanical support. IABP can provide support by reducing afterload and making it easier to pump as well as increasing the coronary blood supply until the heart has recovered from the surgery. IABP therapy is usually only required for 24-48 hours post cardiac surgery.

Coronary Care & Intensive Care Units

An IAB may be inserted at the bedside in a Coronary Care or Intensive Care Unit if the patient is too unstable to be moved to the Cardiac Catheter Laboratory. Ongoing IABP therapy will be managed in the units, with weaning and removal of the IAB being performed there. Assessment of correct timing, management of patient problems, and prevention of complications are of a much higher importance in these units. Patients with many different indications for IABP therapy may be found within these units, with the most common being to support the circulation during shock and after cardiac surgery.

Summary

Through a combination of augmentation of aortic diastolic pressure and a decrease in aortic end-diastolic pressure, intraaortic balloon pump therapy results in decreased afterload, increased cardiac output and increased blood circulation through the coronary vessels. IABP therapy can therefore increase myocardial oxygen supply and decrease demand to redress the imbalance present in left ventricular failure. As a result of these factors the IABP has had significant impact on patient survival in the acute setting.

Test Your Knowledge

Try to answer the questions below without initially referring to the chapter. The short answer questions should be completed in your own words.

Matching Exercises

a. Balloon inflation 1. ___ An indication for IABP therapy.

b. Aortic regurgitation 2. ___ Should occur during isovolumetric relaxation.

c. Balloon deflation 3. ___ Reduces the workload of the heart.

d. Cardiogenic shock 4. ___ A contraindication for IABP therapy.

True/False

Please indicate whether the following statements are true or false.

1. ___ Oxygen is used to inflate the intra-aortic balloon

2. ___ Occlusion of the coronary arteries can lead to a medical crisis and be an indication for the intra-aortic balloon pump.

3. ___ IAB catheters are most commonly inserted in the intensive care unit.

4. ___ Deflation is timed to occur during the period of isovolumetric contraction.

Multiple Choice

Please circle the correct answer.

1. The primary effects of intra-aortic balloon counterpulsation are:
   a. Increased carotid and limb perfusion
   b. Increased coronary perfusion and afterload reduction
   c. Increased afterload and increased coronary perfusion
   d. Reduction in coronary perfusion and afterload

2. Proper positioning of the intra-aortic balloon is:
   a. Below the renal arteries and above the right subclavian artery
   b. Distal to the common carotid and above the diaphragm
   c. Below the left subclavian artery and above the renal arteries
   d. In the aortic arch close to the aortic valve

3. Intraaortic balloon position can be checked by using:
   a. Palpating between the ribs
   b. Fluroscopy (form of X-ray)
   c. Electrogradiograph (ECG)
   d. Auscultation (listening with stethoscope) over the chest and abdomen

4. The major physiological effects of counterpulsation include:

   a. Increased coronary artery perfusion, increased preload, decreased afterload, and decreased myocardial oxygen consumption

   b. Increased coronary artery perfusion, increased preload, increased afterload, and decreased myocardial oxygen consumption

   c. Increased coronary artery perfusion, decreased preload, decreased afterload, and increased myocardial oxygen consumption

   d. Increased coronary artery perfusion, decreased preload, decreased afterload, and decreased myocardial oxygen consumption

5. Which of the following is **NOT** a contraindication of IAB therapy?

   a. Aortic valve regurgitation

   b. Severe peripheral vascular disease

   c. Dissecting aortic aneurysm

   d. Preinfarction angina

   e. Coronary artery disease

Short answer/Fill-in Questions

1. List three indications for IABP therapy.

a.

b.

c.

2. How does intraaortic balloon inflation assist a patient in cardiac failure?

3. How does intraaortic balloon deflation assist a patient in cardiac failure?

Glossary

Acidosis

- A pathologic condition resulting in the accumulation of acid or loss of base in the body.
- Respiratory acidosis is an acidosis caused by the excess retention of carbon dioxide.
- Metabolic acidosis results from excess acid due to abnormal metabolism, excessive acid intake, or renal retention or from excessive loss of bicarbonate (as in diarrhea).
- Lactic acidosis results from the accumulation of lactic acid in body tissues.

Acidemia

- Accumulation of acid, or hydrogen ions, in the blood stream.

Acute

- Having severe symptoms and a short course.

Afterload

- Amount of pressure against which the left ventricle must work during systole to open the aortic valve.
- Amount of wall tension created within the ventricle during the systolic phase of cardiac cycle.
- Clinically measured by systemic vascular resistance or systolic blood pressure.

Anterior

- Situated towards the front of the body.

Atrium

- Upper chamber of the heart.
- Acts as a reservoir to collect blood returning to the heart.
- Actively assists in filling the ventricle.

Autoregulation

- Ability of coronary arteries in the healthy heart to regulate coronary blood flow to myocardial oxygen demand by constricting and dilating.

Balloon Assisted Aortic End-diastolic Pressure (BAEDP)

- The diastolic pressure in the aorta just prior to the onset of systole which is affected by deflation of the intra-aortic balloon.
- Indicates a reduction in Afterload.

Assisted Systole or Assisted Peak Systolic Pressure (APSP)

- Systolic pressure which follows a BAEDP.
- An Assisted Systole (APSP) 5-10mmHg lower than the previous PSP (in 1:2 ratio) is a good indicator of LV workload reduction.

Atrial Systole

- Contraction of the atria. Often referred to as the "atrial kick".
- Contributes 10-30& of total ventricular volume.
- Patients who have heart failure are often dependent on the extra volume contributed by the atrial contraction.

Augmented Diastolic Pressure or Peak Diastolic Pressure (PDP)

- Highest pressure in the aorta during balloon inflation.
- Caused by subsequent displacement of stroke volume.

Blood Pressure

- The measurement of peak and trough pressures in the arterial system.
- Force exerted by the blood against a vessel wall.

Cardiac

- Relating to the heart.

Cardiac Failure

- Failure of the heart to pump adequate amounts of blood to the body to meet metabolic demand.

Cardiac Output

- Amount of blood ejected by left ventricle per minute.
- Determined by multiplying heart rate x stroke volume.
- Expressed as liters/minute (normal values are 4-8L/min).

Cardiac Index

- Cardiac output divided by the patient's body surface area (BSA) expressed in liters/minute/meter^2.

Chronic

- Persisting for a long time.

Coronary Perfusion Pressure

- Pressure required for blood to flow into the coronary arteries, supplying the myocardial cells.
- Determined by pressure in aortic root during diastole minus pressure inside the coronary arteries.

Counterpulsation

- Alternating inflation and deflation of the intra-aortic balloon during diastole and systole respectively.

Deoxygenated

- Blood that has had oxygen removed for use in cellular metabolism.

Diastole

- Relaxation of the heart muscle, begins when the aortic valve closes.

Dicrotic Notch

- Signifies aortic valve closure on an arterial pressure waveform.

Distal

- Situated away from the centre or point of attachment.

Ejection Fraction

- Percent of left ventricular end diastolic volume which is ejected during systole (normal values 60-70%).

Electrocautery

- Device consisting of a needle heated by electric current which decreases tissue bleeding by cauterization – used in Operating Theatre.
- Can interfere with ECG signal for triggering of IABP.

- AP trigger may be used as alternative.

Endocardium
- Internal lining of the heart.

Epicardium
- External lining of heart muscle.

Heart Failure
- See cardiac failure.

Hypoxaemia
- A reduced level of oxygen in the blood. PaO_2 is 55mmHg or below.

Hypoxia
- A reduced level of tissue oxygen.

Infarction
- Permenant damage/death of cells caused by a prolonged period of hypoxia.

Inferior
- Situated below or towards the feet.

Interstitial Fluids
- Fluid between the blood vessels and cells.

Ischaemia
- Reduction in blood supply, usually due to narrowing of arterial lumen from plaque deposition. Commonly known as "angina" .

Isovolumetric Contraction
- The first phase of systole during which 90% of myocardial oxygen consumption occurs.
- All four valves are closed.
- There is no change in blood volume.
- The ventricle is building the pressure it needs to open aortic valve & eject stroke volume.
- Balloon is deflated during IVC.

Isovolumetric Relaxation
- The first phase of diastole when all four valves are closed.
- There is no change in ventricular volume but a rapid decrease in LV pressure. 90% of coronary artery perfusion occurs during this phase.
- Balloon is inflated during IVR.

MAP-Mean Arterial Pressure
- The time-averaged pressure throughout each cycle of the heartbeat.
- Best indicator of organ perfusion.
- Best pressure to use during balloon pumping.

Myocardial Infarction
- Commonly known as a "heart attack". Blood supply to a section of heart muscle is blocked due to a clot in the artery supplying that region. This results in death of heart tissue if not treated immediately to reinstitute blood supply.

Myocardium
- Layer of muscle in heart wall.

Oxygenated
- Blood that contains oxygen.

Patient Aortic End Diastolic Pressure (PAEDP)
- Aortic end diastolic pressure without IABP intervention.

Perfusion
- Blood flow to an organ or tissue bed.

Posterior
- Situated towards the back of the body.

Preload
- The left ventricular end diastolic volume and the amount of pressure it exerts on the walls of the left ventricle.
- Assessed clinically by the PCWP (pulmonary capillary wedge pressure).

Proximal
- Situated next to or near the centre or point of attachment.

Pulmonary
- Relating to the lungs.

Rapid Ventricular Ejection Phase
- Period following opening of aortic valve where approximately 75% of stroke volume is ejected from left ventricle.

Reduced Ventricular Ejection Phase
- Period following rapid ejection where ejection of blood into the circulation slows as pressures inside and outside the ventricle begin to equalise.
- Remaining 60% of blood is ejected from the ventricle during this phase.

Resistance
- Refers to the opposition to flow through a tube or vessel. Affected predominantly by the diameter of the vessel.

Stroke Volume
- Amount of blood ejected by the left ventricle with each systolic event.

Superior
- Situated above or towards the head.

Systemic Vascular Resistance
- Resistance to ventricular ejection.
- Clinical measure of afterload (normal value 900- 1200 dynes/sec/cm-5).

Systole
- Phase of the cardiac cycle in which the ventricles are contracting.

Timing
- Inflation and deflation of the IABP in synchrony with the mechanical cardiac cycle.

Trigger
- Signal used by the IABP to identify the beginning of the next cardiac cycle (R wave) and deflate the IAB (if not already deflated).

Unassisted Systole or Peak Systolic Pressure (PSP)

- Systolic pressure not assisted by counterpulsation.

Ventricle

- Lower cardiac chamber.
- Pumps blood into the circulation.
- Performs most of the cardiac work.

Ventricular Filling

- Passive flow of blood from atria into ventricles.

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IABP Module 1 Evaluation

Please tick the appropriate boxes, complete the questions below and return with your answers to the self-directed learning package.

	Fair	Satisfactory	Good	Very Good	Excellent
Please rate your IABP knowledge and/or skills prior to completing this package.					
Please rate your IABP knowledge and/or skills after completing this package.					
Was the package content relevant?					
Was the content clear & concise?					
Was the topic covered adequately?					
Was the SDLP too comprehensive?					

What part of the package was the most beneficial?

What are they key points you have taken from the package?

If you rated your post SDLP knowledge as Satisfactory or below please make suggestions as to what you need in order to raise your knowledge to *Good* or above.

Please make note of any constructive suggestions you may have for improving future Self Directed Learning Packages:

Thank you for your time in filling out this evaluation. Your comments will assist us in improving future education packages.