Greater Manchester
Critical Care
Skills Institute

Critical Care Programme
Fundamentals Module

Pre-course Workbook
Cardiovascular

Acknowledgements

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Aim of the workbook

- To introduce the reader to concepts of haemodynamic assessment and monitoring supported by the Fundamental Module: Cardiovascular study day
- To provide a revision/follow up resource to the study day and reinforce the instruction provided at that time
- To act as a foundation link for the advanced theory and practice which will be provided on a subsequent Cardiovascular study day.

The workbook provides a basis for the day. Knowledge is then developed throughout the day. Areas that will be developed during the day include the focus of a holistic patient introduction, expand on current evidence when caring for the septic patient, encouraging discussion regarding inotropic and fluid management when caring for patients presenting with shock and lastly discussing evidence regarding invasive monitoring to include a brief discussion regarding cardiac output monitoring relating back to different unit practices. Each part of the book will be preceded by a set of learning objectives for the reader's guidance. It will be beneficial to the reader to have a physiology textbook readily available in order to clarify some of the terms and complete some of the exercises encountered in the text. Recommended reading lists have been provided.

Objectives

On completion of the workbook, the reader will be able to:

- Discuss basic anatomy and physiology of the cardiovascular system
- Discuss the components of blood pressure and physiological control
- Discuss the different types of shock, their management and their effect on the blood pressure equation
- Demonstrate a basic understanding of cardiac ischaemia and its effects
- Discuss the assessment and treatment of cardiac ischaemia and chest pain
- Identify the normal electrical conduction pathways in the heart
- Discuss skin preparation and ECG electrode placement
- Discuss the concept and measurement of central venous pressure (CVP)

Introduction to Haemodynamics and Monitoring

Haemodynamic monitoring is the measurement of haemodynamic status. Haemodynamic status is an index of the pressure and the flow within the pulmonary and systemic circulation. Remember, one of the most important end functions of the systemic circulation is to deliver oxygen (O_2) to cells. Patients with heart failure, fluid overload, shock, pulmonary hypertension, and other such problems have an altered haemodynamic status. Invasive haemodynamic monitoring requires cannulation of the vascular system. Although central venous pressure (CVP) and arterial pressure measurement have been in use for some considerable time, many new advanced haemodynamic monitoring techniques are available the study day will enable the leaner to reflect on their own practice and what techniques are possibly used in practice.

The end purpose of haemodynamic monitoring is to alert the practitioner to changes which, in the acutely ill patient, may allude to shock and thus help guide treatments and care. A patient in shock invariably requires resuscitation measures to return homeostasis and ensure adequate O_2 delivery to the cells. For the nurse to effectively interact with the acutely ill patient an understanding of normal physiological mechanisms is required, as well as an understanding of shock and how available treatments, such as pharmacological, mechanical support and fluid management interact.

Physiology of the cardiovascular system

What is blood pressure (BP)?

We are all familiar with the systolic and diastolic components of blood pressure. However, in caring for the acutely ill patient we need to understand how and why BP is maintained and controlled, and the anatomical structures involved.

What three structures make up the cardiovascular system?

The blood transports nutrients, oxygen, hormones etc. to the tissues and removes waste products such as carbon dioxide (CO_2). To achieve transportation we need a pump, tubes & fluid. Haemodynamics is the study of how well the pump, tubes and fluid are functioning with regard to the transport, principally of O_2. The heart is the pump, the blood vessels are the tubes and the fluid is the blood (figure 1).

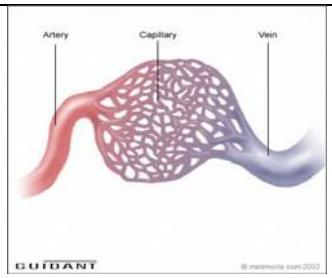
Blood	Heart	Vessels
		

Figure 1

Components of blood pressure:

- Cardiac output (CO) = the amount of blood ejected from the ventricles in one minute
  CO is a combination of stroke volume (SV) and heart rate (HR)
- Stroke volume (SV) = the amount of blood ejected from the ventricles in each contraction
- The HR and the SV force blood into the circulation and together create the Cardiac Output (measured in L/min)
- Systemic vascular resistance (SVR) = the force of the ventricle ejection against the impedance of the arterial vessels
- The CO flows through blood vessels and depending upon the 'tone' of the blood vessels (how constricted or dilated they are) a blood pressure will be generated

From your textbook find a **written** definition of blood pressure

As an equation blood pressure is:

BP = CO (SV x HR) x Systemic Vascular Resistance (SVR)

We thus have **three components of blood pressure, SV, HR and SVR**.

Each of these three components of blood pressure relates to the structures of the cardiovascular system. Which component relates to which structure?		
Blood	Heart	Vessels

There are other factors that govern cardiovascular efficiency termed:

- Pre load
- Contractility
- After load

From your textbook give a definition of these terms:
Pre load: Contractility: After load:

Each of these three factors also relates to the structures of the cardiovascular system. Which factor relates to which structure?		
Blood	Heart	Vessels
Stroke volume	Heart rate	Systemic vascular resistance

If any of these components are altered the body can respond by altering the other component(s) in order to support the others, this is termed 'compensation'. These 'compensation' mechanisms occur to some degree all of the time, to maintain homeostasis. In extreme circumstances, for example when circulating volume is lost following bleeding, there will initially be a reduction in stroke volume. This will cause a 'rebound' tachycardia to occur in an effort to maintain CO at the

'normal' level. This is the reason why specific parameters are monitored post-operatively. An elevated HR may indicate that post-op bleeding is occurring, but an increase in respiratory rate, is an earlier sign of deterioration as the body is trying to get more oxygen into the available blood, (blood loss = loss of oxygen carrying capacity). But, as we shall see, when we examine shock more closely, these compensation mechanisms can be 'self-destructive' rather than helpful.

Physiological control of blood pressure

Neurological control of blood pressure is under the influence of the vasomotor centre and cardiovascular centre of the brain via the sympathetic and parasympathetic nervous system (figure 2)

Pressure Receptors

Baroreceptors in the right atrium, aortic arch & carotid arteries detect changes in pressure

Chemoreceptors in the aortic arch & carotid arteries detect chemical changes in pH, CO_2 and O_2

Both feed back information to the the pons and medulla oblongata, within the vasomotor centre and cardiovascular centre in the brain, which appropriately stimulate the required organ(s), i.e. heart, lungs and vessels.

Peripheral Chemoreceptors

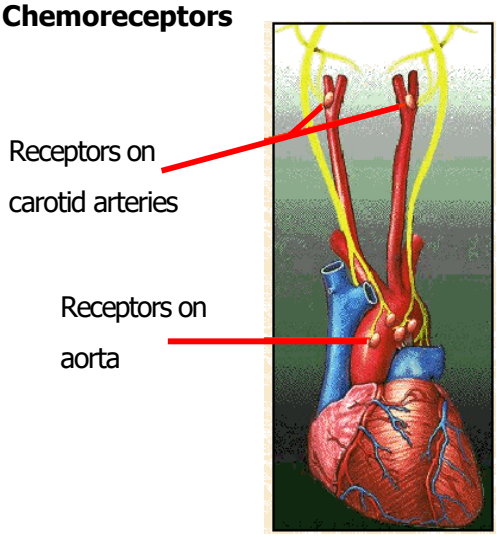


Figure 2

Heart Rate

1. The SA & AV nodes are well innervated by sympathetic and parasympathetic nerve fibres. Control of these autonomic nerves is via the cardiovascular centre in the medulla oblongata in the brain.

a. Sympathetic - nerve discharge to the SA node have a +ve chronotropic effect increasing the heart rate
b. Parasympathetic - vagal stimulation from the vagus nerve, have a -ve chronotropic effect. Main effect is to the AV node, slowing conduction and heart rate

2. Circulating catecholamines, e.g. adrenaline/noradrenaline affect the intrinsic heart rate, +ve chrontropic effect increasing the heart rate.

3. Circulating catecholamines also affect the SA node, +ve chronotropic effect.

Stroke Volume

1. Sympathetic nerve activity – from noradrenaline release, causes the myocardial muscle fibres to shorten even more (the 'inotropic' state of the heart), increasing global contractility and stroke volume. Parasympathetic activity has the opposite effect.

2. General sympathetic activity also causes catecholamine release from intramyocardial catecholamine stores.

Systemic Vascular Resistance (SVR)

Baroreceptors in the aortic arch (Vagus) and common carotid have free nerve endings, and act like a "stretch" receptor. They communicate with the vaso-motor and cardiovascular centre, within the medulla and pons in the brain.

The size (internal diameter) of the blood vessels depends on sympathetic activity (adrenergic pathways - i.e. noradrenaline regulated by the vaso-motor centre (VMC).

- *Hypotension* will cause an increase in VMC activity, i.e. vasoconstrict
- *Hypertension* will cause a decrease in VMC activity, i.e. vasodilate

Parasympathetic activity has a "damping down" effect on the VMC.

Almost all organ systems or tissues have the capacity for intrinsically regulating their own blood flow e.g. hypoxaemia causes vasodilation, either by direct smooth muscle relaxation secondary to a local oxygen deficit or on the basis of vasodilatory substances, such as adenosine, from anaerobic metabolic processes.

Circulating volume

50-60% of the human body is made up of water. This water lies in three distinct compartments, intravascular, interstitial and intracellular. Homeostasis is maintained by constant movement of fluid from one space to another. BP measures intravascular volume so if this volume becomes depleted fluid will move from the other spaces into the vascular system to maintain the blood pressure. This is predominantly managed through a complex series of hormones, pressure receptors, the brain and kidneys, and includes physiological principles, such as osmosis.

Management is vitally important to maintain normal cellular function and, in the case of the plasma volume, to optimize stroke volume and cardiac function.
The amounts of water and sodium lost in the urine (remember, water has a strong affinity for sodium) are under the control of two hormones:

a) aldosterone
b) anti-diuretic hormone (ADH)

Aldosterone

Aldosterone is a hormone secreted by the adrenal cortex. It exerts an effect on the secretion of sodium by the kidney. When secreted, aldosterone increases the activity of the 'sodium pump' and causes sodium to be absorbed from the urine forming in the distal tubule into the bloodstream and taking water with it.

In the absence of aldosterone considerable amounts of sodium are lost in the urine with water. The secretion of aldosterone is stimulated by a fall in the sodium concentration of the blood.

This change is detected by receptors in the kidney. These receptors are situated in the **juxtaglomerular apparatus** at the point where the distal convoluted tubule comes close to the glomerular supply artery (the efferent arteriole).

The juxtaglomerular apparatus release a substance, **Renin**, which combines with angiotensinogen and is converted in the blood stream into a substance called Angiotensin 1. Angiotensin 1 is converted in the lungs into angiotensin 2 via Angiotensin Converting Enzyme (ACE). It acts on the adrenal cortex to cause the secretion of aldosterone.

Angiotensin II is also a potent vasoconstrictor. Thus, if circulating volume is reduced, the kidney, which normally uses about 25% of cardiac output to produce urine, is not only able to conserve fluid but can reduce the circulating volume space to maintain a 'normal' BP.

Anti-Diuretic Hormone (ADH)
ADH is secreted in response to the circulating volume. If there is any sign of hypovolaemia then the ADH cycle is triggered, ADH is released and as the term implies stops diuresis. Fluid will then be diverted back into the vascular system to maintain BP.
In cases of diabetes insipidus when there is excessive diuresis the ADH mechanism has been damaged. ADH is also known as vasopressin and a synthetic analogue (DDAVP) is given in patients with this condition. This is often seen in patients with head injuries.

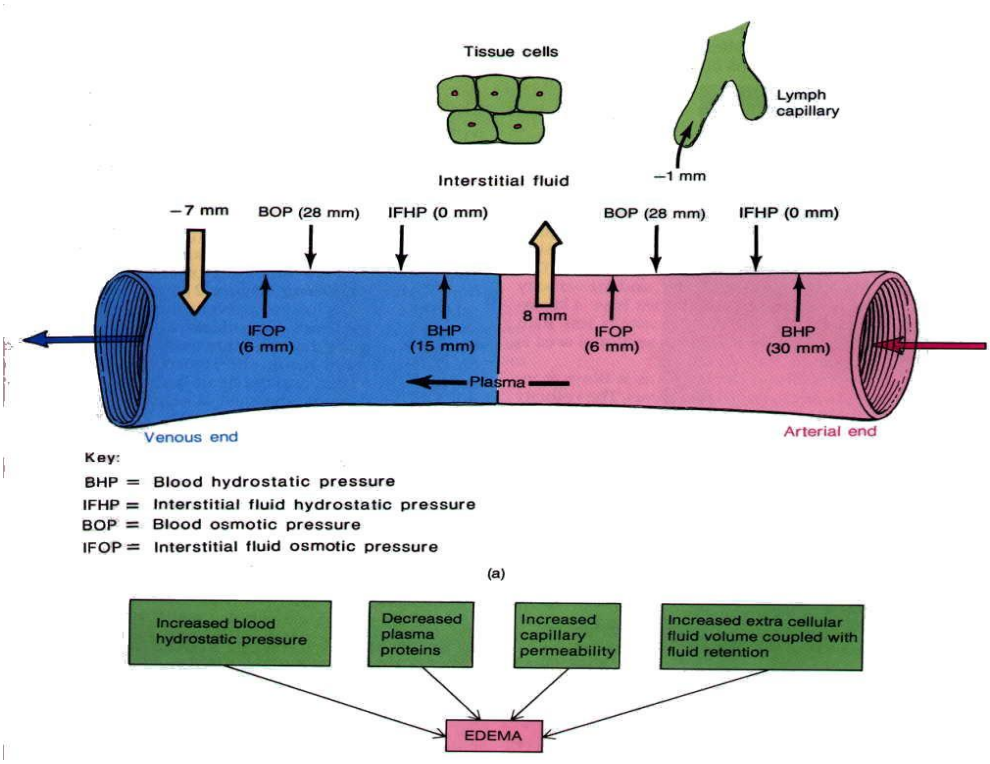
Tissue fluid formation
The capillary networks are permeable in order for oxygen, water, nutrients, hormones, enzymes and electrolytes to bathe the cells (interstitial fluid) and be selectively uptaken by the cells, and waste products to be removed. This is enabled by several pressures.

At the arterial end of the capillary network the plasma is under higher pressure generated by the mean arterial pressure, this squeezing effect is known as the 'blood hydrostatic pressure'. This

squeezes fluid containing nutrients through the pores in the capillaries – a bit like squeezing a tea bag in your hand. Normally, red cells and plasma proteins are too big to pass and so stay in the plasma (although 'micro albumin' can pass through). The cells and interstitial fluid will also exert osmotic pressure upon the plasma – there is normally no perceivable interstitial fluid hydrostatic pressure.

At the venous end of the capillary network plasma proteins exert an osmotic pulling pressure on the interstitial and intracellular fluid. Because blood hydrostatic pressure is less, the effect results in fluid being pulled back into the plasma.

Figure 3. Tissue fluid formation



The green boxes (figure 3) indicate how normal tissue fluid formation can become disordered in the critically ill patient. Haemodynamic instability, hypoxia at tissue level, and production of lactate results in a metabolic acidosis which alters capillary permeability – as seen in shock. Infection, sepsis and Disseminated Intravascular Coagulation (DIC) can also do this. If these conditions are then compounded with malnutrition and a consequential reduction in plasma protein – it can be easily seen that the critically ill patient is at risk of oedema. If this oedema is present within major organs, they will not function as normal – frequently causing pulmonary function to deteriorate as seen in the shocked patient.

Shock

'Shock can be defined as inadequate oxygen delivery to the tissues'

The shocked patient is often, pale, cool, sweaty, tachycardic, hypotensive, oliguric, but may also appear warm and flushed.

Oxygen delivery is essential for cell metabolism. The most common type of shock seen are those when the total delivery of oxygen is reduced. Common causes are haemorrhage, hypovolaemia, myocardial infarction (MI) and/or Left Ventricular Failure (LVF).

Now we understand the components of BP = **CO (SVxHR) x SVR**, we can relate these to the signs and symptoms presented.

Using the following example you can consolidate all you have learned so far:

Reduction in circulating volume will have reduced cardiac output, and reduced oxygen delivery. The baro and chemo receptors will detect this, causing the cardiac and respiratory centres to respond. The respiratory and heart rate increases in an effort to maintain the cardiac output.

Reduced CO and reduced BP will stimulate the juxtaglomerular apparatus in the kidneys to begin the renin-angiotensin cycle, producing aldosterone. This increases vascular resistance and conserves water. Osmo receptors in the hypothalamus are also stimulated to produce ADH, and conserve water. As vasoconstriction begins, blood flow to the periphery is diminished.

Patients will therefore display all the signs of shock. The compensation mechanisms act to maintain oxygen delivery to vital organs. Early detection means simple measures can be taken, rather than crisis management steps.

There are 5 recognised types of shock:

1. **Hypovolaemic**
2. **Septic**
3. **Anaphylactic**
4. **Cardiogenic (dicussed in chest pain section)**
5. **Neurogenic (rare)**

Hypovolaemic shock

Causes often involve haemorrhage, surgery, trauma, poor hydration, high insensible loss, diarrhoea, vomiting and burns.

Signs & symptoms

- Pale
- Cool
- Clammy
- HR elevated
- BP reduced
- CVP down (If in ICU PCWP down)
- Oliguria

How does hypovolaemic shock affect the components of the BP equation? BP = SV x HR x SVR (CO)

Treatment:
Oxygen therapy is always indicated and should be initiated as soon as possible. Oxygen therapy should be guided by saturations as recommended by the British Thoracic Society Guidelines (2017). Treatment following this is to establish the cause of the fluid loss and treat it, i.e. to stop further blood loss.

Depending on the type of loss different fluids are used, for example in haemorrhage plasma volume will need to be increased and so colloids may well be indicated, such as starch or/and gelatin type solutions. If the Hb becomes very low then a transfusion may be indicated. Where intracellular and interstitial fluid is depleted, as in uncontrolled diabetes mellitus, crystalloids may well be indicated. The colloid versus crystalloid debate continues and the study day will support this discussion evidenced by the NICE Clinical Guideline 174 (2017 Intravenous fluid therapy in adults in hospital).

What sorts of volume replacement therapies are used in your area? What is the rationale for their use?

Septic Shock

Septic shock is as prevalent as hypovolaemic and cardiogenic shock, but continues to be poorly recognised, diagnosed and treated. NICE Guideline 51 (2017) Sepsis: recognition, diagnosis and early management recognises the importance of early recognition, diagnosis and treatment.

Causes of septic shock include pneumonia, meningitis, wound infections and bacteraemia, often due to invasive lines. Occasionally no causative organism can be located yet the patient has all the signs and symptoms of septic shock. There have been a few classifications of terms to define Septic Shock more recently discussed by Singer et al (2016) has suggested limiting these terms to **sepsis** and **septic shock**

Septic shock follows a continuum. Briefly define the following terms:
SIRS
Sepsis
Server Sepsis
Septic shock
MODS

Signs & symptoms

- Temp. >38.5˚C, flushed, OR <36˚C
- Heart rate >90bpm, full and bounding
- Tachypnoeic (RR >20)
- WCC<4 or >12 ($x10^9$/l)
- Acutely altered mental state
- Hyperglycaemia in the absence of diabetes
- History of or signs of an infection
- Signs of organ dysfunction

How does septic shock affect the components of the BP equation? BP = SV x HR x SVR (CO)

In septic shock the SVR falls due to vasodilation, and the HR and SV increases to overcome the fall in resistance. However, an increase in CO is insufficient to compensate for the loss of SVR and BP falls as a result. Again, tissues have reduced oxygen supply so will metabolise anaerobically, producing lactic acid as a by-product.

Treatment:

Early goal directed therapy was evidenced initially by recommendation of the 'SEPSIS SIX' following Survive Sepsis 2007 and this has continued to be supported by the United Kingdom Sepsis Trust in The Sepsis Manuel (2019)

What are the components of the 'SEPSIS SIX' and the associated advisory notes?
1 2 3 4 5 6

What do the "NICE Guideline 51 (2017) Sepsis: recognition, diagnosis and early management" recommend for the management of patients with sepsis in people aged 18 and over in hospital?

Anaphylactic shock

Anaphylaxis is a sudden reduction in BP caused by an allergic reaction – the patient can collapse very quickly and suffer cardiac and/or respiratory arrest.

Following initial exposure to something (e.g. penicillin, peanuts, prawns) antibody formation occurs. Upon second exposure an inappropriate systemic allergic reaction occurs, releasing 'massive' amounts of histamine. This results in a sudden drop in SVR and constriction of the pulmonary bronchioles. True anaphylaxis will only occur on 2nd exposure – if there is any suspicion patients will need to be investigated to detect a specific antigen.

Signs & symptoms

- Flushed appearance, warm to touch
- Patient may complain of palpitations, difficulty breathing/ wheeze
- Feeling of 'impending doom'
- BP reduced
- Sudden collapse

How does anaphylaxis affect the components of the BP equation? BP = SV x HR x SVR (CO)

Treatment involves removing the cause (if possible), supporting the blood pressure and evident symptoms.

- Oxygen, adrenaline and volume replacement are primary interventions
- Anti-histamine and steroids may be given to dampen down the inflammatory response and histamine release
- Bronchodilator to alleviate difficult breathing
- If there is any possibility that prescribed drugs may cause anaphylaxis then Piriton and Hydrocortisone should be prescribed in advance

What are the likely causes of anaphylaxis in your area of work?

What equipment/drugs do you have available to treat anaphylactic shock?

Neurogenic shock

Neurogenic shock is usually as a result of spinal cord injury. It occurs when there is a cessation of sympathetic nervous control over the vasculature, causing vasodilation, and a reduction of SVR. Treatment includes the use of vasopressor therapy to restore SVR, however the condition is very rare.

Chest Pain

MANAGEMENT OF CHEST PAIN SUGGESTIVE OF ISCHEMIA

GOALS

Immediate assessment of patients with chest pain
Timely initiation of treatment
Rapid determination of need for transfer to another facility

Immediate Nursing Assessment	Orders for Immediate Treatment
Assess pain level: severity, quality, radiation	**M** Morphine pain relief
Timing	**O** Oxygen (BTS 2017)
Check vital signs, including SaO_2	**N** Nitrates (titrate with B/P)
Obtain 12 lead ECG	**A** Aspirin 300mg
IV access	Anti-emetic if morphine given
Cardiac monitoring	Determine cardiac rhythm on monitor
Bloods: U/E, FBC, glucose and troponin	Beta blockers as per clinician if deemed beneficial

Chest Pain History

Many of our patients will experience chest pain this may be caused by a number of conditions.

Give some examples in the box below:-

Typically ischemic cardiac pain is described as “crushing, vice like and tight”. It may radiate down the arms or up to the neck and chin. Normally the patient would not be able to isolate the pain by pointing to it with one finger. The pain is often associated with nausea, clamminess and shortness of breath, as well as none verbal signs such as clutching the hand across the chest.

The patient who already has pre-existing ischemic heart disease may be able to make useful comparisons to previous pains e.g. “it’s worse than my normal angina”, “it feels like the last time I had a heart attack”. Patients with no cardiac history often state that they thought it was indigestion. Until a diagnosis is made, it should be assumed that chest pain is from cardiac origin.

So what has this got to do with ischemic heart disease?

Ischemia results from a reduced oxygen supply to the heart muscle. The heart receives its oxygen via the coronary arteries. Using an anatomy book of your choice, review coronary circulation, and the area of the heart they supply. Put your findings in the box below

Why is this happening?

Ischaemic heart disease (IHD) is the result of a process of narrowing of the arteries or *atherosclerosis.* This in turn is caused by a number of risk factors. Using available resources make a list of the risk factors for IHD.

This process leads to the development of fatty plaques within the arteries. For a variety of reasons, the plaques may become unstable and *rupture,* when this happens, the damage caused to the artery results in clotting factors being released into the blood, and a blood clot forming. This condition is known as *Acute Coronary Syndrome* (ACS).

NICE Clinical Guideline 95 (2016) Chest pain of recent onset and NICE Clinical Guideline 172 (2013) MI- Secondary Prevention will discussed on the study day

Cardiogenic shock

Cardiogenic shock is caused by impaired cardiac performance. Common causes are left ventricular failure (LVF), myocardial infarction (MI) and cardiac tamponade.

Signs & symptoms

- Pale
- Cool
- Clammy
- HR elevated
- BP reduced
- CVP increased (If in ICU PCWP increased)
- Oliguria

How does cardiogenic shock affect the components of the BP equation? BP = SV x HR x SVR (CO)

The first affected component is the stroke volume (HR will also be affected if MI is the cause, as this affects the conductive pathways). As in hypovolaemic shock, the reduction in CO (and BP) leads to an increase in SVR. However, in cardiogenic shock the CVP rises as the heart fails in its ability to pump the available volume into the systemic circulation, this creates back flow through the lungs and into the right side of the heart.

In cardiogenic shock the compensation occurring to try and maintain a 'normal' BP quickly becomes 'self-destructive'. As the SVR rises, the heart has to work harder in order to pump against an increased resistance (SVR). This results in a vicious cycle of increasing SVR, decreasing CO, and hence a fall in BP. The falling CO impairs tissue oxygen delivery, resulting in anaerobic metabolism and production of lactate. As the heart rate increases the coronary arteries are not perfused adequately and further myocardial insult occurs.

What treatments are available for cardiogenic shock and what is their aim?
Treatment Aim

Treatment aims to 'offload the heart', reduce cardiac workload, reduce SVR and improve oxygenation to the tissues.

The congestion of circulating volume arriving at the right atrium (causing the rise in CVP) can be reduced to a more manageable amount and the increased resistance created by the rise in SVR can also be reduced. Oxygen is administered and drugs to reduce cardiac workload, for example, diuretics and nitrates. Drug therapy needs to be managed carefully as they can exacerbate a further fall in BP.

In some cases continuous positive airway pressure (CPAP) can be used for cardiogenic pulmonary oedema.

If this is unavoidable inotropic support may **cautiously** be instigated as inotropes can increase cardiac workload, rather than reduce it.

What is the definition of an inotrope and a chronotrope?

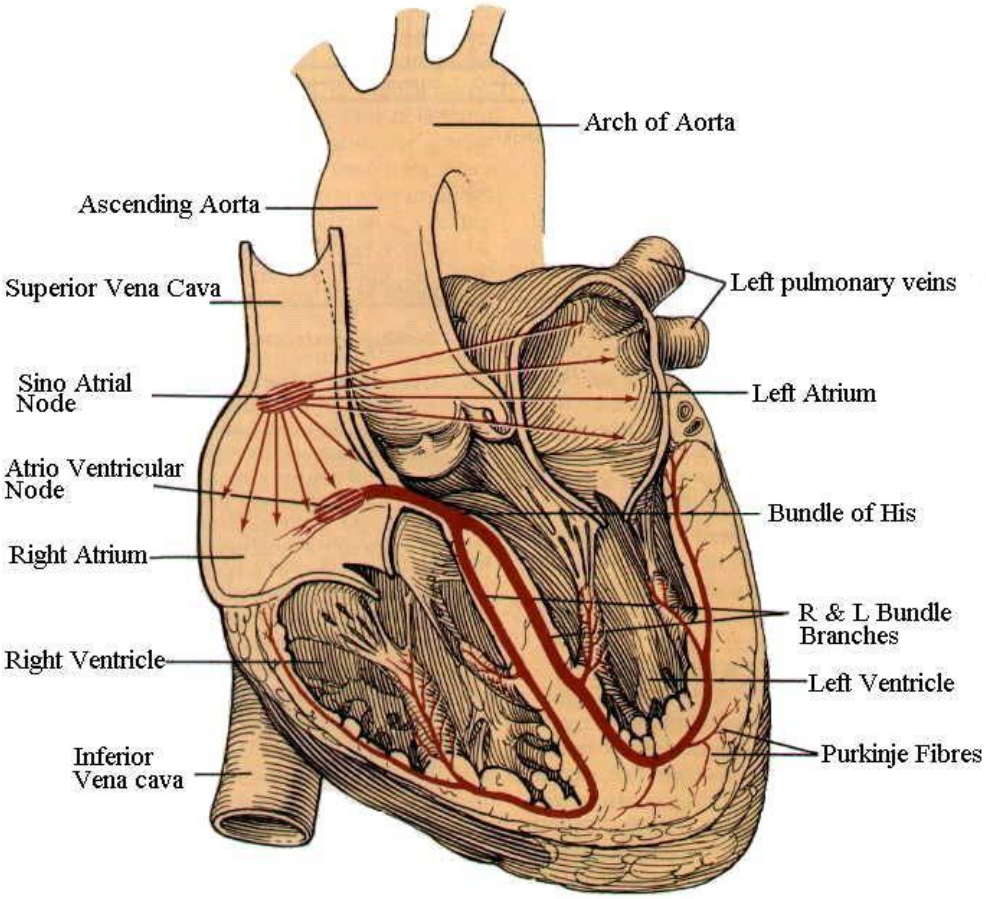
Dobutamine has vasodilatory properties (unlike other inotropes which vasoconstrict and increase SVR) as well as inotropic properties. This means that although dobutamine increases cardiac work, afterload is reduced. The dilatory property also improves coronary perfusion and myocardial oxygen delivery should be resumed.

ECG Monitoring

Poor quality ECG's can delay diagnosis and treatment decisions. ECG tracings arise from the electrical changes that accompany contraction of heart muscle. With knowledge of the normal anatomy and physiology of the heart, the conduction system and correct positioning of the leads and electrodes, you will improve your understanding and monitoring skills.

The Electrical Conducting System

Figure 4. The location of the conduction system in the heart



The heart is typically described as a 'blunt inverted cone', the size of the owner's fist. This means that impulses, triggered from the Sino Atrial (SA) node, travel from the base down to the apex. This is so the impulses travel back up the ventricular walls via the purkinje fibres, causing the heart to depolarise towards the valves at the top of the chamber – so maximising output.

The left ventricle (LV) is larger in volume and muscle mass than the right ventricle (RV) and the conductive system is more extensive. The Left Bundle Branch splits into two branches known as the Anterior and Posterior Fascicles allowing impulses to be spread more quickly through the muscle mass (figure 4).

The SA Node is known as the heart's primary pacemaker.
It is a collection of cells situated near the junction of the Superior Vena Cava (SVC) with the Right Atrium (RA).
It receives its instructions from the sympathetic (Adrenaline response) & parasympathetic (Vagus Nerve) systems depending on the demands placed on the heart by the needs of the body. The normal resting heart rate is approximately 75 beats per minute.

The AV Node is a collection of cells which lies at the junction of the right atrium with the ventricular septum.

The Bundle of His is the continuity of the AV node through the fibrous ring separating the atria and the ventricles. Impulses are 'delayed' by 0.10 sec to allow ventricular filling to be completed by the atria.

The Right Bundle Branch & Fascicles travel down into the ventricles. The anterior fascicle supplies the anterior & superior LV, whilst the posterior fascicle supplies the inferior & posterior LV.

The Purkinje Fibres or the terminating fibres of the conduction system enclose the whole of the ventricular muscle, creating a closed network of conduction.

Why do we need such an elaborate conduction system?

The conduction system can be viewed as a 'motorway network' for electrical impulses. Skeletal muscle and cardiac muscle (myocardium) differ in many ways. The myocardium has the ability to continuously contract and relax; any electrical impulse will travel across the myocardium, causing global contraction.

How and Why Does Electrical Activity Occur?

Electrical activity is produced by the movement of electrolytes across a semi permeable membrane, this is called '**saltatory conduction'.** Electrical activity is then generated and transmitted by the same method, creating a domino effect across the myocardium – similar to a 'mexican wave'.

Adequate electrolyte levels are essential for correct functioning of the myocardium. K^+ is particularly important

- hypokalaemia will increase the electrical potential which could lead to premature contractions;
- hyperkalaemia will reduce the electrical potential – ultimately leading to asystole.

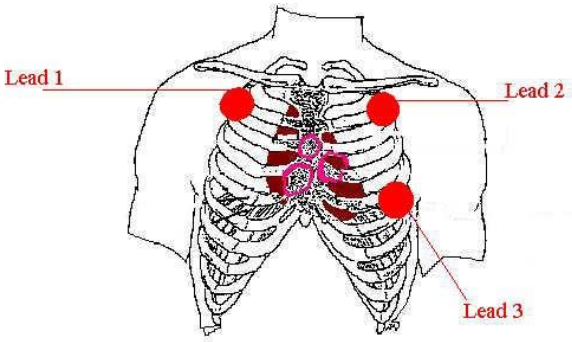
Spurious electrical activity or ectopic activity occurs if the myocardium has experienced an insult causing inflammatory changes. Mechanical, electrolytic, or hypoxia insults allow water and electrolytes to move from an area of high concentration to an area of low concentration, creating abnormal activity in the myocardium.

Continuous Cardiac Monitoring

This can be accessed with 3 or 5 leads. 5 lead monitoring has the advantage of obtaining more information at any one time.

Electrode positioning

Figure 5



Lead 1 is normally positioned to right arm below the clavicle.
Lead 2 is normally positioned to the left arm below the clavicle.
Lead 3 to the 4th intercostal space just left of the patients' nipple.
If 5 lead monitoring is available, the remaining two should be positioned to the right and left lower rib margins. Cables are usually colour coded to identify their appropriate position (figure 5).

Only two of the leads are normally 'active' (electricity basically needs a +ve & -ve electrode), the third is an earth lead to minimize interference. Modern monitors allow the user to manually select their chosen monitoring lead.

When applying electrodes, what would you need to do to obtain an accurate trace, free of artefacts?

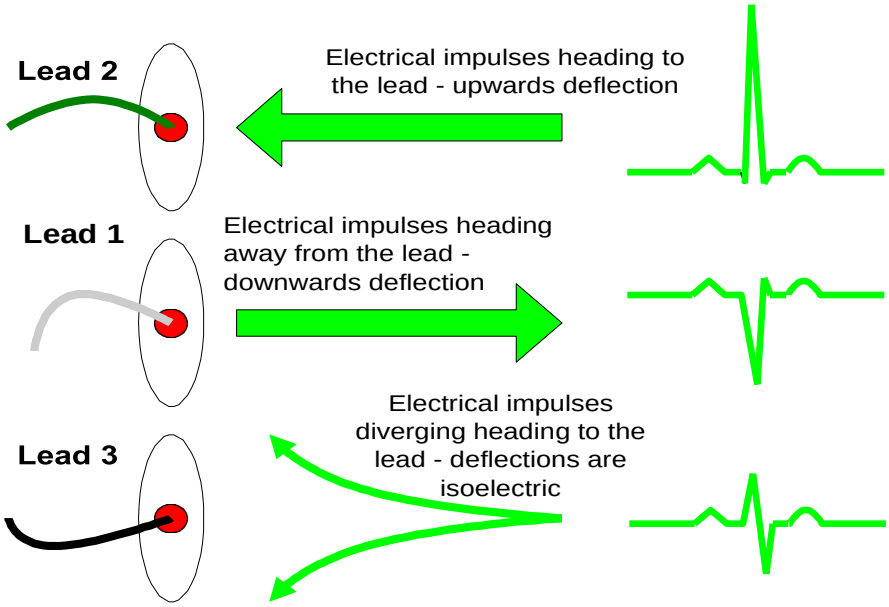
Monitoring leads

It is important to choose a monitoring lead which will show clear atrial and ventricular activity. Cardiac monitors often default to lead 2, because this normally shows a clear upright positive waveform.

Remember:
Most of the electrical energy within the heart is directed towards the left ventricle, so when monitoring electrical activity appears to head in this direction.

Impulses heading towards a lead will produce an upwards deflection, whilst those heading away will produce a downwards deflection (figure 6).

Figure 6.



So, in lead 2 we should have a strong overall upwards deflection.

Central Venous Lines

The recommended insertion site for central venous lines is the subclavian vein. However internal jugular and femoral sites are also used. The number of lumens on a central venous line will be dependant on what the line is to be used for and the condition of the patient.

What are the indications for insertion of a central venous line?
What are the potential complications of inserting a central venous line?

Complications

Some of the complications associated with central venous catheter placement include haemothorax, pneumothorax, nerve injury, arterial puncture and thoracic-duct perforation. The patient is also at risk of systemic or local infection, perforation or erosion of vascular structures, thrombosis, and catheter or air embolism. Central venous catheters should therefore be inserted using an aseptic technique and under ultrasound guidance. When an open vein under negative pressure is exposed to atmospheric air, the potential for air emboli exists. This can be minimised by securing all connections to catheter with leur locks and appropriate positioning for insertion and removal. Avoid unnecessary disconnection by using luer lock needle free connectors.

What anatomical site is most commonly used in your area to insert central lines?	
What are the advantages and disadvantages of this site?	
Advantages	Disadvantages

Central Venous Pressure Monitoring

Central venous pressure (CVP) is measured in the superior vena cava, or right atrium, and is a direct measurement of right atrial (RA) pressure. The CVP reflects the preload of the right ventricle, thereby providing information about the patient's blood volume and right ventricular (RV) performance.

Review the following diagram to establish the relationship between cardiac events and the CVP waveform (figure 7).

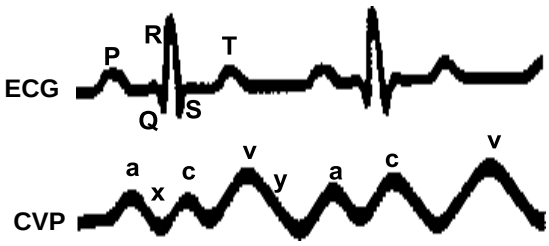


Figure 7

Cardiac events that produce the CVP waveform with a, c, and v waves.
a wave represents atrial contraction
x descent represents atrial relaxation
c wave represents the bulging of the closed tricuspid valve into the right atrium during ventricular systole
v wave represents atrial filling
y descent represents opening of the tricuspid valve and filling of the ventricle

Significance of Values

The CVP may be measured by a pressure transducer in millimetres of mercury (mmHg). Components of this system can be seen in figure 8. Fluid overload produces an elevated CVP, indicating that fluids have been over administered or administered too rapidly. A hypovolaemic patient has a low CVP, indicating that there is insufficient blood volume in the ventricle at end diastole to produce an adequate stroke volume (Fig 8).

CVP values are as follows:

0 – 5mmHg	Healthy ambulatory person lying down
7 – 9mmHg	Upper limit of normal for acutely ill patients
11 – 13mmHg	Upper limit of acceptable CVP values before advanced haemodynamic monitoring may be considered
14 – 18mmHg	Values seen in critically ill patients on mechanical ventilation with positive end-expiratory pressure (PEEP), who require fluid volume to maintain arterial pressure

What are the indications for monitoring a CVP?
What conditions/diseases can affect the CVP reading?

Indications for Monitoring

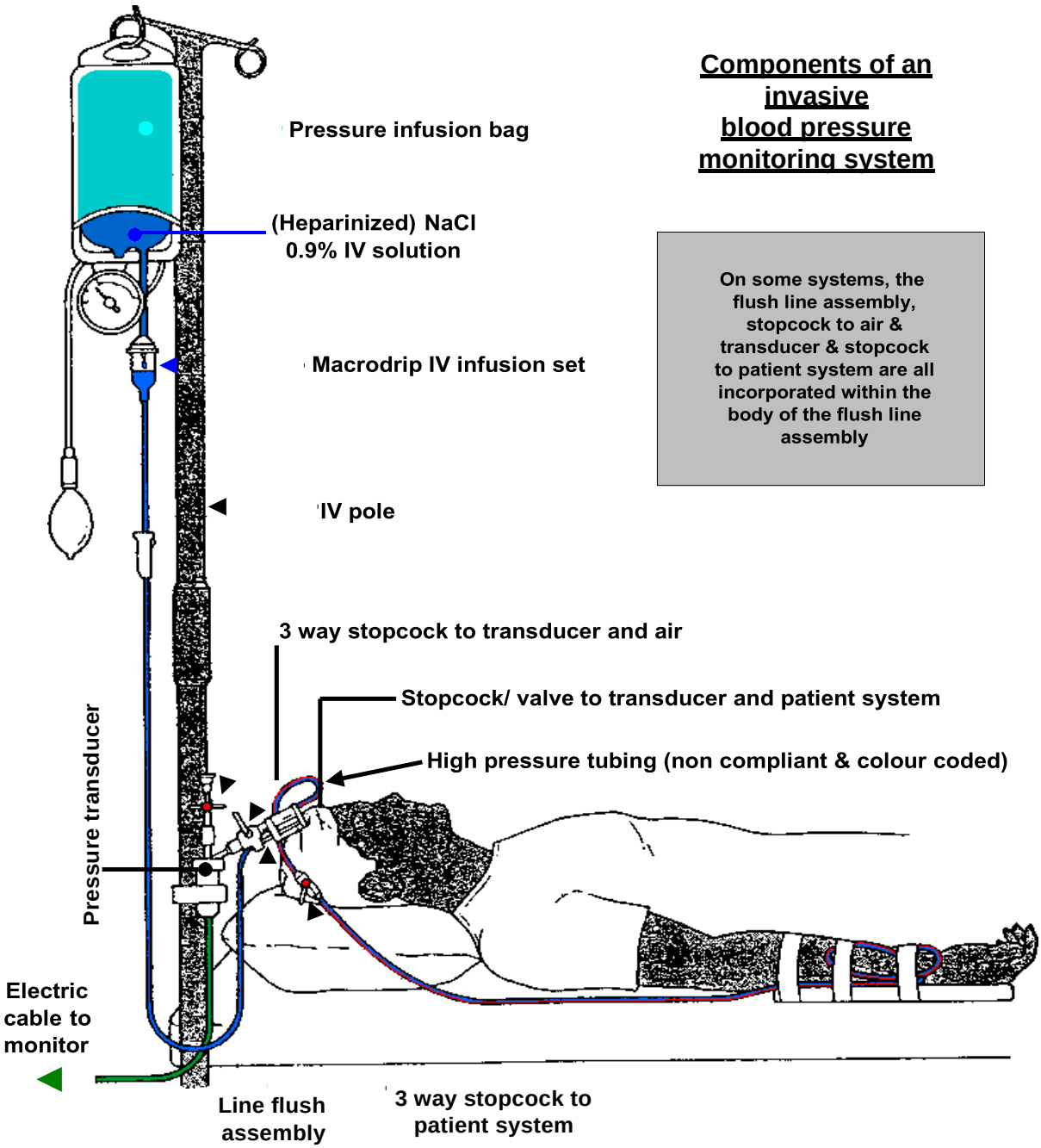
Monitoring of CVP is indicated for patients who have disturbances in circulating blood volume but in whom cardiopulmonary function is reasonably normal. CVP measurements are most useful during early resuscitation and those patients suspected of inadequate circulating volume. The CVP may be used to guide fluid therapy after haemorrhage, accidental and surgical trauma, sepsis, and emergency conditions associated with blood volume deficits. Valuable information can be obtained from the CVP concerning a patient's tolerance to a volume load or fluid challenge over a prescribed period of time. The CVP can indicate the reserve capacity of the heart and vascular tree.

Limitations

The CVP is not a reliable indicator of left-ventricular (LV) dysfunction or preload of the left side of the heart. A patient can have severe deterioration of LV function that will not be reflected in changes in RA or vena cava pressures. CVP measurements are not as accurate at reflecting hypovolaemia as they are at indicating fluid overload. In the hypervolaemic state and in right heart failure, blood accumulates behind the right ventricle, and the venous tree becomes distended. While this venous engorgement increases, the CVP also increases so that high peripheral pressures reflect high CVP values. It is incorrect, however to assess blood volume status from CVP values because there are many factors that influence CVP values, including inotropic therapy, intrinsic venous tone, increased intra-abdominal or intra-thoracic pressures, cardiac performance, and blood volume. Wide variations in the CVP may also occur in cases of tricuspid insufficiency, when the central line slips into the right ventricle, and with severe right heart failure and dilation of the atrio-ventricular ring. Post-partum women and patients with pulmonary disease will also have an increased CVP. Therefore on the study day students will be asked to reflect in practice regarding CVP monitoring and review evidence.

The EPIC 3 (2014) will also be evidenced to discuss care of central lines and encourage the student to reflect on practice

Figure 8



Congratulations you have finished!

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