HAEMODYNAMIC MONITORING SELF DIRECTED LEARNING PACKAGE

By

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1.0 Introduction.

Haemodynamic monitoring is a cornerstone of critical care nursing and management, be it central venous pressure, arterial pressure or the more invasive methods such as pulmonary artery pressure monitoring and or direct measurement of left arterial pressure. It is used by clinitians to obtain and evaluate a patient's baseline cardiac function, circulating blood volume and to assess the patient's physiological response to treatment and trends in the patient's condition.It is important to use haemodynamic monitoring in conjunction with physical assessment of the patient.

2.0 Aims of the Package

To provide the registered nurse with the opportunity to acquire the level of knowledge, through self directed learning, on which to base the nursing skills necessary for beginning and advanced level practice.

3.0 Objectives

At the end of the package, the graduate nurse should be able to:-

- Outline the location, structure and the conduction system of the heart;
- Explain the cardiac cycle in relation to the electrical conduction of the heart;
- State the different types of haemodynamic monitoring, their advantages and disadvantages;
- Understand the physiological basis of haemodynamic monitoring;
- State the reasons for haemodynamic monitoring;
- Set up and assist with the insertion of invasive lines;
- Interprate the haemodynamic monitoring wave forms in relation the cardiac cycle and electrical conduction system of the heart;

- ❖ Distinguish the normal from the abnormal wave forms and their implications on the patient and their family;
- ❖ Understand the nursing considerations of caring for a patient with invasive monitoring;
- ❖ Define the complications of invasive monitoring;

The purpose of haemodynamic monitoring is to obtain information that will indicate whether the conditions that are required to maintain tissue perfusion are being maintained. The body's own regulatory systems do this under normal circumstances. However, there are situations where the body's compensatory ability is exhausted and is unable to establish an adequate homeostatic balance. It is then necessary to intervene to establish a homeostatic balance that is commensurate with tissue health and survival. There are circumstances (eg. Major surgery) where the body's physiological reserve may only be able to deal with an increased demand if that demand is carefully managed.

The basis haemodynamic monitoring is the assessment of the adequacy of tissue perfusion (the balance between tissue oxygen demand and supply, as well the maintenance of the nutritional, temperature, and electro-chemical balances). The most fundermental of haemodynamic observations is an assessment of end organ function. The observations of urine output, mentation, peripheral warmth are the main observations we use to assess end organ perfusion. Measurement of aspects of the internal milieu that are known to affect organ perfusion are also relevant observations. These observations of factors that are known to effect organ perfusion are for example blood pressure, cardiac output, systemic vascular resistance, central venous pressure, pulmonary artery wedge pressure, oxygen delivery,oxygen consumption, and arterial blood gasses.

The following package will review the relevant physiology that is required for

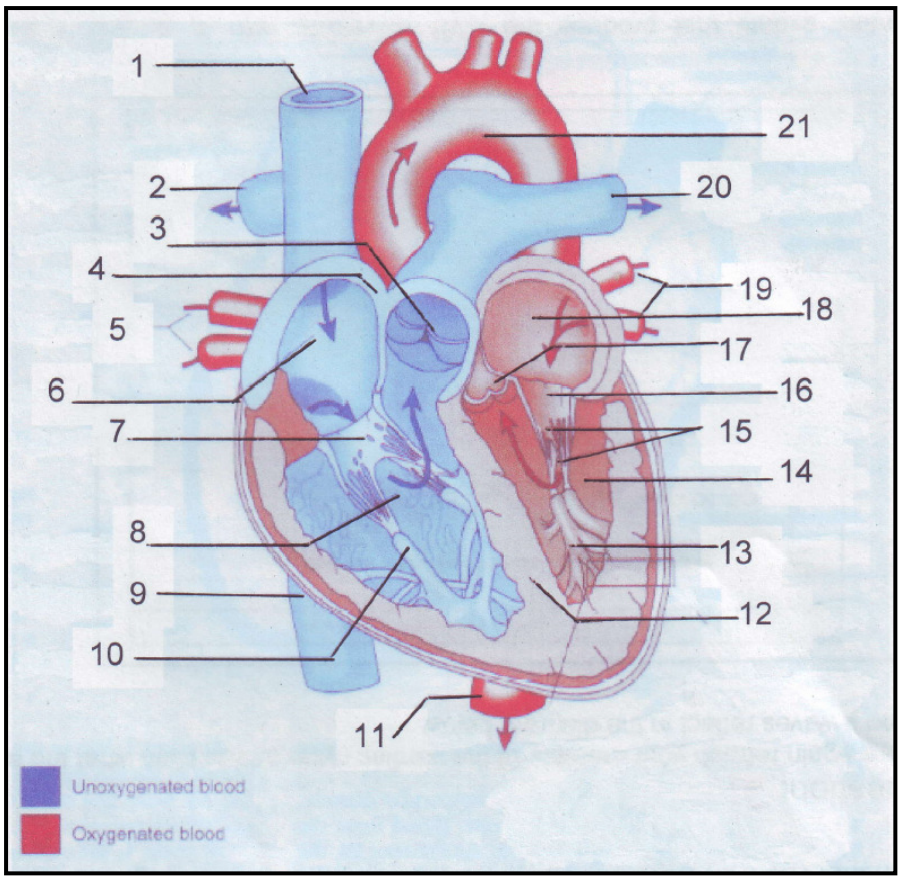
an understanding of the practice of haemodynamic monitoring. It will then cover what is measured in clinical practice, why, as well as look at the "machinery" of measurement.

4.0 Anatomy of the Heart.

To put things in context, we will revise our anatomy and physiology of the heart before we start the haemodynamic monitoring issues

Question 1

Identify the structures of the heart as highlighted by the arrows.



Structure Of The Heart *(Illustration by Hudak, C. M., Gallo, B. M. & Morton, P. P.G. (2005)Critical Care Nursing: a holistic approach (8th ed) Philadelphia: J. B. Lippincott p203)*

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Question 2

a) Describe the heart valves, their location, function and their mechanism of action.

b) What would happen if the chordae tendinae and the papillary muscles were destroyed?

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b) _____

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5.0 Properties of the Cardiac Muscle.

Here, we will concern ourselves with the most important aspects of the cardiac muscle fibres with regard to the cardiac cycle.

In contrast to the skeletal muscles which are long, cylindrical and

multinucleate, cardiac muscles are short, fat, branched and interconnected, with each fibre containing one or at most, two centrally located nuclei. Intercellular spaces are filled with loose connective tissue matrix (endomysium) which contain numerous capillaries and is connected to the fribrous skeleton of the heart that links the cardiac cells together in a coilong array and reinforces the basket like walls of the heart. This skeleton acts as a tendon and as an insertion which gives the cardiac cells something to pull / exert their force against.

We will briefly describe the components of the cardiac cell.

Myocardial cells are long and narrow, branching and overlapping. They are comprised of the following;

- sarcoplasm – a cell membrane which separates the cell from the extracellular space.
- Intercalated discs – the dark staining junction between cells. It provides low resistance to the spread of electrical current and aids in functioning of the myocardial cells as a syncytium.
- Myofibrils –They are composed of sarcomeres which exhibit Z discs, A bands and I bands which reflect the arrangement of the thick myocin and thin actin filaments. These are long rod like structures which contain contractile proteins responsible for generating tension and muscle shortening i.e. contraction.
- Mytochondria – they are sites for energy production required for maintenance of cell membrane barrier Na, K and Ca pumps during contraction.
- Sarcoplasmic reticulum – it stores calcium
- Transverse tubules – they are narrow “tunnels” and are continuous with the cell membrane (sarcolemma). They have pores in the surface which are open to extracellular space and thus allow conduction of action potential deep into the muscle fibre.

Question 3

With help of a diagram, illustrate and identify the cardiac cell and its components as outlined above.

Name the three types of myocadial cells.

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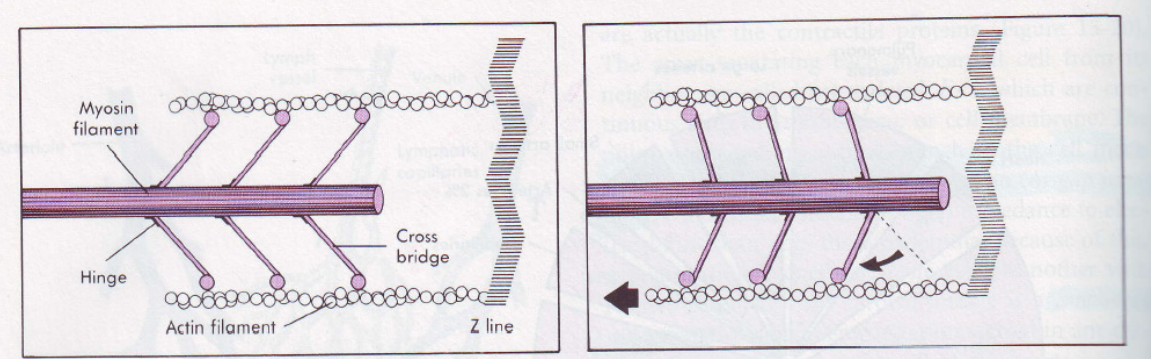
6.0 Myocardial Contraction

The myofibril is comprised of repeating functional units called sarcomeres. Each of which is made up of two protein filaments called **actin** and **myocin** and they are arranged in an overlapping pattern. The proteins **troponin** and tropomycin are located at intervals on the actin filament (thin filament).

The contraction of the cardiac muscle is a result of the shortening of the myofibrils. This happens when the actin and the myocin filaments of the sarcomere pull over each other - a phenomena called the **crossbridge theory of muscle contraction.** Crossbridges are made by the globular head of the thick myocin filaments between the action and the myocin filaments. These globular heads are the sites for breakdown of ATP and thus energy production.

The cross bridge is capable of binding, flexing, releasing and binding again thereby shortening the thin actin filaments towards the centre of the sarcomere and thus shortening the myofibril.



Actin and myocin filaments and cross-bridges for cell contraction (Illustration from Stacy K.L. etal Critical Care Nursing Diagnosis and Management 3rd Ed. p344)

Cross bridge binding is regulated by the function of the proteins troponin and tropomycin found on the action filament. These proteins prevent binding of the cross bridges and effect relaxation.

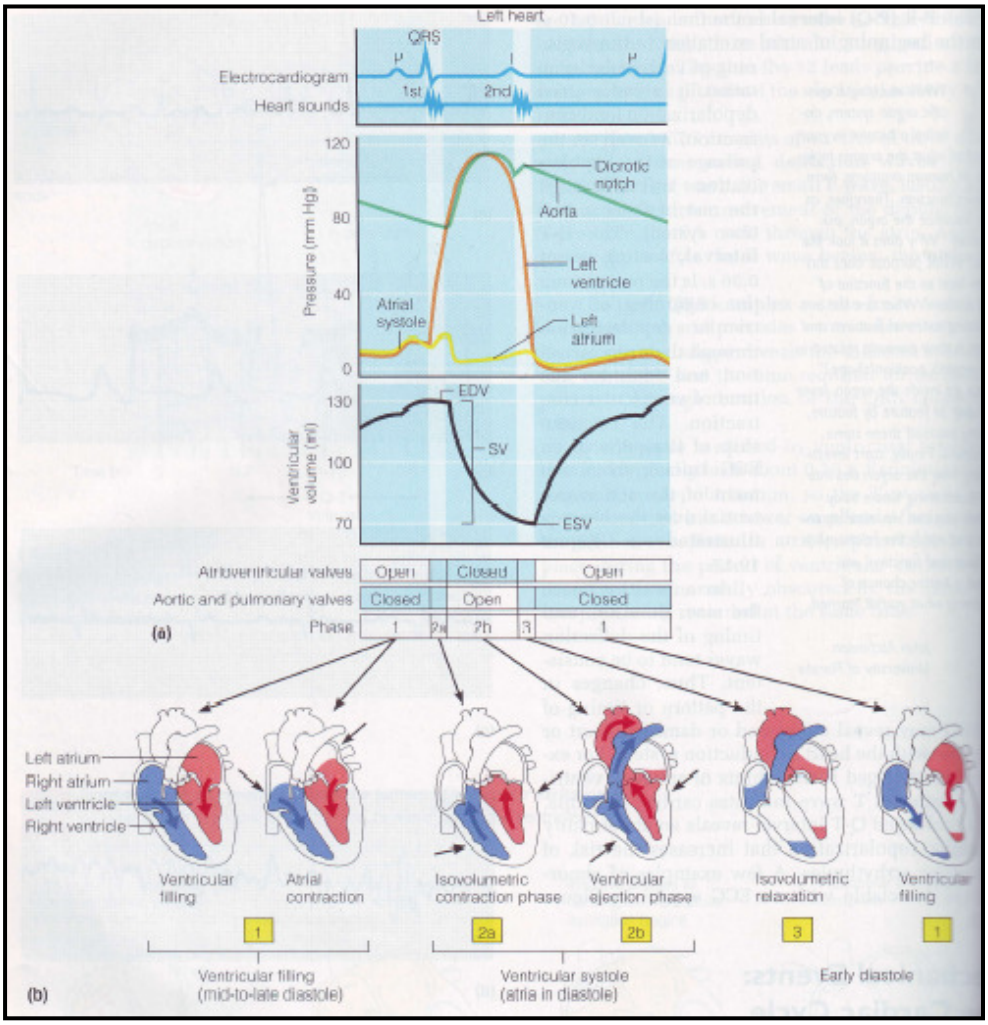
7.0 Electrical Activity.

Electrical signals or action potential passing over the muscle cell cause the movement of calcium from the extracellular space and from the stores of the sarcoplasmic reticulum. This increases the intracellular calcium in the cytoplasm where the calcium concentration is usually low. The increase in calcium in the intracellular space interacts with the actin binding site so that the cross bridges can attach and contraction take place. In the absence of calcium, the binding sites are covered and the fibres are relaxed (relation can only happen if the Ca is removed from the sarcoplasm- inside the myocardial cell).This can only happen through the action of Ca pumps located in the SR and the cell membrane. Contraction is as a result of the action potential causing the influx of calcium and relaxation is a result of ceasation of depolarization and removal of calcium from the sarcomere. This process of action potential/contraction is known as excitation – contraction coupling.

It is important to remember that for contraction and relaxation to occur, there must be ATP derived energy i.e. calcium pumps and relaxation of crossbridges in contraction and calcium pumps in relaxation). This phenomenon is absent in myocardial ischemia because the availability of ATP is limited and the cell is unable to remove calcium resulting in continued coupling of crossbridges. The continued coupling of crossbridges makes the muscle stiff and change in compliance.

8.0 Mechanical Events

Here, we will begin by looking at the diagramatic illustration of the cardiac cycle.



Diagramatic representation of the cardiac cycle. Illustration from Marieb. E. N. (1998) 4th Edition. Human Anatomy & Physiology p676).

Based on the diagram above, answer the following questions.

Question 5

Define the cardiac cyle.

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Question 6

The electrocardiogragh is marked by deflections known as P wave, QRS complex and T wave. What do these deflections represent?

Question 7

Define the following;

a) PR interval

b) ST segment

c) QT interval

a) _____

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b) _____

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c) _____

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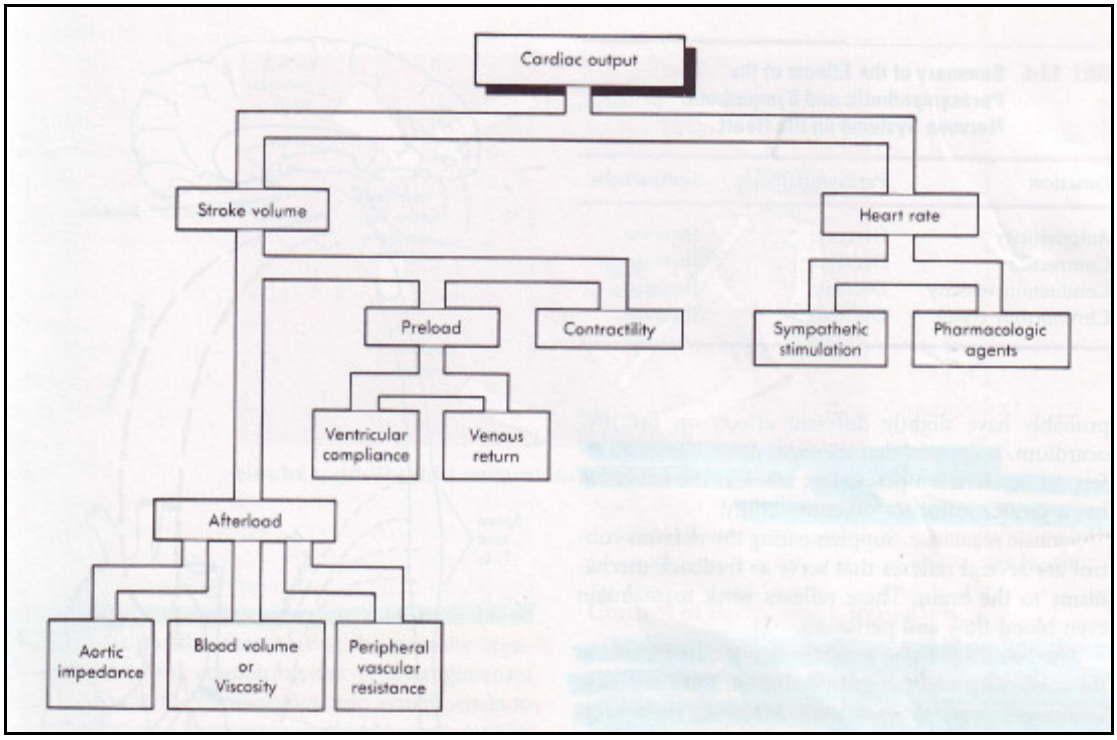
9.0 Interplay of the Heart and Vessels

The function of the myocardium is to provide rhythmic coordinated contraction, pump, add energy to blood and produce flow resulting in **cardiac output**.

Cardiac out (CO) - This is the volume of blood ejected from the heart over one minute. Cardiac output is important in tissue oxygenation. It must be sufficient to carry oxygen to the tissues without which there would be

tissue hypoxia leading to tissue ischemia. CO is determined by heart rate (HR) per minute and stroke volume (SV) in millilitres per beat. The equation is as follows:

CO= HR × SV



Determinants of Cardiac Output. Illustration from Stacey, K. M, et al Critical Care Nursing Diagnosis And Management 3rd Ed. p351

The heart rate is influenced by many neurochemical factors where as stroke volume is determined by preload, afterload and contractility.

Stroke Volume- this is the amount of blood ejected from the left side of the heart with each heart beat. It is usually 70 – 80 mls of blood out of a total end-diastolic volume (EDV) of 110 – 130 mls of blood giving an ejection fraction of 60%. It is regulated by 3 variables namely;

9.1 Preload

The amount of blood returning to the heart (venous return) distending it's ventricles (EDV) just before systole.

9.2 Contractility

The contractile strength of the heart independent of the muscle stretch and EDV. It is the direct consequence of the influx of calcium into the cytoplasm from the extracellular fluid and the SR.

9.3 Afterload

Pressure that the ventricles have to overcome to force open the aortic and pulmonary valves(pressure that must be overcome by the ventricles to eject blood from the heart (vascular resistance).

Now, remember the crossbridge theory? It is useful in conceptualizing **the heart length tension relationship**.

The length of a muscle's contraction is determined by the following factors;

- Number of stimulated muscle fibres
- Thickness of each muscle fibre (the thicker the muscle fibres the more myofibrils – contractile units).
- Initial length of the muscle at rest.

When a fibre is stretched, there is no overlap of Actin and Myocin hence no crossbridges and therefore, no power. When filaments are shortened (completely overlap) there is further shortening resulting in limited contraction. On the other hand, maximum contractile force is attained when the muscle is at a length that optimizes the overlap of actin and myosin and the ability of the fibres to shorten.

Question *8*

Consider what we have discussed above and describe the Frank-Starling Law and relate the property of the ventricle described by this law to the

tension relationship and the cross bridge theory.

Question 9

Define the following:

a) Inotrope

b) Dromotrope

c) Chronotrope

a)

b)

c)

10.0 Regulation of the Heart Beat.

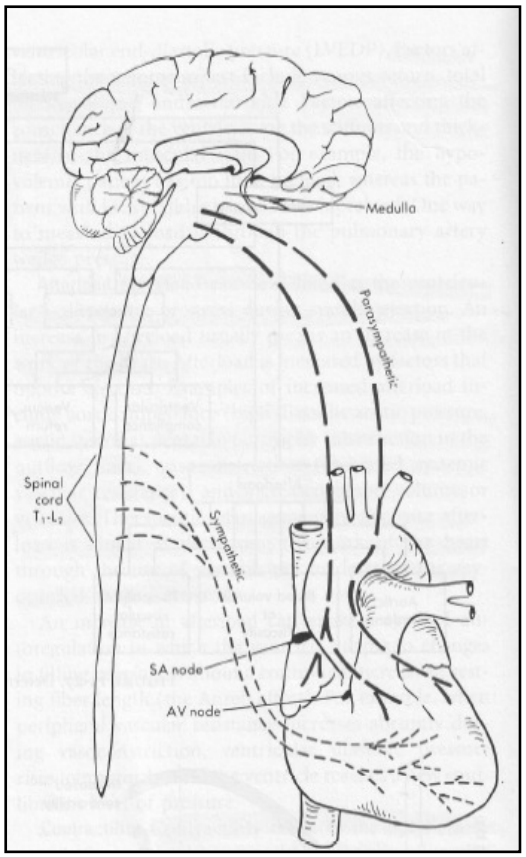
Autonomic nervous system regulation.

Regulation of the heart beat is via nervous control and several reflexes that serve as feedback mechanisms to the brain. The nervous system is divided into the sympathetic and the parasympathetic nervous system.

They normally create balance between maintenance and fight or flight

cardiovascular functions respectively. For example, during excitement, exercise or fright, the sympathetic nerve fibres are activated and they release noradrenaline and adrenaline at their cardiac synapses which bind to *ß1 adrenergic receptors* in the heart, causing the threshhold to be reached faster resulting in the pacemaker firing more rapidly and the heart beating faster. The sympathetic stimulation also enhances contractility by enhancing $Ca^{2}+$ entry into contractile cells .On the hand, the parasympathetic system opposes the sympathetic system in that it effectively lowers the heart rate after the stressful situation is over.

Nevertheless, the parasympathetic system may be persistently activated by emotions such as grief in which case it may produce parasympathetic-activated cardiac responses mediated by ***acetylcholine*,** which hyperpolarises the membranes of it's effector cells by opening potassium channels. At rest, both autonomic divisions send impulses to the SA node of the heart but the inhibitory influence is dominant and thus , the heart is said to exhibit **vagal tone**.



Autonomic nervous system innervation of nodal tissue and myocardium by parasympathetic vagus nerve fibers and sympathetic chains. (Stacy K. M 1998, Critical Care Nursing: Diagnosis And Management 3rd ed. p352)

11.0 Intrinsic Regulation.

Nervous control is supplimented by several reflexes that serve as feedback mechanism to the brain. These reflexes work to maintain even blood flow and perfusion. These are baroreceptors (pressure sensors) located in the aortic arch and the carotid sinuses, Arterial chemoreceptors located in the bifurcation of the aortic arch.

The other intrinsic factors that regulate the heart beat are:

- The bainbridge reflex which is attributed to the pressure in the right atrium. It controls the heartbeat by causing reflex tachycardia when the pressure in the right atrium rises significantly enough to stimulate them. This is so in order to protect the right side of the heart from an overload state and to quickly equalise the right and left sides of the heart filling pressures.
- Atrial natriuretic factor-a hormone secreted by the atria in response to increases in atrial pressure.It causes Na+ and water to be excreted by the kidneys.It is also a potent vasodilator.Thus the excess extracellular volume is excreted and the capacity of the veins to restore total blood volume is increased.
- Calcium ions- low Ca+ ions (hypocalcaemia) depress the heart and high Ca+ ions (hypercalceamia) increase heart irritability.
- Potassium- low K+ ions (hypokalemia) makes the heart beat feebly and a high K+ ions lowers the resting membrane potential resulting in abnormal rythms which can lead to cardiac arrest and death.
- Sodium – Too much Na+ ions (hypernatremia) inhibits transfer of Ca+ ions into the cardiac cells thereby blocking heart contraction.

Effector Organ	Cholinergic Impulses Response	Noradrenergic Impulses	
		Receptor Type	Response
Heart			
Sinoartrial node	Decrease in heart rate; vagal arrest	$ß_1$	Increase in heart rate
Atria	Decrease in contractility and (usually) increase in in conduction velocity	$ß_1$	Increase in contractility and conduction velocity
Atrioventicular (AV) node and conducting system	Decrease in in conduction velocity; AV block	$ß_1$	Increase in conduction velocity
Ventricles	___	$ß_1$	Increase in contractility and conduction velocity
Arterioles			
Coronary, skeletal muscle, pulmonary, abdominal viscera, renal	Dilation	α $ß_2$	Constriction Dilation
Skin and mucosa, cerebral,	___	α	constriction
Systemic Veins	____	α $ß_2$	Constriction Dilation

A and $ß_2$ Effects of the Autonomic Nervous System on the Heart and Vascularity. ***(****Hudak, C. M. et al 2005, Critical Care Nursing. A holistic approach. 8th edn.. P205)*

The law of Laplace states that the wall tension of a sphere will increase as the sphere's diameter increases.

Myocardial oxygen demands are also related to muscle tension.(the greater the tension in the wall at rest the greater the contractile force must be to overcome the wall tension and generate a contraction. Contraction requires ATP derived energy and ATP production requires oxygen.

From what we have discussed above, we can see that as preload increases, so does stroke volume up to a limit. Furthermore, preload increases tension generated by the myocardial cells due to more optimal fibre length. However beyond this point, fibre tension /ventricular wall tension increases as a function of the ventricles geometry.

Increased resistance to ventricular outflow leads to increased ventricular wall strain. Contraction builds pressure within the ventricle and forces the aortic and pulmonic valves to open and then blood is given enough kinetic energy to move through the arterial system. The resistance to the blood flow increases as the blood vessels constrict. This is what is called **vascular resistance.** The increase in ventricular wall strain due to these factors is what is known as the afterload. So, **an increase in after load will result in an increase in myocardial oxygen demand.**

Now that we have covered the anatomy and physiology of the heart, we will proceed to haemodynamic monitoring.

In the Intensive Care setting, there are two types of haemodynamic monitoring.

11.1 Noninvasive

Such as auscultation, automated oscillometry blood pressure monitoring systems, doppler and palpation.

11.2 Invasive

Such as placement of central venous lines for monitoring central venous pressure, pulmonary artery catheters for measuring left heart filling pressure or placement of arterial lines for continuous blood pressure monitoring.

In this learning package we are going to cover noninvasive monitoring of blood pressure; invasive blood pressure monitoring via an arterial line and central venous pressure measurement.

12.0 What Is Blood Pressure?

This is defined as the pressure exerted by the blood against the walls of the vessels - i.e., arteries, veins, capillaries,

The *difference* in blood pressure in arteries, capillaries and veins, or *blood pressure gradient,* is the force that enables blood to flow throughout the body. The further blood flows from the heart, the lower the pressure.

Pressure is highest in arteries, drops significantly in capillaries, and is almost zero in veins.

There are three types of blood pressure namely; arterial, venous and capillary.

12.1 Arterial pressure

Is the pressure of blood against arteries' walls. It has the following components:

a) Systolic pressure

It is the maximum pressure of the blood exerted against the artery walls when the heart contracts. Systolic pressure normally ranges from 110 to 140 mmHg.

b) Diastolic pressure

Is the force of blood exerted against the artery walls when the heart is relaxing. Diastolic pressure normally ranges from 60 to 90 mmHg.

c) Mean arterial pressure

d) Pulse pressure

Is the difference between systolic and diastolic pressures.

Two major factors affect pulse pressure.

i. Stroke volume (output of the heart).

ii. Compliance (total distensibility) of the arterial tree.

Any condition of the circulation that affects either of these two factors will also affect pulse pressure.

Circulatory factors influencing arterial pressure include: -

a) Cardiac output:

Increased output increases arterial pressure, decreased output decreases arterial pressure.

b) Peripheral resistance:

Narrowed arterioles increase blood pressure; dilated arterioles decrease blood pressure.

c) Arterial elasticity:

Elastic vessels accommodate to changes in blood flow, whereas rigid sclerotic vessels cause increases in systolic and pulse pressures.

d) Blood volume:

Decreased blood volume e.g. due to haemorrhage results in decreased blood pressure.

e) Blood viscosity:

Increased blood viscosity, due to overabundance of RBC's or plasma proteins, results in high blood pressure; decreased viscosity from anaemia or lack of RBC's results in

lower pressure.

Other factors influencing arterial pressure are age, weight, emotions and exercise.

12.2 Venous Pressure

Is the blood pressure in the veins. In small veins there are no pulsations; the pressure is about 12 mmHg.

12.3 Capillary Pressure

Is pressure exerted by blood against the capillaries. It is 22 mmHg at the arterial end of the capillaries and 12 mmHg at the venous end.

13 Factors Regulating Circulation

As discussed before there are several factors including the nervous system that regulate heart rate, degree of arteriolar constriction, and arterial blood pressure, in order to maintain homeostasis. Neural reflexes are controlled via the vasomotor centre in the medulla oblongata.

This comprises four centres controlling the heart and blood vessels

1) *Vasoconstrictor centre* - reduces diameter of blood vessels.
2) *Vasodilator centre* - increases diameter of blood vessels.
3) *Cardioaccelerator centre* - increases heart rate.
4) *Cardioinhibitory centre* - decreases heart rate.

The four centres are stimulated or inhibited by:

1) Baroreceptors

Specialised nerve endings affected changes in pressure of blood in arteries. They stimulate the vasodilator and cardio accelerator centres to increase heart rate and vaso-constrict the blood vessels leading to increased blood pressure thereby restoring homeostasis.

a) Arterial pressoreceptors located in the walls of the aortic arch and carotid sinuses.

b) Venous pressoreceptors located in terminal sections of the vena cava and right atrium.

***2)* Chemoreceptors**

located in the aortic arch and carotid bodies. They are sensitive to *oxygen* lack and secondary to *increased blood carbon dioxide and decreased arterial pH. This results in increase of heart rate in order to increase cardiac output which in turn increases delivery of oxygen to the tissues.*

3) The Medullary ischaemic reflex

produces vasoconstriction of small blood vessels in response to stimulation of the vasoconstrictor centre by $C0_2$ excess and decreased or diminishing oxygen.

4) Higher brain centres,

Higher brain centres in the cerebral cortex and hypothalamus, transmit impulses to medullary centres when the individual experiences extreme emotion, e.g, fear, rage, embarrassment.

5) The renal system

Regulates circulation, blood volume and blood pressure by controlling excretion or retention of water in urine. This is done through a system called the Renin- angiotensin system.

Question 10

Describe the Renin-angiotensin system and how it regulates blood pressure.

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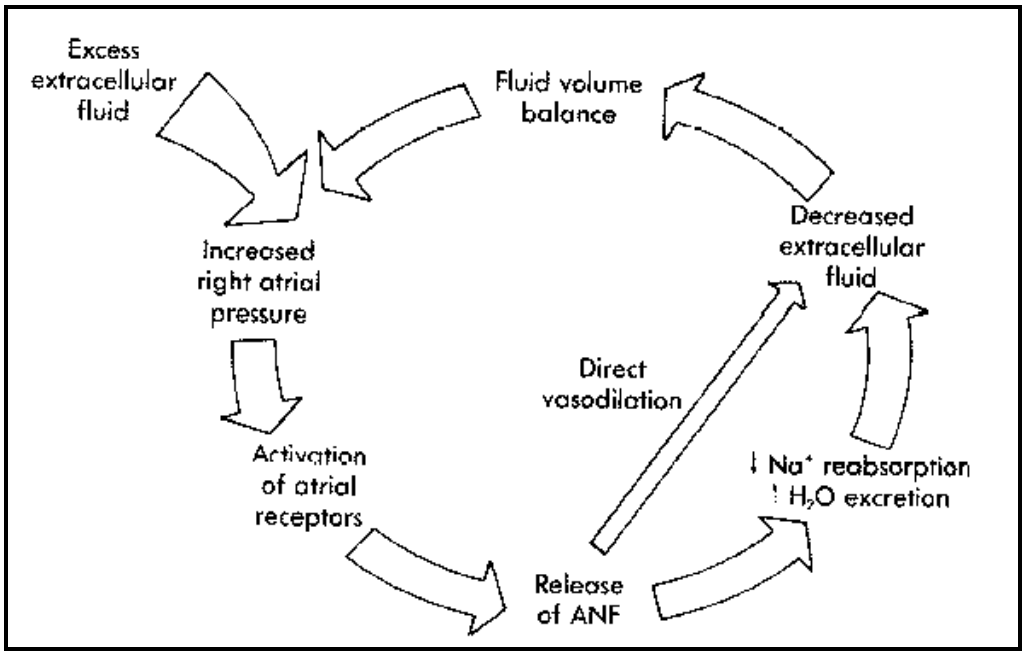
The opposite set of events occurs with an increase in blood volume and pressure. The **Artrial Natriuretic Factor System (ANF)** kicks in as illustrated below**.**



The Artrial Natriuretic Factor System *(Illustration from Stacey, et al Critical Care Nursing Diagnosis and Management , p353)*

Thirst is also stimulated by the hypothalamus to encourage the individual to procure fluids.

With this overview of blood pressure we can now move on to do the three sections of this learning package which have been divided into: -

a) Noninvasive Blood Pressure
b) Arterial Line
c) CVP

14 Noninvasive Blood Pressure Monitoring

14.1 Objectives

At the completion of the package, the registered nurse will be able to: -

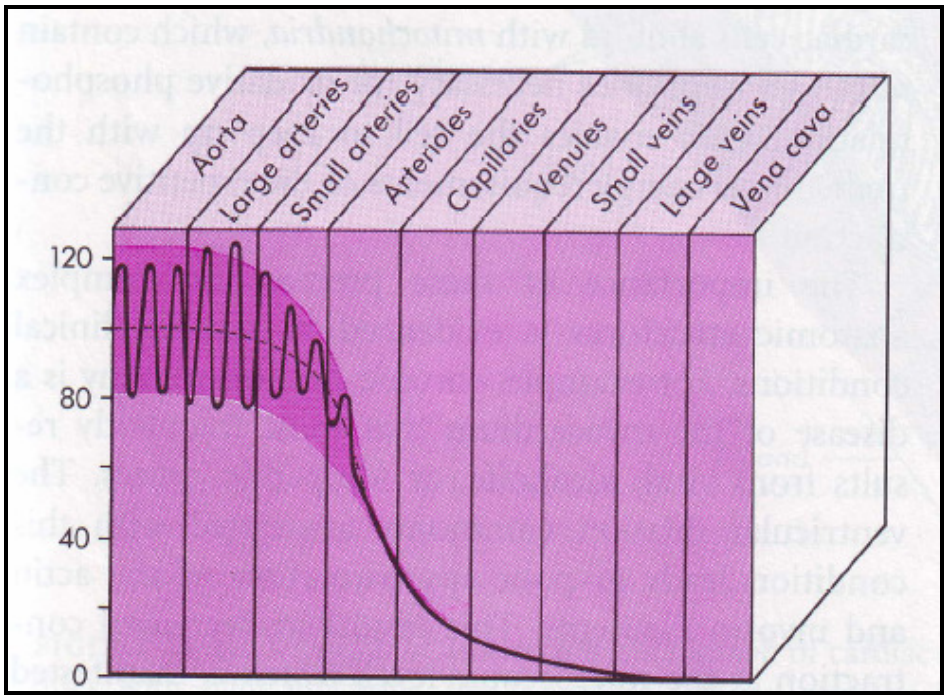
1) List advantages of using noninvasive blood pressure (NIBP).
2) Have gained knowledge of assessment and monitoring technique using the NIBP module for the Spacelab monitor.
3) Be able to troubleshoot an automated vital signs monitor.
4) Have gained confidence in the use of this equipment in a highly technological environment.

14.2 Overview

Blood pressure (BP) is defined as the intra-arterial pressure generated by the pulsatile flow of blood through an artery. The pulsatile nature of arterial flow is caused by intermittent cardiac ejection of blood from the left ventricle and the stretch of the ascending aorta. BP measurements are a reflection of pressures corresponding to various points of the cardiac cycle. It is therefore used to determine cardiovascular function and the state of the peripheral circulation. Noninvasive blood pressure can be taken

from different sites such as the radial and the brachial arteries with the later being the most preferred to the former as it fulfills the necessary requirements for most noninvasive techniques. For instance, the brachial artery lies close to the skin surface, thus allowing for a clear measurement signal to be obtained. Furthermore, the brachial artery is close to the aorta and thereby giving readings that correlate well with the aortic blood pressure. The brachial artery lies on the humerous which allows for uniform compression and occlussion of the artery. Also, the upper arm is the most comfortable and convenient location for routine blood pressure measurement.

Blood pressure differs in different points of the circulatory system as shown below.



Blood Pressure in the different Portions of the systemic circulatory system (Illustration from Stacy K. M Critical Care Nursing: Diagnosis And Management 3rd edn p341)

Blood pressure has a at least three basic components namely systolic blood pressure (SBP), mean arterial blood pressure (MAP) and diastolic blood pressure (DBP).

Question 11

a) What is the significance of the three basic components of blood pressure?

b) Why should we pay attention to pulse pressure?

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b) _____

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Noninvasive blood pressure can be measured in many ways such as air filled cuff to temporarily occlude blood flow through the artery and use of a stethoscope to listen to the characteristic *korotkoff* sounds of the blood flow during cuff deflation to determine the SBP and DBP. This is known as the auscultatory method. The korotkoff sounds are divided into five phases as follows;

- **Phase 1** – starts with the sudden detection of a faint, clear, tapping/thumping sound that gradually in creases in intensity. This corresponds to SBP.
- **Phase 2** - phase 1 ends and phase 2 starts when the sounds

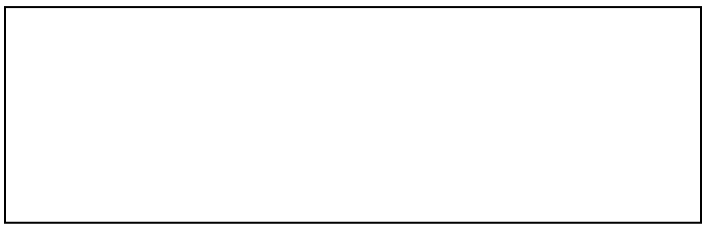
change to a loud “swishing” murmur.

- **Phase 3** – It starts when the sounds change to a loud, distinct, knocking type. However, these sounds are less intense than the ones heard in phase 1.
- **Phase 4** – starts when the sounds suddenly become muffled and have a faint murmur-like or “swishing” quality. This corresponds to DBP.
- **Phase 5** – starts when silence develops.

  NIBP when obtained by the auscultary method can not give a MAP due to several limitations such as hearing differences in individuals.

Question. 12

How is MAP calculated?



The other way to measure NIBP is the oscillometric method. This is the most common way used in the ICU setting. Oscilometric means any measurement of the oscillations caused by the arterial pulse as a result of the coupling of the occlusive cuff to the artery. Here, the artery is inflated until the artery is fully occluded and then the monitor takes the measurements as the cuff deflates. The oscillometric devices examine the pulsatile pressure generated by the arterial wall as it expands and contracts against the cuff with each cardiac cycle. An electrical signal is generated by the pressure transducer based on the distension of the artery. During the measurement process, the magnitude of the pulsatile

signal increases, reaches maximum amplitude, and then decreases. Most of the oscillometric devices use an algorithm whereby MAP is set to equal the point of maximum amplitude and then SBP and DBP are determined by a predetermined systolic and diastolic ratio.

Trouble shooting NIBP

To optimize the NIBP measurement the following things must be considered.

a) To optimize accuracy of the cuff pressure be it auscultatory or oscillometry,
   - Ensure that the cuff is the right size for the patient. Too small a cuff will give a falsely high reading and a too large cuff will give a falsely too low reading. The cuff should be 40% of the mid circumference of the arm.
   - The cuff bladder should be over the brachial artery, approximately 2.5 Cm above the antecubital space. Ensure that all air is removed from the cuff before it is applied. Wrap the cuff snuggly around the arm – if loosely applied, a falsely high measurement will be obtained.

b) The patient's arm must be placed in a relaxed position, approximately at the heart level. If the arm is below the heart level, a falsely low reading will be obtained. Conversely, a falsely high reading will be obtained if the arm is higher than the heart level.

c) If using the auscultatory method, palpate the radial pulse for initial measurement then inflate the cuff 30 mm Hg above the point when the pulse disappears to ensure accurate systolic pressure measurement.

d) In patients with "large cone" shaped arms, uneven pressure can give falsely high readings therefore, forearm

measurements can be tried although this may over estimate the value of systolic BP. Again, palpate the radial pulse before taking a reading.

e) Remember when using the oscillometric method that patient's excessive body movement as in seizures or shivering, arm movement, external cuff compression and irregular heart rhythms may decrease accuracy of the readings.

f) Take blood pressure in both arms at least once to detect any interim differences. The normal difference should be 5 – 10 mm Hg.

Question 13

Define pulsus paradoxus.

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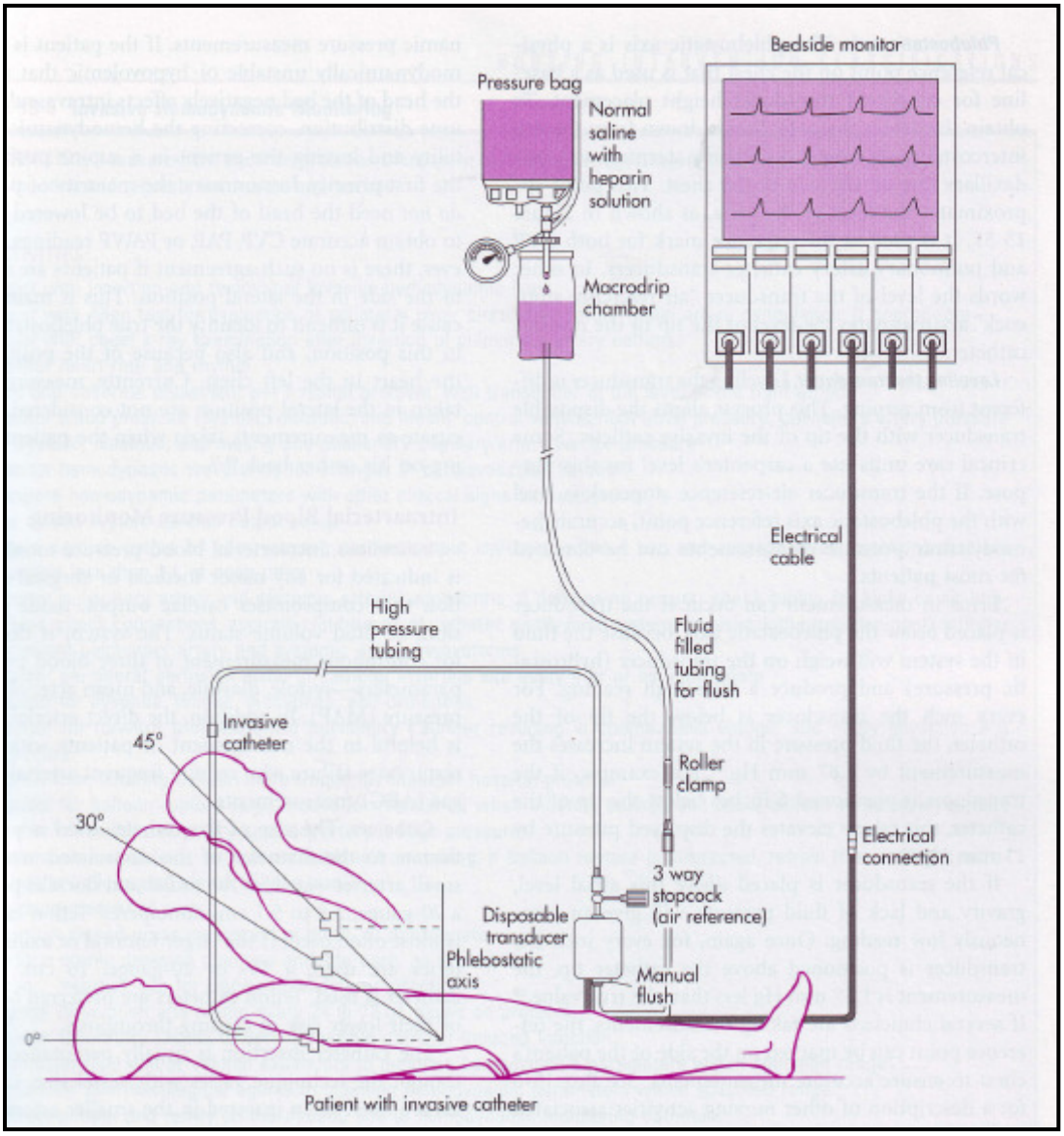
15 Invasive / Pressure Monitoring.

15.1 Overview

In invasive monitoring, haemodynamic pressures are transmitted from the intravascular space or cardiac chamber through the catheter and the fluid in the non compliant pressure tubing to the pressure transducer. A transducer is a device that converts one form of energy into another. In haemodynamic monitoring, the transducer senses changes in the fluid column generated by the pressures in the cardiac chambers or vessels being monitored. When pressure is applied to the diaphragm of the transducer, sensors are compressed, changing electrical flow to the amplifier

or the monitor. Then the monitor converts the electrical signal generated by the transducer to the pressure tracing and digital value. Generally, bedside monitoring systems are capable of displaying several digital readings whereas oscilloscopes display pressure waveforms simultaneously. Also, the monitors have the mechanism to set or adjust alarms, waveform size and zero the system.

The patency of the haemodynamic monitoring system is maintained by a continuous infusion of flush solution such as heparinised saline. The flush solution is placed in a pressure bag which is inflated to 300mmHg to maintain a constant pressure through the transducer and flush device. Hence a constant flow of 3mls/hour of fluid is maintained to prevent back flow of blood through the catheter thus maintaining system patency and accurate transmission of pressures. The system can also be flushed manually by using the fast flush device.



The four parts of haemodynamic monitoring namely, the invasive catheter, transducer, flush system and bedside monitor (illustration from Urden. L etal. (1998) Critical Care Nursing: Diagnosis And Management. (3th ed) St. Louis: Mosby p443).

15.2 Optimal Use of the Monitoring System.

With continuous monitoring of haemodynamic parameters, it is essential that the nurse at the bedside is able to troubleshoot effectively so that inaccurate measurements are identified and excluded. To the critically ill patient inappropriate treatment initiated by inaccurate measurements may be life threatening. We have just

reviewed some of the basic principles of haemodynamic monitoring, and we will now look at how these may affect the accuracy of the measurements obtained.

There are several mechanical and technical factors that can lead to distortion of haemodynamic waveforms and values.

These are:

- ❖ Air bubbles or blood in the tubing or transducer system
- ❖ Continuous pressure of less than 300mmHg on the flush solution bag
- ❖ Non compliant tubing such as soft distensible tubing and catheter (stiffness of catheter and tubing)
- ❖ Length and diameter of the tubing and catheter.

To understand how these factors affect the monitoring system we need to understand how the system works.

This is how it works:

The reaction of the fluid-filled tubing transmitting pressure signal from the patient depends on the **resonant frequency** of the system which is the frequency at which the oscillations have their maximum amplitude.

Basically, we need to construct a fluid-filled system whose resonant (or natural) frequency is very different from the frequency of the pressure waveform being transmitted; otherwise the result is an overshoot of the systolic pressures (accentuation), a lowered diastolic pressure (attenuation), and the presence of artefact on the pressure waveform. We can do this by increasing the resonant frequency through:

- *shortening the catheter and tubing to no longer than 100 cm*

The longer the tubing, the closer the resonant frequency of the fluid-filled system to that of the pressure waveform, causing the alterations mentioned above. Therefore the number of stopcocks and any other connections must be minimized as much as possible and luerlocks must be used to preserve the integrity of the system.

- *using stiff, non-compliant tubing*

  A soft, compliant tubing absorbs some of the energy of the pressure wave being transmitted, resulting in reduced amplitude of the pressure waveform.

- *using large diameter catheters (7 Fr or larger)*

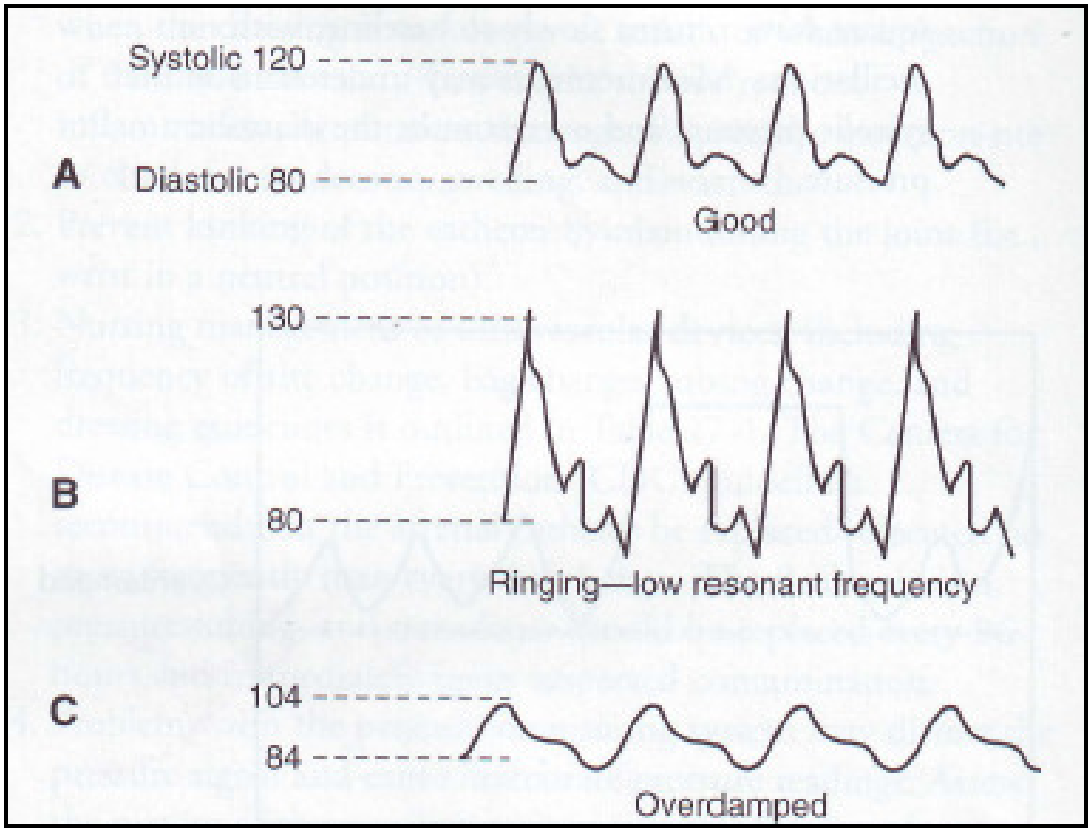
  In small diameter catheters there is an increase in the frictional resistance to movement, which results in some of the energy from the pressure wave being lost.

- *Eliminating air bubbles from the system and preventing clot formation, particularly at the site of stop cocks*.

Air bubbles and clots are compressible; therefore, as with soft tubing, some of the energy from the pressure wave is lost, resulting in a reduced amplitude of the pressure waveform. The amount of reduction is directly proportional to the size of the air bubble or clot. Ensuring that the flush bag is correctly inflated to 300 mm Hg, thereby delivering approximately 3ml of fluid per hour, will help minimise clot formation.

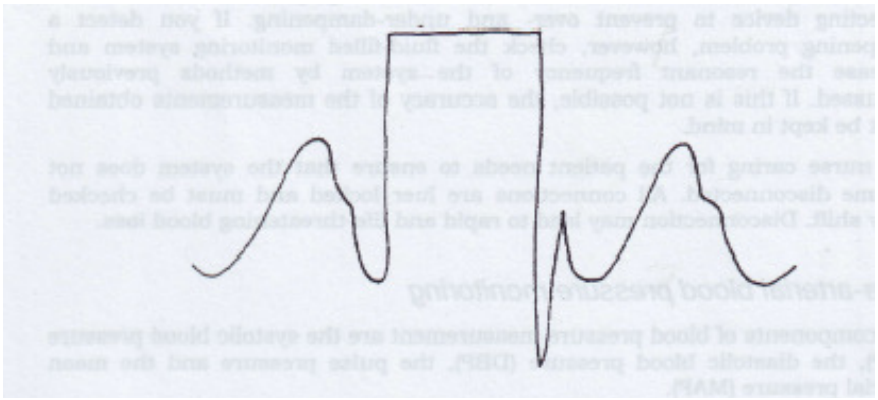
The other factor that can cause alterations to the measurements obtained is the **dampening coefficient.** This describes how quickly an oscillating system comes to rest. Under-dampening may be observed on the pressure waveform; it has a very narrow, high, peaked systolic curve and may have `ringing' or `fling' seen on the waveform.

Over-dampening of the system may appear as a very rounded curve with a low upstroke. Most of the time, however, it is difficult to assess an under- or over-dampened system.

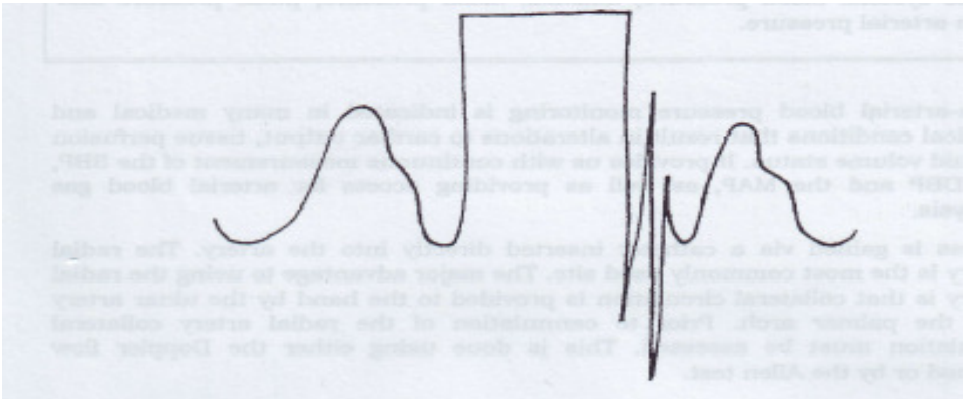


Normal and distorted systemic arterial waveforms (Illustration from Mims et al, Critical Care Skills 2nd Ed. P200)

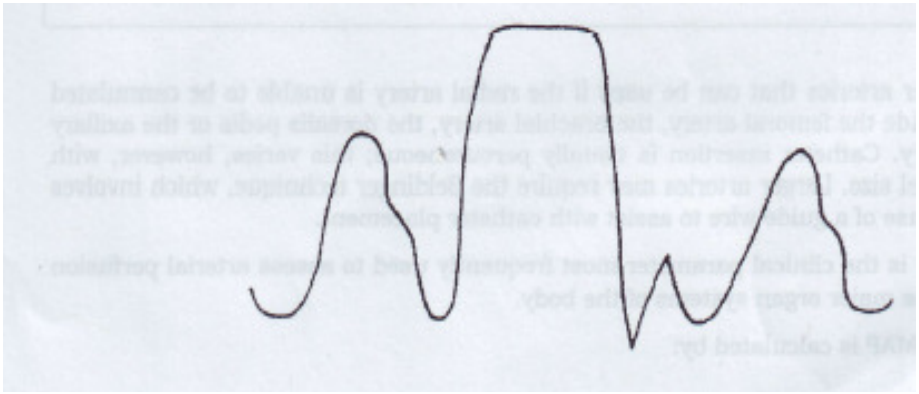
One way to assess the waveform is to test the **dynamic response (square–wave test)** of the system. This can be done at the bedside by using an inline fast-flush device. Activation of the fast-flush device should produce a square wave, followed by one or two oscillations, before returning to the pressure wave.



a) Normal dynamic response



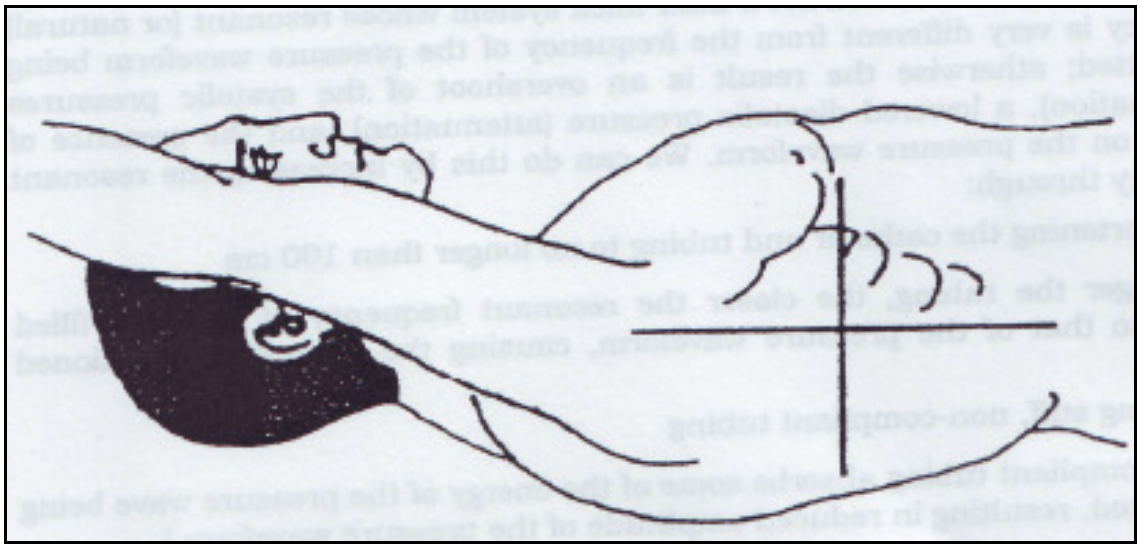
b) Under-dampened response



c) Over-dampened response

Square wave test (illustration from Hudak et al, 2005. Critical Care Nursing. A wholistic approach 8th edn. P 271)

Accuracy of the haemodynamic pressure readings are ensured by first levelling the transducer. There are many different methods utilised in levelling the transducer. One method is attaching the transducer to the insertion site. Another, the most widely recognised method, is levelling the transducer to the phlebostatic axis. This point is obtained by drawing a horizontal line along the fourth intercostal space and a vertical line from the mid-axillary line. The point where they meet is the phlebostatic axis. This point is used as a reference for consistent transducer height, as it approximates the level of the right atria when the patient is lying supine.



Levelling the transducer to the phlebostatic axis. Illustration from evaluation instruments for selection to critical nursing care certificate. Mediclinic limited training & development 1996.

The second factor ensuring accurate haemodynamic pressure readings is the calibration or zeroing of the system to atmospheric pressure. This involves opening the transducer to air as described in the procedure manual. Zeroeing must be done at the beginning of a shift, with any change in the bed or patient's position, elevation of the head of the bed, transport and every 8 hours.

Most of the modern transducers and monitoring systems have a built-in correcting device to prevent over- and under-dampening. If you detect a dampening problem, however, check the fluid-filled monitoring system and increase the resonant frequency of the system by methods previously discussed. If this is not possible, the accuracy of the measurements obtained must be kept in mind.

Question 14

Why should luerlocks be used in the system

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16 Intra-Arterial Blood Pressure Monitoring

16.1 Objectives

At the completion of the package the registered nurse will be able to:

1) List the reasons for insertion of arterial lines.
2) List the advantages and disadvantages of arterial lines.
3) List complications associated with insertion, maintenance and removal of arterial lines.
4) set up for insertion of an arterial line.
5) Implement routine nursing care of an arterial line.

Intra-arterial blood pressure monitoring is achieved through an intra arterial catheter connected to the pressure monitoring system. Thereby, allowing for continuous monitoring of the systemic blood pressure. It also provides vascular access for blood sampling by withdrawing blood from the stop cock in the system.

16.2 Arterial Line Insertion

The commonest sites for arterial line insertion are the radial and the femoral arteries. Alternative and infrequently used sites are the brachial, axillary and the dorsalis pedis arteries in adults as well as the temporal and umbilical arteries in neonates.

However, the following consideration should apply:

- ❖ Size of the artery in relation to the size of the catheter – the artery must be big enough to accommodate the catheter without occluding or significantly impeding on the flow
- ❖ Accessibility of the site – the site must be easy to access and free from body secretion contamination.
- ❖ There should be collateral flow of blood to the limb distal to the insertion site

As we can see from this, the radial artery fulfils these criteria. Hence, it is the most used. Furthermore, it is easy to palpate because it is a superficially located and it poses a risk from infection.

Question 15

Describe the Allens test**.**

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Question 16

What are the indications for insertion of an arterial line?

Question 17

Give 5 disadvantages / complications of arterial line insertion**.**

Question 18

Diagramatically illustrated below is a normal arterial waveform. Recapping the cardiac cycle, identify the marked areas and explain what they depict

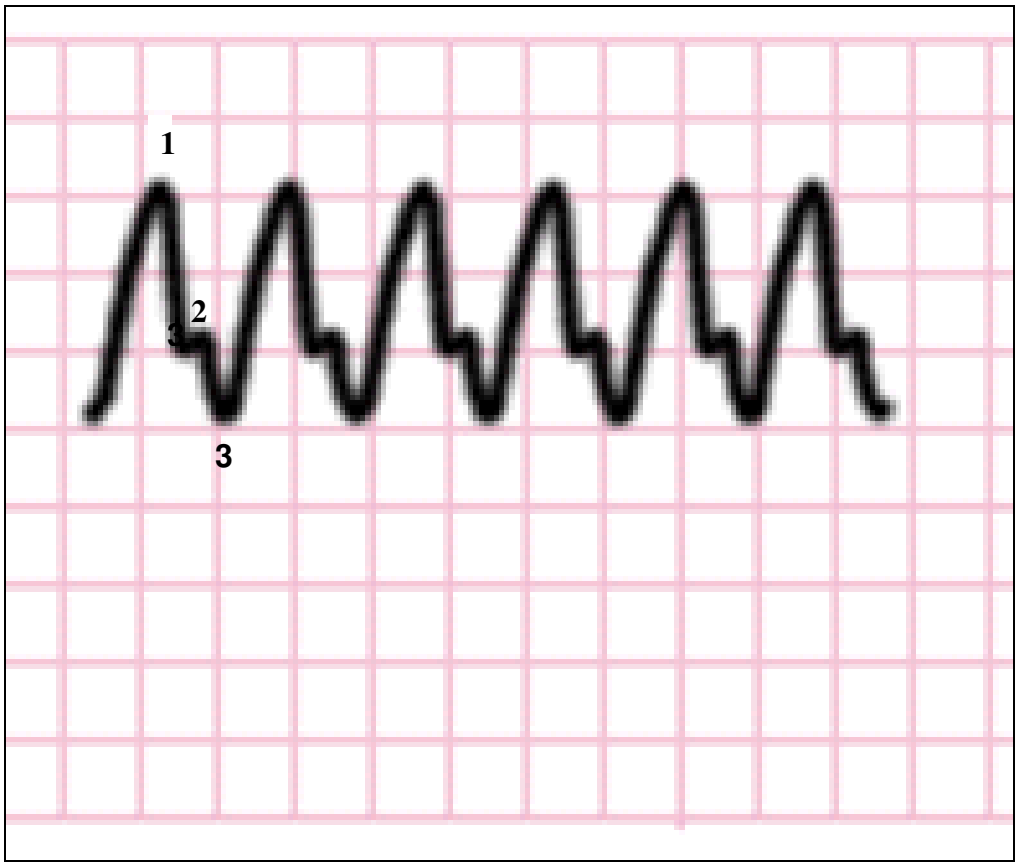


Illustration from classes.kumc.edu/son/nurs420/unit4/hemomon.html

1. _____
   _____
   _____
2. _____
   _____
   _____
3. _____
   _____

17 Nursing Considerations.

- Compare ABP with NIBP whenever the accuracy of the former is questionable. Bear in mind that there is a 5 – 10 mmHg difference between the two but they are correlated.

- Set high and low alarm limits 10 – 20 mmHg above the patient's typical BP

- Perform a nursing assessment whenever alarms are activated. This includes taking the BP, performing the square wave test, checking for kinks, disconnections in the system, insertion site for hemorrhage.

- Remove any unnecessary stopcocks and extension tubings.

- Ensure flush bag is not empty and the pressure bag is inflated to 300 mm Hg and the roller clamp is open.

- Ensure the pressure scale is appropriate for expected readings. If the top number on the pressure scale is too high, it will cause the wave form to appear overdamped.

- Position of the catheter affects the readings. The more peripherally the catheter is located (dorsalis pedis versus radial), the higher the systolic reading will be and the lower the diastolic reading will be.

- Arythmias may produce a beat to beat variation in the arterial tree which may lead to a low blood pressure averaged by the arterial tree.

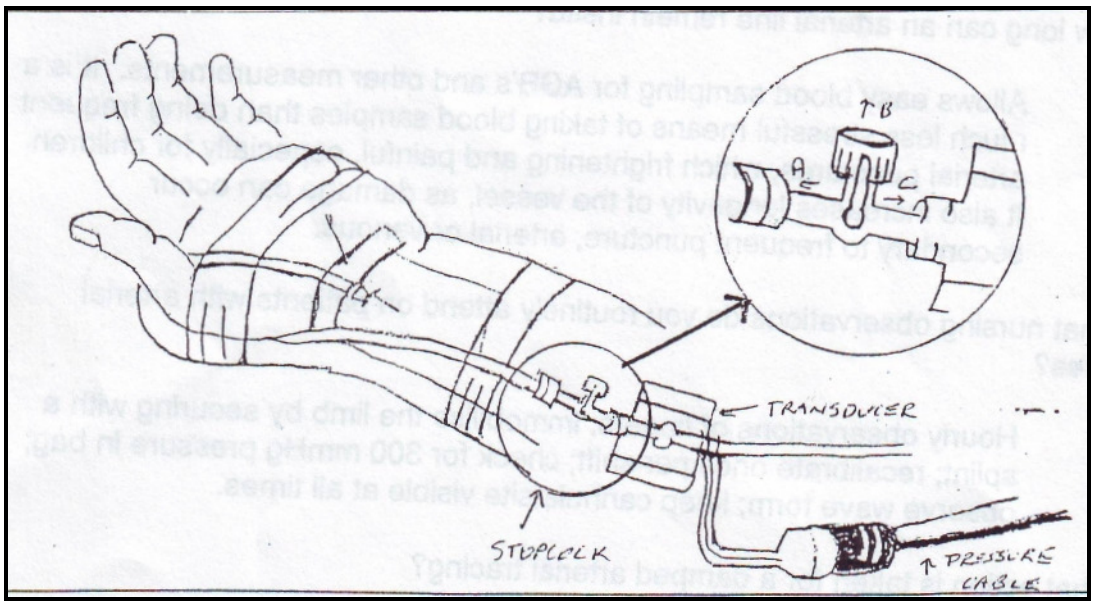
- If catheter patency is in question, aspirate blood and fluid from the

stopcock and then flush the system. Do not use a syringe to flush the catheter as this may damage the artery.

- **Never** inject IV solutions or medication into the arterial line

Question 19.

Look at the diagragm below and identify the direction the stopcock should be turned when zeroing the transducer. (Illustrated by Barbara Rolfe CNE John hunter ICU)



Describe the care of an arterial line with regard the following;

- The insertion site
- Limb observation
- How long the catheter should remain in situ
- Immobilizing the limb with the arterial line.
- Blood sampling

18 Central Venous Pressure Monitoring

18.1 Objectives

At the completion of the package the registered nurse will be able to: -

1) List the reasons for insertion of central venous catheters.
2) Set up for insertion of a central line and implement routine nursing care.
3) Implement nursing care for the patient with central venous catheter.
4) Be aware of nursing actions to minimise complications of central venous lines.

Central Venous Pressure (CVP) reflects the pressure in the right atrium or the vena cava. CVP gives information about intransvascular blood volume, right ventricular end-diastolic pressure and right ventricular function. To some extent, CVP indirectly reflects left ventricular end – diastolic volume and function in that the left and the right sides of the heart are linked by the pulmonary vascular bed. Normal CVP is 4-12mmHg. (Hudak etal 2005) CVP is affected by abnormal alterations in the volume status and ventricular function. Usually, alterations in volume status are responsible for abnormally high and low CVP measurements. CVP measurement on its own is meaningless, it should be used in conjunction with other parameters such as breath sounds, heart and respiratory rate, neck vein distension and urine output. The following are other pathologies responsible for alterations in CVP values.

- Decreased CVP more often than not reflects hypovolemia requiring fluid administration. Other factors that lower CVP are diuretic therapy, vasodilatation due to sepsis or vasodilating drugs because they both expand the vascular space while the blood volume

remains the same. Haemorrhage and dehydration are other factors that lower CVP.

- Increased CVP requires more scrutiny as there are a number of complex and interrelated factors that can cause increased CVP values. Right ventricular failure and mechanical ventilation are the commonest causes of increased CVP values. However, Intravascular volume overload (hypervolemia) rarely causes increased CVP values on it's own in which case, diuretics have to be used.

- Mechanical ventilation increases CVP values because it increases intrathoracic pressure which is transmitted to the pulmonary vasculature, the heart and the great vessels. Furthermore, increased intrathoracic pressure compresses the pulmonary vessels, leading to resistance to blood flow from the right side to the left side thus causing blood to back up in the right ventricle, right atrium and the vena cava. In extreme cases, this could result in right ventricular dysfunction.

- Right ventricular failure resulting from coronary artery disease or left ventricular failure causes increased CVP values because the right ventricle fails to pump blood through to the pulmonary vasculature resulting in increased pressure and volume in the right atrium. Left ventricular failure causes congestion of blood in the pulmonary vasculartur and impairs blood flow from the right ventricle leading to subsequent right ventricular dilation and failure.

- Other factors that increase CVP values are vasoconstriction, hypertension, and tension pneumothorax.

Question 20

Name 5 indications for insertion of a CVP line.

_____

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_____

18.2 Catheter Insertion.

The CVP catheter is long and fliable and it is inserted under sterile conditions. The sites of insertion are the subclavian, internal jugular, antecubital, or femoral vein and it is threaded into position in the vena cava close to the right atrium. If the catheter migrates into the right atrium, the haemodynamic waveform will be more pronounced and this should prompt the physician to withdraw the catheter a few centimetres until the fluctuations cease.

Insertion Sites for Central Venous Catheters

Site	Advantages	Disadvantages
Subclavian	Large vessel with high flow rate (decreases risk of thrombotic complications) Low infection rate Easy to dress and maintain sterile intact dressing Less restricting for patient	Lies close to the lung apex (pneumothorax risk) Close proximity to subclavian artery Difficult to control bleeding (noncompressible vessel-artery under clavicle)
jugular	access Short, straight path to superior vena cava (right side) Low rate of complications-, lower incidence of arterial laceration or pneumothorax than subclavian site Large vessel with high flow rate	Hard to maintain sterile, intact dressing Close proximity to carotid artery Highest infection rate of insertion sites Possible contamination by hair, oral secretions Problematic in patients with tracheotomies, cervical spine precautions
Femoral	Easy access Large vessel Advantageous during resuscitation No risk of pneumothorax	Decreased patient mobility Increased rate of thrombosis, phlebitis, and infection Can be contaminated by urine and feces Risk of femoral artery puncture Difficult to maintain sterile intact dressing Difficult to locate in obese patients
Brachial	No risk of pneumothorax or major hemorrhage Bleeding from site more easily controlled in patients with coagulopathies	Increased incidence of phlebitis, thrombosis Possible catheter tip movement related to arm movement by patient May be difficult to locate in obese or edematous patients

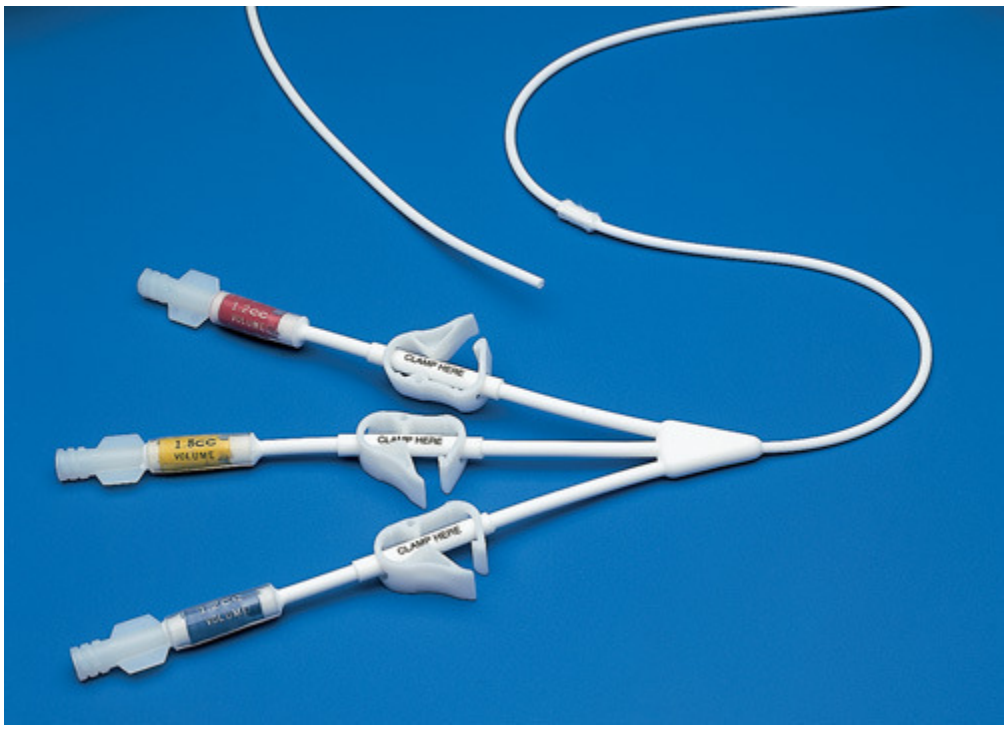
Data from Arrow International (1996). Arrow Multi-Lumen Central Venous Catheter nursing care guidelines. Reading, PA: Arrow International; Darovic, G., & Kumar, A. (2002). Monitoring central venous pressure. In G. Darovic (Ed.), Hemodynamic monitoring: Invasive and noninvasive clinical application (3" ed.). Philadelphia: W. B. Saunders; and Velmahos, G. (2002). Central venous catheterization. In W. Shoemaker, G. Velmahos, & D. Demetriades, Procedures and monitoring for the critically ill. Philadelphia: W B. Saunders.

There are two ways of measuring CVP, the water manometer and the haemodynamic monitoring technique. For the purpose of our unit, we will only discuss the latter as we do not use the former.

Activity

Refer to the procedure manual located at the nurses' desk and practice assembling the equipment required for insertion of a CVP line.

The arrow triple lumen is like a colour coded piggy tail for rapid identification. It shows the gauge and position - proximal (longest), medial and distal (shortest).



Adapted from Cook Medical Supplies @ www.cookmedical.com

Question 21

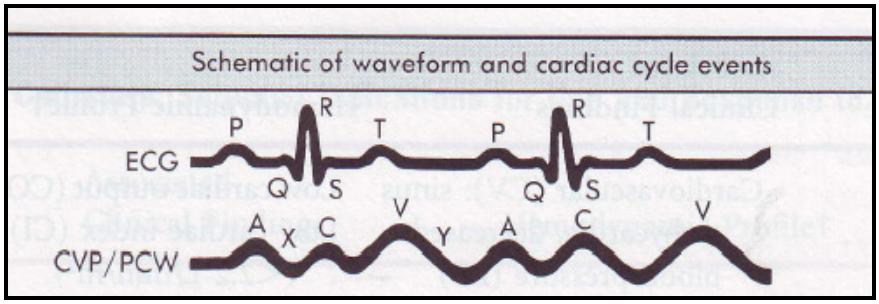
What is the recommended use for each of the lumena on the triple lumen

catheter?

As you may have discovered by now, the transducer system is just the same as the one for arterial line insertion except for the catheter and the two are connected to the monitor pretty much the same. However, their waveforms are different. The CVP waveform has three positive waves and two negative waves.

Question 22

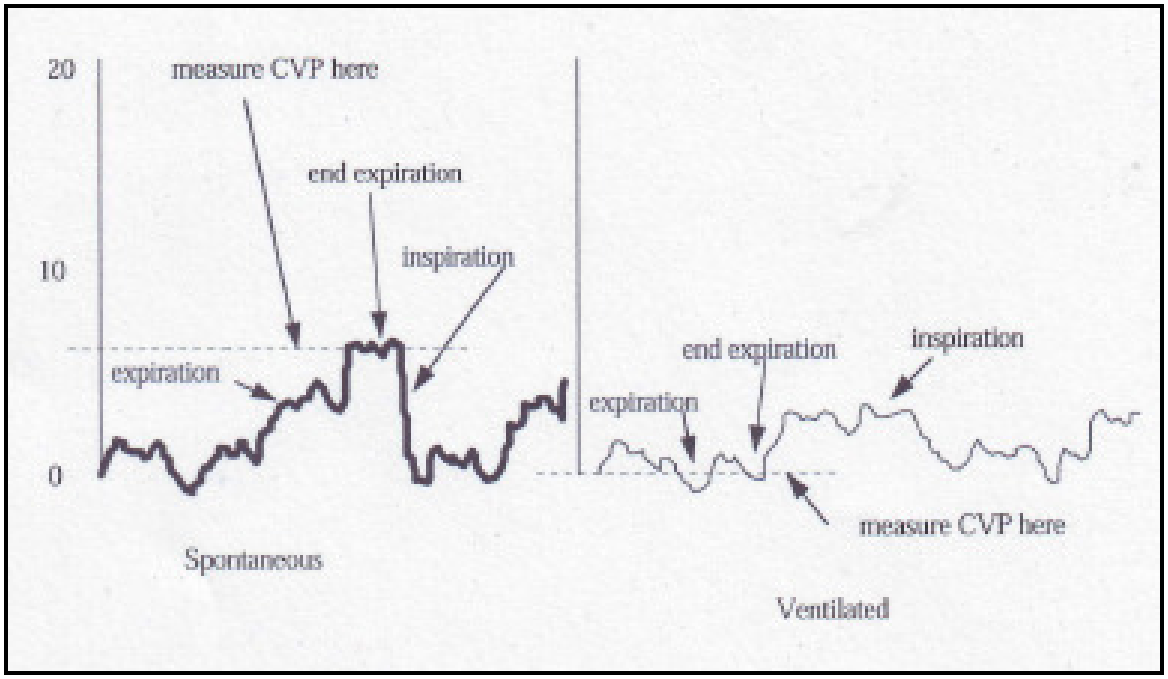
Once again refresh your memory of the cardiac cycle and explain what the a,c,x, v and y waves reflect in the diagram below.



Cardiac events that produce the CVP waveform with a, c, and v waves. (Illustration from Stacy K. M , Critical Care Nursing Diagnosis And Management 3rd Ed., p457)

Remember that because the central line lies within the thorax, central

venous pressures and waveforms are influenced by changes in intrathoracic pressure during respiration. These changes are small, but may be observed on the waveform. CVP decreases slightly with spontaneous inspiration, and increases slightly with forced exhalation as well as positive pressure mechanical inspiration. A patient on PEEP (positive end expiratory pressure) greater than 7.5cmH_2 may also have an elevated CVP reading.



Differences of Central Venous Pressure in Ventilated and non ventilated patient (Illustration from M Boyle and R Butcher, Haemodynamic Monitoring http://intensivecare.hsnet.nsw.gov.au/doc/Haemodynamic.PDF#search=haemodynamic monitoring, p44)

Activity

Find a chest X ray of a patient with a central venous line in situ, ask one of the CNs or doctors to show you where the tip of the central venous line is located . Let them explain to you the land marks and their significance .

Question 23

What changes in the CVP waveform can be seen in the following conditions?

ii. Atrial fibrillation

_____

_____

_____

iii. Tricuspid regargetation.

_____

_____

_____

iv. Tricuspid sternosis

_____

_____

_____

v. Cardiac tamponade

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_____

_____

Question 24

Name 5 complications of CVP.

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_____

For air embolism to occur, minimum of 10 – 20mls of air must enter the

system before the patient becomes symptomatic. Physiologically, foam is created in the right ventricle with each heart contraction resulting in loss of stroke volume due to air instead of blood in the ventricle resulting in a sudden decrease in cardiac output which may lead to cardiac arrest.

Question 25

What are the signs and symptoms of air emboli as a result of CVP disconnection? What nursing action can you take in this case?

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Question 26

Critically ill patients are at risk for catheter-related blood stream infections (CR-BSLs) and sepsis. How can this be minimised?

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Question 27

Short Essay

Mrs. Green is brought to following a motor vehicle accident. She is haemorrhaging her right leg where she sustained a fractured femur. Her pulse is rapid and thready 120bpm but her blood pressure is 110/ 90 mmHg (note the pulse pressure), she is cold and clammy to touch. Calculate the MAP for Mrs. Green and describe the compensatory mechanisms that are keeping Mrs. Green's blood within normal limits. What is Mrs. Green's long term management?

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