Westmead Intensive Care Unit

HAEMODYNAMIC MONITORING LEARNING PACKAGE

Ain 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 safe practice.

Objectives of the Package
1. Understand gross cardiac anatomy and how it relates to the cardiac cycle
2. Demonstrate knowledge of the principles of invasive pressure monitoring
3. Be able to demonstrate knowledge and skill in the management of arterial and central venous pressure lines

What to do with the Package
1. Attempt all questions
2. You will need to exceed 80% correct answers to pass the package, if this is not achieved you will need to repeat the package
3. Your package will reviewed by a CNE and then you will be provided with the package answers to allow further review

Useful Resources
- Textbooks are available in the CNE nook
- CIAP can be access on all the computers through the intranet site
- Policies, procedure, and guidelines may be useful to access

Question 1
Identify each of the numbered structures in the diagram.
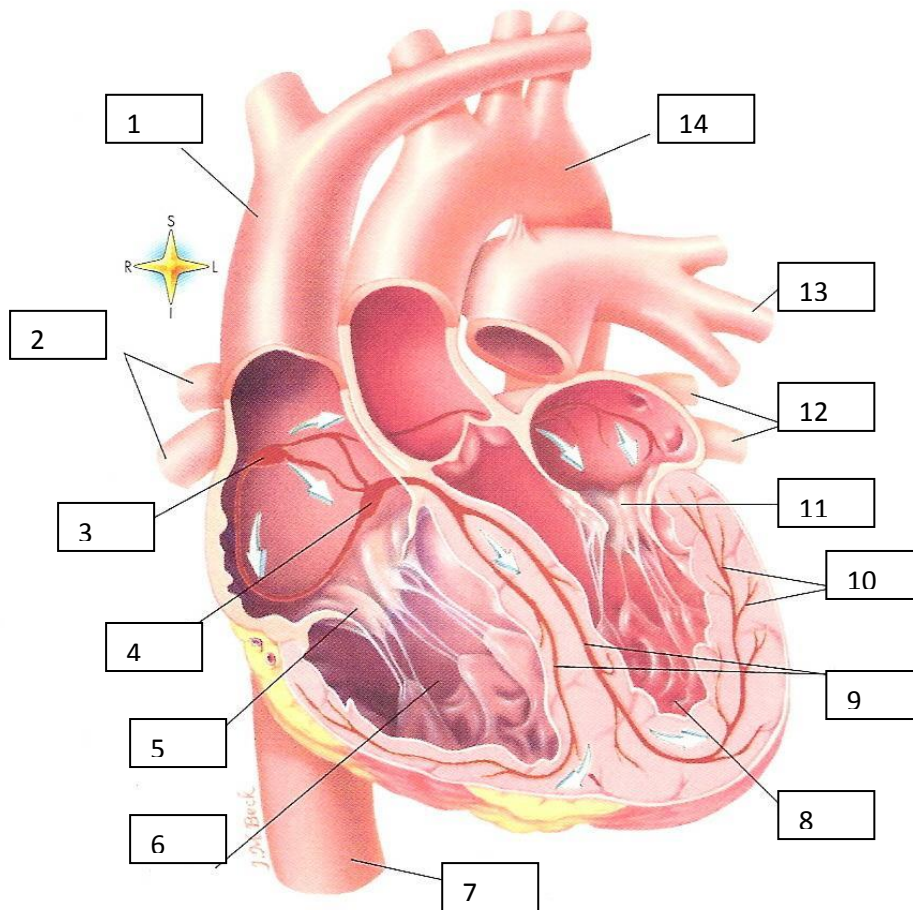
1.   10.
2.   11.
3.   12.
4.   13.
5.   14.
6.
7.
8.
9.

Urden: Thelan's Critical Care Nursing: Diagnosis and Management, 5th ed., Copyright © 2006 Mosby, An Imprint of Elsevier

CARDIAC CYCLE

Question 2

Look carefully at the diagram on the next page. It is a diagrammatic representation of the following:

a) systole and diastole of both the atria and ventricles related to time;
b) pressures in the aorta, atria and ventricles;
c) the ECG

Once you are familiar with the diagram complete the following sentences by circling the correct answer from the alternatives provided in brackets.

A) The average cardiac cycle is **(0.8, 0.6, 0.12)** seconds in duration. Approximately **(54, 60, or 75)** complete cardiac cycles or heartbeats occur each minute.

B) As the P waves occur, nerve impulses spread across the **(ventricles, atria, septum)**. During this initial phase, the duration of which is **(0.1,0.3, 0.8)** seconds, the atria begin their contraction. During this phase they are said to be in **(systole, diastole).** As a result, pressure within the Atria **(increases slightly, decreases slightly, is unchanged),** as shown at point a in the diagram.

C) The QRS complex signifies **(contraction, repolarisation, depolarization)** of the ventricles. Note that although the QRS complex itself only occurs over a brief period, the ventricular contraction that follows has duration of approximately **(0.3, 0.5, 0.7)** seconds.

D) Trace a pencil lightly along the curve that shows changes in pressure within the ventricles during ventricular systole. Note that intraventricular pressure shows the progressive increase that by point **(a, b, c, d, e)** it has surpassed intra-atrial pressure which results in the AV valves being forced **(open or closed)**

E) A brief time later ventricular pressure becomes so great that it surpasses the pressure within the great arteries. This pressure forces blood against the undersurface of the **(tricuspid, semilunar, mitral)** valves, causing them to **(open or close)** at point *c.*

F) During ventricular systole blood is ejected from the heart into the great vessels. In the aorta this creates a systolic blood pressure of approximately **(15, 80, 120)** mmHg.

G) During ventricular systole blood is ejected from the heart into the great vessels. In the aorta this creates a systolic blood pressure of approximately **(15, 80, 120)** mmHg.

H)	Following ventricular depolarization and the subsequent **(contraction, relaxation)** of the myocardium, repolarisation of the muscle occurs which is represented by the **(P, T, R)** wave. This causes the ventricles to enter **(systole or diastole)** which results in a drop in ventricular pressure.

I)	When intraventricular pressure drops below intra-atrial pressure, the pressure exerted by the blood in the atria forces the AV valves to **(open, close)**, which occurs at point **(a, b, c, d, e)**

J)	Note the AV valves now remain open until point *b*, well into the next cardiac cycle. This ensures that the ventricles are adequately filled before their next contraction. Notice also that the only period during which the A.V. valves are closed corresponds with ventricular **(relaxation, contraction).**

Seconds-- 0.1/---0.2/---0.3/---0.4/---0.5/---0.6/---0.7/---0.8//
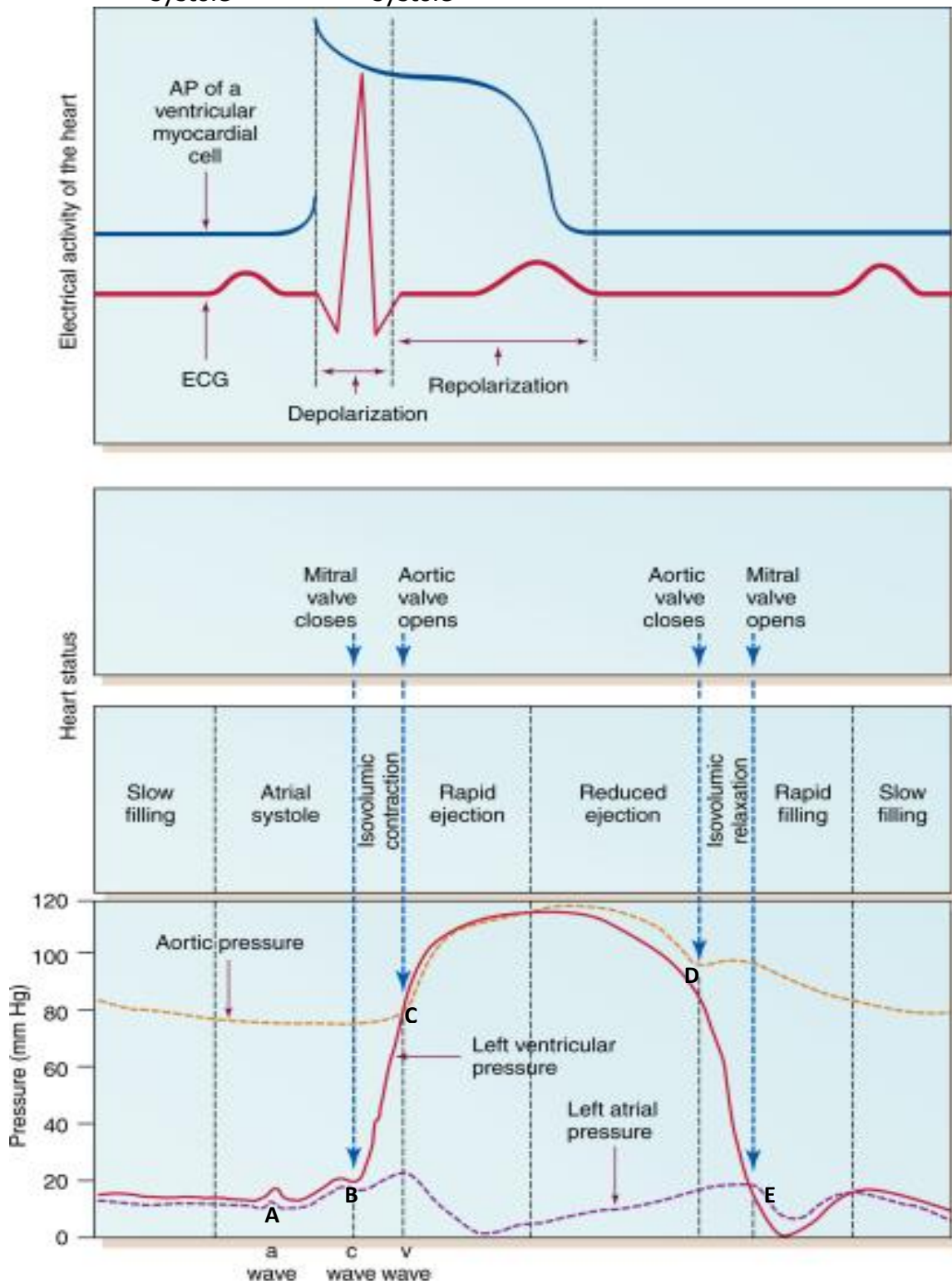< Atrial > < Ventricular > < Atrial & ventricular diastole >
systole systole

Electrical activity of the heart

AP of a
ventricular
myocardial
cell

ECG

Depolarization

Repolarization

Heart status

Mitral
valve
closes

Aortic
valve
opens

Aortic
valve
closes

Mitral
valve
opens

Slow
filling

Atrial
systole

Isovolumic
contraction

Rapid
ejection

Reduced
ejection

Isovolumic
relaxation

Rapid
filling

Slow
filling

120

Aortic pressure

100

80

C

60

Left ventricular
pressure

40

Left atrial
pressure

20

B

D

E

0

A

Pressure (mm Hg)

a
wave

c
wave

v
wave

Fig. 15-26 The cardiac cycle.

Urden: Thelan's Critical Care Nursing: Diagnosis and Management, 5th ed.,

Types of Myocardial cells

1. Working myocardial cells make up the bulk of the wall of the atria and ventricle responsible for contraction
2. Nodal cells specialized pacemaker function – clustered around SA & AV node
3. Purkinje cells rapid conduction cells – found in conduction pathways

Myocardial Contraction

The myofibril is composed of repeating functional units called sarcomeres. Each sarcomere is made up of two protein filaments – Actin and Myosin. The proteins troponin and tropomysin are located at intervals on the actin filament (or thin filament) the actin and myosin filaments are arranged in an overlapping pattern.

Contraction of cardiac muscle occurs as a result of the shortening of myofibrils. This shortening is the result of the actin and myosin filaments of the sarcomere pulling over each other. This is called the **Crossbridge theory of muscle contraction.** Crossbridges are formed between the actin and myosin filaments by the globular head of thick myosin filament (see following illustration)

The globular heads are the sites for the breakdown of ATP and thus energy production. This energy is used to produce contraction.

The globular head of the myosin filament contains actin binding sites that can form cross bridges between the actin and myosin. The cross bridge is capable of binding, flexing, releasing, and binding again. By doing this the thin actin filaments are pulled towards the centre of the sarcomere thus shortening the myofibril.
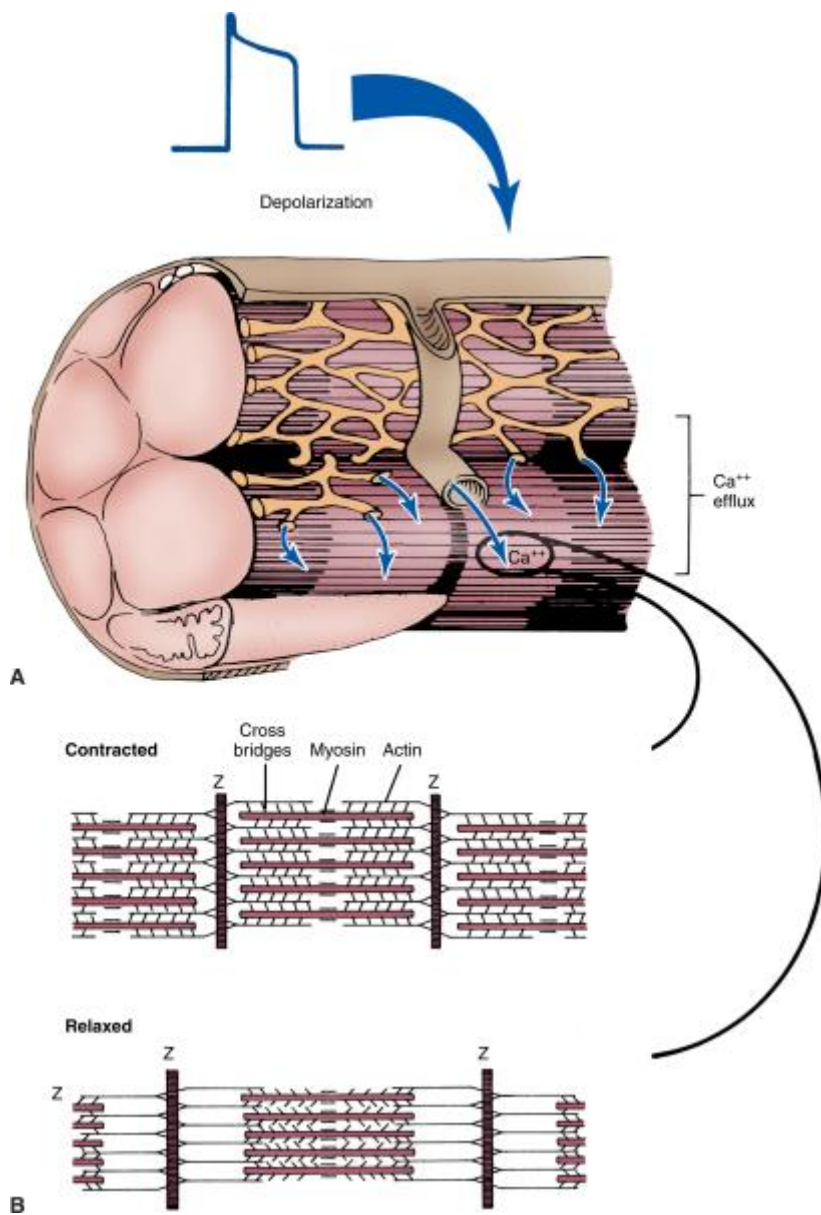
Fig. 15-22 A, Depolarization of a myocardial cell causes release of calcium from the sarcoplasmic reticulum and the transverse tubules. **B,** Calcium release allows for the cross-bridges on the myosin filaments to attach to the actin filaments to effect cell contraction. *(From Quaal S: Comprehensive intraaortic balloon pumping, ed 2, St Louis, 1993, Mosby.)*
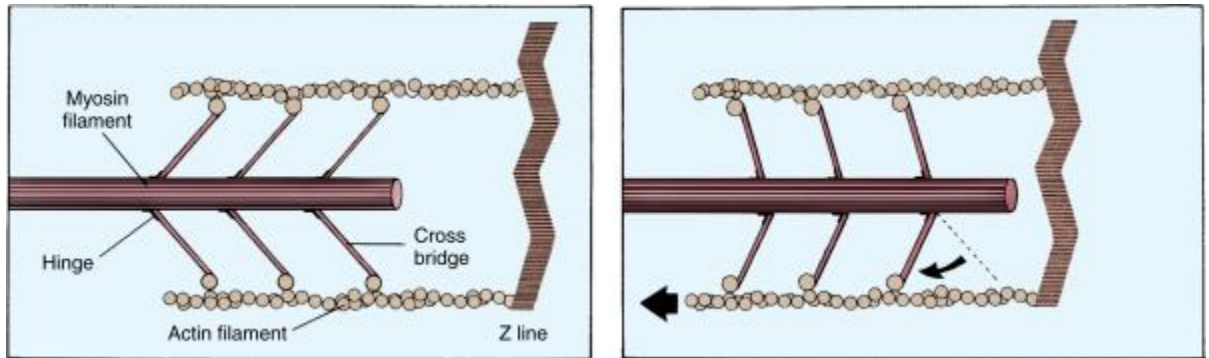
Fig. 15-21 Actin and myosin filaments and cross-bridges responsible for cell contraction.

Question 3
Briefly describe – Preload, Contractility, Afterload.

Question 4
Describe the Frank-Starling law in relation to the property of the ventricle.

Question 5
Which haemodynamic measurement gives an indication of Right Ventricular
End Diastolic Volume?

Question 6
Which haemodynamic measurement gives an indication of Left Ventricular
End Diastolic Volume?

The end result of the cardiac cycle – the excitation contraction coupling – is
the generation of cardiac output – delivery of oxygen to the tissues of the
body. **Cardiac output** is the product of **Stroke Volume** and **Heart Rate.**

Question 7
Describe the regulation of heart rate.

Question 8
Complete the following table

Region Affected	Sympathetic effects	Parasympathetic effects
SA Node		
AV node		
Atrial Muscle		
Ventricular muscle		

Question 9

What are the factors that affect venous return? Draw a flow chart and show how an increased venous return can result in an increased CO.

Question 10

Illustrate diagrammatically the control of cardiac output and blood pressure.

Question 11

Define tissue oxygen extraction

PRESSURE MONITORING – GENERAL PRINCIPLES

This section will present some basic concepts that help in the understanding of the monitoring systems used in ICU as well as providing a rationale for basic clinical trouble shooting.
 Intravascular pressures can be measured by means of a fluid column or electronic transducer. In this unit an electronic transducer is used to measure arterial and venous pressures.

The intra-arterial pressure may vary from anatomical point to anatomical point as a result of;

> 1. Hydrostatic pressure variations
> 2. Interaction of the arterial pressure wave and the arterial tree.

The venous pressure will vary from point to point primarily as a result of hydrostatic pressure variations.

Hydrostatic pressure is produced by a column of fluid and is the result of gravity.

The arterial pressure is a dynamic pressure that has volume displacement and energy wave components. The arterial pressure wave is a result of the pressure and volume changes produced by the cardiac cycle. The pressure wave interacts with the arterial tree (a system of decreasing diameter and branching tubes) such that the pressure measured at different points varies. Generally the systolic pressure rises and the diastolic pressure falls the more distal from the heart. The mean pressure does not tend to change (see illustration next page)

You will have noticed that the arterial trace is a complex wave. This complex wave is made up of a number of components (frequencies) called Harmonics. The fundamental frequency is the heart rate. (see the following illustration). The overall balance of frequency components produces the final picture or
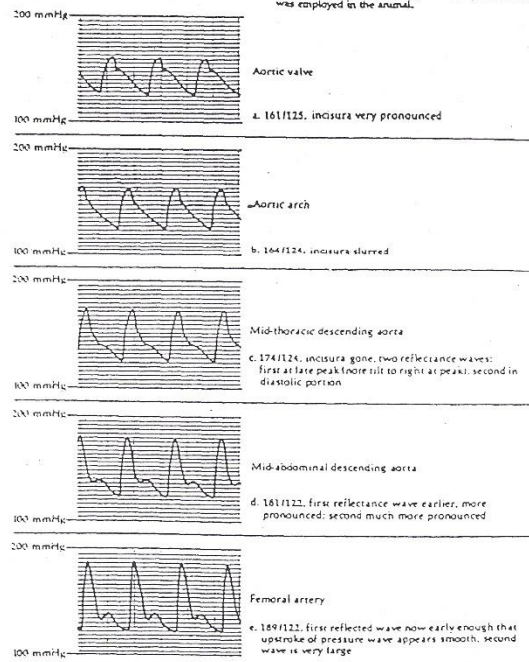
waveform. The frequency components that make up the arterial wave are determined by the heart rate, the strength of contraction, and the interaction of the wave with the arterial tree.

The electronic transducer is a device designed to respond to the frequency components that make up the arterial pressure wave. However, the transducer communicates with the arterial tree by way of an intravascular device (cannula), three way taps, and fluid filled connecting tubing. The addition of these components reduces the monitoring systems ability to respond to all the frequency components necessary to reproduce a clinically accurate pressure measurement. Frequency components of the arterial signal may be either lost or exaggerated (amplified) giving rise to an inaccurate measurement.

— A Fourier analysis of a pulmonary arterial pressure tracing. The bottom curve represents the addition of the various sinusoidal wave components.
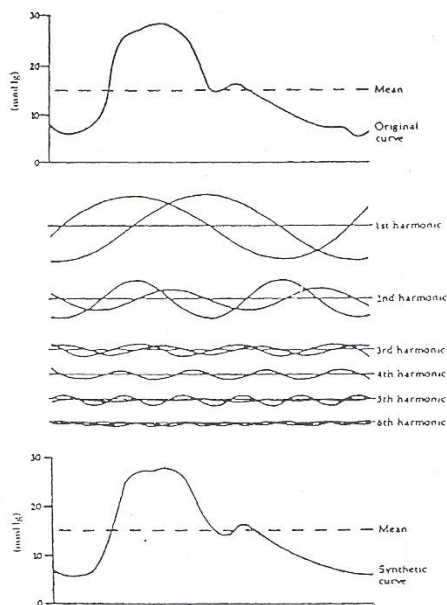
— Series of tracings obtained by gradually pulling a catheter back from the aortic valve to the femoral artery of a dog. A high fidelity catheter-tip manometer was employed in the animal.

Aortic valve
a. 161/125, incisura very pronounced

Aortic arch
b. 164/124, incisura slurred

Mid-thoracic descending aorta
c. 174/124, incisura gone, two reflectance waves: first at late peak (more tilt to right at peak), second in diastolic portion

Mid-abdominal descending aorta
d. 161/122, first reflectance wave earlier, more pronounced; second much more pronounced

Femoral artery
e. 189/122, first reflected wave now early enough that upstroke of pressure wave appears smooth, second wave is very large

Aortic valve

Aortic arch

Mid-thoracic descending aorta

Frequency Response and Damping

For accurate pressure measurement to occur all the significant pressure "information" must be communicated from the arterial intravascular site to the monitor that reads and displays the information. The transducer translates the message into information the monitor understands. The transducer is able to do this by being connected to the arterial tree via a fluid filled system. This fluid filled system and transducer is caused to vibrate in response to the arterial signal. If it is to convey an accurate message it must vibrate in exactly the same way as the original signal.

Any vibrating or oscillating system displays certain characteristics.

1. Natural frequency
2. Damping

1. The natural frequency of an oscillating system is that frequency that it oscillates at when set in motion. If an oscillating system is forced to oscillate at a frequency near or at its natural frequency it's amplitude of oscillation increases greatly i.e. the input signal will be amplified.

2. In any system there will be a loss of energy such that the second oscillation will have smaller amplitude than the first.

To use a system for monitoring that has little or no damping means that the system must have a high natural frequency (or frequency response), compared to the range of frequencies being measured, if there is to be no overshoot. If this is not the case then a monitoring system can be used for measuring a pressure wave with frequency components near the systems natural frequency only if the system is appropriately damped.

The damping also effects how well the different frequency components stay together (called phase different). As damping increases the phase difference of the frequency components increases.

Both the issues of resonance or overshoot and phase difference are dealt with by optimally damping the system. Optimal damping is about 64% of critical damping, or that damping results in no overshoot at all. This amount of damping results in an overshoot of about 6% at the system's natural frequency.

Practical Considerations.

1. The performance of the transducer monitoring system is maximized by;
   (a) Using short non-compliant manometer tubing
   (b) Avoid tubing that is too thin – this increases resistance, and the mass of the system which increases damping
   (c) Prevent kinks and clotting in the monitoring line
   (d) Ensure the system is free of air bubbles
2. Assess the dynamic response of the monitoring system. This can be done by the snap test. The damping of the system can either be adjusted at the monitor or mechanically (see unit protocol and practice)
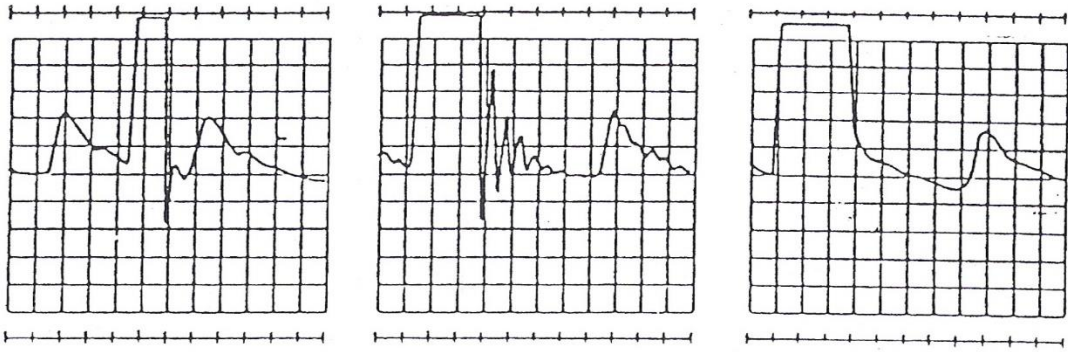
OPTIMALLY DAMPED SYSTEM	UNDERDAMPED SYSTEM	OVERDAMPED SYSTEM
An optimally damped system can be verified by observing the pressure trace on the monitor following a fast flush, square wave test.	(OVERSHOOT) This is the typical monitoring system. It is free of air bubbles & compliant components, but it has such a low natural frequency & damping coefficient that the pulse pressure is exaggerated. The oscillating effect can be seen following	When the fast flush square wave returns slowly to the baseline, and the patient trace resumes without any hint of an oscillation, the system is overdamped. The most likely cause is air trapped in the system, or a compliant component.

(Illustration taken from Spacelabs Biomedical Measurement Series)

3.   Check transducer-monitoring system against another measure. E.g. cuff pressure on the same. Note that the cuff pressure will be generally about 10% less than the direct pressure.

4.   Static Calibration. The transducer can be tested against mercury column to see that the reading it gives corresponds to a referent mercury column value. Adjustments can be made in the monitor to adjust for pressure discrepancies (see unit protocol and practice)

5.   The pressure monitoring system uses atmospheric pressure as its reference pressure. I.e. the system is zeroed at atmospheric pressure.

6.   Hydrostatic errors are minimized by using an anatomical reference point at which the pressure is relatively independent of body position. This point is the area of the tricuspid valve. A plebostatic axis can be determined by either the midaxillary method or the angle of Louie method (refer to unit practice)

7.   The mean pressure tends to be stable despite excessive damping or overshoot.
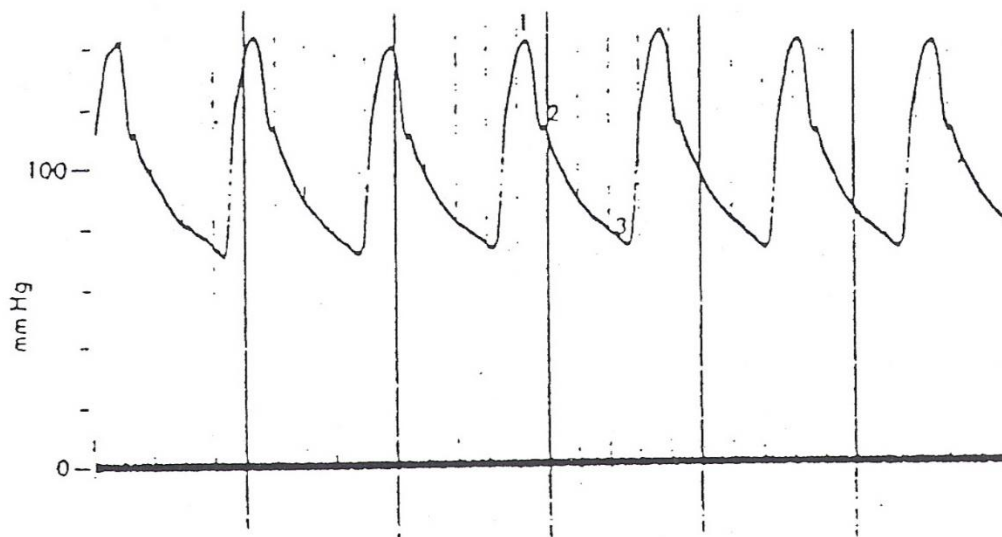
Arterial Lines

Question 12
What are the advantages of having an arterial line sited in a critically ill patient?

Question 13
Identify any risks and/or complications associated with the insertion of an arterial catheter?
Clinical Implications

Illustrated below is the characteristic waveform of an arterial pressure trace.

. Normal arterial pressure tracing. 1, Systole: 2, dicrotic notch: 3, diastole. (From Daily, E.K., and Schroeder, J.S.: Hemodynamic waveforms: exercises in identification and analysis, St. Louis, 1983, The C.V. Mosby Co.)

The slope (the rate of rise) of the initial pressure upstroke is an indication of the strength of contraction. The second positive "bump" occurring on the downstroke side is called the dicrotic notch when the pressure trace is from a measurement near the aortic root. The dicrotic notch is the result of the pressure change associated with aortic valve closure. In a radial artery trace the second pressure rise is not called the dicrotic notch but is called the incisure and represents the pressure rise caused by the rebound of the pressure wave of the periphery. If the circulation is "shut down" the incisure occurs higher up the pressure downstroke – if peripherally dilated the incisure occurs further down or may appear as a second smaller pressure wave. This latter situation can result in the pulse oximeter counting this pressure rise as

another heart beat and thus displaying the heart rate as being twice the ECG determined heart rate.

Question 14
What effects will the following cause on an arterial waveform? Provide a rationale for your answers.
An air bubble in the line between the cannula and the transducer / Blood or air bubble in the transducer / A deflated pressure bag.

Question 15
What observations should be maintained whilst an arterial line is insitu?

Central Venous Pressure Lines

Morphology and ECG Correlation of CVP or Right Atrial Waveform

Notes from Daily EK., Schroeder JS. Techniques in Bedside Haemodynamic Monitoring. Fourth ed. 1989. C.v. Mosby Co.

The right atrial or CVP waveform usually consists of 3 positive waves, the "a", "c", and "v" waves. The a wave is followed by the "x" descent whilst the c and v waves are followed by the "x" and "y" descents respectively. (see illustration). The positive deflections represent;

A wave - represents atrial systole – the x descent follows this wave and results from atrial relaxation.

C wave – results from the closure of the tricuspid valve (it may or may not be evident) – the x decent follows this wave

V wave – results from right atrial filling (same time as right ventricular systole) – the descent follows this wave and is caused by the opening of the tricuspid valve and the emptying of right atrial blood into the ventricle.

Note: when correlating pressure waves to ECG waves you must allow the difference in transmission speed. The pressure wave is a mechanical signal and is transmitted more slowly than the electrical signal. Therefore when looking at recordings of both waveform taken simultaneously the pressure wave will occur after its corresponding electrical wave.

The a wave corresponds with atrial depolarization. It appears after the P wave. The c wave reflects closure of the tricuspid valve – corresponds to the RST junction of the ECG – follows a wave by same time as PR interval. The v wave succeeds ventricular systole – anytime in TP interval.

B

Right atrium
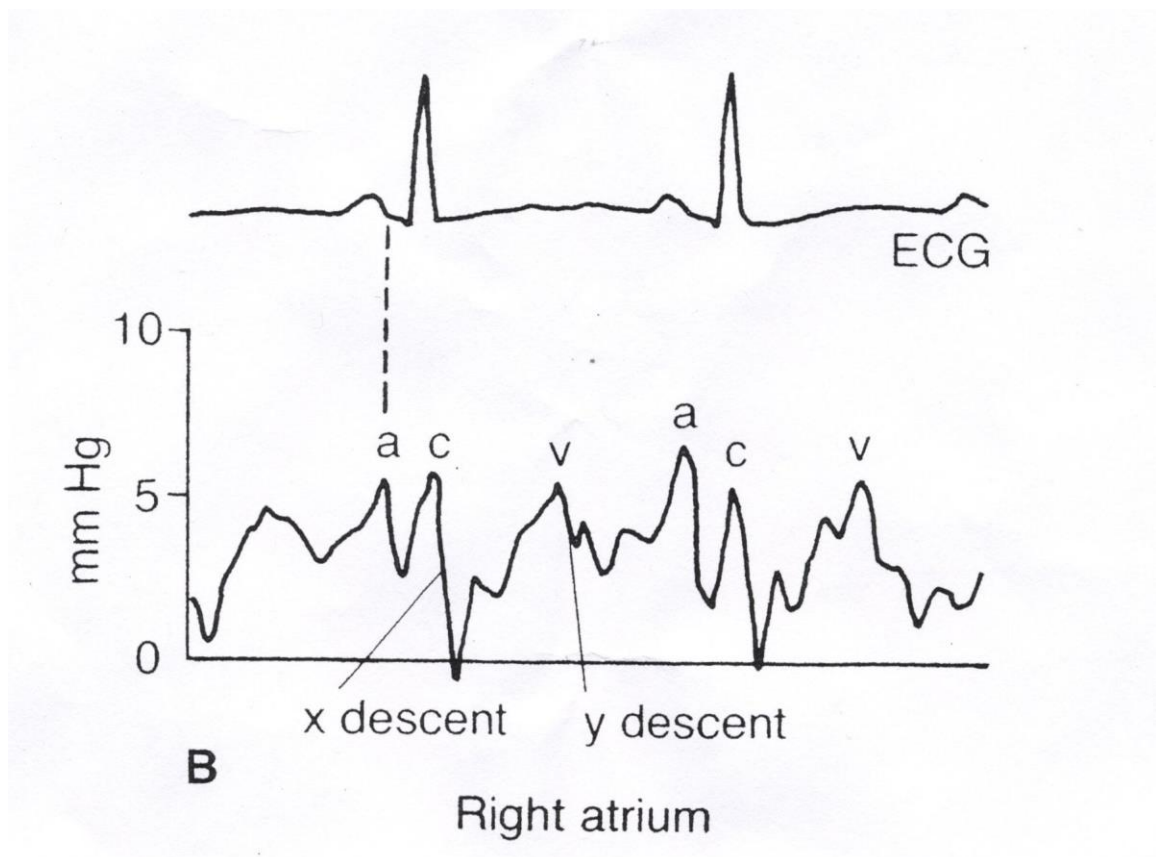
Clinical Implications

Central Venous Pressure Reflects right ventricular end diastolic pressure (RVEDP) which reflects right ventricular end diastolic volume (RVEDV). Normal 4 – 12 mmHg. The actual number measured as a CVP is unimportant by itself. CVP like all measured parameters need to be interpreted with other clinical data. For instance a patient may have a CVP of 5 which is considered "normal", however if this same patient is cool around the peripheries and has a poor urine output then this normal CVP of 5 is inadequate fro this patient. If this same patient is given a bolus of 500mls of colloid in order to increase the CVP to 8 and in doing so the patient becomes peripherally warm and has a good urine output, then this measured CVP of 8 is adequate for this patient.

Question 16
What is the normal range for CVP measurement?

Question 17
List three clinical conditions where the management of a critically ill patient might be guided by CVP measurement.

Question 18
List three clinical conditions that will decrease a patients CVP below accepted limits. Briefly discuss the rationale for your answer.

Question 19
What change would you expect to see in the CVP waveform as a result of
   a) Tricuspid Valve incompetence
   b) Tricuspid Valve stenosis

Question 20
List three clinical conditions that will increase a patients CVP above accepted limits. Briefly discuss the rationale for your answer.

Question 21
You are nursing a patient who has a long history of emphysema and has recently been diagnosed with a bleeding disorder (coagulopathy). Discuss why insertion of a central venous catheter (CVC) in the subclavian site would be contraindicated in this patient.

Question 22
How long should a central venous line remain insitu

Question 23
What landmarks on the chest x-ray are used to determine the correct placement of the CVC for CVP measurement?

Generally in ICU we use QUAD lumen CVC lines, and we dedicate lumens:

   Brown (distal lumen, 16Ga) – CVP measures, maintenance fluids, sedation, medications.

   Grey (medial 1. 14Ga) – Resuscitation fluids, any incompatible drugs, etc

   Blue (medial 2. 18Ga) – Inotropes

   White (proximal, 18Ga) – TPN/Actrapid

Question 24

What changes in the following parameters are typically seen in the following shocked states.

	Cardiogenic	Septic	Hypovolaemic
Systemic BP			
Heart rate			
Cardiac Output			
PAWP			
SVR			

CASE STUDY

68 year old gentleman presents to ED with shortness of breath, central chest pain.

Past history
Smoker, HT, hypercholesterolemia, .

Diagnosis

Acute M.I. – Transferred to cardiac cath lab for stenting of coronary vessels. Transferred to ICU in cardiogenic shock.

ICU
Patient intubated and fully ventilated. Arterial line and a Pulmonary artery catheter insitu.

On assessment
Patient pale, peripherally cold and shut down.
BP: 85/48, MAP: 57
HR:120 Sinus Tachycardia with multiple focal ectopics
CVP: 20
C.O. 3.8, SVR: 2880. PAWP: 24
Temp: 36.2
U.O. 10 – 20mls/hr

Question
   a) What would be your initial priority in the treatment of this patient? Give rationale.
   b) What are the possibilities for treatment to increase the Cardiac output and decrease afterload? (drugs or other therapies)