Additional information to accompany presentations

PACU Learning and Teaching Resource
CIRCULATION : BASIC SCIENCES

Presentation : Basic sciences

Introduction : The Circulatory System

The circulatory system is a network of blood, blood vessels, and the heart. It delivers oxygen and nutrients to body tissues. It transports waste products from metabolism, principally carbon dioxide to the lung for exhalation.

The heart and circulation : an overview

- the heart lies in the mediastinum in close proximity to the lungs
- it is about the size of an adult fist
- it is made up of specialized muscle tissue which allows it to pump blood within the circulatory system
- it is divided into 4 chambers : the right atria and ventricle and the left atria and ventricle. The atrium ‘receive blood into the heart. The ventricles pump blood out of the heart.
- Deoxygenated blood from the body tissues returns to the right atria and is transferred to the right ventricle. It is pumped from the right ventricle to the lungs via the **pulmonary circulation**. In the lungs the blood gives up carbon dioxide and receives oxygen. From here it returns to the left atrium via the pulmonary veins. It passes into the left ventricle and from here oxygenated blood is delivered to the aorta and the arterial circulation for delivery to the body tissues via the **systemic circulation**.
- **Coronary artery circulation** delivers blood to the heart muscle

Role of cardio-vascular system in metabolism

- Metabolism takes place in all body cells when oxygen and nutrients [glucose] burn to form heat and energy
- The energy created enables all body activities [hormonal – electrical]
- Without oxidative metabolism body cells malfunction and die

- The pulmonary circulation transports deoxygenated blood to the lungs where external respiration takes place [oxygen and carbon dioxide transfer across the alveolar-capillary membrane].
- The systemic circulation [arterial] delivers oxygenated blood to the body tissue cells where oxygen and nutrients diffuse into the cells and waste products back into the venous capillary end [internal respiration]. The venous circulation delivers blood back to the right heart.

Circulatory system – the 3rd priority in ABC

- If the airway is not clear, oxygen and carbon dioxide would not be transferred between atmosphere and lungs. If the lung function is impaired oxygen and carbon dioxide cannot be transferred from the alveoli into the circulation. Airway and breathing are therefore the first and second priorities in ABC. If the circulation is impaired oxygen & carbon dioxide cannot be delivered to and removed from tissues. Aerobic metabolism cannot take place. Body cells will die.
  Airway : Breathing : Circulation systems form the cardio-pulmonary integrated unit and work together.

Gross anatomy and physiology of the heart

- The heart lies proximal to the lungs in the mediastinum. This is essential because the two work simultaneously to deliver oxygen to the tissues, and carbon dioxide away.   The volumes of fresh air and blood equate : around 5 litres of blood leave the heart over one minute for delivery to the lungs. About 5 litres of air pass through the lungs over one minute. If the one fails, the other compensates up to a point. For example, if the BP falls [and content of blood to tissues is diminished] the respiratory rate goes up in an attempt to load more oxygen into the failing blood supply.

- The heart comprises two pumps, the right and the left, although they work simultaneously. The right heart [atrium and ventricle] pumps blood to the lungs, the left heart pumps blood to the body. The work of the left heart is much greater than that of the right, therefore the chamber pressures are higher in the left heart.

The chambers of the heart

- a. the upper chambers : left atrium receives blood back from the lungs [via pulmonary veins]. The right atrium receives blood back from the body [via the superior and inferior vena cavae]. Their work is to pump on this blood to the lower chambers, the ventricles. In reality they 'top up' blood already in the ventricles [which never empty]. If they do not beat regularly [condition known as atrial fibrillation] this 'atrial kick' or top up is impaired and blood pressure may fall.

- b. the lower chambers : left ventricle pumps blood into the systemic circulation [via the aorta]. The right ventricle pumps blood into the pulmonary circulation [via the pulmonary artery]. Remember at this point that blood always passes from a higher to lower pressure down a pressure gradient.

The valves of the heart

- there are 4 heart valves : in the left heart [mitral or bicuspid valves] is the 'gateway' between the left atrium and left ventricle. The aortic valve is the gateway between the left ventricle and the aorta. On the right side the tricuspid valve lies between the atrium and ventricle and the pulmonary valve is the gateway from left ventricle to the pulmonary artery.

- The function of the valves is to open and close allowing blood to be shunted from one chamber to the next. When pressure builds in one chamber which is greater than the next chamber the valve is pushed open. It closes by natural reflux. It is important that they close tightly to prevent blood backflowing in the previous chamber. The mitral and tricuspid valves have chordae tendinae [thread like structures attached to the valve and the papillary muscles] – these tendon and muscles ensure tight closing of the valves.

- Valve disease can result in leakage of blood from one chamber to the back chamber – eventually leading to cardiac muscle stress and enlargement.

Layers of the heart

- There are 3 layers [tunica] to the heart
    a. Pericardium – the external layer of the heart, a fibrous layer which protects the organ. Pericardial effusion occurs when fluid accumulates between the peri and myocardial layers. It can constrict heart muscle contraction.
    b. Myocardium – the heart muscle. Made up of myofils, intercalculated discs which allow movement and energy. Sheets of cardiac muscle cells allow the ventricle to squeeze in several directions simultaneously : top to bottom, side to side and twisting to squeeze the maximal amount of blood out of the heart on each contraction [stroke volume]. Myocardial efficiency is vital for life – the muscle works without stop to deliver blood to the systemic circulation. It has automaticity properties, in other words it is not dependent on the voluntary nerve system. The muscle wall is thickest in the left ventricle where blood is pumped out against a resistance in the systemic circulation. The pressure in the left ventricle is highest.
    c. Endocardium – smooth inner lining of the heart. Connects with valves.

Coronary artery circulation

- The heart **must** be supplied with blood and oxygen continuously – it therefore has its own supply. The **coronary arteries** stem from the aortic root and straddle the heart as follows:
    a. the left coronary artery supplies the hard working left myocardium. The left anterior descending is particularly important since it feeds the left ventricle. Diminished blood supply to this portion of the heart can lead to ischaemia, and if severe, to infarction [death] of left ventricular muscle mass with serious consequences.
    b. the right coronary artery supplies the right heart
    c. the posterior artery – supplies the posterior wall of the heart muscle
- Coronary artery blood supply to the muscle is vital. Cardiac muscle work is strenuous, and requires a constant supply of oxygen and nutrients. The heart muscle will become ischaemic if deprived of blood from the coronary circulation. This can lead to infarction of the myocardial cells. General anaesthesia and surgery can stress the heart muscle : for anyone with a history of

ischaemia or myocardial infarct, the anaesthetist will use a mixture of cardiac friendly drugs, advanced monitoring and meticulous fluids management to protect cardiac function.

Heart chamber pressures

Chamber	Pressure
Right Atrium	0-4 mmHg
Left Atrium	8 – 10 mmHg
Right Ventricle	25/4 mmHg
Left Ventricle	120/10 mmHg

- The atrial pressures are lower than ventricular ; atrial contraction, which propels blood to ventricles is much less forceful than ventricular contraction. Atrial contraction serves to ‘top up’ the ventricles just prior to systole where blood is ejected into the circulation. The right heart pressures are lower than left heart pressures. The right heart only pumps blood to the lungs under low pressure. The left ventricle has the highest pressure, pumping blood into the circulation.

Electrical conduction system of the heart

- Mechanical contraction of the chambers is fired by electrical stimulus via the hearts conduction system. The **sino-atrial node** in the right atria is a group of specialized cells which act as the primary pacemaker. They kick start the cardiac cycle by stimulation of the atrium, to contract [‘p’ wave on ECG]. Waves of excitation spread though the intra atrial pathways so fast that 100 million myocardial cells contract in less than one third of a second. Atrial contraction causes blood to be ejected into the ventricles. The conduction chain then passes to the **atrio-venticular node** [at the junction between atria and ventricles] – here there is a short delay. The **Bundle of His** receives the stimulus, the right and left bundle branches fire off ventricular contraction – the excitatory wave being carried down and around the ventricular muscle by the **Purkinje fibres** [QRS wave on ECG]. The ventricles, topped up with fluid from the atrium are now at their highest pressure and on contraction eject blood into the system and pulmonary circuits. Ventricular contraction of 400 million ventricular cells takes one third of a second. This is known as **systole**. The resting period [**diastole**] where the ventricles fill. This is followed by atrial contraction 'p' wave on ECG] and the whole cycle starts again.

Sinus rhythm is so called because the conduction system is stimulated by the **SA node**. The cardiac cycle [atrial and ventricular contraction] [PQRST] takes 0.8 sec – over one minute there are around 70 contractions.

Cardiac Cycle

- as stated above each **cardiac cycle takes 0.8 sec** for adult at rest. The **cardiac cycle denotes systole : diastole**. In clinical practice this is measured by blood pressure with systolic and diastolic components. Systole equates with the actual contraction of the ventricle forcing blood into the aorta at high pressure. Normal adult systole is around 120 mmHg. Diastole equates with the relaxed heart, where the atria are filling with blood and the ventricular muscle mass is resting. Pressure is still significant in the heart and the blood vessels however, and the diastole 'resting' pressure reflects this.
- The difference between systolic and diastolic pressure = **the pulse deficit**. If the deficit is small and the diastolic pressure high this indicates the heart is under considerable pressure, the ventricle having to push blood out against a high systemic vascular pressure.
- **MAP : Mean arterial pressure** is calculated in clinical practice to reflect the average pressure combining both systolic and diastolic pressure which constantly perfuse the tissues : it is calculated as follows: Systolic Pressure + Diastolic Pressure x 2 divided by 3.
- The rate and strength of ventricular contraction are both vital in determining how much blood will be ejected on each beat : usually around 70ml for the adult at rest. Given the fact that the heart at rest beats 70 beats a minute – that means around 5 litres of blood is pumped into the circulation per minute. This is known as the **Cardiac Output** [to be discussed later].
- The **autonomic nervous system** [sympathetic and parasympathetic] play a vital role in controlling the heart rate and force of contractions. The parasympathetic chain slows down the intrinsic heart rate to around 70 beat per minute via the Vagus Nerve. Sympathetic nerves [cardiac nerves] speed up the rate and force of contraction and increase the cardiac output.

Structure and function of the circulatory system

Remember there are 3 systems to be studied here:

a. coronary artery system [already studied]
b. pulmonary system
c. arterial-venous system [systemic]

Before examining the systemic system we must consider the pulmonary circuit.

Pulmonary circulatory system

- this carries de-oxygenated blood from the right heart to the lungs via the pulmonary artery, the vessels divide to become capillaries with thin walls [one cell thick]. This network straddles the pulmonary alveoli [the walls of which are also one squamous cell thick]. The space between the air sac and blood vessel is minute – all these factors allow for FAST transfer of gases, oxygen and carbon dioxide to diffuse from an area of higher to lower pressure across the cell membranes.
- As stated before the volume of blood and gas delivered over one minute equates : **5 litres of air to 5 litres of blood**. In normal health and regular activity the ratio of blood available in the capillaries is equal to that of fresh gas in the alveoli for gas exchange throughout the lungs. However, in perioperative practice, this equilibrium is disturbed, alveoli collapse leaving unequal distribution of blood and fresh air.

Systemic circulatory system : Arterial-venous system [we will compare and contrast these two, very different systems].

- The arterial system [as with the left heart] works under much greater pressure to deliver oxygen to tissues than the venous system [right heart]. The arterial system contains 13% of the blood volume. It meets **resistance in the arterioles**. The venous system is known as the capacitance system because it stores great quantities of blood ready for use in emergency, the veins contain 64% of blood volume.

- The major vessel leaving the heart is the **aorta, which acts like a motorway**, enabling mass transport of blood away from the left ventricle. The aorta contains fibrous tissue and elastic tissue so that ventricular contraction is carried along with the vessels dilating and contracting [pumping] to propel blood along.
- The smaller arterial vessels contain less elastic tissue and more connective tissue and smooth muscle.
- The **arteriole system is where resistance to blood flow happens**. The arterioles help to control blood pressure. They constrict or dilate in response to sympathetic nervous impulses. Baroreceptors in the aortic arch and carotid bodies sense the blood pressure and alert the cardio-vascular centre in the brainstem. This activates the sympathetic nervous system. If pressure is high – the vessels dilate, if pressure is low, they constrict.
- The pressure gradient is essential in allowing the transport of blood at high pressure from the heart, to diminish as it travels around the arterial system. By the time the veins transport blood back to the right heart, the pressure is very low. **Pressure of gas and fluid ALWAYS flow down a pressure gradient from high to low.**
- The **capillary network** – this is the 'microcirculation'. This is a vast network which ensures that all body tissues receive blood supply essential for cell function. At the arterial capillary end oxygen diffuses across the membrane into the cell. At the venous capillary end carbon dioxide diffuses from cell to blood vessel [internal respiration]. The capillary and cell walls are only one cell thick to facilitate diffusion. Gases diffuse across the cell membranes from high to lower pressure until equilibrium is reached.
- The **microcirculation plays an important role in 'shock'.** Where circulation is failing small pre capillary sphincters shut off the microcirculation to conserve blood for major vessels and organs. Lack of oxygen means that anaerobic metabolism sets in : the cells become acid and dysfunction.
- **Veins** have to return **blood back to right heart overcoming gravity**. Semi-lunar valves in their lumen prevent back flow of blood. Calf muscles help to pump the blood back up. Change in intra-thoracic pressure on breathing in and out also aid in propelling blood back to the heart.
- By the time the blood reaches the right atria – pressure is at an all time low. The highest pressure in the systemic system is in the aortic arch [around 100 mmHg] the lowest is at the right atrium [3-5mmHg].

Haemodynamics : the forces or mechanisms involved in circulation

- Blood pressure = the pressure exerted by circulating blood flow on the walls of the vessels.
- Pressure is the relationship between flow and resistance. Blood 'flow' is the cardiac output [CO] which is about 5 litres over one minute.
- Resistance to flow is set up in the arterioles which vasoconstrict, or dilate in response to autonomic nerve activity.
- Resistance has no bearing on output, which is constant. In clinical practice we call this resistance 'systemic vascular resistance' [SVR].
- In perianaesthesia practice many variables [drugs, anaesthetic techniques, fluid loss due to surgery, pain] impact on CO or SVR or both. We need to understand how they work together to create blood pressure.

BP = CO X SVR

- **Blood pressure = Cardiac Output X Systemic Vascular Resistance**
- **Cardiac output [CO] = amount of blood delivered into the circulation in one minute**
- **CO = Stroke volume [SV] x Heart Rate [HR]**

Stroke volume = amount of blood ejected on one contraction = 70mls [adult at rest]

- Heart contracts around 70 times a minute
- Over one minute 4,900 mls [5L] of blood are delivered into the circulation [70mls x 70 contractions]

Stroke Volume : 3 main factors affect stroke volume. **Preload, contractility, and afterload**. We will take each one in turn:

1.Preload or venous return : the volume of blood returned to the right heart is the preload or venous return. This will work its way round [via the lungs] to the left ventricle for expulsion. If diminished, then the left ventricle will have less fluid to pump out – cardiac output will fall. While blood pressure may be maintained by vasoconstriction, tachycardia, eventually the blood flow and oxygen to tissues will diminish.

2.Contractility : this is the pumping power of the ventricles in systole. The force of myocardial contraction is dependent on the volume of fluid it pumps. The greater the fluid volume = the greater the pressure – the greater the force of myocardial contraction [Starlings Law]. Up to a point [if the heart is weak, then too much fluid load will cause it to fail]. If venous return is reduced, volume will diminish and myocardial contractility will also be impaired, resulting in fall in CO.

3.Afterload : This is the resistance the ejected blood meets in the great vessels. If resistance is high then the left ventricular muscle has to work harder to eject blood out of the heart. If low, then the heart work diminishes.

Stroke volume is a key factor in blood pressure and directly affects the cardiac output.

Remember SV x HR = CO. Heart rate is also a key factor in affecting CO.

- **The heart rate** for adult at rest is around 70 beats per minute. If the heart rate is diminished – then given a constant stroke volume the CO will be diminished.

Consider this scenario :

SV = 70mls X HR = 40 bpm *[consider that the patient is elderly]*

The CO will be 70 x 40 = 2,800mls [normal CO = 5 litres]. This profound bradycardia is compromising the blood pressure [BP = CO X SVR]. What is physiological natural response? The cardiovascular centre will stimulate the sympathetic nerves to vasoconstrict the arterioles and increase the SVR so that the blood flow meets a tighter resistance and pressure increases.

The anaesthetist could deliver a large fluid bolus so that the stroke volume is increased to 100mls on each ejection. This would mean that :

CO = SV [100mls] x HR [40 bpm] = 4000mls – almost normal.

In practice the first intervention would be to increase the HR [with atropine] back up to 70 beats per minute.

** Note that the patient is elderly here. This information is given because young fit athletic people often have HR of around 40 bpm and deliver healthy stroke volumes delivered by a strong, healthy heart muscle.*

- A fast heart rate can lower the BP. If the heart is beating over 100 contractions a minute, this means that the diastolic filling time [when atria are collecting blood] is shortened – less fluid is passed onto the ventricles – SV is diminished with a loss of CO leading to hypotension.

- Consider also **arrhythmias** [electrical conduction that does not arise form the SA node.] **Atrial fibrillation** is a common disease in elderly people. It may require treatment with anti-coagulation and, or B blockade but usually patients tolerate this rhythm throughout peri-anaesthesia. **Ventricular ectopii** need investigating as they are often due to hypoxia, or potassium imbalance, both of which irritate the myocardium. Life threatening arrhythmias such as **ventricular fibrillation** should be recognized as haphazard weak, non coordinated muscle contractions which cannot deliver stroke volume or cardiac output. Along with loss of consciousness it will be evident that this calls for cardiac arrest alert. A**systole** also should be recognized as life threatening.

- We have considered the factors underpinning Cardiac Output. We will now consider **Systemic Vascular Resistance.**

- **Systemic Vascular Resistance [SVR] :** As stated the SVR does not affect the CO, however, it determines the BP by its action on vasodilation or constriction of the arteriole bed.

Consider the following:

- *Given a CO of 3 litres, if the vessels are constricted, [i.e. SVR elevated], the BP will may be normal. If the vessels are dilated [SVR diminished] the BP will be low.*

- *If the CO is 5 litres but the patient has had a spinal which vasodilates the vessels, the BP will be low. If fluids are administered so that CO is 7 litres, the BP will recover even though vessels may still be dilated.*

- The systemic vascular resistance plays a vital role in determining BP and keeping it within normal limits. It is controlled by the autonomic nervous system – the action of the cardiovascular centre in the medulla and the sympathetic and parasympathetic chains of the autonomic nervous system which we will now explore.

The activity of the ANS [cardio-vascular centre/sympathetic and parasympathetic nervous systems].

- The autonomic nervous system ANS is not under voluntary control. It automatically adjusts body functions by stimulation of sympathetic or parasympathetic chains. Generally **sympathetic activity 'speeds up'** body processes and **parasympathetic 'slows down'.** In doing so they counter balance each others effects.

- The parasympathetic nervous system directly affects heart rate. The **vagus [parasympathetic nerve]** slows down the intrinsic heart rate to around 70 bpm. In clinical practice stimulation of the vagus nerve will cause bradycardia. Atropine is given to treat bradycardia. Atropine blocks the vagal response.

- The sympathetic nerve chain is more complex [arises from the spinal cord at various levels] and directly affects the heart muscle through the action of the **cardiac nerves** which increase heart rate and increase the strength of myocardial contraction. Cardiac output is improved as stroke volume and heart rate are both increased.

- The sympathetic nerve chain and its **neurotransmitter, noradrenaline** acts directly on the diameter of the arteriole vessels, causing dilation or constriction. As seen already. In reality the blood vessels are in a state of permanent semi - constriction.

Action of the cardio-vascular centre [CVC] in the medulla.

- The CVC is a group of sensitive cells in the **medulla oblongata,** situated near to the Respiratory Centre. It receives information about the blood pressure status from the **baroreceptors**.
- The baroreceptors are sensitive cells in the aortic arch and the carotid bodies which monitor the blood pressure. If low or high – they signal the CVC – which in turns signals the sympathetic nervous chain to :

    - increase or decrease the HR and ventricular contractions to increase or decrease the CO
    - vasoconstrict or vasodilate the blood vessels to increase or diminish SVR

- This is known as a ‘negative feedback response’.

Summary : to understand how to manage the circulation in clinical practice you need a thorough understanding of the anatomy and physiology of the heart and circulation, constituents of haemodynamics together with knowledge of how anaesthesia and surgery destabilise their normal function.

Trainer’s notes on power point presentations

Circulation : Basic Sciences

TRAINER'S NOTES

CIRCULATION : Basic Sciences Presentation

Introduction

These notes are designed for the mentor to inform how they use the presentations.

Slide 3: ABC - re-establish why Circulation is 3rd priority. Reminds students of A+B and how closely the 3 'systems' work together to deliver oxygen to tissues – this should be VERY familiar by now!

4: A reminder about metabolism and the role of circulation in enabling this. While this should be very familiar, it is worth reminding students again of the fundamentals of life and the way all systems ABC are integrated towards enabling metabolism.

6: Co-dependence of lungs and heart is an important principle – they work together : failure of one causes compensation by the other and 'integrated cardio-pulmonary unit' describes this well. Concept should be familiar to students by this stage. Open a discussion about how and when students have witnessed this in practice.

8: Heart comprises 2 pumps : an important principle to establish from the start as it makes sense of blood flow, and explains the difference in pressures + work of the left and right heart chambers.

9: While this is revision of basic A+P it remains important to remind students of how blood moves from R to L heart and how oxygenated en route.

10 & 11: Useful to pair function and pressures in [upper V lower chambers and right side V left side heart chambers]. Understand how upper chambers only collect and deliver blood to ventricles [final top up on atrial contraction is the stimulous to ventricular contraction] and therefore work under lower pressure. Ventricles pump blood into lungs and around body –volume and pressure correspondingly higher. Left heart pressures higher reflect harder work on contraction.

12-14: Emphasise the work of myocardium, the composition of muscle cells which allows it to beat for a lifetime. Stress the importance of the left myocardium, why it is the thickest part of heart muscle and how it forces blood to systemic circulation against a pressure [afterload]. Stress again importance of coronary artery blood flow to this part of muscle and consequences of it being damaged - slides 21 & 22].

15-17: Stress how valvular incompetence where the valves fail to completely close, can lead to back flow of blood and cardiac instability. Again valvular stenosis [particularly aortic stenosis] leads to build up of pressure as blood is forced through narrow valve with associated loss of cardiac output delivered to systemic circulation. Discuss pressure gradient here – blood moves from chamber with higher pressure to one with lower pressure via valves – a basic, vital concept.

19 & 20 : Coronary artery blood supply. Simple explanation of blood flow to heart itself – again stress the importance of the left coronary artery and left anterior descending – supplying blood, nutrients and oxygen to important left myocardial muscle mass.

21 & 22 : Two slides to demonstrate ischaemia and infarct in the left ventricle. Ischaemic heart disease is a risk factor in peri-anaesthesia and accounts for a high proportion of peri-anaesthetic incidents. Discussion on how to protect the frail heart in peri-anaesthesia would be useful here.

24. The cardiac cycle : Simple explanation of the cycle : atrial contraction followed by ventricular contraction. This looks back to slide on chamber pressures.

25-27: Introduction of blood pressure with focus on diastole and systole. Connects with previous slides [atrial/ventricular contraction] so follows through logically. Give systolic and diastolic pressures. Important to explain more fully diastolic pressure as pressure held in arteriole system at end of systole – more difficult to understand than systolic pressure.

28: Explanation of Pulse Pressure and Mean Arterial Pressure. Pulse pressure : if difference is narrow i.e. 105/95 - this means resistance to ejection of blood into circulation is high –the heart is working under pressure.   MAP is the average pressure of blood flowing to the tissues – a measurement used in advanced clinical practice – to provide parameters for blood pressure.

29-31: Electrical conduction system. Logical explanation of sequence of electrical impulse, relating it to mechanical action diastole and systole. Slide 30 diagram demonstrates conduction really well. Slide 31 demonstrates electrical sequence 'pqrst' with exact timing of mechanical events in systole and diastole. This is very useful. If students connect electrical and mechanical events they will understand sinus rhythm and from there be able to unravel ECGs'.

32: Classic 'pqrst' with timing. One cardiac cycle = one 'pqrst' configuration – which takes 0.8sec. From now they will see that HR works out at 70 beats per minute [0.8sec X 70 = 60 seconds]. The presence of 'p' wave here denotes 'sinus rhythm' – stress the important of this. This beat starts off in sino-atrial node [primary pacemaker] – if no 'p' wave, heart rhythm is not sinus – this is fundamental to study of arrhythmias. Also discuss the height and width of the 'qrs' and discuss how this demonstrates much higher electrical energy in ventricular contraction.

33: Heart rate : role in delivering 5 litres [cardiac output] to body over one minute. Bradycardia will diminish this volume. Tachycardia will put coronary artery supply to muscle at risk. Important concepts that will occur repeatedly in this section.

35-41: Arrhythmias can compromise cardiac output and blood pressure. Stress that this is an only an introduction to the topic. Focus on examples of major arrhythmias : sinus bradycardia, sinus tachycardia, atrial fibrillation : ventricular fibrillation, asystole. Also an example of ventricular extra-systoles. A rhythm strip [lead II] is given to demonstrate all of above. At this stage get students to identify SINUS rhythm, brady and tachy sinus rhythms easy to understand. Atrial and ventricular fibrillation are also logical. Asystole – ensure they think to check for lose leads!! Ectopii occur often in practice and should alert nurse to instability. Remind them that muscle tremor can through up what looks like arrhythmia – beware of these. All patients have ECG monitoring in PACU – a basic knowledge of ECG is very important to use this valuable monitoring tool.

42-43: Introducing here the components of blood pressure and BP equation in slide 42.   This will be analysed at length in 'haemodynamic section' further on. These are fundamental concepts that the students must understand to analyse blood pressure in practice.

45: Reminder of how vast is the distribution of blood vessels in the circulation.

46: Stress the delivery to the brain [15%] : mental status is so important in diagnosing hypoperfusion and hypoxia. Also 25% of output goes to drive the kidneys. Loss of output and pressure to these organs quickly leads to acute renal failure.

47: This slide explains the diminishing pressure as blood flows through arteriole side of circulation. Blood always flows from high to low pressure. This is a very important physiological concept. As the vessels get smaller, so the pressure drops. The venous system works under much lower pressure and higher volume than the arterial side.

48 : Demonstrates that 64% of blood volume is held in storage in the veins – and only 13% in the arteries.

49: A diagram to show relationship of elastic to smooth muscle of the arterial circulation. The elastic properties of the aorta and large arteries set up a 'pumping action' to ensure blood propelled along. Smooth muscle replaces this elastic component as vessels get smaller. Stress strongly the arteriole vessels as key to resistance in the system.   This network responds to sympathetic nervous stimulation in vasodilating or constricting the arteriole bed and controlling blood pressure.

50: Compare the thickness of the walls of venous to arterial vessels.   This is a very good diagram illustrating the muscle power of the arteries. Discuss why veins have valves.

51-52: Two slides which reinforce the previous pictures. Discuss the differences between the arterial resistance and venous capacitance systems. Resistance set up by small arteriole system – which is manipulated by the autonomic nervous system and plays a direct role in regulation of BP. These are very important facts, worth spending time over before going onto haemodynamic section.

53 -54: Good chance to revise Breathing Module and re-examine the micro-circulation with regard to it's importance for transfer of gas and nutrients to and from the cells.

55 : Reinforcing the key role of the arterioles in controlling blood pressure.

57-59 : Haemodynamics -this is straightforward if taken slowly. Build up the equations bit by bit giving lots of examples. These three slides confirm the relationship of **BP = Cardiac Output V Peripheral Vascular Resistance**. This is a fundamental concept – they should remember this equation if nothing else. It will help them to make sense of BP in clinical practice and analyse hypotensive states more accurately.

60: The next slide introduces stroke volume. Spend a little time discussing this – ask the group what factors they think will be important in delivering stroke volume on each heart beat.

61-63 : These slides unravel ‘stroke volume’. 3 determinants : preload, contractility and afterload. The diagram is useful here [slide 61]. Again logical information but need to take it slowly. Of the three concepts – afterload is possibly the most difficult to understand. Slide 62 is straightforward and gives more explanation about preload : contractility and afterload.

64-65 : Now we consider pre-load as a constituent part of stroke volume. Preload [venous return] – use both expressions. Important to establish that volume of fluid = pressure here. So CVP denotes right atrial pressure which denotes fluid volume. Also consider that poor venous return will impact on the cardiac output ejected by the left heart if not corrected. The importance of fluids in peri-anaesthesia care makes this factor understandable and important. Discuss how you would know if patient is volume deplete with no monitoring equipment. Then discuss the use of CVP monitoring in practice.

66 : Afterload : the pressure [or resistance to ejection of blood] against which the left ventricle has to pump. The greater the resistance, the more the ventricle fails to contract fully – the smaller the cardiac output. Resistance is determined by the calibre of the arterioles which is governed by sympathetic nervous control.

67 : Contractility : the force of contraction of the myocardium. Discuss Starlings Law here, it is important. The greater volume of fluid the pump has to eject – the more the muscle is stretched, the greater the power of contraction. So hypovolaemia will lead to smaller, less powerful contractions. If the heart is damaged however, this law does not apply – too much volume and the left ventricle [already damaged] will fail. So the anaesthetist has to ensure that in the patient with a history of ischaemic heart disease, he does not overload with fluid.

68-69 : Some of the other factors that impact on contractility related to perianaesthetic practice. This is inserted mainly to demonstrate all the variables that can affect blood pressure in perianaesthetic practice. A discussion about these factors [drugs, anaesthetic technique, surgical factors] will make this slide very relevant.

70-71: Finishing off the equation : CO = SV X HR – we must not forget about HR. We have already discussed this earlier and seen some examples of arrhythmias. Stress again here that a heart rhythm irregularities can impair CO which will impair BP in turn.

72 : We now have to remind the students about the whole equation : **BP = CO X SVR**. We are now looking at the SVR [systemic vascular resistance].

73: Simple introduction that SVR is governed by degree of vasoconstriction or vasodilation of the arterioles.

74: Reminded that the sympathetic nervous system controls diameter of blood vessels [arterioles]. Given a constant CO – the diameter of the vessels will determine if pressure high or low. Again a logical concept. You can play around with this and give the students some made up scenarios

a. High CO [8L] with dilated vessels –will BP be maintained ?
b. Low CO [3L] and constricted vessels : likely BP?
c. High CO [8L] with constricted vessels : likely BP?
d. Low CO [3L] with dilated vessels : likely BP?

76-78 : Autonomic nervous system : a reminder that it consists of two involuntary chains – sympathetic and parasympathetic – one speeds up, the other slows down. Sympathetic chemical transmitter = noradrenaline. Parasympathetic transmitter = acetylcholine. Discuss the activity in stress response [fear, flight].

79-80 : Cardiovascular control centre [CVC] in the medulla controls BP. Baroreceptors sense low blood pressure – relay information to CVC which then activate cardiac and sympathetic nerves to increase cardiac output and vasoconstrict arterioles : pressure up. If pressure high, this feedback system works the other way – sympathetic and cardiac nerve activity is withdrawn, cardiac output falls, and vasodilation occurs – blood pressure falls. Not a difficult concept but take time to explain.

81 : A reminder of the peri-anaesthesia factors that can impact on BP

82 : Finish by putting up the **all important equation** again.

83-5 : Remind students that many factors determine BP and that peri-anaesthetic practice can disturb normal homeostasis. If the student begin to apply this knowledge to their practice it will be very helpful – and make practice much more interesting.