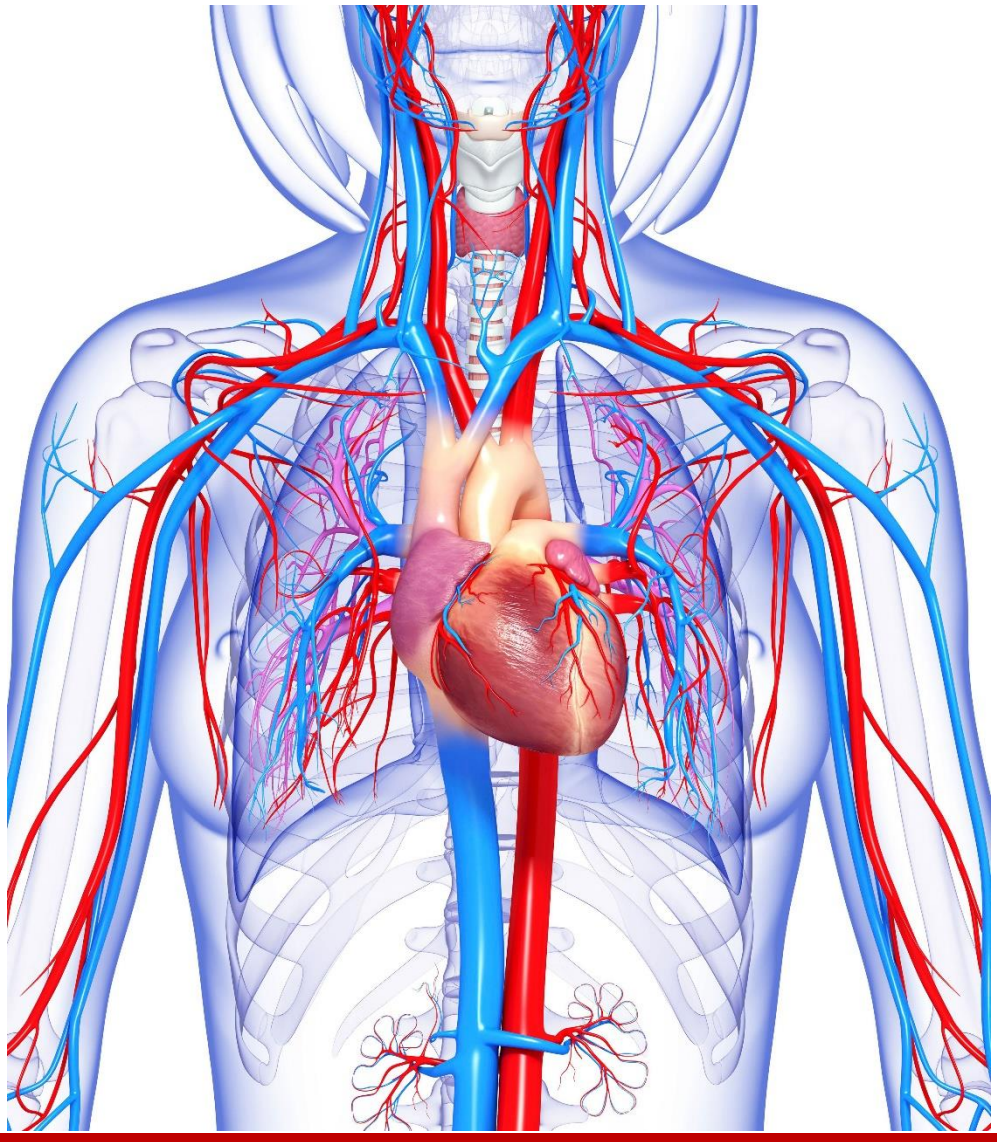
Sheffield Teaching Hospitals NHS
NHS Trust

CRITICAL CARE DIRECTORATE

Supporting Staff Nurse Step Competencies

Cardiovascular Guide



Version Two - Written by:

Samantha Cowan

Rebecca Parkinson

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Introduction

Haemodynamics involves studying the physiology of blood flow and circulation, with a particular focus on homeostatic mechanisms that maintain perfusion to major organs and tissues within the body. Under normal physiological conditions the heart should be able to maintain optimal function, adapting to increasing oxygen demand, based on physical activity. However, in critical illness, this system can be potentially compromised. This has particular importance in critical care where adequate cardiovascular function is vital in sustaining perfusion to all organs and tissues. It is important to understand the principles that effect cardiovascular performance and how cardiovascular function is maintained during critical illness.

The purpose of this document is to provide entry level and refresher information on cardiovascular support provided in general critical care and to outline some key principles relating to cardiovascular monitoring. This document is not intended to be a comprehensive guide to cardiovascular management and is designed to be used in conjunction with other educative resources and guidelines that are available.

Other Resources can include….

-Taught Induction Day Two Powerpoint on the Cardiovascular elements of an A-E assessment. Accessible on Learning Zone.

-Greater Manchester Critical Care Skills Institute Anatomy and Physiology Workbook.

-Resources under the ‘Cardiovascular System’ Topic, found on Learning Zone by clicking on ‘Clinical Information’.

-LiDCO Guidelines.

Cardiovascular System

Key Parts and Functions of the Circulatory System

Key Parts

- **Blood:** Made up of cells and plasma.
- **The Heart:** A muscular organ that pumps blood to all parts of your body.
- **Blood Vessels:** A network of arteries, veins and capillaries to carry blood pumped by your heart.

Functions

- **Transport oxygen**, nutrients, and hormones to your body's cells to use for energy, growth and repair.
- **Remove carbon dioxide** and other wastes your cells do not need.
- **Perfuses** major organs and tissues

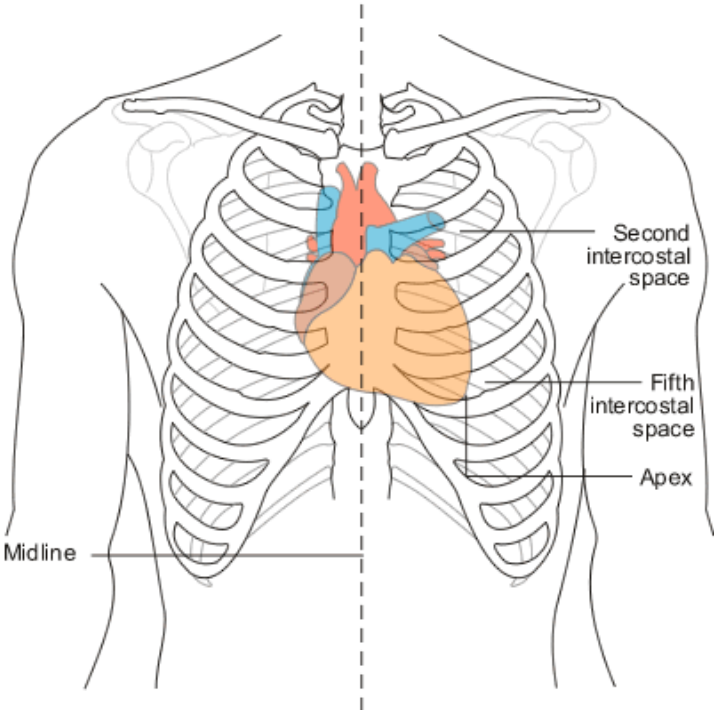
There are two blood circulatory systems in the body:

Systemic Circulatory System: Main circulatory system that transports blood to the organs, tissues and cells throughout the body.

Pulmonary Circulatory System: This system moves blood between the heart and lungs. It is where oxygen enters the blood and carbon dioxide leaves the blood. This happens through a process known as diffusion – Please see Respiratory Guide for more information.

Respiratory Package Version Three

Anatomy and Function of the Heart



Position of the Heart

-Sits off-centre, to the left of the sternum.

-Located within the mediastinum, sitting on the diaphragm.

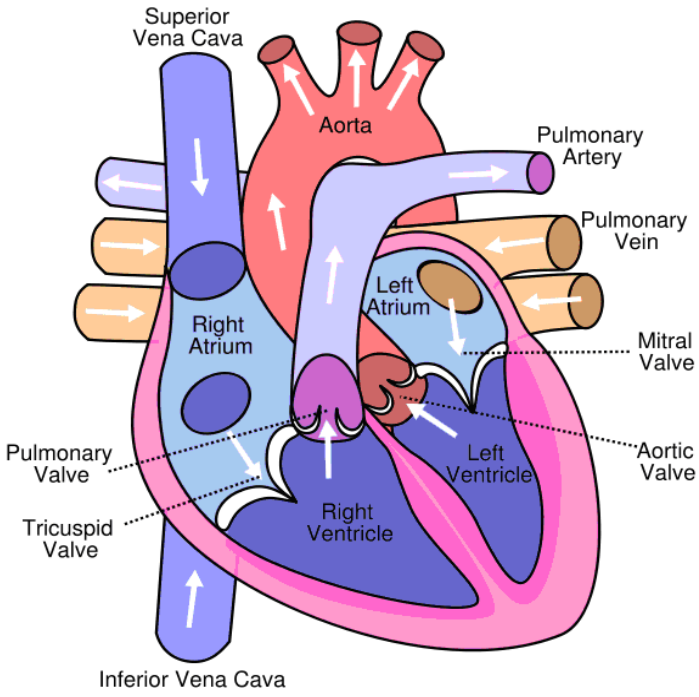
-Overall diagonal shape narrowing towards a tip or apex.

-This tip or apex lies in the 5th intercostal space in most individuals.

Structure of the Heart

The heart has **four chambers.**

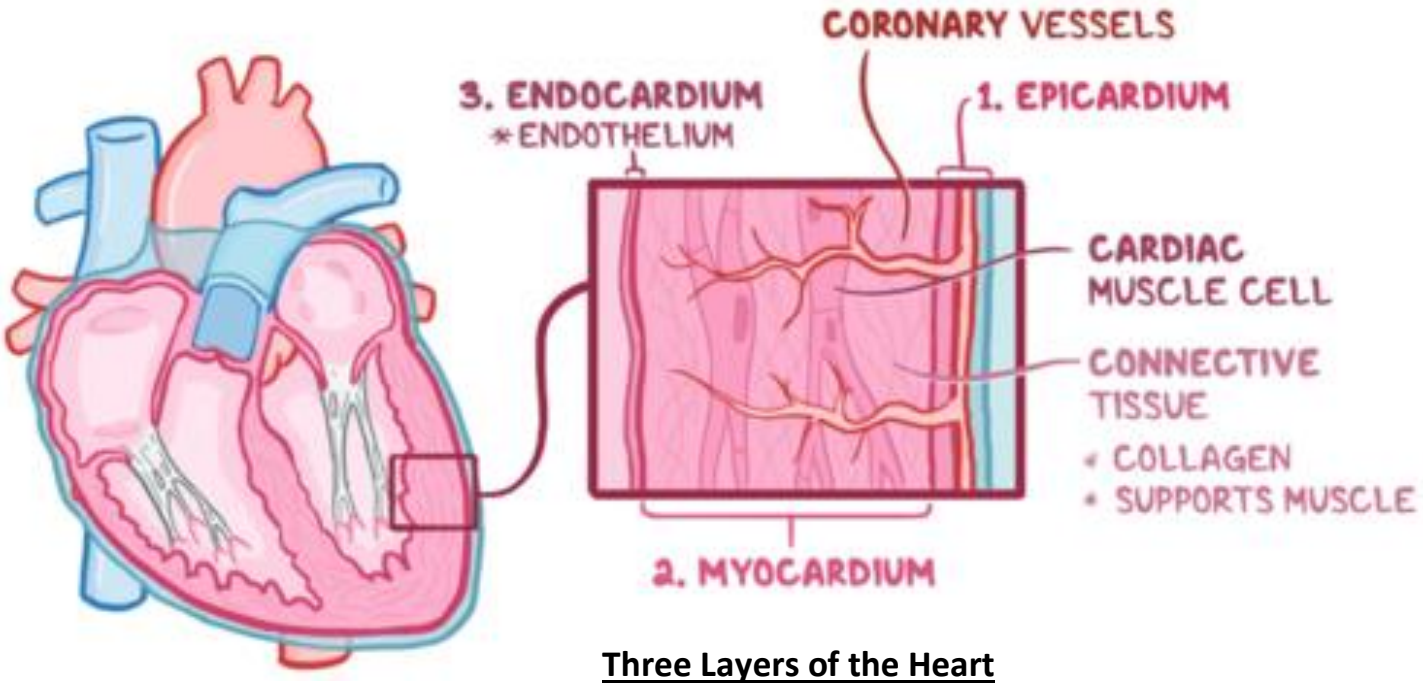
-Right Atrium
-Left Atrium
-Right Ventricle
-Left Ventricle



The heart has **four valves**.
-Tricuspid Valve:
Rests between the right atrium and right ventricle.
-Pulmonary Valve:
Rests between right ventricle and pulmonary artery.
-Mitral Valve (Aka Bicuspid Valve):
Rests between left atrium and left ventricle.
-Aortic Valve: Rests between left ventricle and aorta.

-The valves are thin but strong flaps of tissue, keep the blood flowing at the right time and in the right direction. Each valve has flaps that open and close once per heartbeat.
-The valves create the sound of a heartbeat: Often described as a Lub and a Dub.
-If a heart valve does not work correctly, your heart might have to work harder to pump blood. This can be related to age-related changes, congenital heart disease, infections or underlying conditions such as diabetes.
-If valve problems occur, such as regurgitation which is the backward flow of blood due to a valve that doesn't close properly, they will likely need cardiac surgery and will be cared for on CICU.

-The heart is composed of **three layers**; the epicardium, the myocardium, and the endocardium.



-The heart has its **own blood vessels** that supply the heart muscle with blood.
-The **coronary arteries** branch from the aorta and surround the outer surface of the heart like a crown.
-They diverge into capillaries where the heart muscle is supplied with oxygen before converging again into the coronary veins to take the deoxygenated blood back to the right atrium where the blood will be re-oxygenated through the pulmonary circuit.

The heart muscle will die without a steady supply of blood.

Three Layers of the Heart

1 - Epicardium

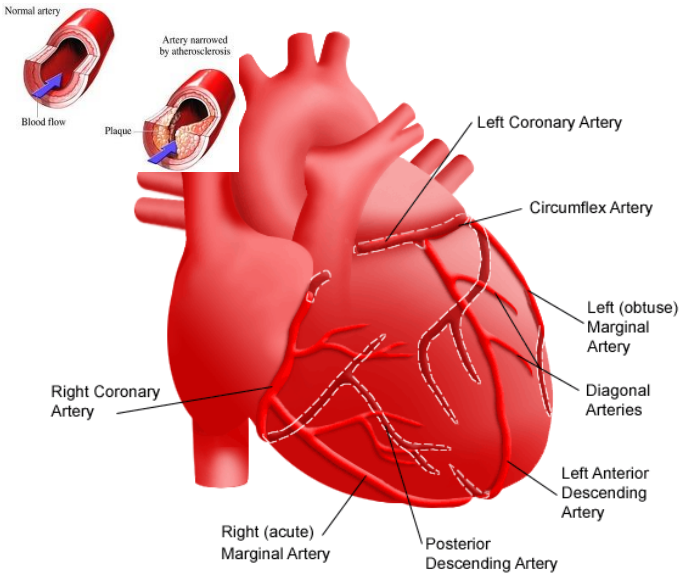
- The outer layer of cells.
- The second layer is a membranous layered structure called the pericardium that surrounds and protects the heart.
- The Pericardium allows enough room for vigorous pumping, but also keeps the heart in place to reduce friction between the heart and other structures.

2 - Myocardium

- Consists of the heart muscle cells that make up the middle layer and the bulk of the heart wall.

3 - Endocardium

- The inner wall of the heart lining.



-**Atherosclerosis** is the **blockage of an artery** by the build-up of fatty plaques. This can be **deadly in the coronary arteries**. The slowdown of blood flow and subsequent oxygen deprivation causes severe pain, known as **angina**, and complete blockage of the arteries will cause **myocardial infarction**: the death of cardiac muscle tissue.
-**Percutaneous Coronary Intervention (PCI)** is a procedure performed in the **cath lab** which helps open a clogged or blocked artery. A long, thin flexible tube (catheter) is inserted in a blood vessel, usually in the groin or wrist, and guided to the blockage. Then, a balloon on the tip of a catheter can be inflated to open the artery. A **mesh tube (stent)** is typically used to keep the artery open.

Major and Minor Blood Vessels and Blood Flow

Major artery of the body that takes blood away from the heart

Major blood vessels that stretch from your head to your upper chest. Direct blood from head to heart. Common CVAD site.

Major blood vessels in the neck that supply blood to the brain, neck, and face.

Drains blood from the jugular vein that comes from the brain and from the veins that come from the arms

Aorta

Jugular veins

Carotid artery

Carries deoxygenated blood from the right side of the heart to the lungs.

Drains blood from the veins that come from the lower organs and the legs

Superior vena cava

Pulmonary arteries

Inferior vena cava

Pulmonary veins

Carries oxygen-rich blood from your lungs to your heart

Brachial artery

Heart

Major blood vessel supplying blood to your upper arm, elbow, forearm and hand

Basilic vein

Thoracic aorta

Hepatic artery

Soft oxygenated blood vessel that supplies oxygen-rich blood to the liver, duodenum, and pancreas.

Gastric vein

Superior mesenteric artery

Main blood vessel that carries blood from the kidney and ureter to the inferior vena cava

Renal veins

Renal artery

Large blood vessels that carry blood from your heart to your kidneys

Abdominal aorta

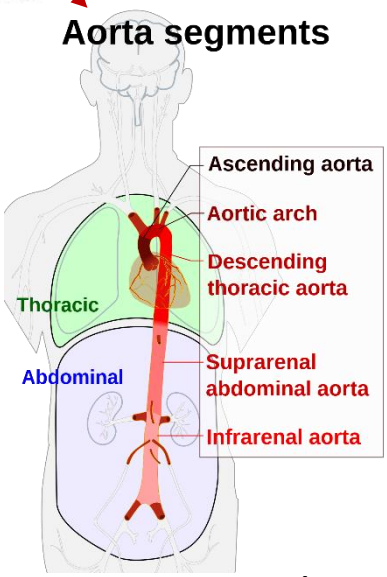
Iliac vein

Common iliac artery

Blood vessels in your pelvis

A short artery transporting blood from the aorta towards the pelvic region and lower extremity.

Aorta segments



Function of the Heart - Oxygenated and Deoxygenated Blood Flow

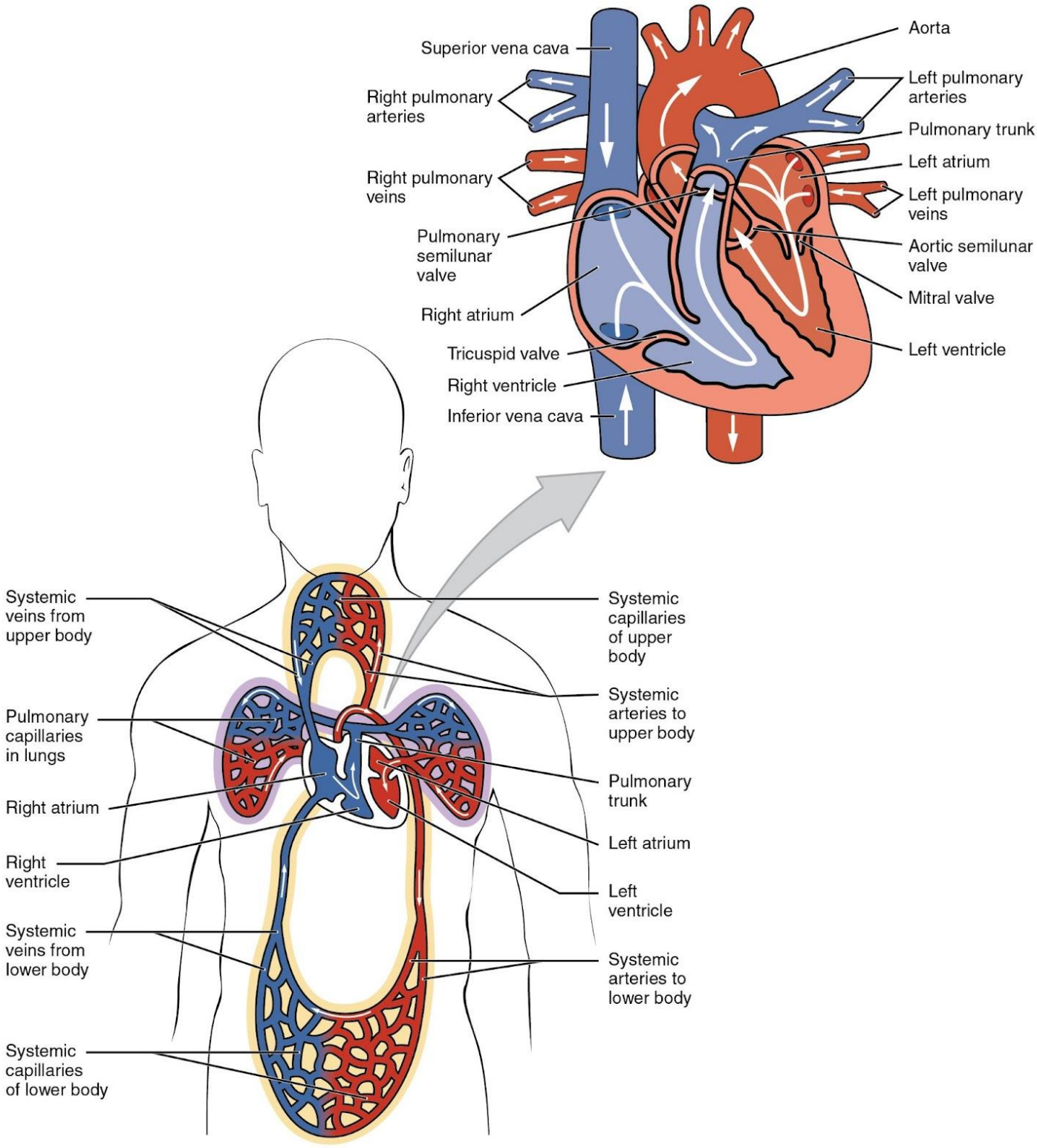
The cardiovascular system contains a 'double circulation'.

Systemic Circulatory System

Delivers oxygenated blood to organs and tissues and returns blood to the heart.

Pulmonary Circulatory System

Delivers de-oxygenated blood to the lungs and freshly oxygenated blood back to the heart.



Description of Blood Flow

Pulmonary Circulation	Systemic Circulation
De-oxygenated blood enters the heart through two large veins, Inferior and Superior Vena Cava	Pulmonary Vein empties oxygen rich blood from the lungs
Into the Right Atrium... Atrium Contracts	Into the Left Atrium... Atrium Contracts
Blood flows from the Right Atrium to the Right Ventricle through the Tricuspid Valve	Blood flows from the Left Atrium to the Left Ventical through the Mitral Valve
When full, the valve shuts, preventing backflow	When full, the valve shuts, preventing backflow
Right ventricle contracts De-oxygenated blood then leaves the heart through the Pulmonary Valve into the Pulmonary Artery	Left ventricle contracts Oxygen rich blood then leaves the Left Ventricle/Heart through the Aortic Valve into the Aorta
Flows through the Pulmonary Artery into the lungs, where it is oxygenated	The Aorta then transports the oxygen rich blood to the organs and tissues through a series of large and small blood vessles

Simultaneously...

Cardiac Cycle and Conduction

Normal Cardiac Cycle and Conduction

Electrical conduction of the heart begins in the Sinoatrial (SA) Node in the Right Atrium, which acts as the hearts natural pacemaker.

→

Impulse from SA Node causes the Atriums to contract, pushing blood through the valves into the ventricles (Atria Depolarization).

→

Electrical signals arrive at Atrioventricular (AV) Node, which is located between the two Atriums.

↓

The signal then travels down the Bundle of His, which divides into the Left and Right Bundle Branches.

→

Signal continues to travel through the Left and Right Bundle Branches and through the Purkinje Fibers.

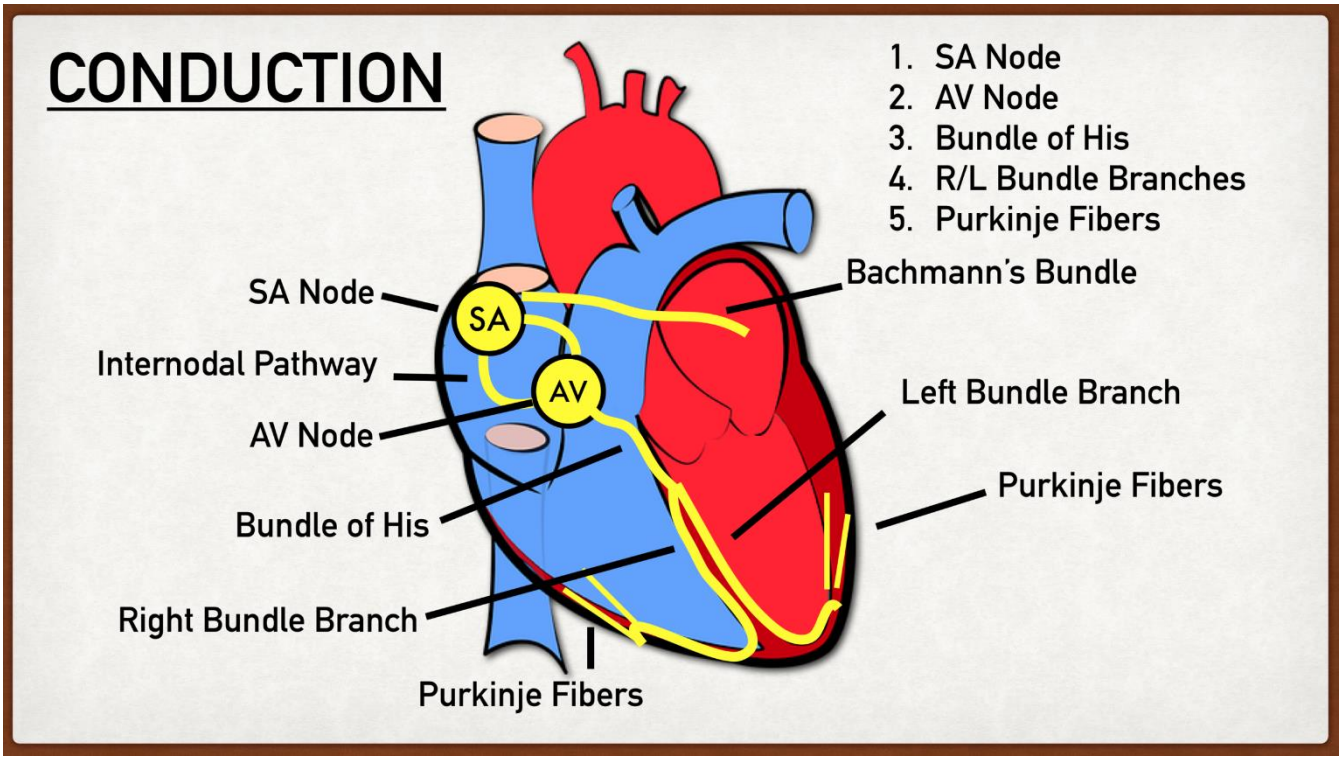
→

This causes the ventricles to contract (Ventricle Depolarization).

↓

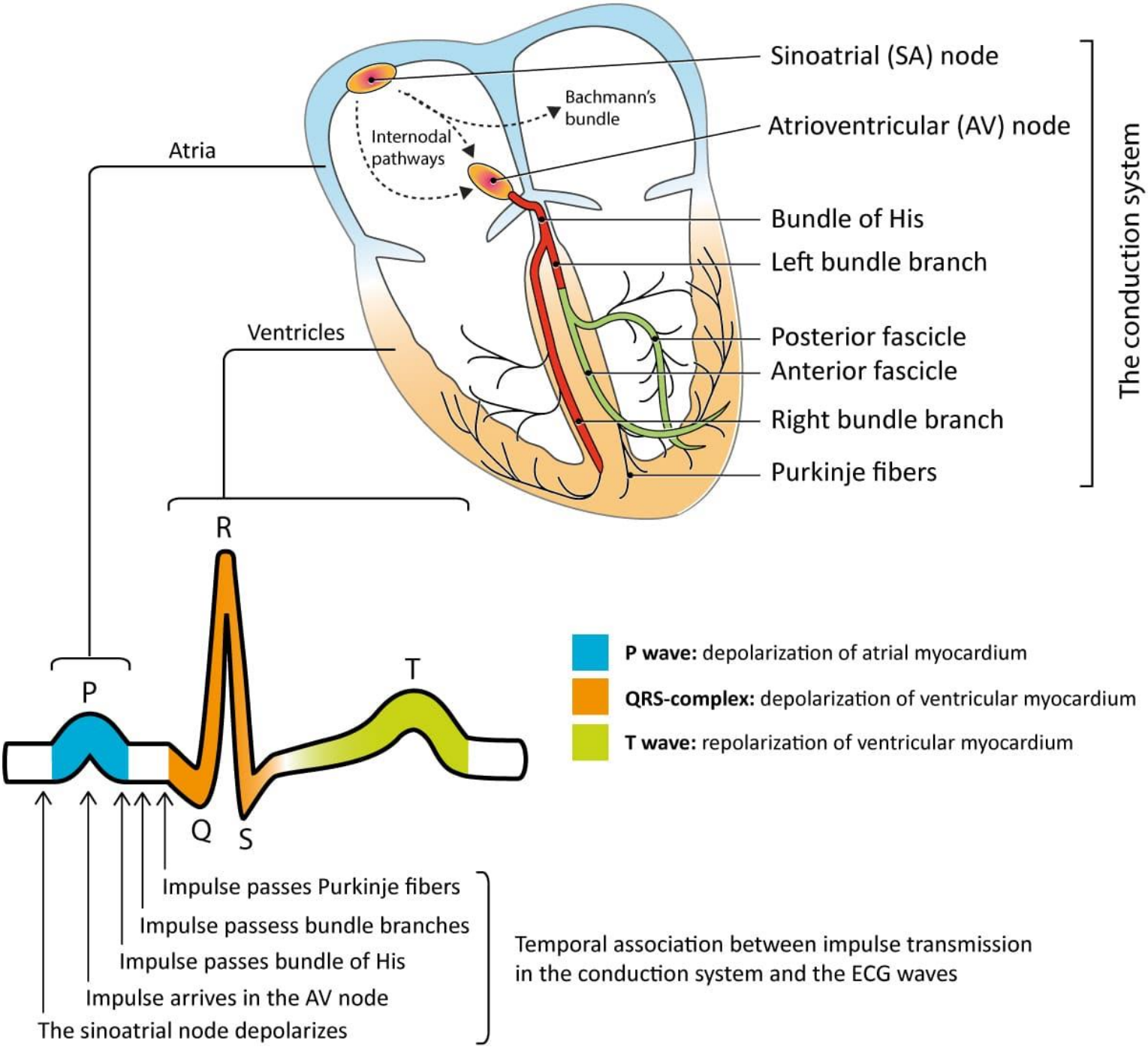
While the ventricles are being depolarized, the atria are regaining their internal electrical negativity, a process known as Repolarization. This allows them to relax. Once the ventricles have fully depolarized, they too become Repolarized, at which point they relax as well.

Left Ventricle contracts slightly before right. Blood from Left Ventricle is pumped through the Aortic Valve to the rest of the body. Blood from Right Ventricle is pumped through the Pulmonary Valve and into the lungs.



Electrocardiography (ECG)

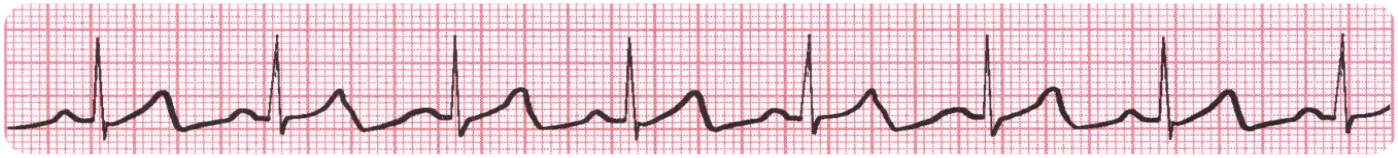
Electrocardiography is a method used to analyse the heart's conduction system, which provides us with information about the heart's electrical activity.



-The P wave represents the depolarization (contraction) of the atria.
-The PR segment the transmission of the electrical impulse to the ventricles.
-The QRS complex represents depolarization (contraction) of the ventricles
-The T wave shows the repolarization (relaxation) of the ventricles.

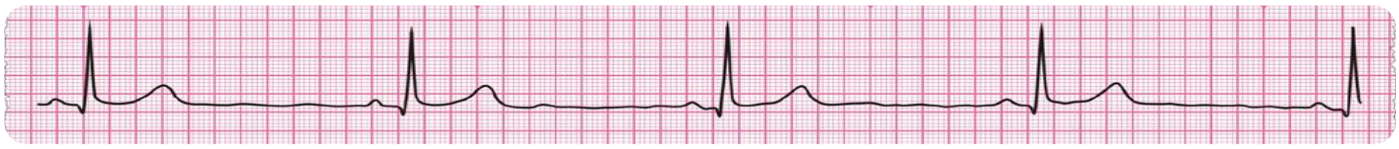
When disturbances of the conduction system are present, this can be detected via abnormalities of the spikes and waves on an ECG.

Cardiac Rhythms



Normal Sinus Rhythm

- Regular.
- All aspects of P-Q-R-S-T present.



Bradycardia

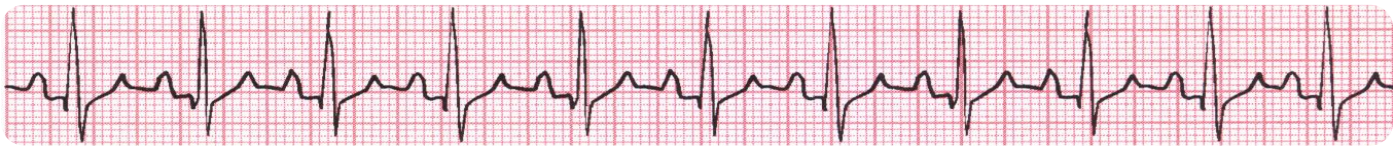
- Regular, Rate <60bpm.
- All aspects of P-Q-R-S-T present.
- Narrow QRS Complex.

Causes:

- Sleep
- Medications (Clonidine)
- Myocardial Infarction (MI)

Treatment:

- Treat underlying cause
- Medication (Glycoprolate)
- Pacemaker (PPM)



Tachycardia

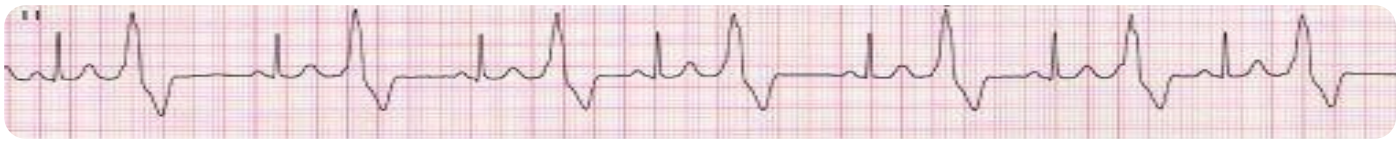
- Regular, Rate >100bpm.
- All aspects of P-Q-R-S-T present.
- Narrow QRS Complex.

Causes:

- Pyrexia/Infection
- Anxiety/Pain /Medications
- Hypovolemia (Hb/Fluid)

Treatment:

- Treat underlying cause
- IV Fluid



Ectopic Beats

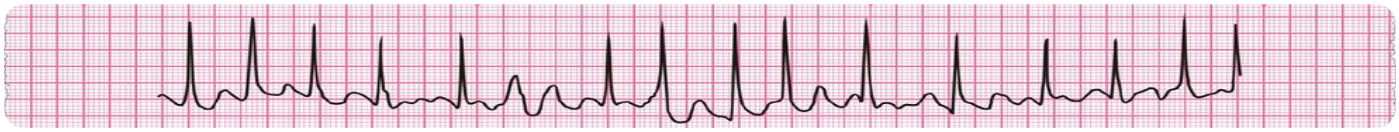
- Indication of cardiac irritation (Electrolyte, Fluid, or pH imbalance).
- Could be Artial or Ventricular related.

Causes:

- Hypoxia
- Electrolyte Imbalances
- Metabolic Acidosis (Drug Toxicity)

Treatment:

- Treat underlying cause



Atrial Fibrillation

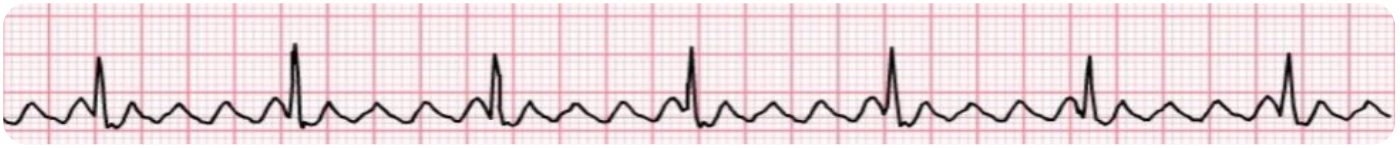
- Irregular, Rate 100-180bpm depending on AV conduction.
- Absent P Waves.

Causes:

- Hypoxia/Infection/PE
- Electrolyte Imbalances
- Chronic HTN / Diabetes / COPD

Treatment:

- Anticoagulant Therapy
- Rate Control (Digoxin/Amiodarone)



Atrial Flutter

- Saw-tooth appearance.
- Rate >100bpm.

Causes:

- Hypoxemia
- Pulmonary Embolism (PE)
- Myocardial Infarction
- COPD

Treatment:

- Anticoagulation
- Rate Control Medication
- Rhythm Conversion

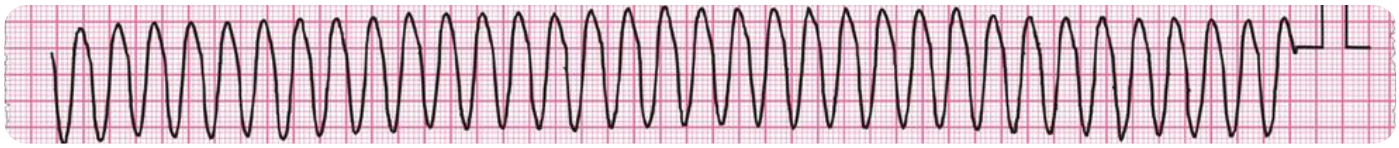
Cardiac Arrest

Life Threatening Cardiac Terminology

-Myocardial Infarction (MI) or 'Heart Attack' is the process of myocardial cell death due to ischemia, or perfusion imbalance between supply and demand within the coronary arteries, resulting from acute thrombosis.

-A Cardiac Arrest is where the HEART STOPS contracting effectively and ceases to pump blood.

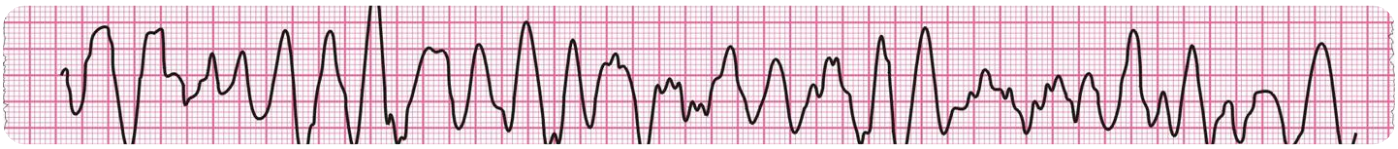
Life Threatening Cardiac Dysrhythmias



Ventricular Tachycardia (VT)
- Pulsed or Pulseless.
- Absent P Waves.
- Unstable Rhythm.

Causes:
- Electrolyte Imbalance
- Drug Intoxication
- Myocardial Infarction (MI)
- 4 Hs & 4 Ts (Pulseless)

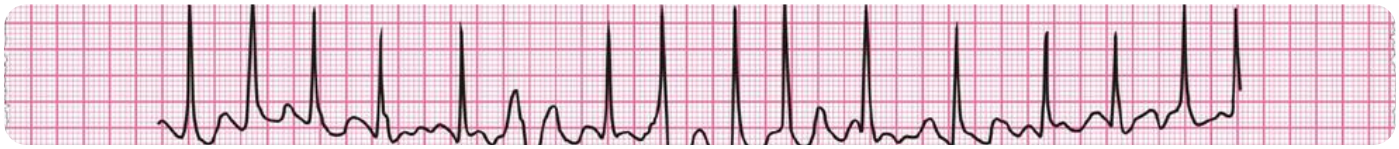
Treatment:
- Pulsed: Medication or cardioversion.
- Pulseless: BLS/ALS Algorithm.



Ventricular Fibrillation (VF)
- Irregular and Chaotic.
- Pulseless.
- Absent P-Q-R-S-T.

Causes:
- 4 Hs & 4Ts

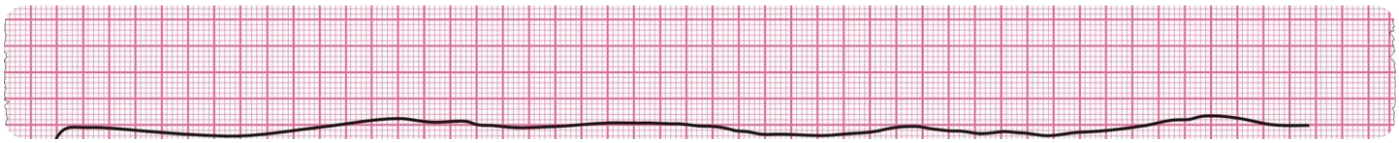
Treatment:
- Treat underlying cause
- BLS/ALS Algorithm



Pulseless Electrical Activity (PEA)
- Rhythm that is normally associated with an output.
- PULSELESS.

Causes:
- 4 Hs & 4Ts

Treatment:
- Treat underlying cause
- BLS/ALS Algorithm



Asystole
- Undulating baseline.
- No evidence of electrical activity.

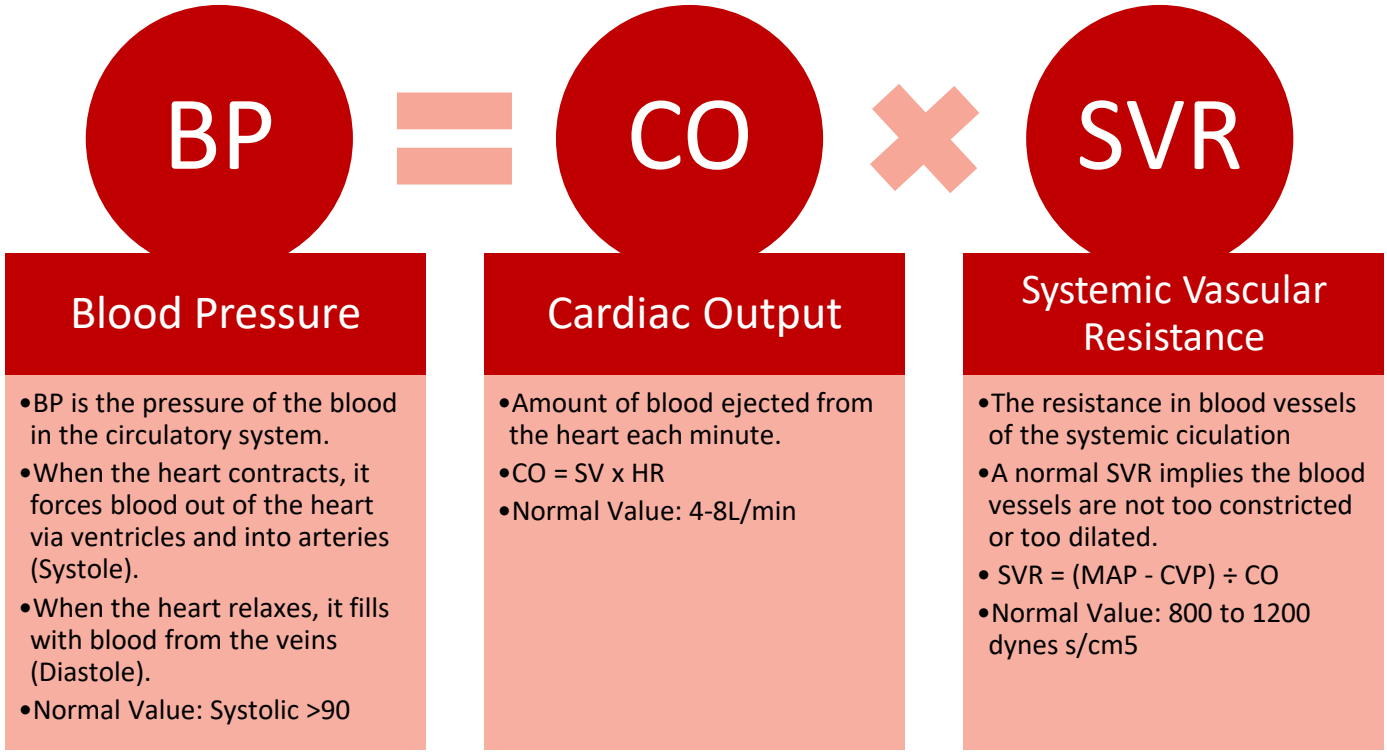
Causes:
- 4 Hs & 4Ts

Treatment:
- Treat underlying cause
- BLS/ALS Algorithm

4 Hs	4 Ts
• Hypoxia • Hypovalemia • Hypothermia • Hypo/hyperkalemia	• Tamponade • Thrombosis • Toxins • Tension Pneumothorax

Determinants of Blood Pressure

-The hemodynamic determinants of Blood Pressure (BP) include cardiac output (CO), which is governed by stroke volume (SV) and heart rate (HR), and systemic vascular resistance (SVR).



-Advanced haemodynamic monitoring can help inform your clinical decisions and optimise your patient from a cardiovascular perspective.

Mean Arterial Pressure (MAP) measures the flow, resistance, and pressure in your arteries during one heartbeat. The Normal MAP is 65–100 mm Hg.

A MAP of at least 60 mm Hg is necessary for adequate cerebral perfusion.

$$\text{Map} = \frac{\text{Systolic BP} + 2 \times \text{Diastolic BP}}{3}$$

-MAP is a product of flow (CO) and resistance (SVR).

-It is important to know that MAP is not always the sole indicator for perfusion or the delivery of oxygen.

-This can be demonstrated using the adjacent table.

	CO x SVR = MAP
Patient A	6 x 1200 = 72
Patient B	2 x 3600 = 72

-Patient A has a normal CO and normal SVR.

-However, despite achieving the same MAP, Patient B has a low CO and high SVR. This would indicate that they are on too many vasoconstrictive drugs and would benefit from some fluid.

Physiology Tree

-Follow the numbers to see what factors must be considered when assessing cardiovascular status.

② Perfusion is the delivery of oxygenated blood. When your patient is not perfusing, you may see ↑HR, ↑Lactate, ↓Urine output. This therefore affects your CO.

Delivery of Oxygen (DO2)

Oxygen delivery (DO2) refers to the amount of oxygen that is actually delivered to the tissues by the blood per minute.

Hb

③ Haemaglobin (Hb) is a protein found in RBCs. Hb contains iron, which allows it to pick up oxygen and transport it around the body. Particulary important for perfusion.

Oxygen

④ Oxygen is required for tissue perfusion. If there is evidence of hypoxia, there is likely evidence of poor perfusion. Oxygen interventions may be required.

CO

① CO = SV x HR.

⑥ Stroke Volume is the amount of blood ejected from the ventricle during systole per heartbeat. This can be affected by the three determinants below.

SV ✖ **HR**

⑤ HR is number of heart beats per minute.

Preload

Preload refers to the amount of myocardial fibre stretch or the amount of volume in the ventricles at the end of diastole.

Contractility

Contractility is the strength of the heart's contraction during systole.

Afterload

Afterload is the amount of pressure the heart must overcome to eject blood into the systemic circulation during systole.

A number of factors can influence these determinants of SV. See next page.

Intervention or Cause

FLUIDS

Intervention or Cause

DRUGS (Inotropes / Electrolytes)

Intervention or Cause

DRUGS (Vasopressors / Vasodilators)

Determinants of CO (CO = HR x SV (Preload, afterload, contractility).

-CO is determined by SV and HR.

-Manipulation of either of these factors will result in alteration to CO.

-CO can increase fivefold in healthy individuals to meet the metabolic demands of the body.

-CO can be assessed clinically as part of the ABCDE approach;

-Common signs of low cardiac output include; (C) cold clammy skin, prolonged capillary refill, tachycardia, and poor urine output (<0.5ml/kg/hour); (D) Confusion, restlessness and dizziness.

-Common signs of high cardiac output include; (C) warm / flushed appearance, tachycardia, bounding pulse, brisk capillary refill; pyrexia.

Low cardiac output will result in poor organ perfusion and lead to single/multi-organ failure if left untreated.

① Heart Rate

-Under normal circumstances the heart rate is determined by the rate of impulse generation in the sino-atrial (SA) node, this is the hearts pacemaker. This is in turn regulated by autonomic control.

② Preload

-Preload refers to the amount of myocardial fibre stretch or the amount of volume in the ventricles at the end of diastole.

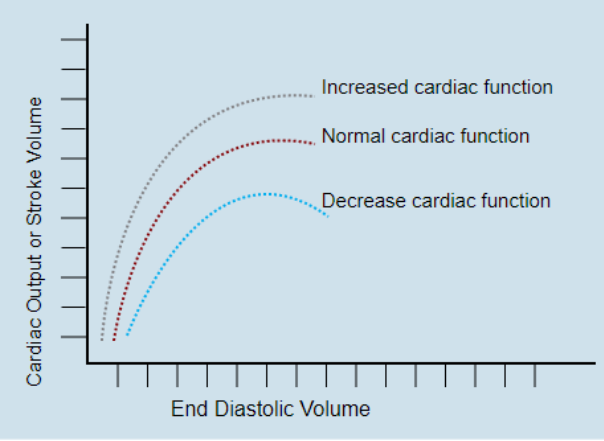
-A reduced volume in the ventricle at the end of diastole correlates with a reduced degree of stretch within the cardiac muscle fibres. This in turn leads to a decrease in cardiac output.

-An increase in preload has the potential to increase cardiac output.

-Factors influencing Preload;

Factors causing a reduction in Preload (Decreases in circulating volume)	Factors causing an increase in Preload (Increases in circulating volume)
• Hypovolemia • Vasodilation • Tachycardia • PEEP (Raised Intra-Thoracic Pressure)	• Hypervolaemia • Vasoconstriction • Bradycardia (Increases ventricular filling time) • Heart Valve regurgitation

Frank-Starling Law



-Frank and Starling identified the relationship between myocardial fibre length and force of contraction.

-The more the diastolic volume or fibres stretch at the end of the diastole, the stronger the next contraction during systole up to a physiological limit.

-It is important to remember that there is an optimal range of stretch, beyond which, force of contraction is reduced rather than increased e.g. fluid overload.

-In a clinical situation, when increased quantities of blood flow into the heart (increasing preload), the walls of the heart stretch.

③ Afterload

-Afterload is the amount of pressure the heart must overcome to eject blood into the systemic circulation during systole.

-Clinical conditions that lead to vasoconstriction will increase afterload as the left ventricle needs to generate a higher pressure in order to eject blood into the systemic circulation.

-Clinical conditions that lead to vasodilation will reduce afterload.

-Factors influencing Afterload;

Factors causing a reduction in Afterload (Vasodilation)	Factors causing an increase in Afterload (Vasoconstriction)
• Vasodilator Medication (e.g. Labetalol, GTN) • Sepsis • Anaphylactic Shock • Neurogenic Shock	• Vasopressors (e.g. Noradrenaline, Vasopressin) • Hypovolemia • Cardiogenic Shock • Arteriosclerosis • Heart Failure • Aortic Valve Stenosis

④ Contractility

-Contractility is the strength of the heart's contraction during systole.

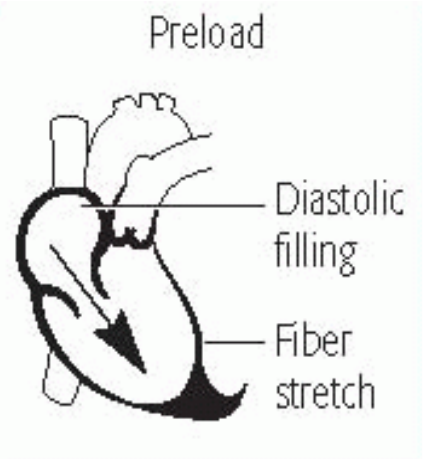
-Factors that influence contractility can either be;

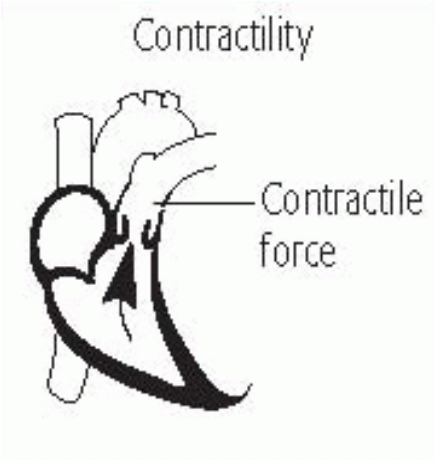
-Intrinsic (e.g. preload and afterload)

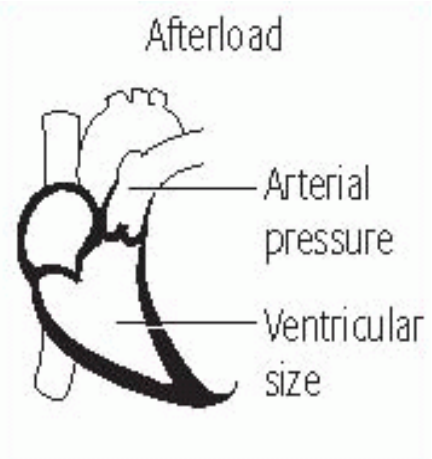
-Extrinsic (e.g. sympathetic responses from physical and emotional experiences, pathological effects such as ischaemia or chemical effects such as acidosis).

-Contractility of the heart may also be influenced by prescribed pharmacological agents.

Factors Decreasing Contractility	Factor Increasing Contractility
• Myocardial Ischemia / Damage • Hypocalcaemia • Hypovalemia • Hypoxia	• Inotropic drug therapy (e.g. Milrinone, Dobutamine) • Hypervolaemia • Hyperthyroidism • Catecholamine Release



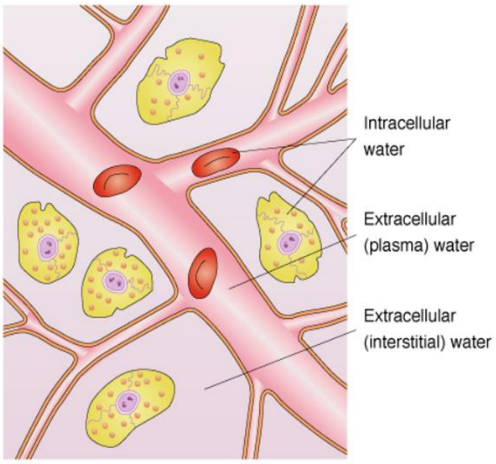




Fluid and Electrolytes

Fluid Compartments

Approximately 60-70% of Human Body Weight is composed of water.



-There are two major fluid compartments: Intracellular and Extracellular.

-Extracellular is then broken down into two compartments:

Intravascular and interstitial.

-Intracellular fluid is defined as all the body water within cells and are made up of protein, water, electrolytes, and solutes.

-The extracellular compartment is the fluid that lies outside of the cells.

-The extracellular compartment is further divided into two areas - intravascular (fluid inside the blood vessels) and interstitial (fluid outside the blood vessels and between the cells).

-Fluid movement is controlled through osmosis. Osmosis is water movement through a semipermeable membrane, from an area of lesser solute concentration to an area of greater solute concentration, in an attempt to equalize the solute concentrations on either side of the membrane.

Types of Fluid

Crystalloid

- First choice in fluid replacement therapy research has shown that crystalloids are superior in initial fluid resuscitation.
- Small molecules that can easily cross cell membrane.
- Up to two thirds of the infused volume will move into the tissues.
- Carry an increased risk of oedema.
- 0.9% Sodium Chloride, Hartmanns, Plasmalyte or Glucose Solutions

Colloid

- Colloids contain large molecules that don't pass through semipermeable membranes. Colloids stay in the intravascular spaces for longer than crystalloids.
- Because colloids pull fluids from the interstitial space to the vascular space, the patient is at risk for developing fluid volume overload.
- Albumin

Altered Fluid Status: Two Types of Fluid Imbalances that may necessitate Fluid Intervention.

Excessive Fluid Volume (Hypervolemia)

- Heart Failure
- Renal Failure
- Cirrhosis
- Pregnancy
- Symptoms include: pitting oedema, ascites, dyspnea and crackles from pulmonary oedema.

Deficient Fluid Volume (Hypovalemia)

- Diarrhoea
- Vomiting
- Pyrexia / Excessive Sweating / Sepsis
- Poor Fluid Intake
- Symptoms include: Headache, Dry Mouth, Dry Skin, Dark Concentrated Urine and Tachycardia.

Electrolytes and their Roles

Calcium

-Helps with muscle contractions, nerve signalling, blood clotting, cell division and forming/maintaining bones and teeth.

Electrolyte (Normal Adult Range)	Reasons for Abnormal Findings	
	Deficiency	**Excess**
Calcium ABG: 1.12-1.29mmol/L Lab: 2.20 – 2.59mmol/L	Hypocalcaemia • Hypoparathyroidism • Acute Pancreatitis • Hyperphosphatemia • Thyroid carcinoma • Vitamin D deficiency	Hypercalcaemia • Prolonged immobilisation • Hyperparathyroidism • Malignancy of bone

Potassium

-Keeps blood pressure levels stable, regulates heart contractions and helps with muscle functions.

Electrolyte (Normal Adult Range)	Reasons for Abnormal Findings	
	Deficiency	**Excess**
Potassium ABG: 3.70-5.30mmol/L Lab: 3.50 – 4.69mmol/L	Hypokalemia • Excessive loss through vomiting, urination, perspiration or diarrhoea • Use of Drugs • Poor intake of potassium	Hyperkalemia • Renal failure • Use of drugs • Excessive intake of potassium

Magnesium

-Needed for muscle contractions, proper heart rhythms, nerve functioning, bone building and strength, reducing anxiety, digestion and keeping a stable protein-fluid balance.

Electrolyte (Normal Adult Range)	Reasons for Abnormal Findings	
	Deficiency	**Excess**
Magnesium Lab: 3.50 – 4.69mmol/L	Hypomagnesemia • Excessive loss from GI tract • Use of Drugs • Chronic alcoholism • Diabetic ketoacidosis	Hypermagnesemia • Renal disease and failure • Treatment with magnesium and magnesium-containing medications

Sodium

-Helps maintain fluid balance, needed for muscle contractions, and helps with nerve signalling.

Electrolyte (Normal Adult Range)	Reasons for Abnormal Findings	
	Deficiency	**Excess**
Sodium ABG: 135-144mmol/L Lab: 135-144mmol/L	Hyponatremia • Excessive loss through diarrhoea, sweating, vomiting and diuretics. • Excessive water intake • Heady Injury	Hypernatremia • Deprivation of water • Excessive salt intake • Diabetes insipidus • Heat stroke

Chloride

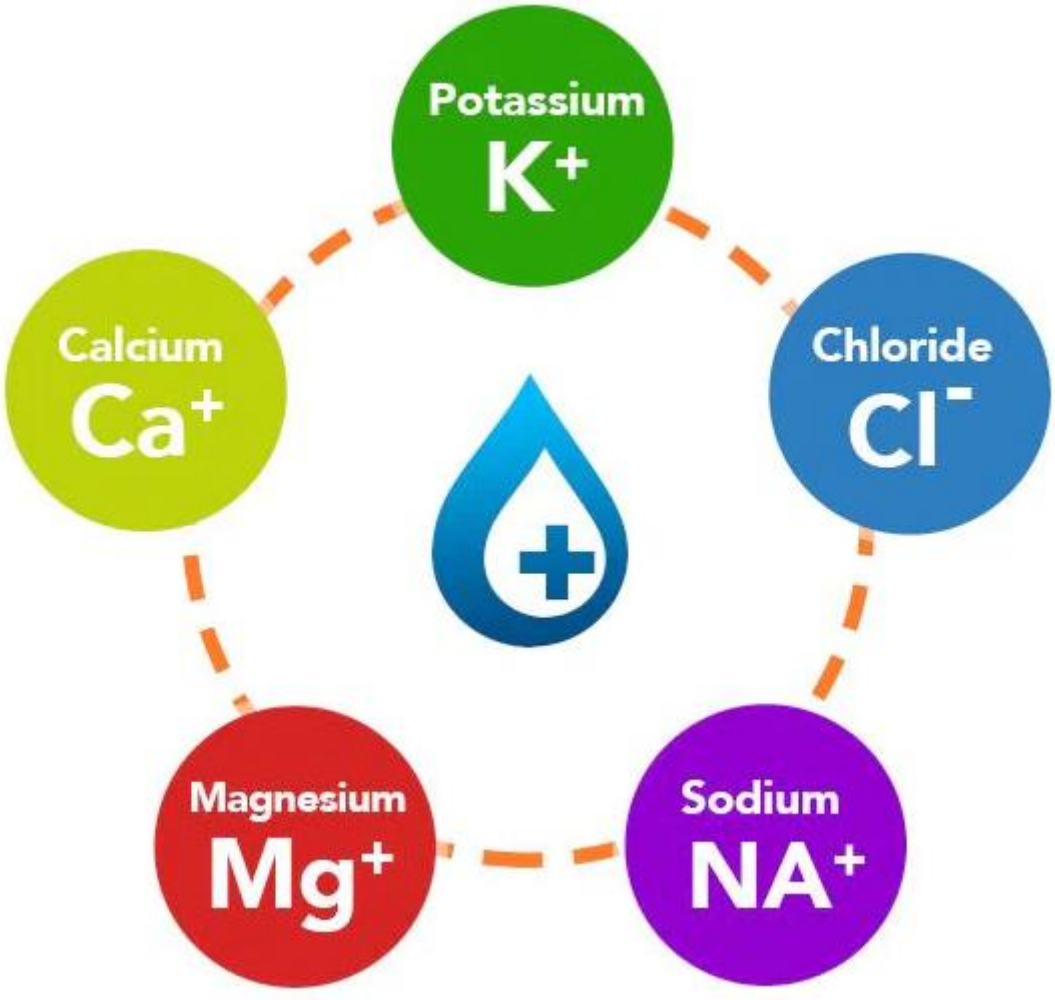
-Maintains fluid balance.

Electrolyte (Normal Adult Range)	Reasons for Abnormal Findings	
	Deficiency	**Excess**
Chloride Lab: 91-107mmol/L	Hypochloraemia • Diarrhoea • Vomiting. • Metabolic Alkalosis • Respiratory Acidosis	Hyperchloremia • Cardiac decompensation • Uraemia • Metabolic Acidosis • Respiratory Alkalosis

Phosphate

-Build and repair bones and teeth.

Electrolyte (Normal Adult Range)	Reasons for Abnormal Findings	
	Deficiency	**Excess**
Phosphate Lab: 0.69-0.93mmol/L	Hypophosphatemia • Alkalosis • Diabetes • Chronic alcoholism • Recovery from malnutrition	Hyperphosphatemia • Renal Failure • Hypoparathyroidism • Trauma • Heat Stroke

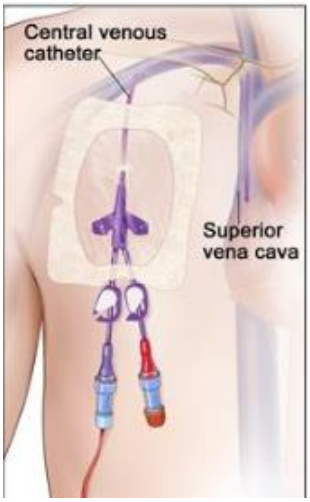


Central Venous Access / Pressure

-A Central Venous Access Device (CVAD) provides access to central blood vessels and are used for the delivery of IV medication, fluids, parenteral nutrition, and central venous pressure monitoring.

-Insertion Sites for CVADs include:

Right or Left Internal Jugular Vein	Right or Left Subclavian Vein	Right or Left Femoral Vein



-Placement via femoral veins avoided where possible. This is due to the increased risk of infection.

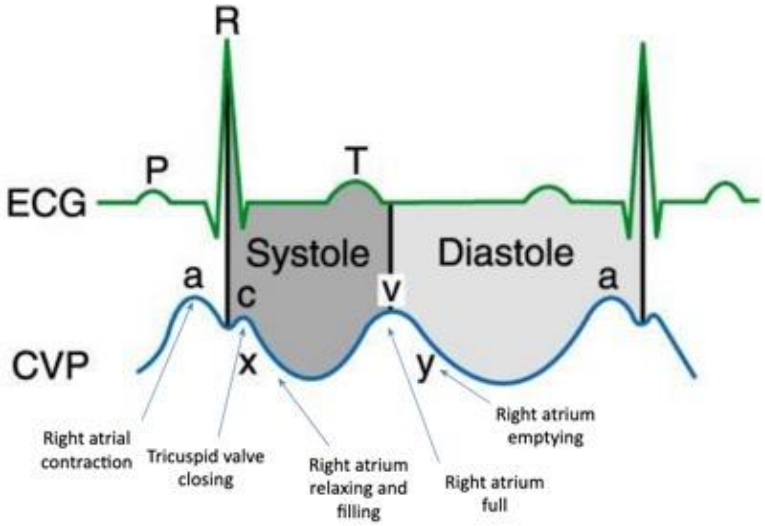
-PICC lines may also be inserted via the antecubital area or veins in the upper arm.

-The CVAD tip will always be placed in the proximal or distal superior vena cava, or proximal right atrium.

Central Venous Pressure (CVP)

- CVP is the systemic venous pressure at the level of the superior vena cava just before it enters the right atrium. It shows the mean right atrial pressure and it can indicate how well 'filled' your patient is, however it should not be used in isolation as can be inaccurate.

-Measured by lying patient flat and 'zeroing' CVP line. Should be measured at least once per shift.



Normal CVP Range: 2-16mmHg

CVP Waveform

-The peaks and troughs of the CVP waveform represent pressure changes in the right atrium.

-See adjacent image to indicate the appearance of a normal CVP waveform and what each section represents.

Determinants of Central Venous Pressure

-Its principal determinants are intravascular volume, right heart function, and venous tone.

Cardiac Competence	Blood Volume	Intra Aortic and Intra Peritoneal Pressure	Systemic Vascular Resistance
• Reduced Ventricular Function raises CVP • E.g. Heart Failure	• Increased Venous Return raises CVP • E.g. Over Hydration / Vasodilation • Decreased Venous Return lowers CVP • E.g. Hypovalemia / Fluid Depletion	• Tricuspic and Pulmonary Valve Disease • Raises CVP	• Increases in pressure / vasoconstriction • E.g. Vasopressors • Raises CVP

Associated Pharmacology

-There are several medications that help the cardiovascular system in different ways.
-First line treatment for hypotension is the administration of IV Fluids to increase circulatory flow.

Inotropes (Positive)

-Drugs that improve heart performance through increasing contractility.
-Positive inotropes make the heart muscle contractions stronger, raising cardiac output to a normal level and thereby increasing the amount of blood the heart can pump out.
-**Effects:** Increased CO | Increased Myocardial oxygen demand | Tachycardia | Does not necessarily ↑BP.
-**Cautions:** Active Ischemia | Can worsen hyperglycaemia | Lactic Acidosis.
-**Indications:**

Cardiogenic Shock	Septic Shock	Heart Failure	Pulmonary Hypertension	Post-Operatively after open-heart surgery	Bradycardia

-**Examples include:**

Dobutamine	Milrinone	Dopamine

Vasopressors

-Increase blood pressure through vasoconstriction.
-Vasopressors increase systemic vascular resistance (SVR), reducing the diameter of blood vessels and therefore reduces the amount of blood required to fill them to achieve an acceptable BP.
-**Effects:** Increased Preload / Afterload | Increased MAP | Increased Myocardial oxygen demand.
-**Cautions:** Hypovolemia | Peripheral Ischemia | Excessive vasoconstriction.
-**Indications:**

Vasodilation (E.g. Sedation / SIRS)	Cardiogenic Shock	Management of Raised ICP	Anaphylaxis

-**Examples include:**

Noradrenaline	Vasopressin / Argipressin	Metaraminol	Adrenaline

MAP = CO x SVR

Inotropes increase CO by increasing HR and Stroke Volume

CO = HR X SV

SV depends on preload and afterload.
(SV will increase to a certain point, before the muscle fibres become overstretched and there is a failure (i.e. Starling's Law)).

Vasopressors increase SVR/afterload by causing resistance to flow.

Vasodilators

-Reduces blood pressure through vasodilation.

-Vasodilators affect the muscles in the walls of the arteries and veins, preventing the muscles from tightening and the walls from narrowing. Blood flows more easily through the vessels and the heart doesn't have to pump as hard, reducing blood pressure.

-**Effects:** ↓SVR | Reduced pressure in blood vessels or on heart.

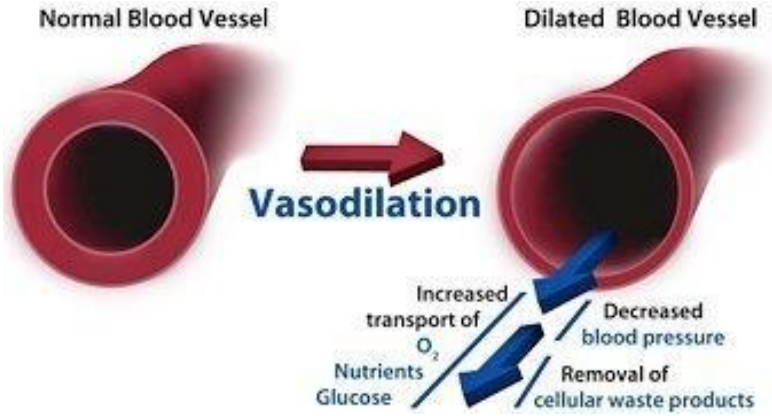
-**Cautions:** Hypotension | Bradycardia

-**Indications:**

Angina	Hypertension	Preeclampsia or Postpartum Preeclampsia	Pulmonary Hypertension	Heart Failure

-**Examples include:**

Labetalol	GTN	Nicardipine	Esmolol	Hydralazine



Anti-Arrhythmic Medication

-Drugs that prevent and treat a heart rhythm that's too fast or irregular.

-They reset your heart to a normal rhythm or prevent episodes of arrhythmia by acting on the heart's various electrical channels.

-**Effects:** If effective, controlled heart rhythm.

-**Cautions:** Other Arrhythmias | Cardiogenic Shock.

-**Indications:**

Atrial Fibrillation (AF)	Atrial Flutter	Ventricular Tachycardia (VT)	Ventricular Fibrillation (VF)

-**Examples include:**

- Beta Blockers slow down the heart rate, often by blocking hormones such as adrenaline. They reduce the heart's workload and the hearts output of blood and so can also reduce a patient's blood pressure.
- Potassium Channel Blockers prevent potassium from getting through cell membranes, which can slow down electrical impulses in all the heart's cells.

Beta Blocker: Atenolol	Beta Blocker: Bisoprolol	Beta Blocker: Metoprolol	Potassium Channel Blocker: Amiodarone	Digoxin (↓HR & ↑Contractility)

Anti-Hypertensive Medication

-Drugs that reduce blood pressure.

-**Cautions:** Hypotension | Monitor electrolytes | Monitor HR.

-**Indications:**

Hypertension

-**Effects and Examples include:**

- ACE inhibitors help the body produce less angiotensin, which helps the blood vessels relax and open up, which, in turn, lowers blood pressure.
- Alpha Blocker drugs reduce the arteries' resistance, relaxing the muscle tone of the vascular walls.
- Calcium Channel Blockers prevent calcium from entering the smooth muscle cells of the heart and arteries. When calcium enters these cells, it causes a stronger and harder contraction, so by decreasing the calcium, the hearts' contraction is not as forceful. Calcium channel blockers relax and open narrowed blood vessels, reduce heart rate and lower blood pressure.

Beta-Blocker: Atenolol	Beta-Blocker: Bisoprolol	Beta-Blocker: Metoprolol	ACE Inhibitor: Ramipril	Calcium Channel Blocker: Amlodipine	Calcium Channel Blocker: Nicardipine
	Alpha Blocker: Doxazosin	Combined Alpha/Beta Blocker: Labetalol	GTN	IV Hydralazine (Vasodilator)	

Diuretics

-Drugs that help the body get rid of excess sodium (salt) and water and help control blood pressure.

-**Effects:** Increased Urine Output | Reduced Potassium level.

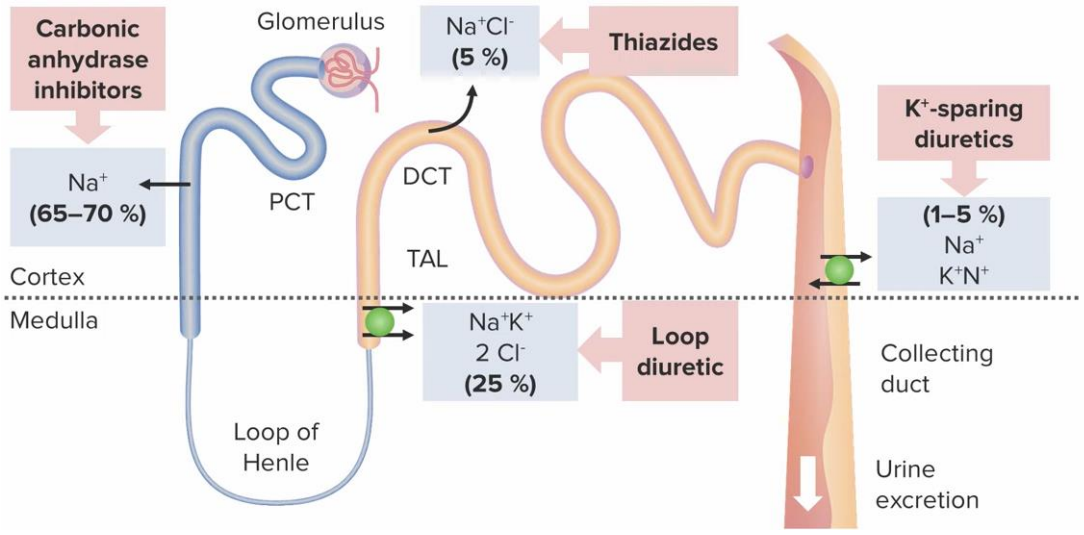
-**Cautions:** Hypotension | Electrolyte Imbalance.

-**Indications:**

Post Acute Phase Shock (Fluid Overload)	Heart Failure	Pulmonary Hypertension	Post-Operatively after open-heart surgery

-**Examples include:**

Loop Diuretic: Furosemide	Loop Diuretic: Bumetanide	Thiazide Diuretic: Bendroflumethazide	Thiazide Diuretic: Metalozole	Potassium-Sparing Diuretic: Spironolactone	Potassium-Sparing Diuretic: IV Potassium Canrenoate



Advanced Haemodynamic Monitoring - LIDCO

Heart Rate

- The rate at which the myocardium contracts as determined by the rate of impulse generation in the sinoatrial node.
- Normal Range: 60-100bpm

CVP

- Systemic venous pressure at the level of the superior vena cava just before it enters the right atrium.
- Normal Range: 2-16mmHg

Delivery of Oxygen (DO2)

- The amount of oxygen in the blood leaving the heart and is dependent upon
  ① Gas exchange
  ② Haemoglobin level
  ③ Cardiac output
- An inadequate DO2 can lead to organ dysfunction, multiorgan failure and death.
- Normal Value: 900-1100ml/min

Oxygen Consumption (VO2)

- Oxygen taken up by the cells to sustain cellular metabolism and is dependent upon DO2
- Ability of cells within the tissues to extract the oxygen.
- Normal Value: 200-290ml/min
- During critical illness value can rise to 700-800ml/min.

Stroke Volume (SV)

- The amount of blood ejected from the ventricle during systole per heartbeat.
- Normal Range: 60-100mls/beat

Stroke Volume Index (SVI)

- Stroke volume indexed to patient body surface area.
- Normal Range: 33-47ml/m2/beat

Cardiac Output (CO)

- This is the volume of blood from each ventricle every min.
- 4-8L/min

Cardiac Index (CI)

- Cardiac output is commonly individualised to body size by dividing the cardiac output by the body surface area.
- 2.5-4l/min/m2

Systemic Vascular Resistance (SVR)

- The resistance that is generated by the systemic vasculature (incl pulmonary circulation). Increase in SVR = increase afterload = increase workload = decrease in stroke volume.
- Normal Range: 800-1200dynes.sec/cm5

Systemic Vascular Resistance Index (SVRI)

- Individualised (patient specific) index of systemic vascular resistance generated by dividing the SVR by CI.
- Normal Range: 1970-239dynes.sec/cm5/m2

Stroke Volume Variation (SVV)

- Is the change in the amount of blood ejected from the left ventricle into the aorta with each heartbeat.
- This is reflected by arterial blood pressure changes in relation to the pattern of respiration (arterial swing being a key factor)
- ***For ventilated patients only.***
- Determinant of fluid responsiveness.
- Normal Range: <10% unlikely to be preload responsive. >13-15% likely to be preload responsive.

Effects of Ventilation on the Cardiovascular System: Delivery of Oxygen (DO2)

-The prime function of the cardiovascular and respiratory system is the transportation of oxygen and nutrients to organs and tissues to meet metabolic demand.

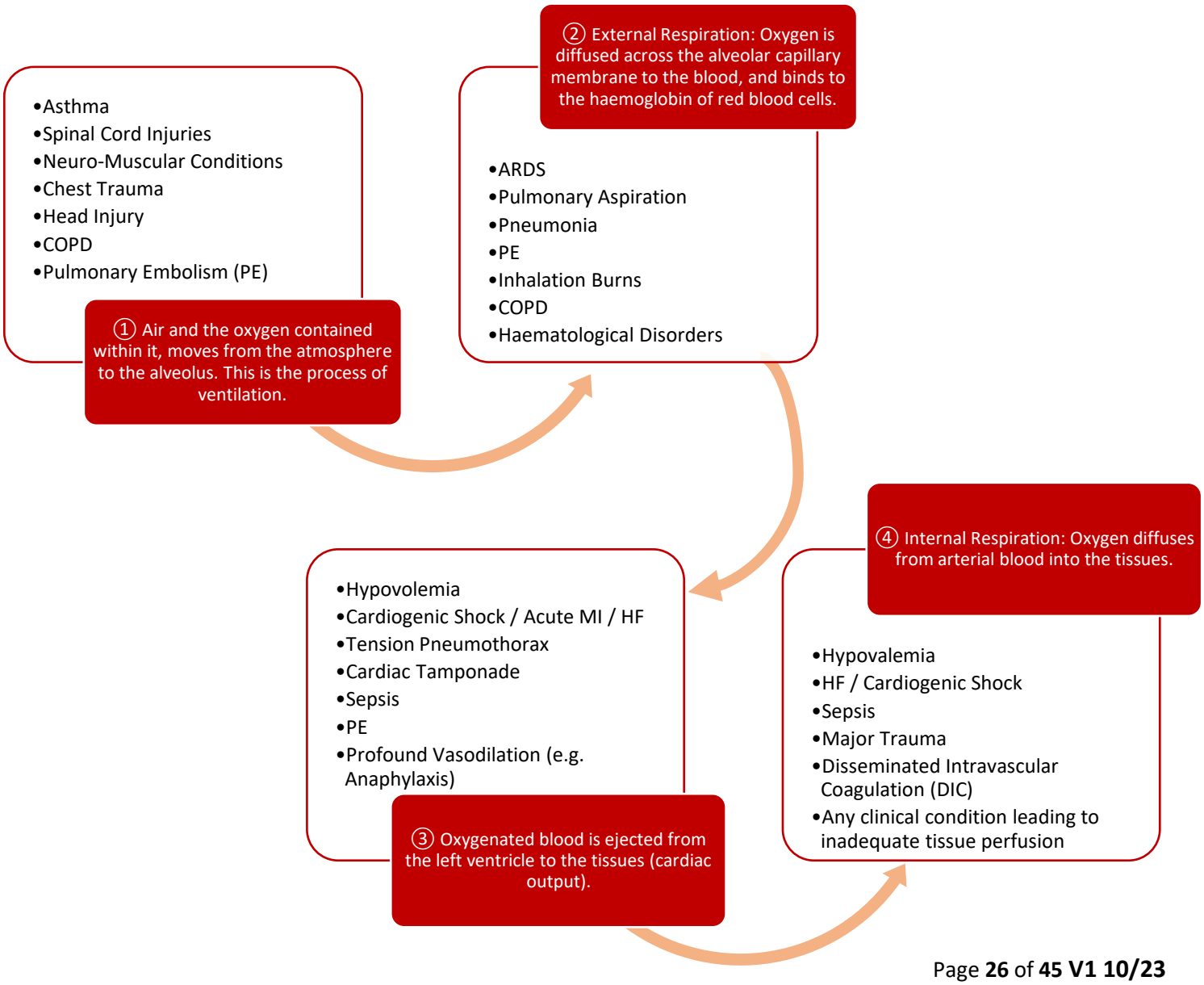
-Any patient with a compromised cardiovascular system may have poor tissue perfusion and inadequate tissue oxygenation. Ensuring that tissues are well perfused with oxygen should be a target for treatment in the critically ill patient to prevent tissue hypoxia and organ failure.

-In health, oxygen supply and demand are well matched. Increased demand, for example during exercise, is rapidly balanced by an increase in oxygen delivery through raised cardiac output.

-Oxygen debt occurs when cell oxygen requirements are greater than delivery, or an impaired cell ability to extract oxygen.

-Optimisation is the prevention of a prolonged oxygen debt by maximising oxygen delivery and consumption.

<u>The process by which oxygen moves from the atmosphere and into the tissues and conditions which affect each stage and may lead to impaired oxygen delivery.</u>



Delivery of Oxygen (DO2) refers to the amount of oxygen that is delivered to the tissues by the blood per minute. It depends on blood flow and oxygen carrying capacity of the blood.

Oxygen delivery is affected by pulmonary gas exchange, haemoglobin levels, oxygen saturation and cardiac output.

The Normal DO2 is 90–1100 ml/min.

$DO_2 = CO \times CaO_2 (1.34 \times Hb \times SaO_2) \times 10$

-CaO_2 is the arterial oxygen content.

-It considers the following:

-1.34 is the amount in mls of oxygen carried by 1 g of haemoglobin (Hb).

-SaO_2 is the oxygen saturation of arterial blood.

-Multiplying by 10 ensures oxygen content is in ml/litre and not ml/dl of blood.

From this equation, there are three physiological variables; cardiac output, haemoglobin count and arterial oxygen saturation, which can be manipulated to improve oxygen delivery.

Optimisation

Respiratory	Cardiovascular
• Maintain Arterial SaO_2 >94% • Regular Chest Physiotherapy • Clearance of Bronchial Secretions • Humidification of Oxygen • Drug Therapy, such as Nebulisers	• Optimise Preload • Reduce Afterload • Increase Contractility • Maintain Haemoglobin >85g/L

Ventilation / Perfusion Mismatch

-Gas exchange in the pulmonary capillaries is not always efficient.

-Blood may return to the left side of the heart without being adequately oxygenated.

-This may be caused by several conditions that affect either ventilation or pulmonary perfusion, such as those identified on the previous page.

-When blood returns to the left side of the heart without being oxygenated, it is referred to as a ventilation/perfusion mismatch or shunt.

-This has implications for oxygen delivery

Oxygen Consumption (VO2) refers to the amount of oxygen removed from the blood for use by the tissues per minute.

The Normal VO2 is 200–290 ml/min.

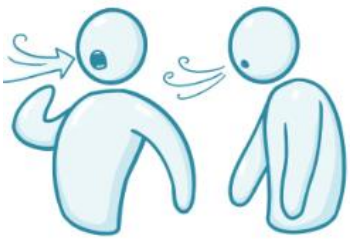
VO2 = DO2 – Venous Oxygen Return

-To assess the balance between oxygen supply and demand, consideration has to be given to consumption or utilisation of oxygen by the tissues.
-In a healthy person, the total consumption of oxygen per minute (VO2) is constant over a wide range of oxygen delivery values. This value is determined by measuring the amount of oxygen delivered on the arterial side compared to the amount returned on the venous side.

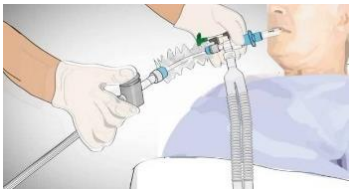
Venous oxygen return is determined by measuring the O2 saturation of a mixed venous blood sample from a central venous catheter.

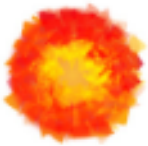
-Normal venous saturations should be 75%.
-Patients in a shocked state may have reduced venous saturations (<75%) due to oxygen demand being greater than oxygen delivery.

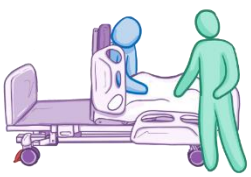
-It is important to understand the conditions and activities that can affect oxygen demand and consumption, and take steps to alleviate these in the critically ill patient.
-The table below highlights some examples.

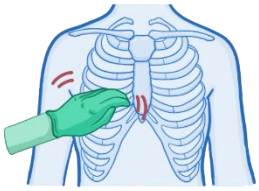


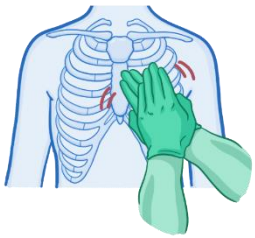
Condition and Activities	% Increase in Oxygen Demand
Shivering	50-100%
Sepsis	50-100%
Work of Breathing	40%
Chest Physiotherapy	35%
Position Change	31%
Endotracheal Suction	27%
Visitors	22%
Fever (of 1°C)	10%











Sepsis

'Sepsis is characterised by a life-threatening organ dysfunction due to a dysregulated host response to infection.'

Sepsis Pathophysiology

-Widespread activation of inflammatory cascades and cytokines in response to infection.
-This causes vasodilation and increased vascular permeability resulting in a redistribution of circulating blood volume and subsequent hypotension.
-There is also widespread activation of the clotting cascade with depletion of clotting factors and subsequent disturbance of coagulation.

****SEPSIS CLAIMS MORE LIVES THAN LUNG CANCER, AND MORE THAN BOWEL, BREAST AND PROSTATE CANCER COMBINED****

PREVIOUS Sepsis Terminology / Guidance

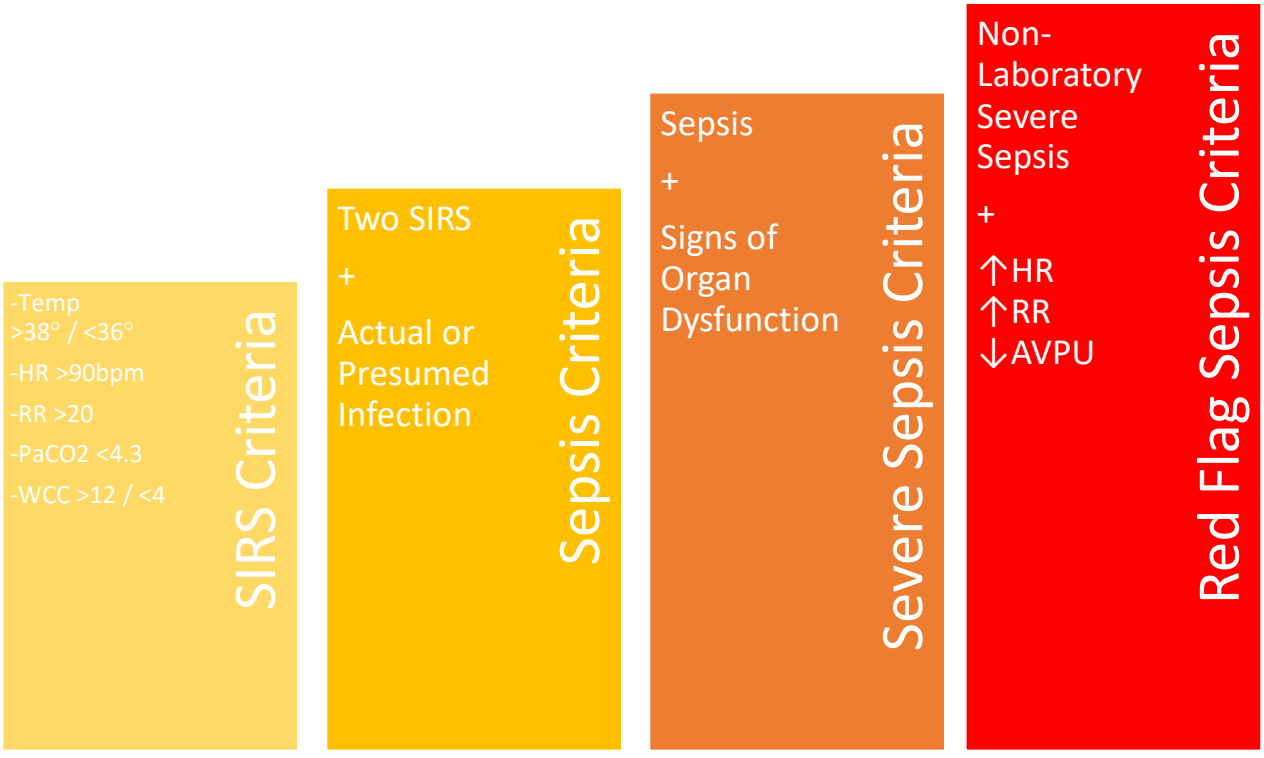
(Currently in Step Competency Framework but will be updated to reflect 2016 Sepsis Guidance (See next page)).

-Systemic Inflammatory Response Syndrome (SIRS).
  -Characterised by ≥2 criteria exceeding thresholds for temperature, HR, RR and WCC.
  -Formerly used in combination with infection to identify 'sepsis' but now discarded as often represents an appropriate (i.e. non-pathological) host response to any inflammatory (i.e. non-specific for infection) insult.

-Sepsis - Life-threatening organ dysfunction caused by a dysregulated host response to infection.

-Severe Sepsis - Outdated terminology combining SIRS + organ dysfunction; now replaced by 'sepsis'.

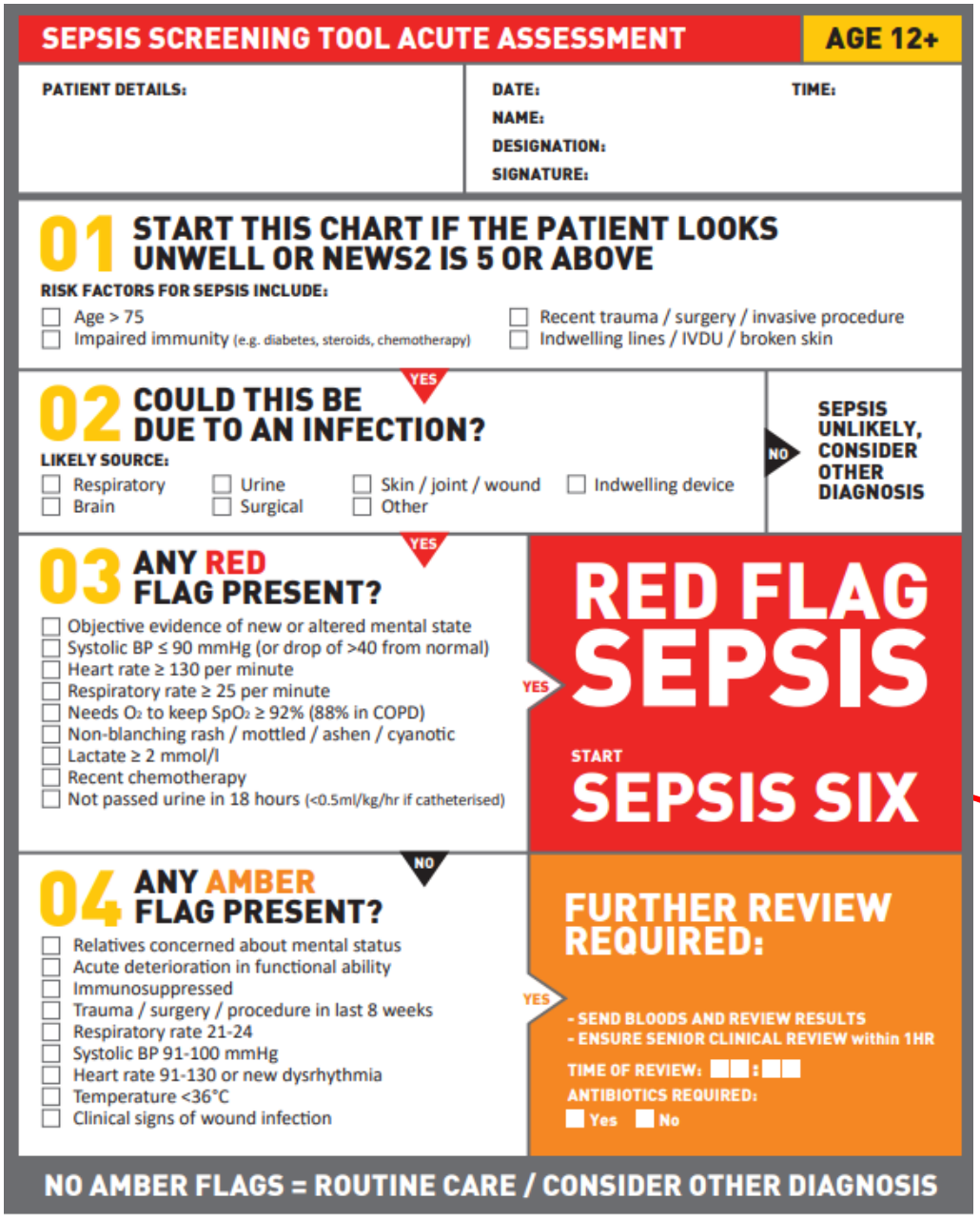
-Red Flag Sepsis - A term used to describe the presence of any one or more Red Flag criterion.

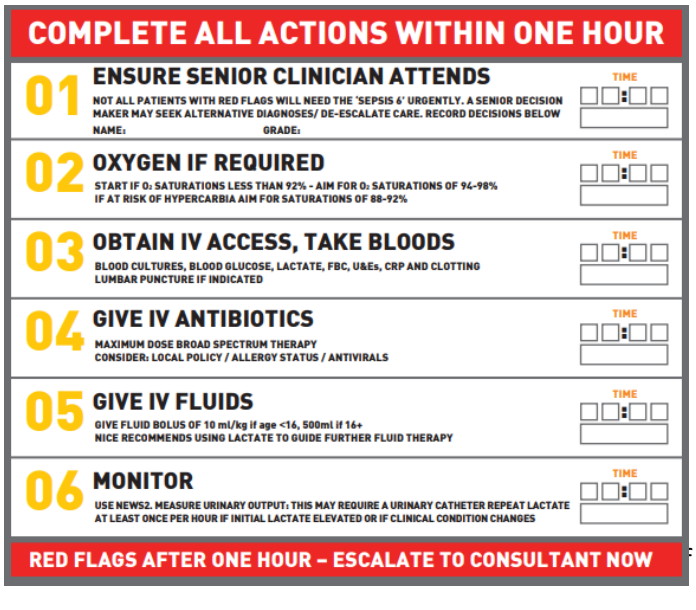


2016 Guidance for Sepsis Screening / Treatment

(Please visit [Sepsis-Manual-Sixth-Edition.pdf (sepsistrust.org)] for more information).

Red Flag Sepsis is not a formal 'diagnosis' of sepsis: it is a bedside tool that suggests it is highly likely the patient has a degree of organ dysfunction, and which empowers health professionals to act.





Shock

-Shock occurs when there is inadequate blood flow (CO) and oxygen delivery (DO2) to meet demands.
-Shock can be broken into four categories: Distributive, Obstructive, Cardiogenic, and Hypovolemic.

Distributive Shock

-Distributive shock or vasodilatory shock is a medical emergency where the body can't get enough blood to the heart, brain, and kidneys. This happens because blood vessels are extremely dilated, which causes hypotension and reduces how much blood can get to organs (affecting SVR).

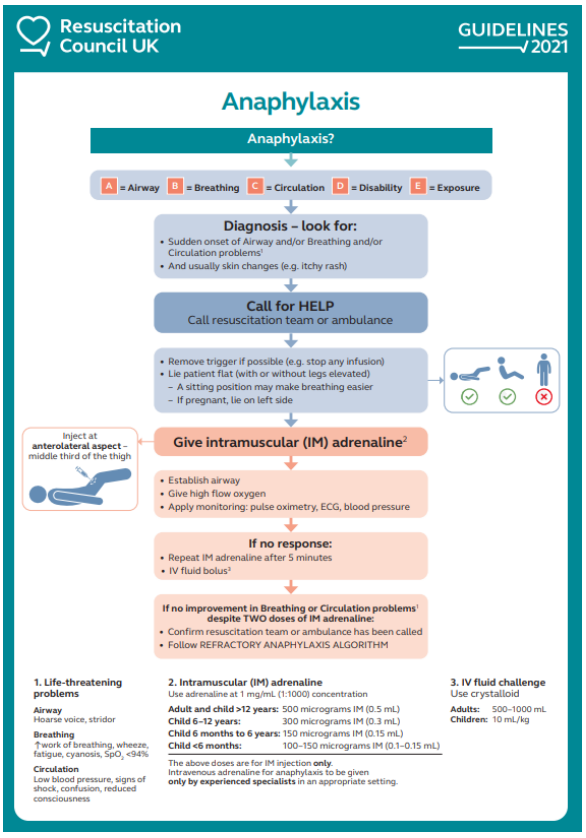
There are Three Types of Distributive Shock:

Anaphylactic Shock | Septic Shock | Neurogenic Shock

Anaphylactic Shock

-Anaphylactic shock is a complication of a severe allergic reaction known as anaphylaxis.
-Allergic reactions occur when your body mistakenly treats a harmless substance as harmful, and this triggers a dangerous immune response.
-**Anaphylaxis is characterised by:**
•Sudden onset and rapid progression of symptoms.
•Airway and/or Breathing and/or Circulation problems.
•Usually, skin and/or mucosal changes (flushing, urticaria, angioedema).
-**Treatment:**
-Call for Help Early.
-Use the ABCDE approach to recognise and treat problems.
-Resuscitation Council UK Anaphylaxis Algorithm.

Emergency treatment of anaphylactic reactions: Guidelines for healthcare providers | Resuscitation Council UK

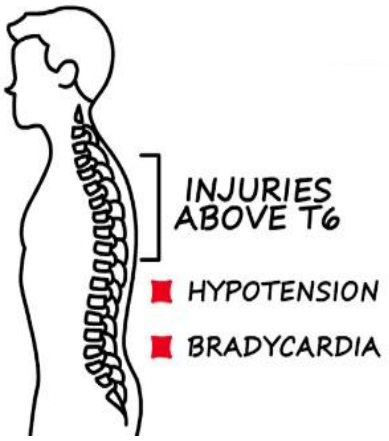


Airway problems	Breathing problems:	Circulation problems:
• Airway swelling (throat and tongue swelling causing difficulty in breathing/swallowing; patients may feel their throat is closing) • Hoarse voice • Stridor (a high-pitched inspiratory noise caused by upper airway obstruction)	• Increased work of breathing • Bronchospasm (wheeze) and/or persistent cough • Patient becoming tired with the effort of breathing (fatigue) • Hypoxaemia (SpO_2 <94%) which may cause confusion and/or central cyanosis • Respiratory arrest	• Signs of shock: o pale, clammy o significant tachycardia (increased heart rate) o hypotension (low blood pressure) • Dizziness, decreased conscious level or loss of consciousness • Arrhythmia • Cardiac arrest

Septic Shock (see pages 29 and 30).

-Septic shock occurs when bacteria and their toxins cause serious damage to tissues/organs in the body.
-**Treatment:** Sepsis Six.

Neurogenic Shock



-Neurogenic shock is caused by damage to the central nervous system.
-The autonomic nervous system is damaged resulting in the blockage of the sympathetic nervous system, which is supposed to speed up the vitals and vasoconstrict. Only the parasympathetic system is intact, which puts the breaks on the vitals, causing widespread vasodilation and hypotension.
-We see hypotension and bradycardia as vasodilation makes it difficult for blood to return to the heart and leads to decreased blood flow out of the heart (↓CO).
-Decreased CO means less oxygenated blood is being delivered to organs, leading to poor organ perfusion and impaired cell metabolism which if left untreated can result in multi-organ failure and death.

-**Symptoms:**

- -Hypotension
- -Bradycardia
- -Flushed, warm skin that gets cold and clammy later.
- -Blue Lips and Fingernails.
- -Lack of full consciousness.
- -Body Temperature Changes.

-**Causes:**

- -Spinal Cord Injury (Most common cause).
- -Spinal Anaesthesia.
- -Cerebral Ischemia / Subarachnoid Haemorrhage.
- -Guillain-Barré Syndrome.
- -Autonomic Nervous System Toxins.
- -Meningitis.

(A CT or MRI is likely required to diagnose neurogenic shock).

-**Treatments:**

- -Stabilise the spine. Cervical/neck collar to avoid further spinal injury.
- -Fluids for hypotension / Medication for bradycardia.

Neurogenic shock symptoms can last four to five weeks.

Obstructive Shock

-Obstructive shock is a condition that prevents blood and oxygen from getting to your organs.

-**Symptoms:**

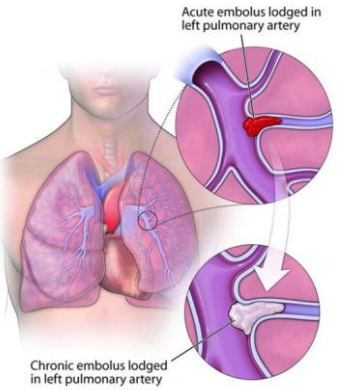
- -↑RR.
- -Tachycardia.
- -Cool and clammy skin.
- -Hypotension.
- -Altered consciousness.
- -Chest or Abdominal Pain.

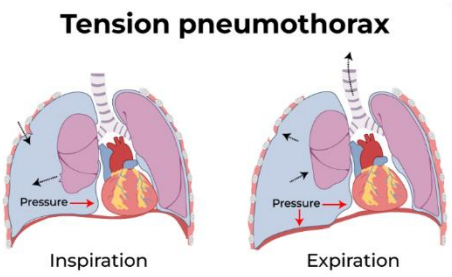
-**Causes:**

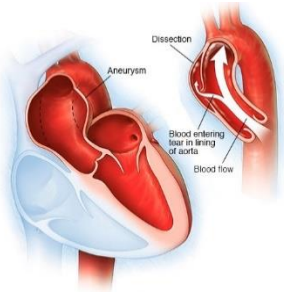
- -Tension Pneumothorax.
- -High PEEP.
- -Pericardial Tamponade.
- -Vena cava compression syndrome.
- -Thromboembolism in the pulmonary artery.
- -Aortic Dissection.

-**Treatments:**

- -Physical Exam / ABCDE Assessment. Treat cause of obstructive shock.







Cardiogenic Shock

-Life- threatening condition where the heart is unable to pump enough blood to rest of the body.

-The hearts lack of ability to pump the blood properly will lead to the back up of blood and congestion in the lungs and right side of the circulatory system. This affects CO and tissue perfusion, despite adequate intravascular volume in the cardiovascular system.

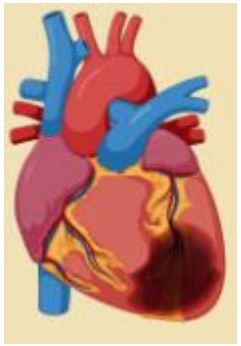
-**Symptoms:**

-Chest Pain that lasts for more than a few minutes or goes away and comes back.

-Pain or discomfort in upper body / down left arm.
-Sweating or cold sweats.
-Feeling very weak, lightheaded, confused / anxious.
-Trouble Breathing.
-Fast or Irregular HR.
-Hypotension / Tachycardia.

-**Causes:**

-Myocardial Infarction (Most common cause).
-Endocarditis.
-Cardiac Tamponade.
-Myocarditis,
-Arrhythmia.
-Pulmonary Embolism.



-**Treatments:**

-Fluids and medications to improve contractility and CO (e.g., Inotropes and vasopressors).
-Diuretics to remove excess fluid and help improve blood flow.
-Potential surgical procedure depending on initial cause of cardiogenic shock.

Hypovolemic Shock

-Hypovolemic shock is a dangerous condition in which there is a decrease in the intravascular blood volume to such an extent that effective tissue perfusion cannot be maintained.

Symptoms:

-Weakness / Fatigue / Dizziness.
-Tachycardia (Weak and Thready Pulse).
-Confusions / Restlessness / Anxiety.
-Hypotension.
-Rapid / Shallow breathing.
-Oliguria.

-**Causes:**

-Bleeding/Trauma.
-Burns.
-Vomiting / Diarrhoea.

-**Treatments:**

-Treat underlying cause of the severe bleeding or fluid loss.
-Fluid resuscitation / O2 therapy / Blood transfusion if applicable.
-Position: Supine with the legs elevated.

LiDCO

Lithium Dilution Cardiac Output Monitoring

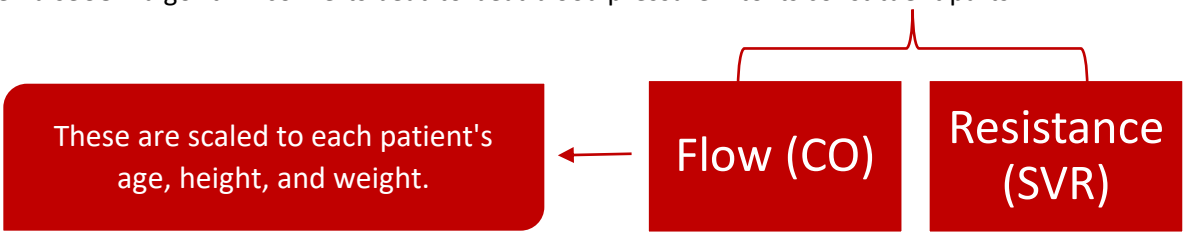
LiDCO Monitors

LiDCO
-Lithium Dilution Cardiac Output Monitoring.

What is LiDCO?

- Non-invasive cardiac output monitoring is the concept of analysing cardiovascular function and performance to optimize fluid status and guide titration of pharmacological management.

LiDCO Monitors within Critical Care
-There are two modes to choose when considering LiDCO; Non-Calibrated and Calibrated.
-The PulseCO™ algorithm converts beat-to-beat blood pressure into its constituent parts.



Trend Notifications

Alerts user to significant haemodynamic changes (>10%) to encourage an immediate response to patient deterioration

Internal Battery

For portability around the bed space and seamless transition to different clinical areas

Short-term Trend

2-minute window for greater focus on the immediate response to interventions

Day/Night Mode

Switch between day and night mode to best suit your environment

Guided Protocols

To help you assess fluid responsiveness (Fluid Challenge, Passive Leg Raise and New Ventilator Tests)

Long-term Trend

Facilitates interpretation of trends from the start of monitoring, which can be customised to show only the parameters you need

Education

On-screen educational screens for calibration

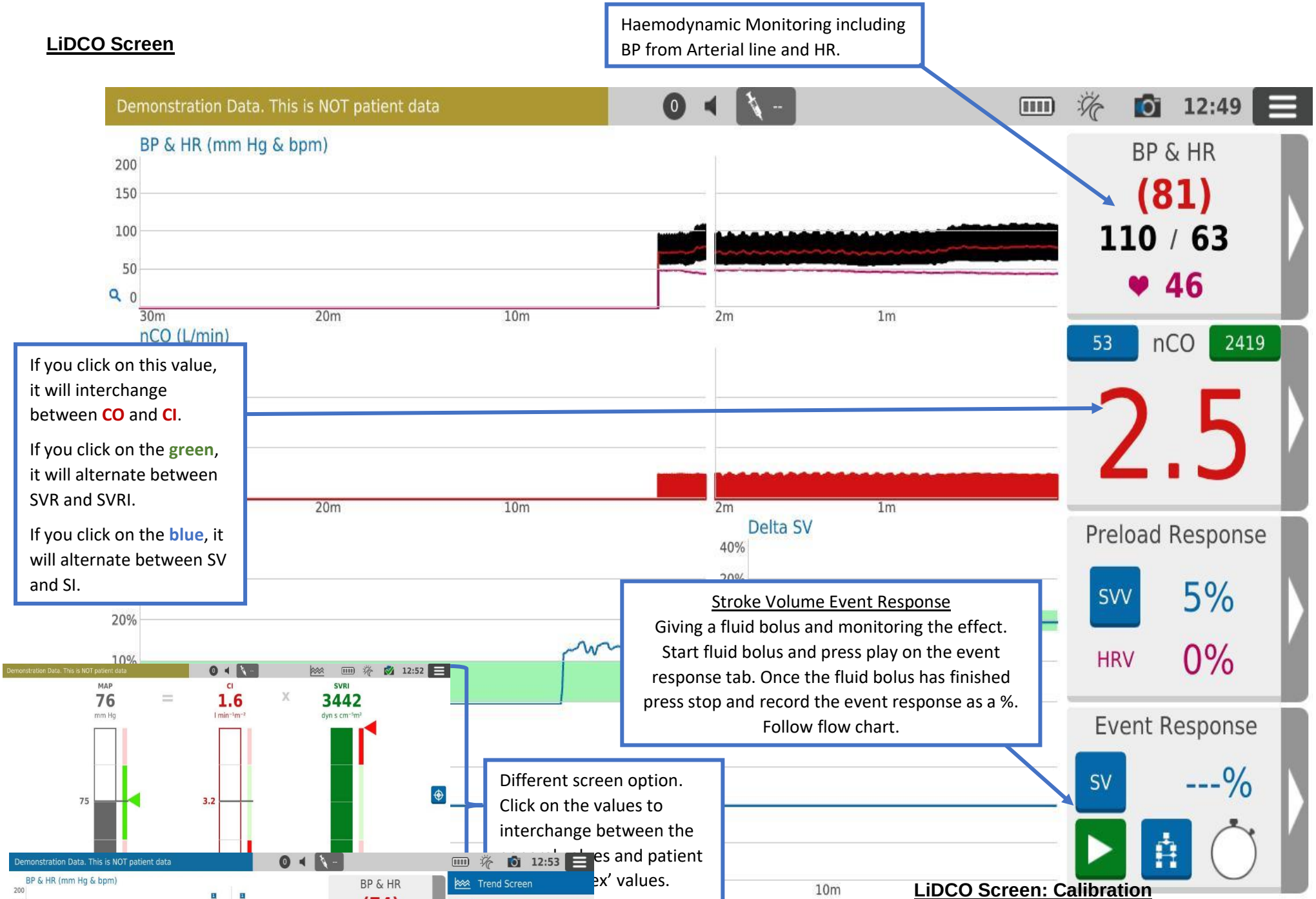
Event Response

Allows you to mark and monitor specific events, like a fluid challenge

Preload Response

Displays volume status indications for Pulse Pressure Variation (PPV%) and Stroke Volume Variation (SVV%)

LiDCO Screen



LiDCO Screen: Calibration

Uncalibrated LiDCO

Uncalibrated LiDCO

- Uses arterial waveform analysis and the PulseCO algorithm to convert blood pressure to its constituent parts of flow (CO, SV) and systemic vascular resistance (SVR).
- Displays nominal values since they are estimated rather than directly measured.
- May not be as accurate when used in patients with past medical history such as chronic hypertension, arteriosclerosis and diabetes mellitus.
- Generally used to monitor trends following an intervention such as a fluid bolus or vasopressors.
- MUST have a reliable Arterial Line.
- Used predominantly in the initial 12-24hrs after high-risk surgery.

Calibrated LiDCO

Calibrated LiDCO

- Combines single point lithium indicator calibration with arterial waveform analysis.
- Lithium is used as a bolus indicator to calibrate the PulseCO software.
- A small dose of lithium chloride is injected via a designated lumen on the central line, using the park and ride line. This results in a lithium concentration-time curve which is recorded by withdrawing blood past the lithium sensor attached to the patients existing arterial line.
- Provides a real-time continuous display of haemodynamic parameters and enables effective and accurate titration of fluids and drugs.
- MUST have an Arterial Line and reliable central (preferable) or peripheral venous access.

*Lithium calibration should not be performed within 120 minutes after either an atracurium or rocuronium bolus. Calibration should not be performed for at least two hours after stopping the infusion and the presence of a train-of-four count checked.

Key Points

-Lithium calibration should be avoided during haemodynamically unstable periods.
-LiDCO recommends that re-calibration should be considered every 24 hours.
-In more stable patients, this is probably sufficient. However, more frequent recalibration should be considered if the patient is unstable or if the haemodynamic parameters do not match clinical/echocardiography assessment.
-LiDCO haemodynamic parameters are more accurate within the 4-hr period after calibration.

When to use LiDCO – summary

Consider LiDCO in the following:		
Patients requiring frequent fluid boluses. (1.5L in 2hrs)	Patients with escalating cardiovascular support	Patients who do not meet contraindications

Relative Contraindications for LiDCO – D/W with Cons/Reg/ACCP			
Severe peripheral vascular disease	Severe structural heart disease	Aortic valve regurgitation	Tachyarrhythmia (E.g., uncontrolled AF, frequent ectopics)
Patients with a highly dampened arterial line	An intra-aortic balloon pump if ratio is not 1:1	Severe peripheral arterial vasoconstriction.	Hypothermia (<34^{0}C)

Indications for:			
Uncalibrated LiDCO		**Calibrated LiDCO**	
Noradrenaline 0.15-0.3mcg/kg/min Metaraminol 4-6mgs/hr	When use of lithium for calibration is contraindicated (See pg.6 of guideline for details)	Noradrenaline >0.3mcg/kg/min	Patient on more than 1 vasopressor.
Patients requiring frequent fluid boluses. (1.5L in 2hrs)	Increasing vasopressor requirements	Patients on Inotropes (Dobutamine or Milrinone)	OOHCA hypotensive patients from the Cath-lab

Contraindications to Lithium (Calibrated LiDCO)		
Muscle relaxants* (See Pg. 6 of guideline for details)	Weight <40kgs	Patient on Lithium therapy
First trimester of pregnancy	Aortic valve regurgitation	Ketamine infusion / IV Lidocaine

LiDCO Calibration Step-by-Step Guide

LiDCO
Hemodynamic Monitoring

CALIBRATION
STEP-BY-STEP GUIDE

1 Before calibration

- Lithium chloride must be prescribed. Standard dose is 0.300mmol, **2.00ml.**

Please note: Dose should not be limited to 2ml (locked), as additional doses may be needed in one calibration. Maximum dose allowance is 20ml in 24 hours (10 calibrations).

2 Initiate calibration on screen

- Using the Navigation Menu ☰ select Inject
- Enter recent Hb, sodium and press large green tick ✓

3 Collect items

- Flow regulator.
- Lithium.
- Sensor (30 minutes before to reach room temperature).
- Injectate kit.
- 4 x luer lock syringes (1 already in kit).

4 Fill syringe

- Fill all four syringes each with 20ml saline.
- Label and set aside on clean area.

(If your unit does not have luer lock syringes you can fill the one provided and refill when needed, keeping on clean area.)

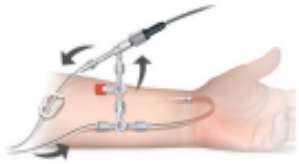
5 Prepare lithium

- Withdraw all 10mls lithium from vial, using 10ml syringe and safety straw.
- Attach safety valve as shown and add 3ml syringe.
- Draw up the standard dose 0.300mmol, 2.00ml lithium into 3ml syringe.
- Label (provided) and set both syringes aside on clean area. You may need to draw a second dose.

6 Prime sensor

- Attach 20mls syringe of saline to opposite end of red cap and leave red cap on (as shown in picture).
- Keeping sensor upright, flush with saline to wet and remove air bubbles. Keep red cap on.
- Catch saline in dish.

7 Connect and flush sensor with saline



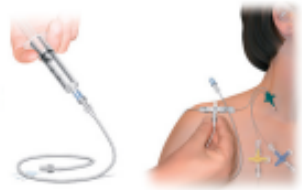
- Remove syringe and attach drain bag to sensor.
- Attach other sensor port to 3 way tap on arterial line.
- Close 3 way tap to patient and flush sensor from transducer bag, to remove any new air bubbles.
- Turn tap back to patient and re-establish BP trace.
- Remove red cap and attach black sensor interface cable from LiDCO (push fit). Sensor interface cable connects to interface pod and then to monitor, you will now notice 2 green ticks on screen.

Please note: you do not need an additional 3 way tap, as shown in picture.

8 Fit tube to pump

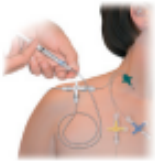
- Fit tube of drain bag to pump, as shown in picture.
- Insert large red lug into large slot on pump with prong facing up.
- Press clamp and push tubing around wheel.
- Insert small red lug into small slot, prong up.

9 Prime park & ride



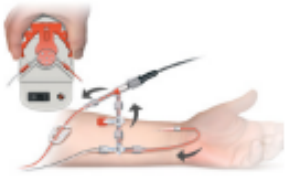
- Remove tubing labelled 'park & ride' from kit.
- Attach second 20ml syringe of saline and flush.
- Attach other end of tubing to venous access port.
- Ensure no other pharmacological substance is in the port/line.

10 Inject lithium into line



- Remove 20ml syringe from park & ride and attach 3ml syringe, containing **2ml** lithium previously prepared.
- Push lithium into park & ride (which will now sit inside the park & ride).
- Attach 3rd 20ml saline syringe to park & ride—**DO NOT INJECT YET.**

11 Prime sensor with blood



- Open the 3 way tap to allow blood to pass over the sensor.
- Squeeze clamp on flow regulator and allow blood to pass over the sensor for a few seconds.
- You will notice the screen on LiDCO temporarily displays 'stabilising'. This will return to 2 green ticks once clamp is released.

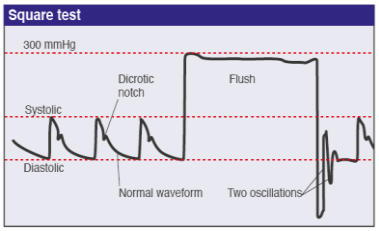
12 Injecting the lithium

- Turn on the flow pump which will draw blood over the sensor and wait for the signal to stabilise and display inject.
- Press Inject and inject the full 20ml of saline which will push the 2ml of lithium into the patent.
- You will see the curve start to appear.
- Flush the sensor and arterial line when instructed remembering to squeeze the flow pump clamps to allow saline to flow. Then re-establish the arterial trace.
- Press 'Auto Calibrate' and wait. You may be required to repeat the calibration to confirm the readings. Go back to step 10.
- Press 'Finish Calibration'.
- Calibration recommended again in 24 hours.

Assessing the arterial line tracing

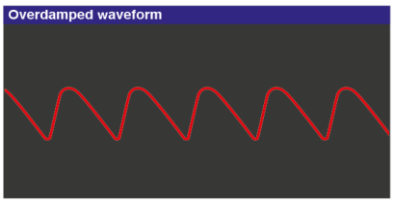
The accuracy of the haemodynamic values obtained using LiDCO are dependent upon the arterial line trace. If the arterial waveform is severely damped, the values obtained may be misleading.
It is recommended that a square test is performed in order to check the system is correctly damped. Damping is required in order to reduce natural vibrations and friction of the fluid system, but overdamped and underdamped traces can give incorrect results and may impact on patient management.

How to perform a square test

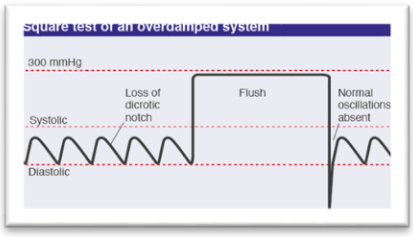


Pull the tab on the arterial line transducer for one second and then release it. The pressure will rise rapidly then will instantly fall, creating a square waveform. There will be two oscillations before the arterial waveform resumes.

Square test on an overdamped trace

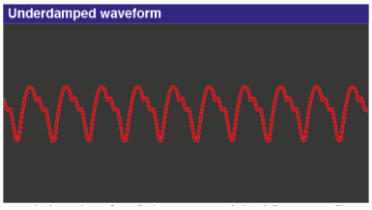


With an overdamped trace the waveform loses its characteristic landmarks and appears unnaturally smooth, with a diminished or absent dicrotic notch. This can lead to a falsely low systolic and falsely high diastolic reading.

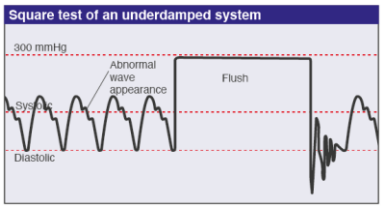


When performing a square test on an overdamped system, there will only be only one oscillation.

Square test on an underdamped trace



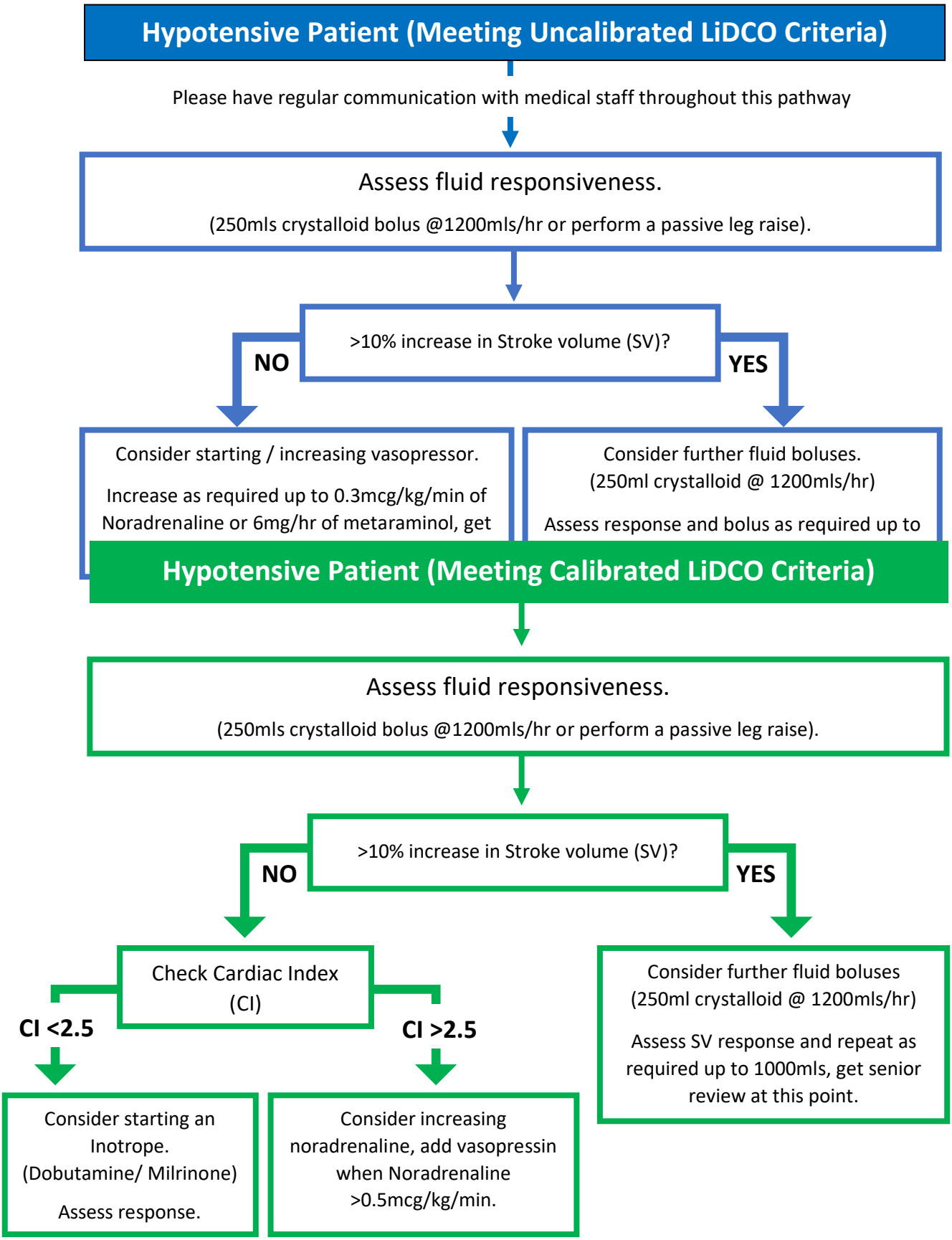
With an underdamped trace the waveform displays an exaggerated systolic pressure with a rapid systolic slope. This results in an overestimated systolic pressure and an underestimated diastolic pressure.



In an underdamped system there will be multiple oscillations after the square wave,

If the waveform is found to be either underdamped or overdamped following a square test, then please check the following. The arterial cannula positioning (if the arterial line is in the radial artery, is the wrist bent?). Check for air bubbles or clots in the system, remove if any are identified. Check the patency of the arterial line. Check that the transducer is set up correctly.

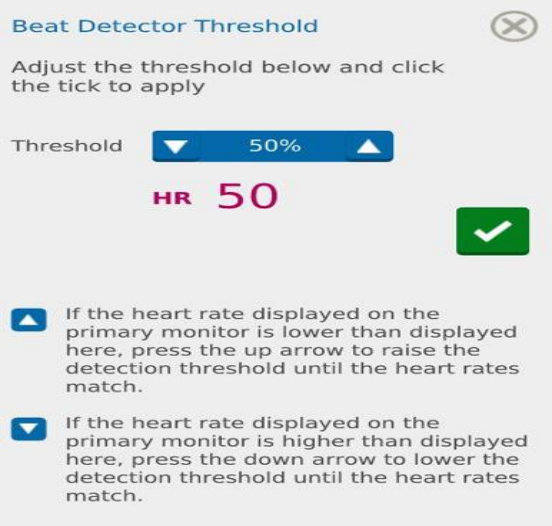
LiDCO Guided Treatment Algorithm



*Senior review = ACCP/ICM REG/Consultant.

Using LiDCO when a patient is in Atrial Fibrillation (AF).

LiDCO can be used in those patients with either rate-controlled AF (Heart rate less than 100) or controlled AF (if the heart rate variance on the LiDCO screen is not above 10%). However, to ensure the data provided by LIDCO is as accurate as possible consider adjusting the beat detector threshold (found on the main menu) by adjusting the averaging time within in the LiDCO settings. See below for instructions on how to do this.



SVV and PPV should not be used to assess fluid responsiveness for those patients in AF as the additional variance of AF, in addition to the variance of SV during a ventilation cycle, affects the accuracy of this data.

Please use '*Haemodynamic Monitoring in General Critical Care using calibrated Lithium Dilution Cardiac Output (LiDCO) and uncalibrated LiDCO*' Guidelines for more information regarding;

-LiDCO Monitors within Critical Care

-Lithium Calibration-How does it work?

-Lithium Chloride Dose

-Lithium Toxicity

-Nursing Care

-Assessing the arterial line tracing

-Assessing Fluid Responsiveness

-Volume Responsiveness v Volume Tolerance

-Administering a Fluid Challenge

-Performing a Passive Leg Raise

-Effects of Mechanical Ventilation

-End- Expiratory Occlusion Test

-Advanced Haemodynamic Variables

-Central Venous Saturations ($ScVO^2$)