Hemodynamics

Hemodynamics:

Hemodynamic Monitoring:measurement of pressure, flow, and oxygenation within the cardiovascular system. Monitoring is used to asses heart function, fluid balance, and the effects of fluids and drugs on cardiac output.

Indications for hemodynamic monitoring include: shock, sepsis, anaphylaxis, MI with right/left ventricular failure, Hypo/Hypertension, Cardiac tamponade, ARDS, Post open heart surgery, fluid resuscitation, and evaluation of cardiac output

Mean Arterial Pressure (MAP): the average pressure within the arterial system that is felt by organs in the body. **Essentially how hard the blood is flowing when hitting the organs.**

Calculating MAP 2xDiastolic + Systolic = #. # divided by 3. **Example:** BP:120/60: 2x60+120=240. 240/3= 80. MAP of 80.

Pulse Pressure: the difference between the systolic blood pressure (SBP) and diastolic blood pressure (DBP). **Narrow/decreased:** shock state or HF- meaning insufficient preload leading to reduced cardiac output (CO). **Wide/increase:** occurs during exercise or in atherosclerosis- Increased ICP.

Calculating Pulse Pressure: SBP-DBP=Pulse Pressure

Stroke Volume: the amount of blood pumped by a ventricle with each **beat.**

Calculating Stroke Volume: Cardiac Output/ HR= stroke volume

Cardiac Output: Amount of blood ejected from the heart into circulation each **minute**

Calculating Cardiac Output: HRxSV= Cardiac Output

Hemodynamics (cont)

Cardiac Index: cardiac out that is adjusted for each individual based on the body surface area (BSA).

Ranges:
MAP: >60mm Hg. <65 needs to be addressed. <60 inadequate tissue and organ perfusion
Pulse Pressure: 40-60mm Hg
Stroke Volume: 60-150mL/beat
Cardiac Output: 4-8L/min
Cardiac Index: 2.2-4L/min

Autonomic Nervous System

Autonomic Nervous System:ANS

ANS consists of the Sympathetic Nervous System (SNS) and the Parasympathetic Nervous System (PNS)

SNS: this is the bodys' **fight of flight**. It controls blood flow by constricting the arteries throughout the body and increases blood pressure and blood flow.

How does SNS work?: The body senses a low arterial pressure. The SNS is then stimulated to maintain CO. Norepi and epi (catecholamies) is released. The receptors respond the the norepi and epi by increasing the HR, contractility, and conduction (chronotropic, inotropic, dromotropic effects). This then increases myocardial oxygen demand.

SNS Effects: Chronotropic- increases the **rate.** Inotropic- increases the **contractility.** Dromotropic- cardiac **conductivity time**.

Chronotropic: responds to norepinephrine and epinephrine by **increasing the HR.**

Inotropic: responds to norepinephrine and epinephrine by **increasing the contractility.**

Dromotropic: responds to norepinephrine and epinephrine by **increasing the speed of conduction.**

PNS: this is the bodys' **rest and digest.** It controls blood flow by dilating the arteries which decrease blood pressure and blood flow.

Autonomic Nervous System (cont)

How does PSN work? Happens after SNS response. It "reverses" the SNS effects. Acetylcholine is released which then decreases HR, conduction and irritability. Stimulates the GI tract.

Monitoring

Four Parameters of Hemodynamics: Preload (think volume), Afterload (think resistance), Contractility (thing ability to pump), and Cardiac Output (think perfusion status).

Contractility: Hypokinesia: reduced movement or contraction of a segment of the heart muscle. **Dyskinesia:** abnormal movement, myocardium bulges out in systole. **Akinesia:** lack of movement or contraction of a region of the heart muscle.

Preload: Amount the ventricles stretch at the end of diastole (the filling phase). Preload is once the ventricles are completely filled.

How to increase preload? (pt in hypovolemic shock) give fluids (increasing the amount of volume to fill the ventricle). Stimulate SNS - give vasopressors (causing vasoconstriction- making the area to fill "smaller").

How to decrease preload? (pt is fluid overload) give diuretics (excreting excess fluids). Give vasodilators (creating more space for fluid to flow).

Afterload: the pressure the ventricles have to work against to get valves to open to pump blood out of the heart. Afterload is affected by **vascular resistance**.

Vascular Resistance in the right side of the heart: Right ventricle pumps blood to pulmonic valve. Pulmonic valve has to fight against **pulmonary vascular resistance (PVR)** to open and pump blood to the lungs.

Monitoring (cont)

Vascular Resistance in the left side of the heart: Left ventricle pumps blood to aortic valve. Aortic valve needs to fight against **systemic vascular resistance** to open and pump blood to the rest of the body (systemically).

How to increase afterload? (pt is hypotensive) Can increase naturally by vasoconstriction such as **pulmonary HTN** for left sided or *HTN* ***for right sided. This happens due to the increase vascular resistance. Valve problems cause an increase in after load*** *(aortic stenosis)** due to the constriction of the valve increasing pressure on the ventricles.

How to decrease afterload? Give vasodilators. This will decrease vascular resistance resulting in a decrease of pressure to work against.

Effects on Cardiac Output: If HR, Preload, contractility is increased/decreased CO will follow suite