

Definitions

Blood pressure – measurement of the force against the walls of the arteries (mmHg)

Systolic pressure - maximum aortic pressure during LEFT ventricular contraction

Diastolic pressure – minimal aortic pressure during left ventricular relaxation

Pulse pressure – strength of contraction (systolic – diastolic = pulse pressure)

Mean arterial pressure – diastolic pressure – 1/3 pulse pressure

Importance of MAP

MAP is important as the heart does NOT spend an equal amount of time in systole and diastole.

- + 1/3 systole contraction
- + 2/3 diastole relaxation + filling

This allows the heart to be more efficient at filling with blood

Formulae

Blood pressure = Cardiac output x peripheral resistance

Cardiac output = stroke volume x heart rate

Stroke volume = EDV – ESV

Ejection fraction = stroke volume / EDV

+ Normal EF = 55%-75%

Stroke volume

Stroke volume = amount of blood transferred from left ventricle to aorta in SYSTOLE

3 factors affect SV:

- 1. Preload volume of blood that ventricles can pump
- 2. Contractility force that the muscle can contract with
- 3. Afterload arterial pressure against which the ventricle will contract against

(left ventricle <u>NEVER</u> completely empties at the end of systole)

LEARN WHAT CAUSES INCREASE/DECREASE IN THE 3 FACTORS

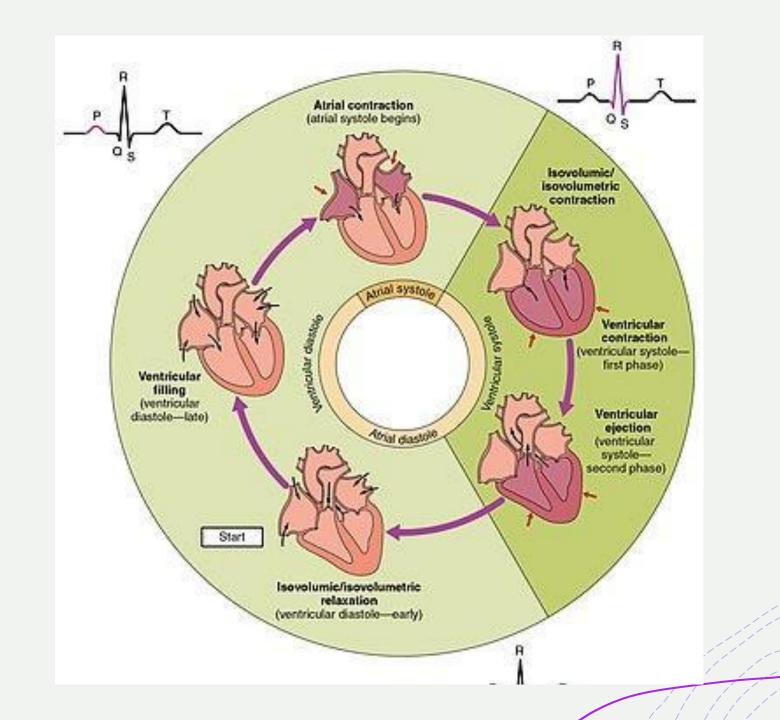
Cardiac cycle

1) // Aţrial systole

- 2) Isovolumetric ventricular contraction pressure increases, NO change in volume
- 3) Rapid ventricular ejection
- 4) Isovolumetric ventricular relaxation pressure decreases, NO change in volume
- 5) Rapid ventricular filling

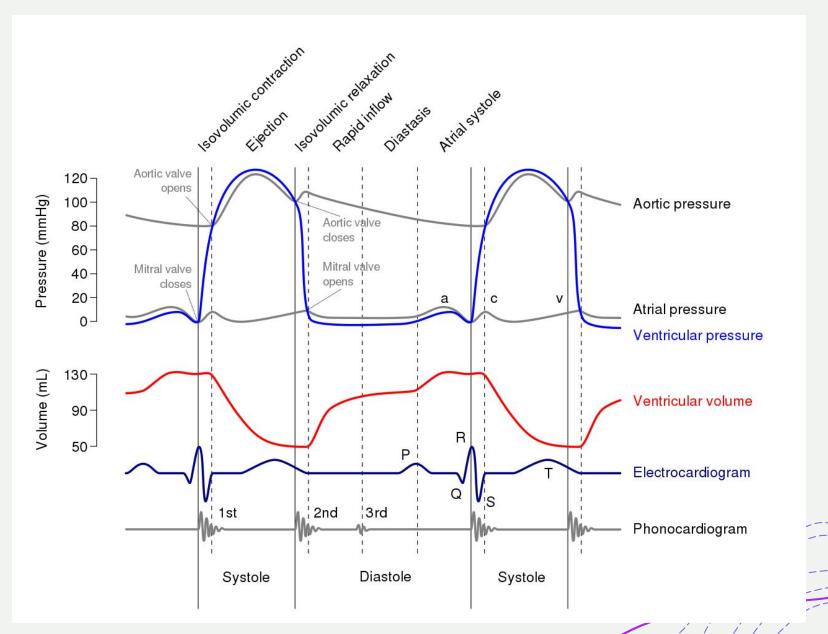
Valves open - high volume behind them increases pressure, causing valves to open

Valves close – high pressure in front of them



Learn when valves open and close – HIGH YIELD

Wigger's diagram



Control of the cardiac cycle

Controlled by 2 mechanisms:

- + Endocrinal long term
- + Neuronal short term

Endocrine - RAAS

- 1) Macula densa cells detects low Na+ = low BP
- 2) Juxtaglomerular cells secrete renin
- 3) Renin converts angiotensinogen (released by liver) to angiotensin I
- 4) Lungs release ACE to convert angiotensin I into angiotensin II
- 5) Angiotensin II increases blood pressure

LEARN THE RAAS SYSTEM

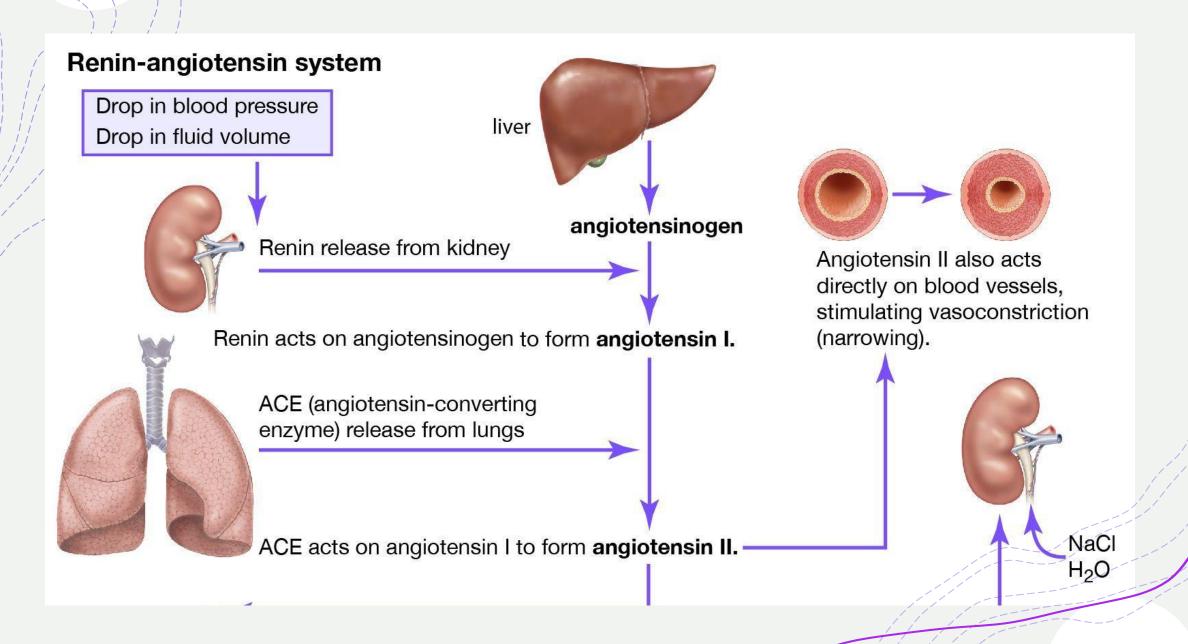
Functions of angiotensin II

SYSTEMIC VASOCONSTRICTION - main function

2) Cardiac and vascular hypertrophy

3) Acts on adrenal cortex – aldosterone – increases NaCl retention – increases BP

4) Acts on posterior pituitary gland – ADH – increase H2O retention



Neuronal

Cardiovascular centre:

+ located in medulla – regulates HR and SV

Sensory receptors:

- + Proprioceptors increase HR if there is anticipation exercise
- + Baroreceptors detect change in blood pressure
- + Chemoreceptors detect chemical changes

Baroreceptors – aortic arch and carotid sinus

+ prevent excessive fall in blood pressure when you stand up quickly

Chemoreceptors – carotid sinus + aortic body

Neuronal- ANS

Sympathetic nervous system:

+ Increases BP – cardiac accelerator nerves – noradrenaline is released and acts on Beta 1 adrenoreceptors

3 effects:

- 1. Positive chronotropic effect increase HR
- 2. Positive ionotropic effect increase in contractility increase CO
- 3. Peripheral vasoconstriction via alpha 1 adrenoreceptors increase in péripheral resistance

Neuronal - ANS

Parasympathetic control:

*Decreases BP – vagus nerve – acetylcholine acts on M3 receptors

Main effect:

1. Negative chronotropic effect – decreases HR

(Little effect on contractility)

Autonomic control

Beta 1 adrenoreceptors:

Increase HR + contractility

Alpha 1 adrenoreceptors:

Vasoconstriction

Beta 2 adrenoreceptors:

+ Vasodilation

Alpha 2 adrenoreceptors:

Vasodilation

Questions ©

1) What releases RENIN?

2) How do you calculate CO?

3) What 3 things does SV depend on?

4) What is the main function of AGII?

5) What do baroreceptors do?

Questions ©

What is the main function of the sympathetic nervous system regarding blood pressure?

2) What nerve is involved in the parasympathetic control of BP?

3) Where is the cardiovascular centre located?

ANSWERS

- 1) Juxtaglomerular cells
- 2) SV x HR
- 3) Preload, afterload, contractility
- 4) Peripheral vasoconstriction
- 5) Detect changes in BP
- 6) Increase BP
- 7) Vagus nerve (CNX)
- 8) Medulla (oblongata)

Cordiac cycle:	
Olorial Systone:	
* Okria contract	
isovolumetric ventricular controction:	
A-V+S-L values = Shut	
Volume remains the same	
* Ventricular contraction = 1 pressure	
rapid ventricular ejection:	
Tapid ventricular ejection: 1 pressure in ventricus > Pressure of blood in S-2 cusps	
1 pressure in ventrales > Pressure of blood in S-L cusps	
1 pressure in ventrales > Pressure of blood in S-L cusps	
1 pressure in ventrales > Pressure of blood in S-2 cusps 1 volume as blood enters aorta + P.A.	
† pressure in ventrales > Pressure of blood in S-2 cusps + volume as blood enters aorta + P.A. isovolumetric ventralar relabation:	
T pressure in ventricles > Pressure of blood in S-2 cusps + volume as blood enters aorta + P.A. sovolumetric ventriclar relaxation: diastole	
1 pressure in ventricles > Pressure of blood in S-2 cusps 4 volume as blood enters agree + P.A. isovolumetric ventricular relaxation: diastole 5-L, A-V values = closed	
T pressure in ventricles > Pressure of blood in S-2 cusps + volume as blood enters aorta + P.A. isovolumetric ventriclar relaxation: diastole S-L, A-V values = closed volume remains same	
T pressure in ventricles > Pressure of blood in S-2 cusps + volume as blood enters aorta + P.A. isovolumetric ventriclar relaxation: diastole S-L, A-V values = closed volume remains same	
† pressure in ventroles > Pressure of blood in S-L cusps † volume as blood enters agree + P.A. isovalumetric ventrollar relaxation: diastole S-L, A-V values = closed volume remains same blood enters atria	

