Renal Physiology

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Renal blood flow

- Renal fraction is the fraction of the cardiac output that supplies the kidneys.
- It equals 1200 ml/min (1/4 COP).
- Significance: good processing of plasma.
- Renal cortex receives 4-5ml/min/1gm while medulla receives 0.25 -1 ml/min/1gm.

Renal Oxygen consumption:

- the renal Oxygen consumption is 6 % of the total body consumption.
- Oxygen utilization by the kidneys is blood dependent; increased blood flow → increased renal oxygen consumption → increased Na^+ reabsorption by the renal tubules.
- Other tissues like brain and heart; blood flow is oxygen depepdent.

Causes of decreased medullary blood flow?

- Decreased medullary blood vessels.
- Increased length of the blood vessels.
- Increased blood viscosity.

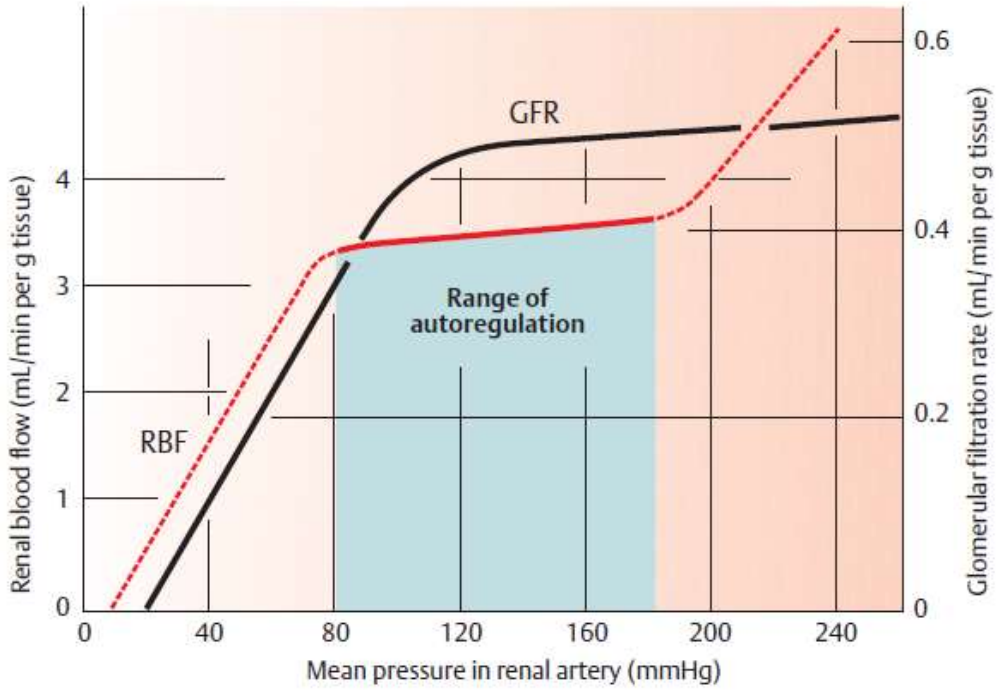
Regulation of the renal blood flow:

1- Autoregulation.

2- Extrensic regulation

Autoregulation

- Certain mechanism produced b the kidney to keep RBF and GFF constant with blood pressure changes ranging from (80 -180 mmHg).
- Prevent changes in blood pressure from affecting Na and water excretion.



Mechanism

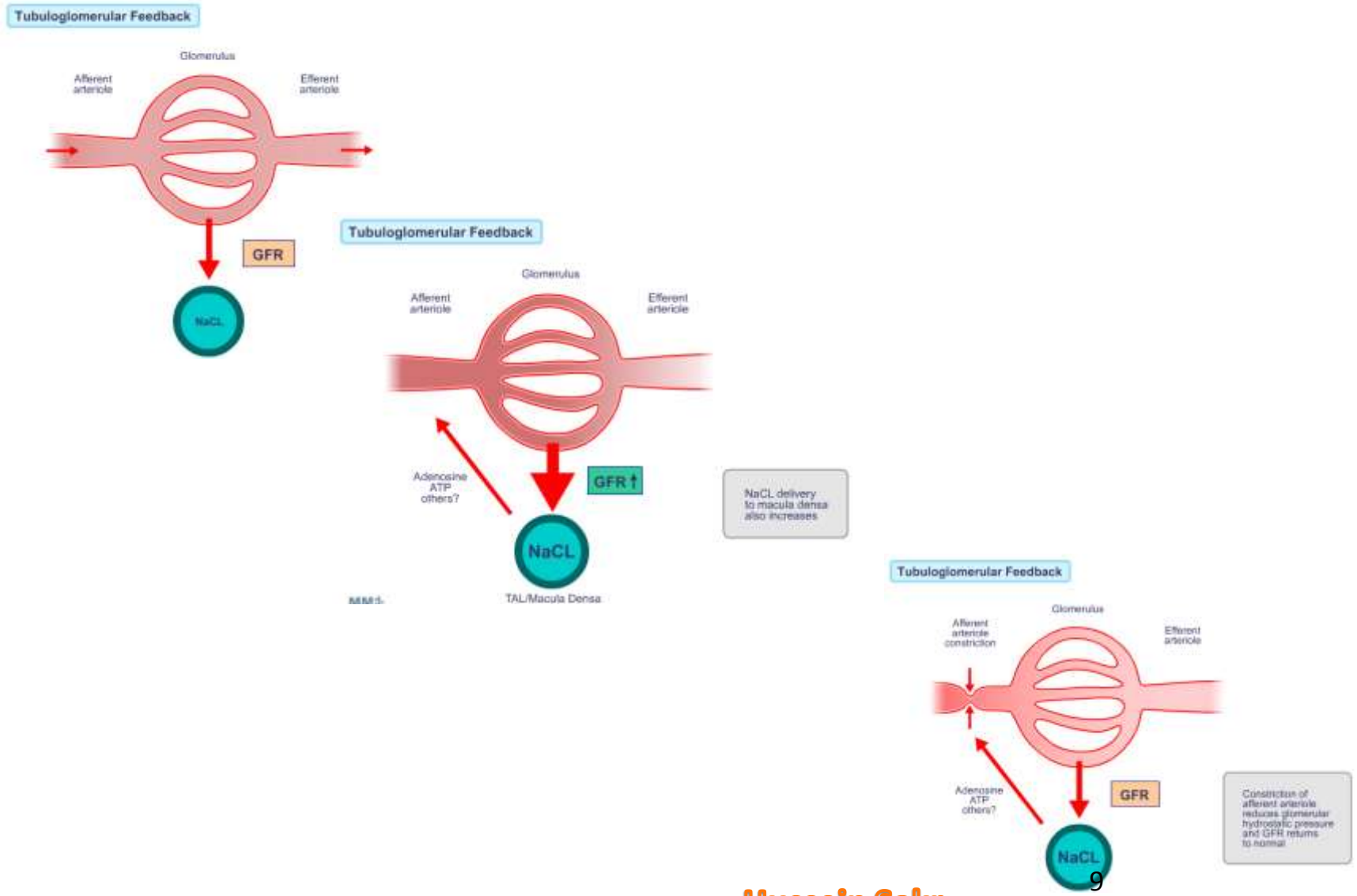
Myogenic theory	Tubulo-glomerular feed back mechanism
• Increased arterial blood pressure → increased Renal blood flow → stretch of the arterial wall and smooth muscles → increased Ca influx → increased contraction of smooth muscles → decreased RBF and GFR to normal.	• Delivery of NaCl to the macula densa sends a signal that affect the resistance of the afferent arteriole

Tubulo-glomerular feed back mechanism

- Decrease in blood pressure → decrease RBF → decreased GFR → decrease Na and Cl delivered to macula densa → release of PGs from macula Densa cells → stimulate JGG cells to release renin → formation of AII → constriction of the efferent arteriole only → increased capillary hydrostatic pressure → increase GFR to normal.

- Increase in blood pressure → increase RBF → increased GFR → increased NaCl delivered to the macula densa → release of Adenosin → Vasoconstriction of the renal artery → decreased RBF & GFR.

Tubulo-glomerular feed back mechanism



Extrinsic regulation

Nervous:

- sever sympathetic stimulation produces vasoconstriction and decreases renal blood flow.

Humoral:

- Nitric oxide and prostaglandins increases renal blood flow.
- Endothelin and thromboxane A_2 decreases renal blood flow.

Kidney & PGs

Vasodilator PGs: PGE2 & PGF2α

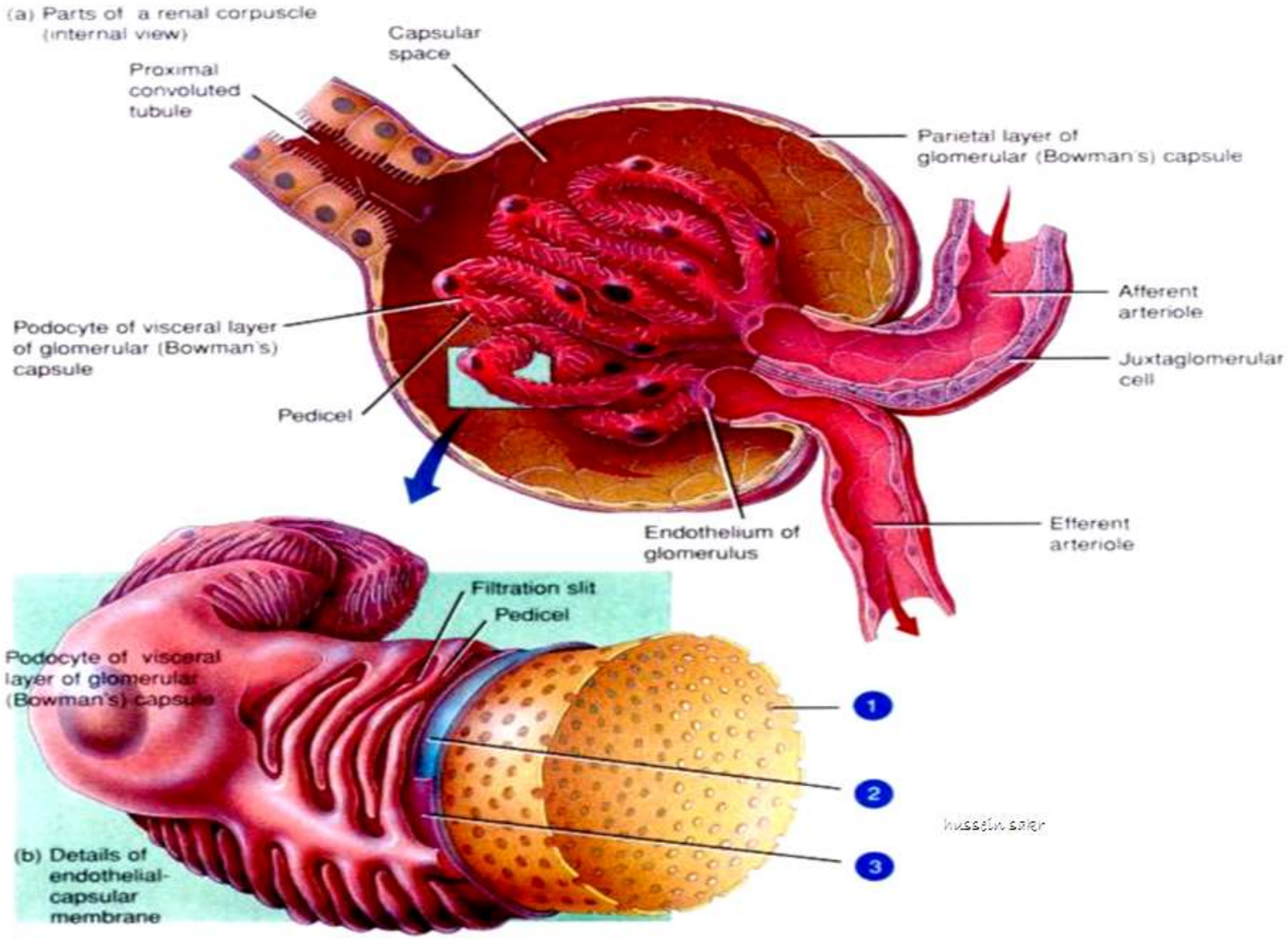
Are essential in the process of regulation RBF. Intake of analgesics such as NSAIDs → decreases PGs synthesis which may depress renal function in patients with reduced RBF.

Vasoconstrictor PGs: Thromboxane A2

Are released in response to ureteric obstruction to decrease RBF and GFR → obstructive uropathy.

Glomerular Filtration

- It is the bulk flow of solvents carrying with it solutes that are small enough to pass through the glomerular membrane.
- The filtrate is ultrafilterate (plasma – plasma proteins).


(a) Parts of a renal corpuscle (internal view)
Capsular space
Proximal convoluted tubule
Parietal layer of glomerular (Bowman's) capsule
Afferent arteriole
Podocyte of visceral layer of glomerular (Bowman's) capsule
Juxtaglomerular cell
Pedicel
Endothelium of glomerulus
Efferent arteriole
Filtration slit
Pedicel
Podocyte of visceral layer of glomerular (Bowman's) capsule
1
2
3
(b) Details of endothelial-capsular membrane

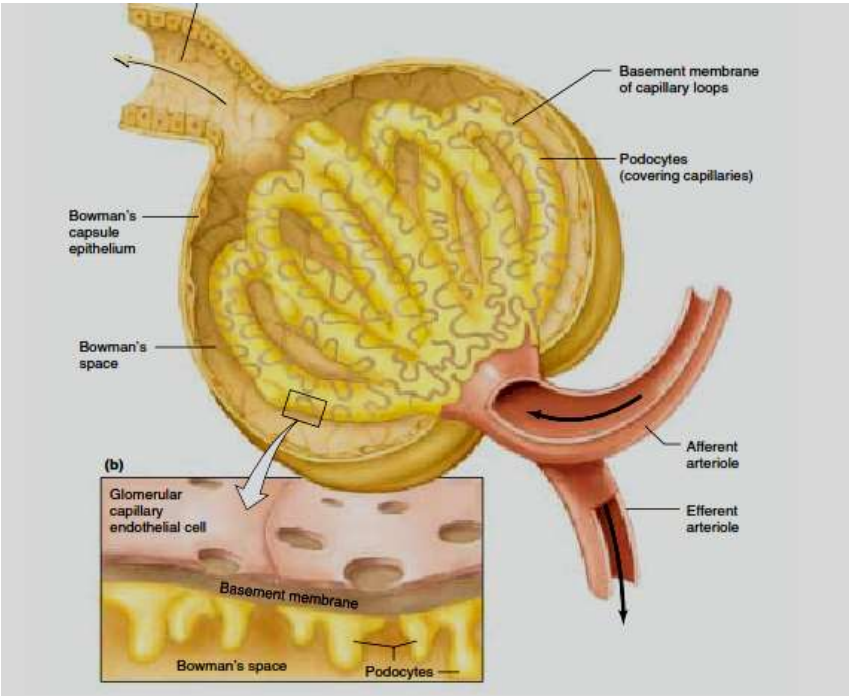
Filtering Membrane

- The filtering membrane is formed of the following layers:

1- Bowman’s capsular epithelial cells.

2- Basement membrane.

3- Endothelial cells lining the glomeruli



Filtering Membrane

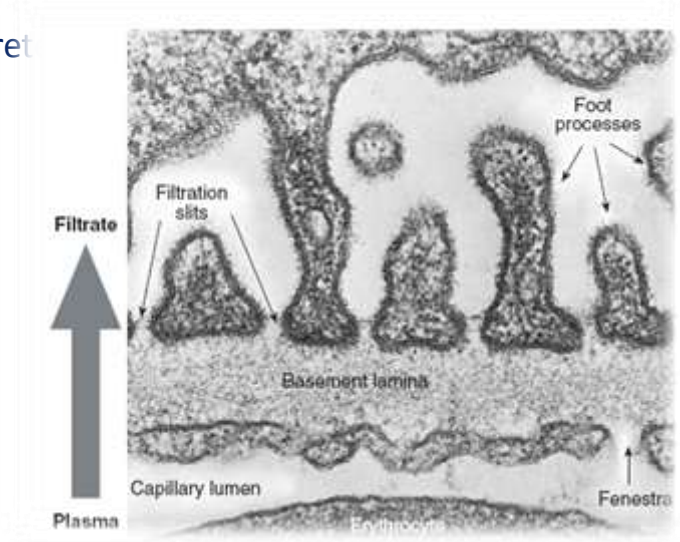
- Capillary endothelial cells with fenestrations →prevents the passage of blood cells into urine.
- Basement membrane formed of a network of negatively charged proteins.
- Bowman’s capsular epithelium with finger like processes called podocytes

Filtering Membrane

- High permeability: the pores in the bowman's capsular epithelial cells and the endothelial cells increased the permeability of the glomerular membrane.
- High selectivity: caused by size of the pores and negative charge present on the basement membrane.

Mesengial cells :

- are stellate cells :
- It holds delicate structure together.
- Present between the capillary loops.
- Has contractile nature.
- Secret



The following are not freely filtered

- Albumin and other plasma proteins.
- Lipid soluble substances transported in the plasma attached to plasma proteins; soluble bilirubin, T4, other lipid soluble hormones.

Causes of high filtration:

1- High Glomerular hydrostatic pressure (45-60 mmHg).

2- High filtration coefficient (4.2 ml/min/mmHg/100 gm renal tissue).

3- High renal plasma flow (650 ml/min).

Comparison between systemic and glomerular filtration

Glomerular filtration

- Surface area of the glomerular membrane 1.6 m
- 180 L/day
- Glomerular hydrostatic pressure = 60 mmHg
- Filtration coefficient = 4.2 ml/min/1mmHg for each 100 gm renal tissues.

Systemic filtration

- Surface area of the systemic capillaries 1000 m.
- 20 L/day.
- Systemic capillary hydrostatic pressure at arterial end = 30 mmHg, at venous end =10mmHg.
- Filtration coefficient = 0.01 ml/min/1mmHg for each 100 gm systemic tissues.

The four factors determining the net filtration pressure

- Forces Increasing filtration:

1- Hydrostatic pressure of the glomerular capillaries.

2- Oncotic pressure of the Bowman's space.

- Forces decreasing filtration:

1- Hydrostatic pressure of the Bowman's space.

2- Oncotic pressure of the glomerular capillary.

Forces Increasing flitration:

- **Hydrostatic pressure of the glomerular capillaries: (60 mmHg)→ PGC**
- The only force that promotes filtration.
- Under normal condition this is the main factor that determine GFR.

- Oncotic pressure of the Bowman's space → πBS.
- This represent the protein or oncotic pressure in Bowman's space.
- Very little if any protein is present
- For all practical purposes this factor can be considered zero.

Forces decreasing filtration

- **Oncotic pressure of plasma (32 mmHg)→ πGC.**

- The oncotic pressure of the plasma varies with the concentration of the plasma protein.
- Sa the fluid is filtred the plasma volume decrease → oncotic pressure increase by the end of the glomerular capillaries.

- **Hydrostatic pressure in Bowman's space (18 mmHg) → PBS:**

- It opposes filtration.
- It increased with ureteric obstruction → decreased GFR.

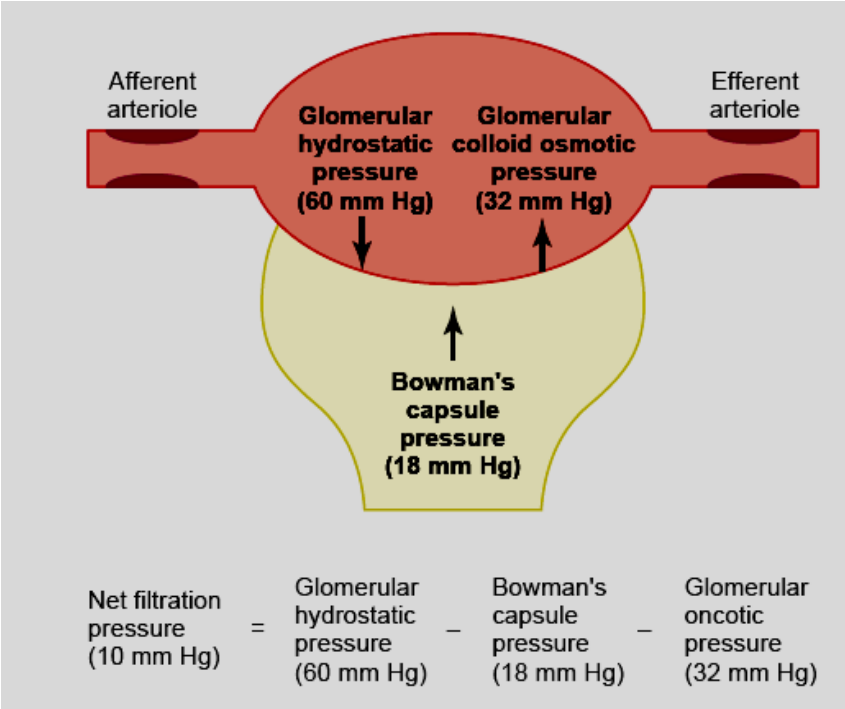
Net filtration force

filtering forces= capillary hydrostatic pressure + Bpwman's osmotic pressure.
Forces opposing filteration= osmotic pressure of plasma proteins + Bowman's hydrostatic pressure.

Filteration coeffeicient = it is fluid filtered by all nephrons in both kidneys by net filtering pressure 1 mmHg.
12.5 ml/min/1mmHg.

GFR= Net filtering force X filtration coefficeint.

GFR= 10 X 12.5= 125 nl/min.



Assignment

- **From the following data calculate the GFR?**
- PGC= 48mmHg, πGC= 24mmHg, PBS= 12 mmHg.
- Filtration coefficient= 8 ml/min/1mmHg

Factors affecting GFR:

1- Glomerular hydrostatic pressure.

2- Oncotic pressure of the plasma proteins.

3- Renal plasma flow.

4- Filtration coefficient.

5- Balance between afferent and efferent arteriolar resistance.

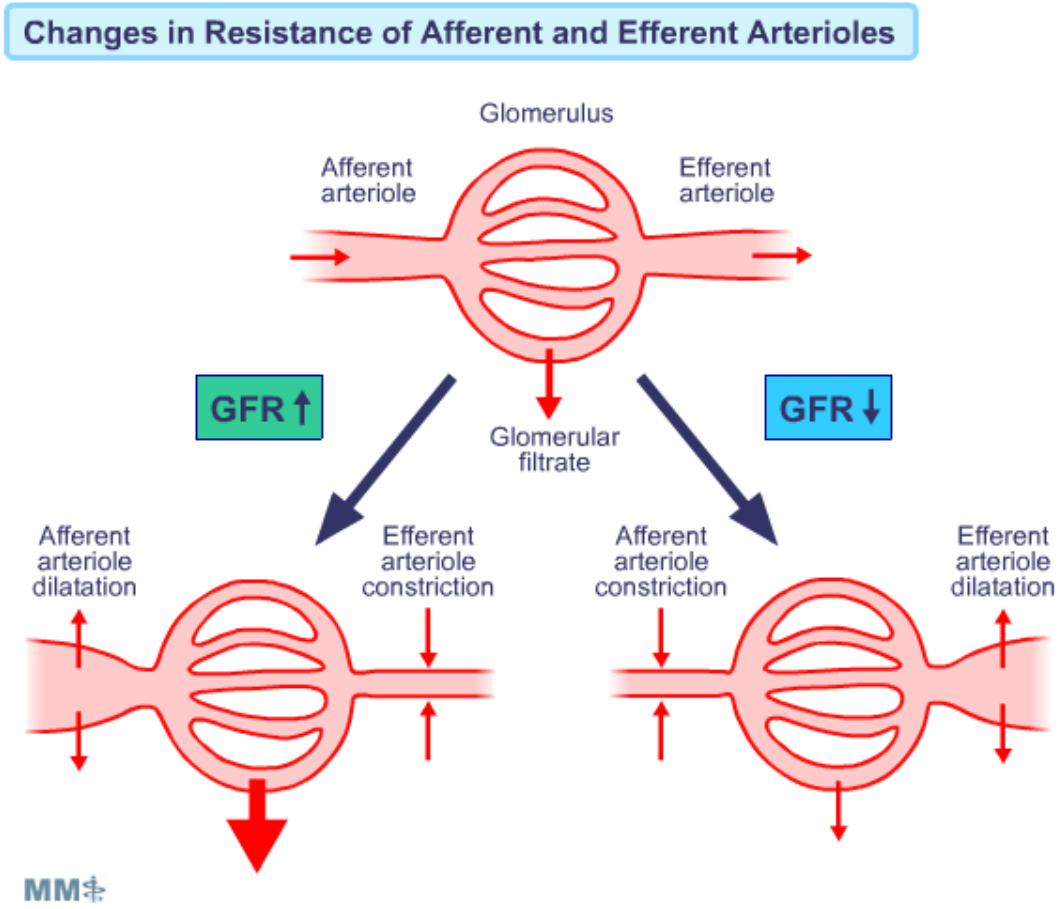
Afferent and efferent arteriolar resistance

- Changes in the afferent and efferent arteriolar resistance determine the glomerular hydrostatic pressure and GFR.
- Afferent arteriolar resistance changes the RBF.
- Efferent arteriolar resistance changes the PGC.

Effect of Angiotensin II

- In small doses it constricts the efferent arteriole → increases the glomerular hydrostatic pressure → increases GFR.
- In large doses it constricts both afferent and efferent arterioles → decreases both renal blood flow and GFR.

Afferent and efferent arteriolar resistance



Afferent and efferent arteriolar resistance

	GFR	RPF	Filtration fraction (GFR/RPF)
Afferent dilatation	↑↑	↑↑	Constant
Afferent constriction	↓↓	↓↓	Constant
Efferent constriction	↑↑	↓↓	↑↑
Afferent dilatation	↓↓	↑↑	↓↓