RENAL BLOOD FLOW

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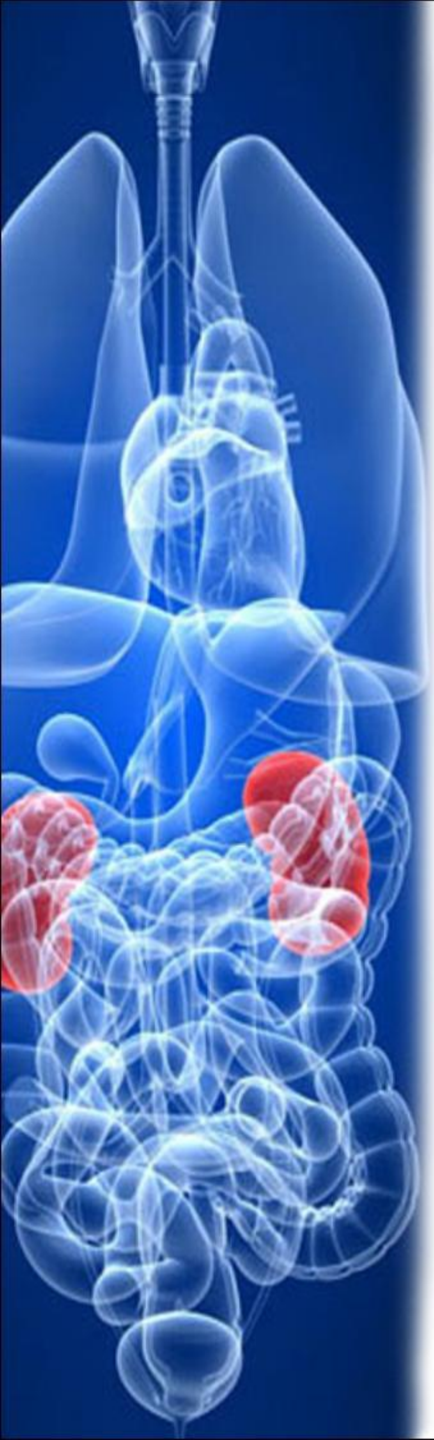
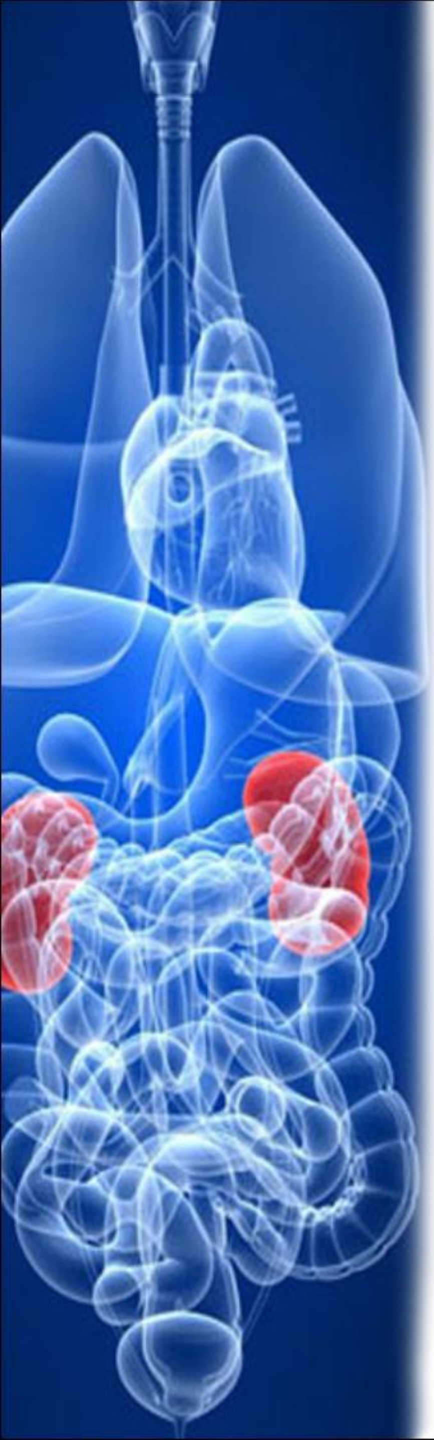


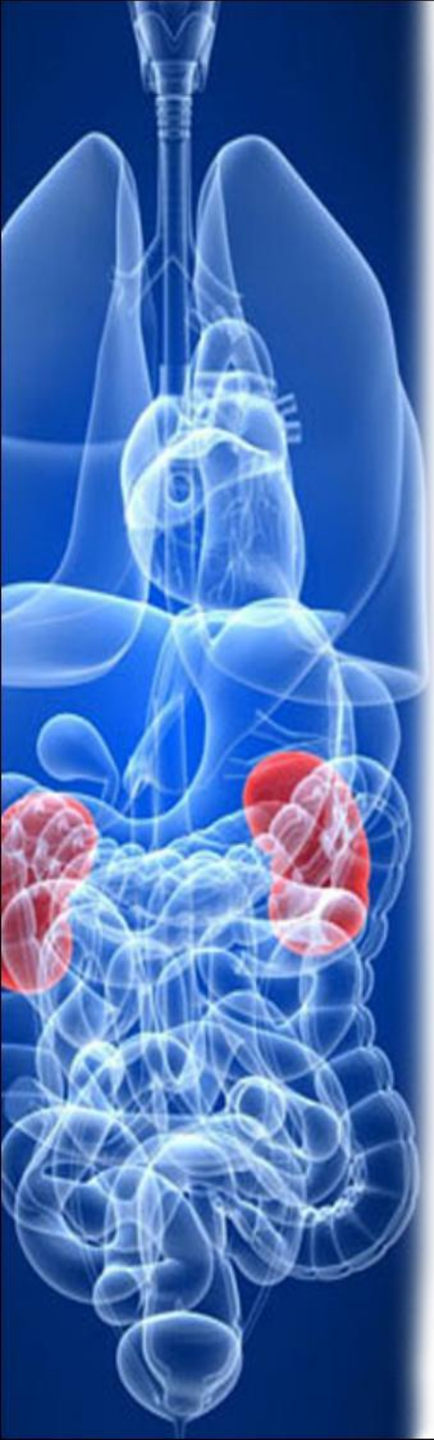
Renal circulation

Although the mass of the both kidney's is less than 0.5% of total body weight yet, they receive more than **22% of the cardiac output (1200 ml/minute)**. This huge blood flow of course is not for the high metabolic rate of that organ but because this blood is "Cleared" during its passage through the kidney tubules from the wastes and excess salt and water. It was estimated that about **180 Litres of plasma** are cleaned/day by the kidney or in other word the whole plasma is cleared **60 times/day.** It is interesting that the O_2 consumption by the kidney tissue is low and renal vein is one of the veins that contain relatively high O_2 concentration among the other body veins.



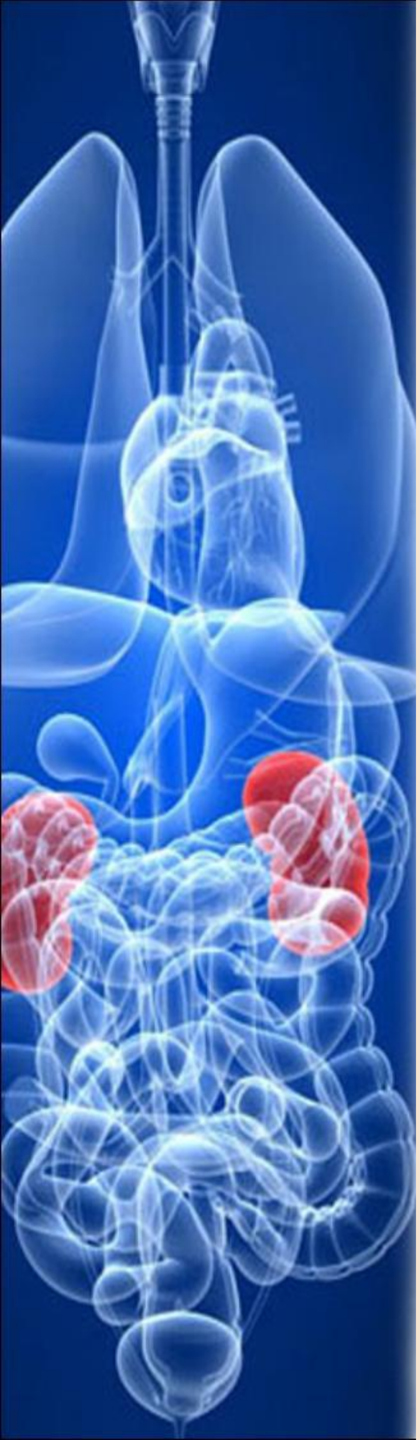
origin and distribution of renal blood vessels:-

The renal artery arises directly from the abdominal aorta in perpendicular manner to ensure nearly the same pressure as in the aorta i.e., **100 mmHg** and enters the kidney lobes, where it divides into interlobar arteries then they give the arcuate arteries which curve over the medullary pyramids at the junction of the cortex with the medulla. The arcuate artery then breaks to the interlobular arteries (**pressure= 85 mm Hg**) which then further subdivided into afferent arterioles (**pressure 80-60 mm Hg**).



- The afferent arterioles give the glomerular capillaries. They have the highest pressure than any other capillary in the body=**60 mmHg**, because it end by the efferent arterioles which show great resistance to blood flow (1/3 diameter of afferent arteriole).
- The pressure drops inside the efferent arterioles from 60 to only **18 mm Hg** where they breaks to another seat of capillaries, these are the Peritubular capillaries in the cortical nephrons or to a series of long straight thin capillary plexuses called the Vasa recta in the juxta medullary nephrons.

- These vase recta descend then ascend together with the long Henle’s loop
- The great length of vasa recta results in high resistance to the blood flow in the medulla and that is why it is less vascular than cortex.
- **After that,** the vasa recta or Peritubular capillaries give the interlobular vein then arcuate vein then interlobar veins then renal vein in which the pressure is only **4 mmHg.**



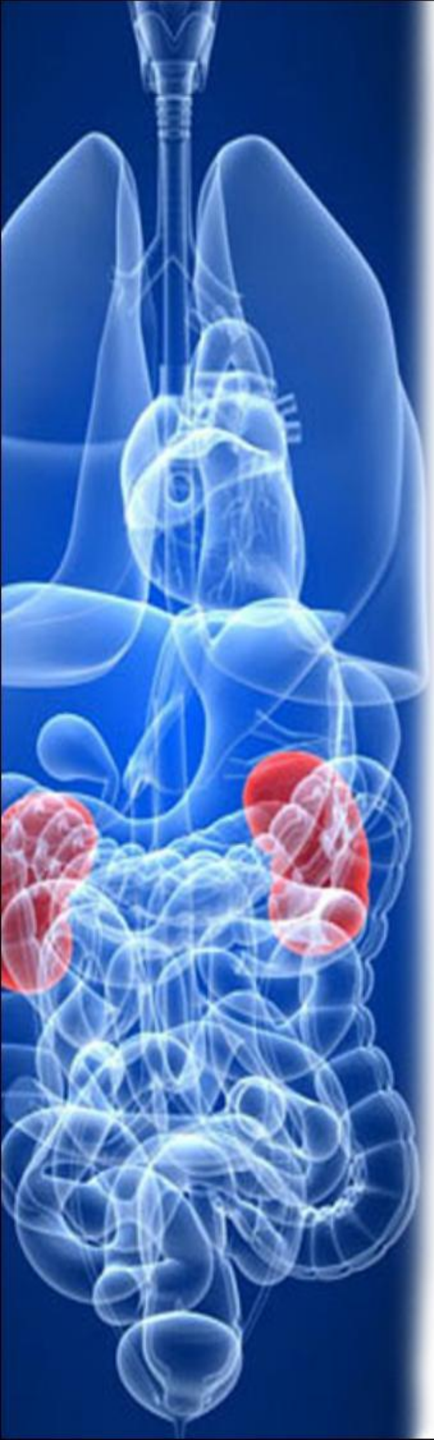
Origin & distribution of renal blood vessels:

Renal artery (<u>**100**</u> mmHg) ⇩ Inter-lobar artery ⇩ Arcuate artery ⇩ Inter-lobular artery (<u>**85**</u> mmHg) ⇩

Afferent arteriole (<u>60</u> - 80 mmHg) ⇨ Glomerulus (60 mmHg) ⇨ **Efferent arteriole** (18 mmHg) ⇨ ***Peritubular capill.***(13 mmHg

Vasa recta in JM nephron

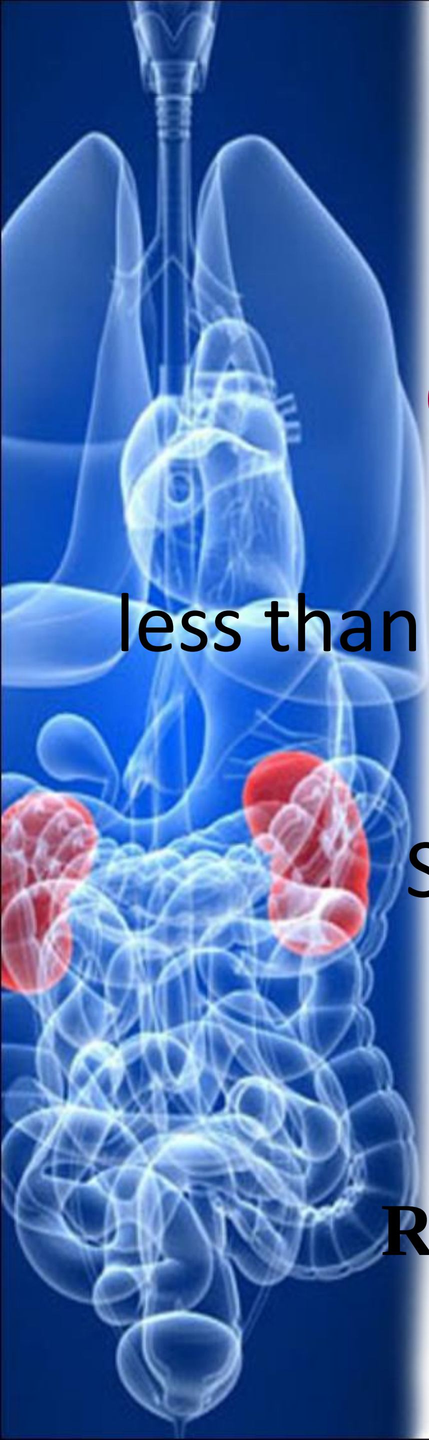
⇧ Inter-lobular vein ⇧ Arcuate vein ⇧ Inter-lobar vein ⇧ Renal vein (<u>4</u> mmHg)



Measurement of the renal blood flow.

1. This can be done by application of the electromagnetic flow meter directly over the renal artery.
2. The most common method used to determine RBF is the estimation of renal plasma flow firstly by calculation of paraminohippuric acid clearance (PAHA) .

N.B: clearance of PAH underestimates true RPF by **10%** which supply nonfunctioning structures



Calculation of RPF

PAHA clearance = effective RPF (10% less than the actual RPF).

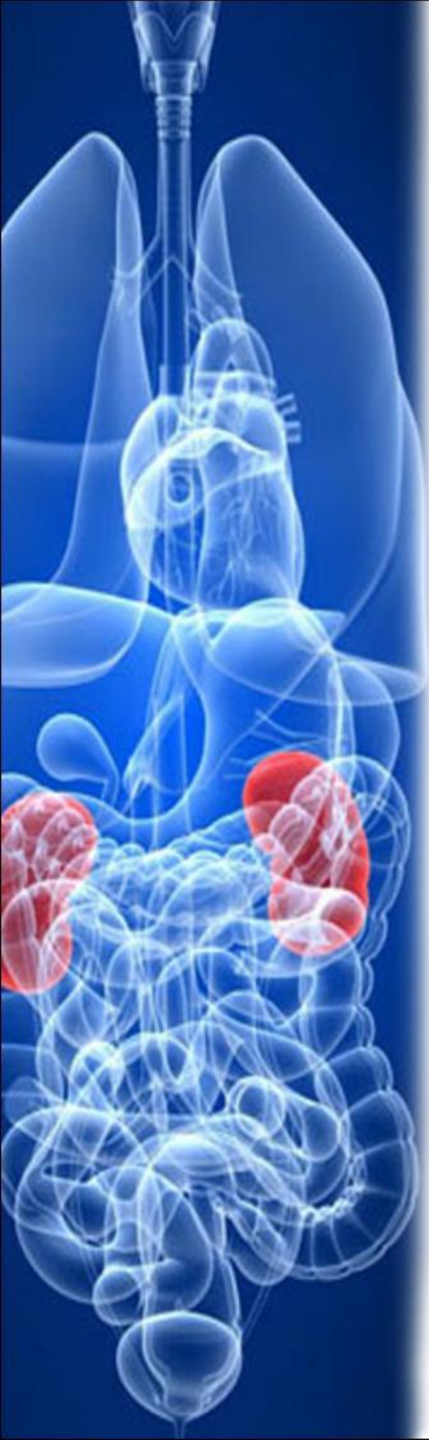
The effective RPF = 630 ml / min.

So, the actual RPF = effective RPF / 90%

= 700 ml / min.

Calculation of RBF

RBF = Actual RPF X (100/100-haematocrite value) = 1250 ml /min

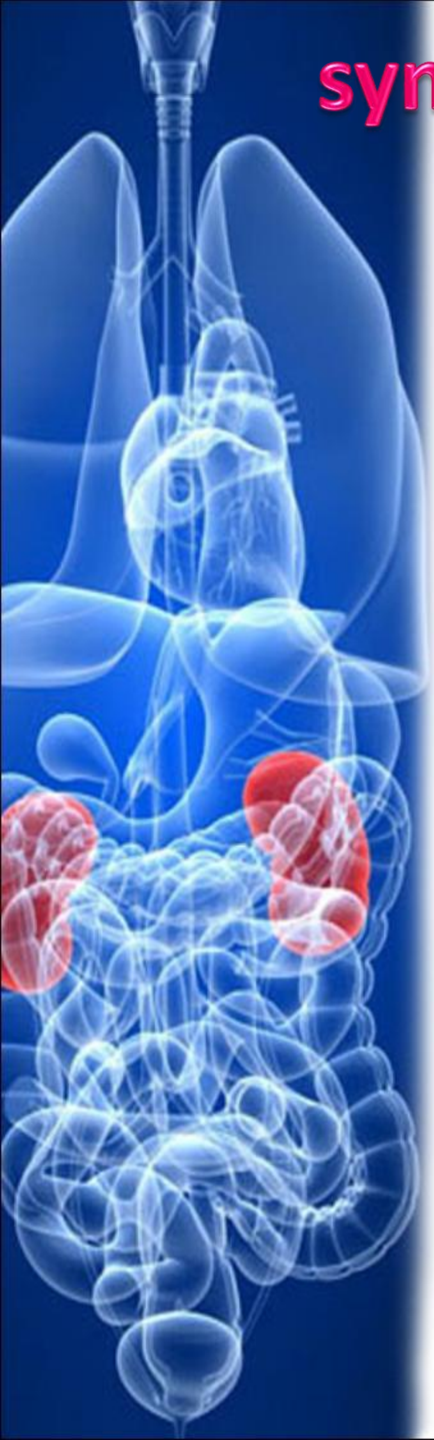


Innervation of the Renal Vessels

The renal nerves travel along the renal blood vessels as they enter the kidney. They contain:

A. Efferents (to kidney):-
   1. Many post-ganglionic sympathetic efferent fibres.
   2. Cholinergic innervations via the vagus nerve.

B. Afferents (from kidney):-
   1. Few sympathetic afferent fibres.
   2. afferents that carry pain sensation.



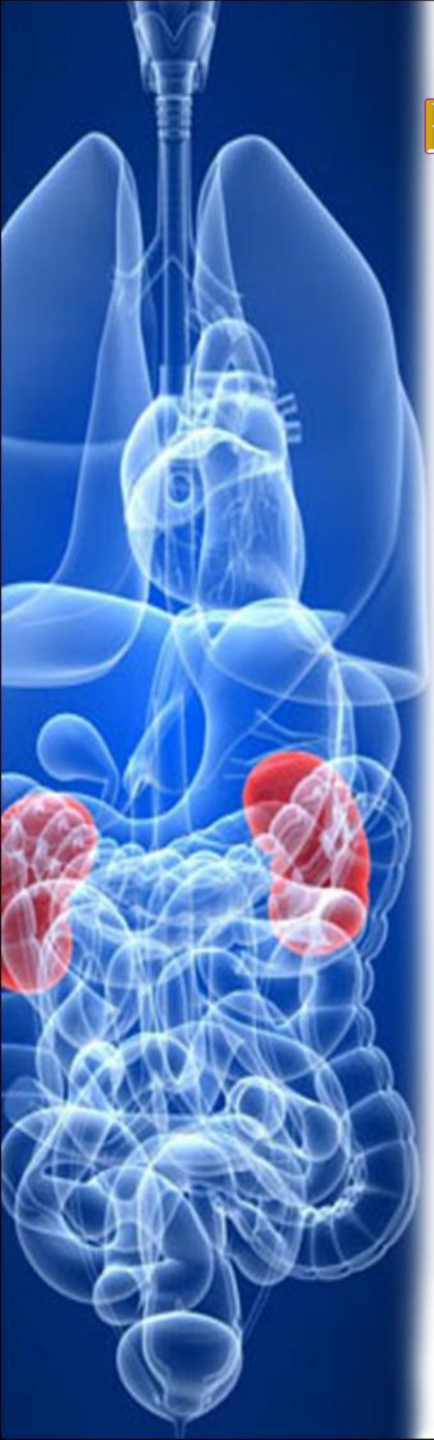
sympathetic innervations to kidney and its effects ?

A. Pathway:-

The sympathetic preganglionic fibers come from the lower thoracic and upper lumbar segments of the spinal cord.

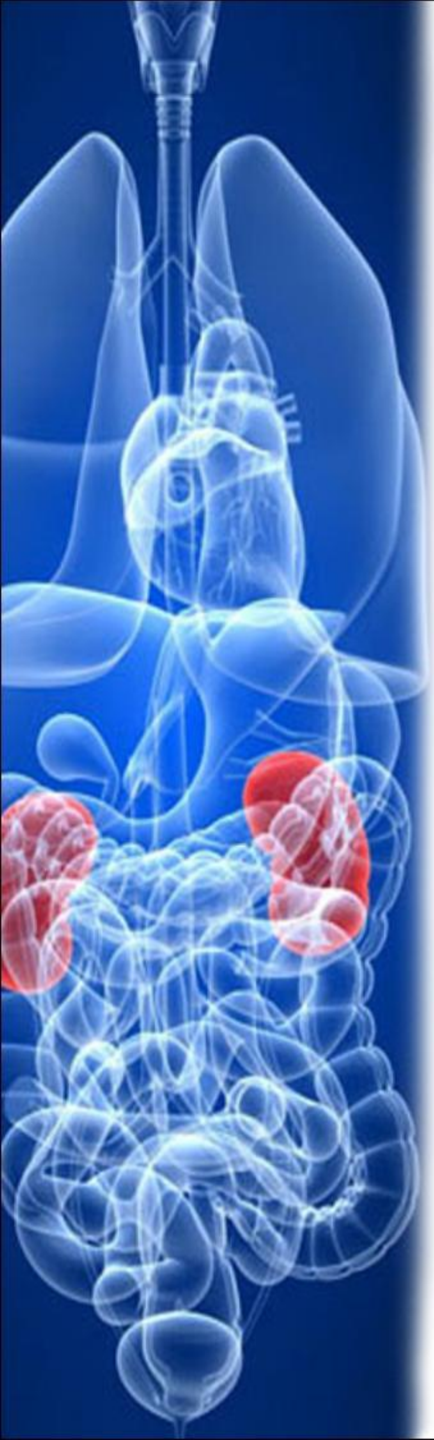
The postganglionic sympathetic fibres supply:

- The afferent and efferent arterioles.
- The proximal and distal tubules.
- The juxtaglomarular cells.
- The Loop of Henle(Thick part)



B. Effect of sympathetic stimulation:

1. V.C of afferent arterioles ($\alpha 1$ receptors) → decreased glomerular filtration and renal blood flow.
2. Increased renin secretion (β_1 receptors at the juxtaglomerular cells).
3. Increased Na+ reabsorption by the renal tubular cells →decreases Na + execration.

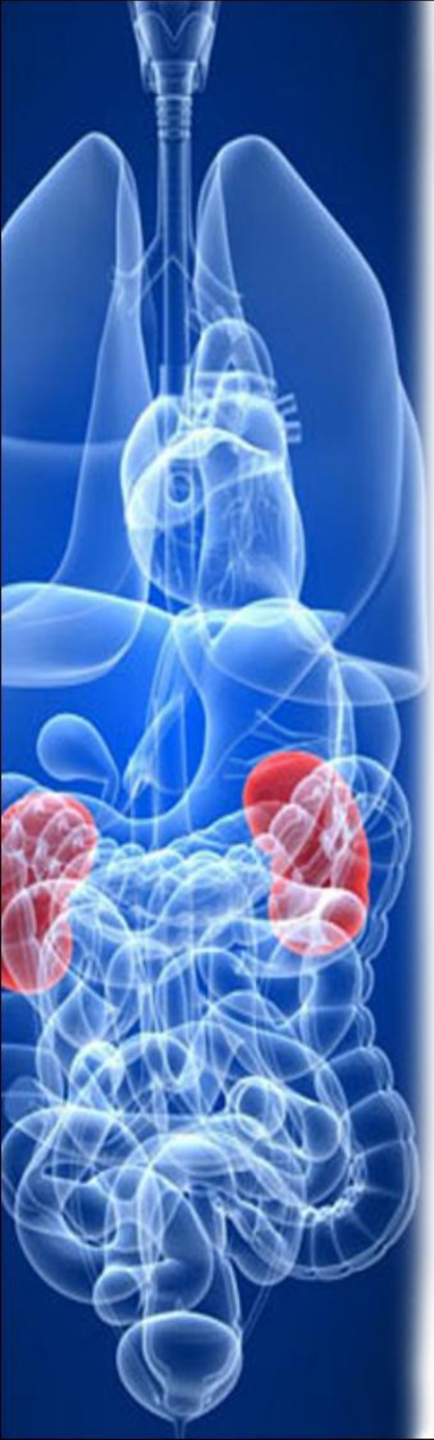


Factors controlling renal blood flow (RBF)

A. Nervous factors

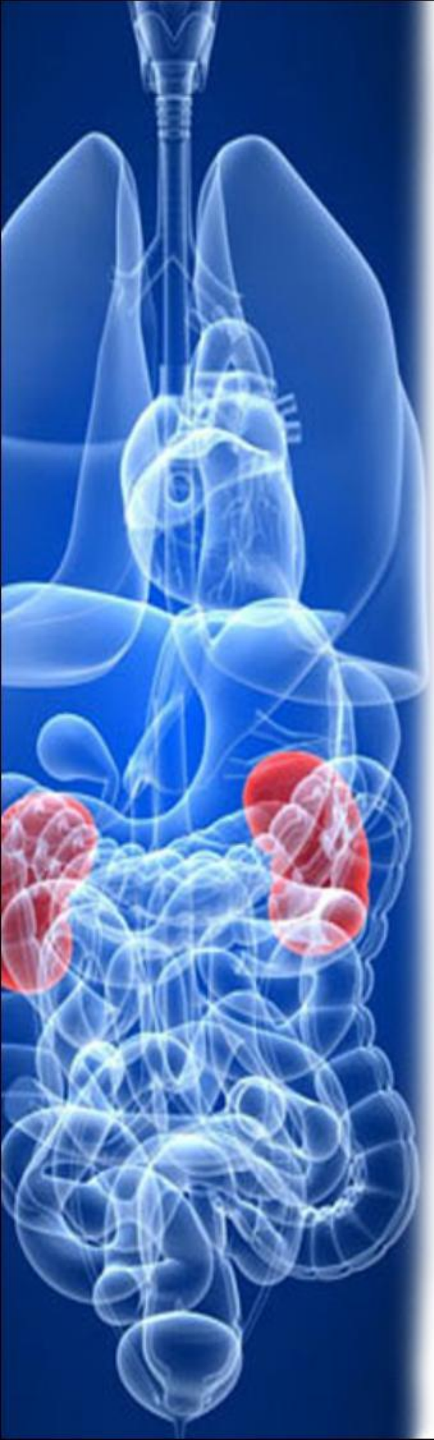
Stimulation of the sympathetic supply to the kidney diminishes RBF through constricting renal artery. and this greatly reduces GFR & urine formation.

e.g. muscular exercise



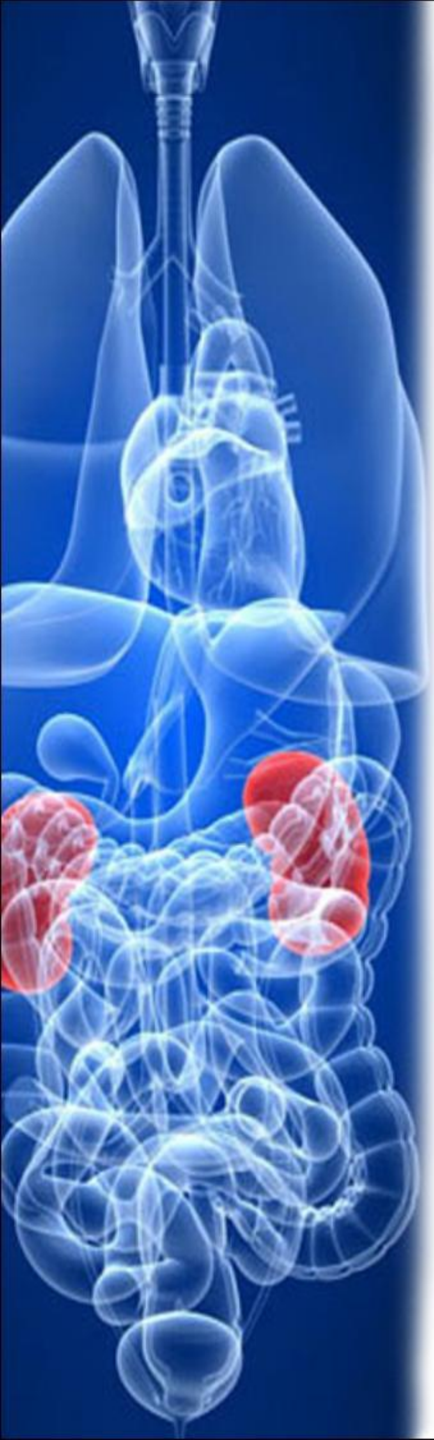
B. Haemodynamic factors

1. Drop in blood pressure as in haemorrhage or severe shock markedly reduces RBF to the degree that causes acute renal failure.
2. During pregnancy renal blood flow may be increased by 50% due to increase Blood volume & Progesterone which causes VD.
3. Sudden standing from recumbent position diminishes RBF by about 20%.



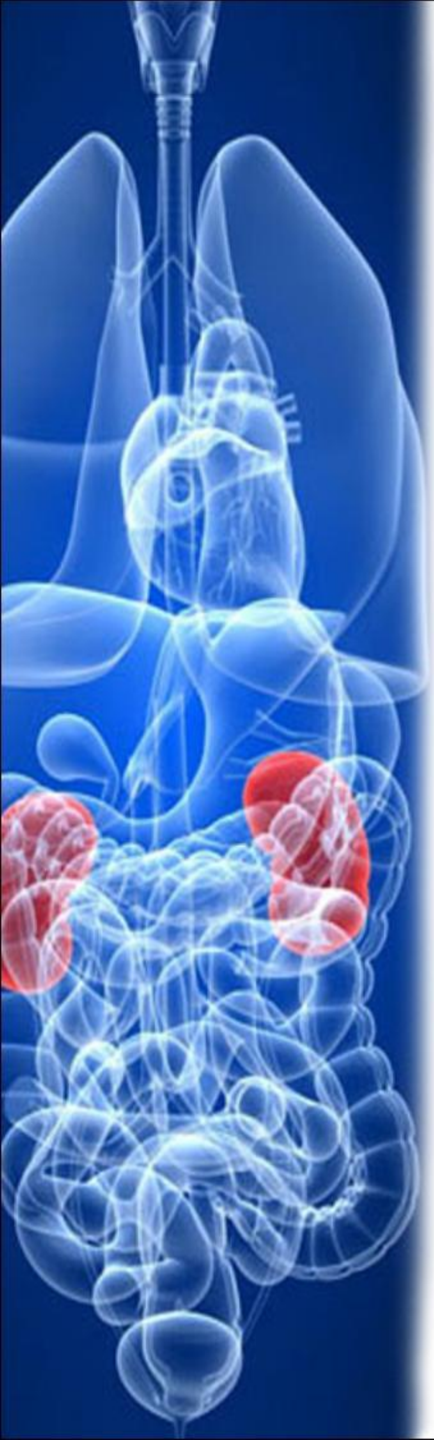
C. Hormonal factors

1. Angiotensin II and norepinephrine diminishes RBF.
2. Endothelin cause renal VC and decrease RBF.
3. Acetylcholine and dopamine cause renal vasodilatation.
4. Nitric oxide (NO) → renal VD



D. Auto-regulation of the renal blood flow:

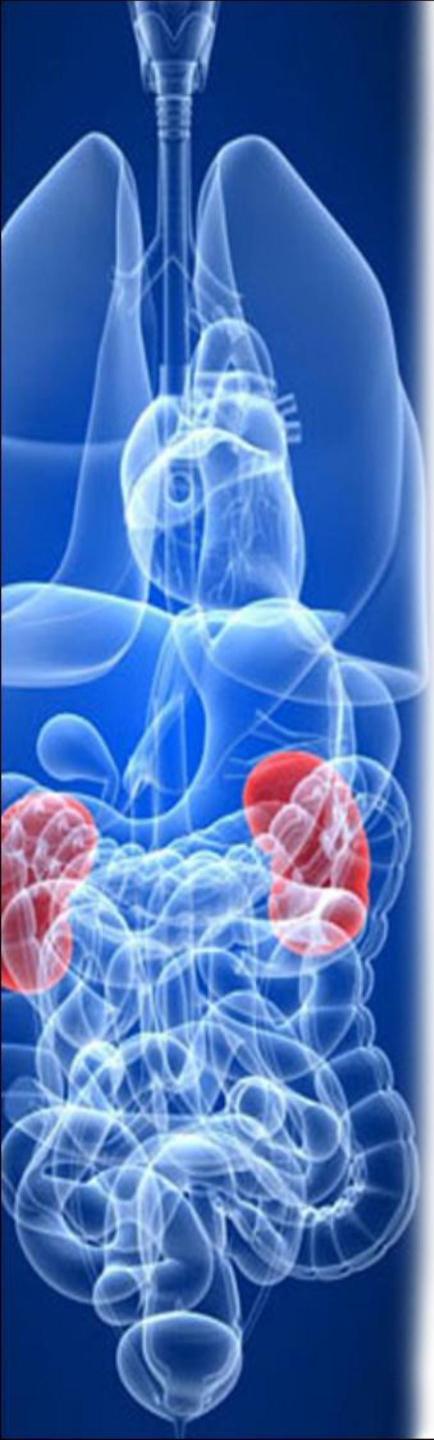
- A fall of ABP to **50 mmHg** may completely stop urine output, while a rise to **210 mmHg** may increase the urine output **7 – 8 times.**
- The kidneys have an auto regulatory mechanism, and the blood pressure may vary from 70 to 180 mm Hg with little change in RBF or GFR.



The mechanism may be:

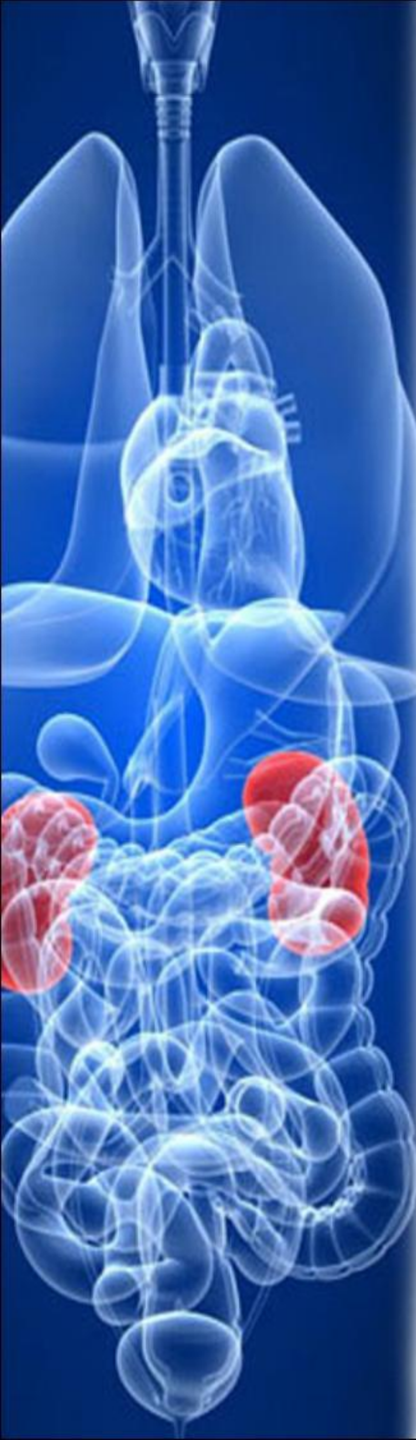
a- Myogenic: an increase in ABP → stretch of arterial wall → increase rate of depolarisation in its smooth muscles → constriction of the arterioles → diminish blood flow.

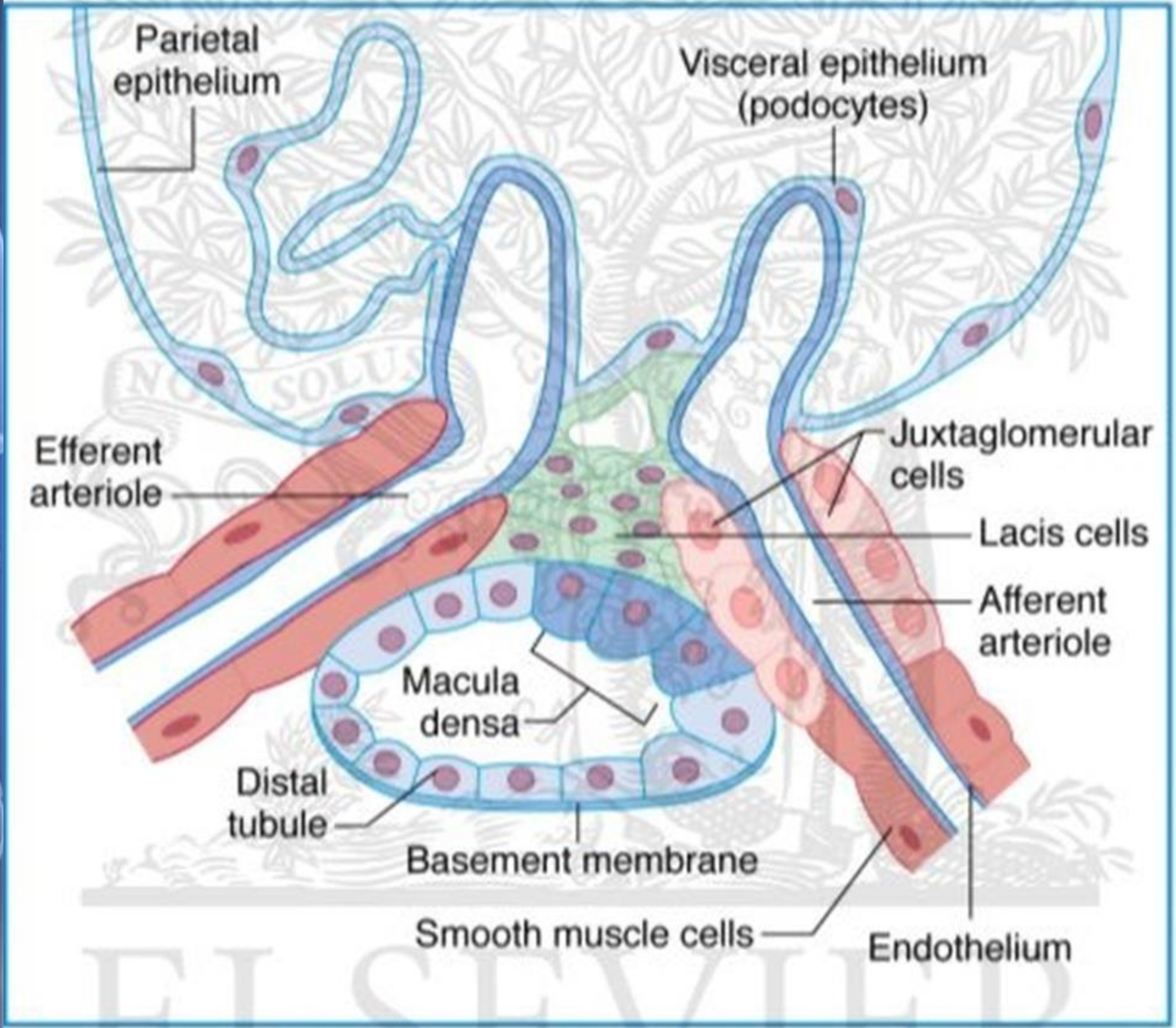
b- Intra-renal tissue pressure theory: the kidney is surrounded by tight capsule, any increase in blood flow → increase in the intrarenal pressure that diminishes excess blood flow.



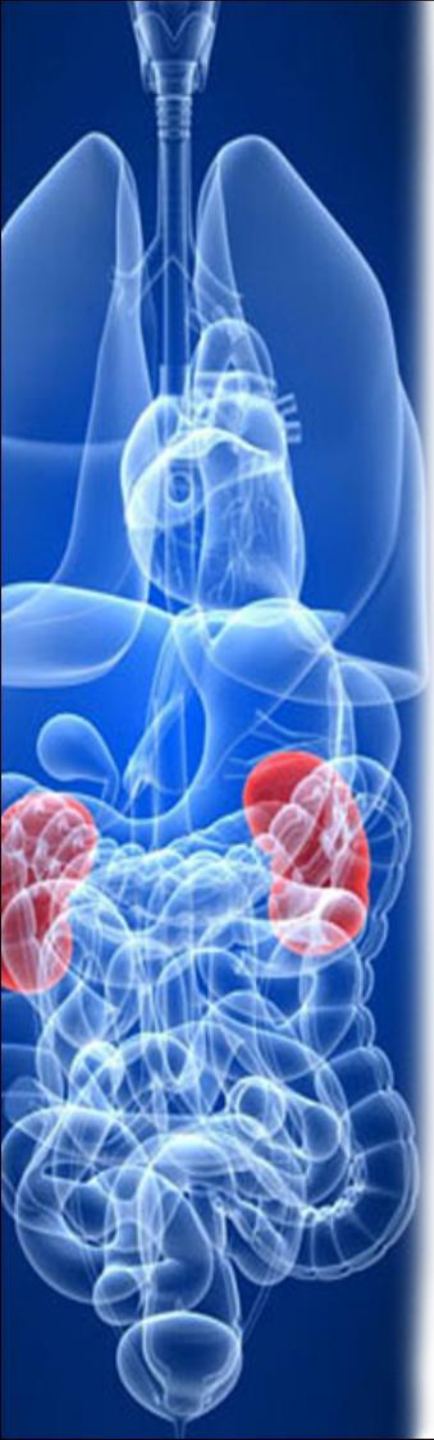
C- Tubulo-glomerular feedback:-

If rate of flow through the first part of DCT is increased, the glomerular filtration decreases (and vice versa).





Parietal epithelium
Visceral epithelium (podocytes)
Efferent arteriole
Juxtaglomerular cells
Lacis cells
Afferent arteriole
Macula densa
Distal tubule
Basement membrane
Smooth muscle cells
Endothelium



Mechanism of tubuloglomerular feedback.

Increase NaCL in the DCT
in front of macula densa cells

Decrease renin secretion by JG cell
in the afferent arteriole

↓

Abolish V.C effect of renin angiotensin
system on efferent arteriole

Increase Na-K ATPas activity

↓

Increase ATP hydrolysis

↓

More adenosine is formed

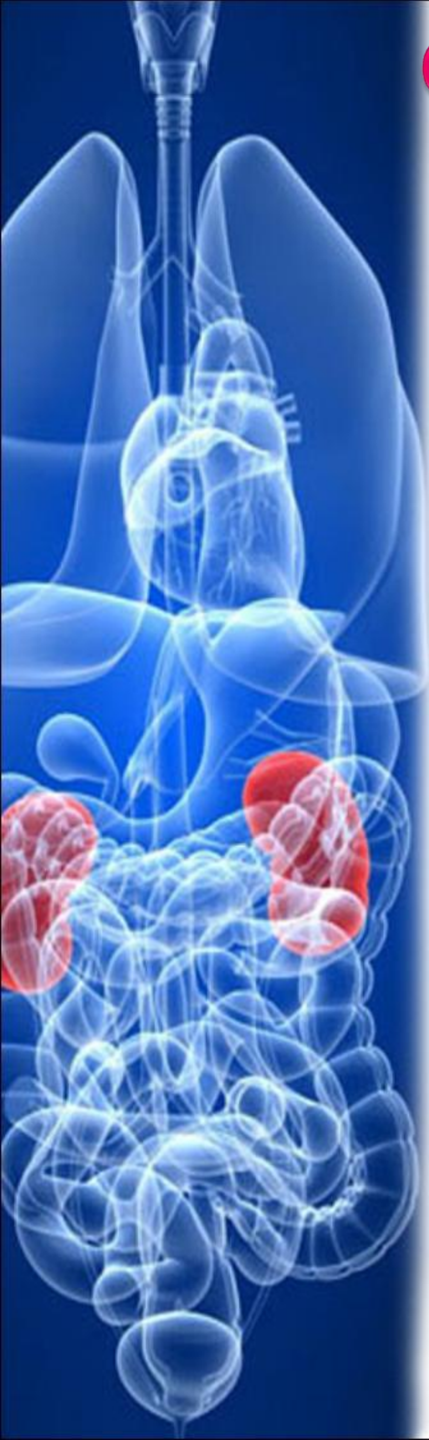
↓

Adenosine is secreted from the basal membrane of the cells and act via adenosine A1 receptors on the macula densa cells to increase their release of Ca^{2+} to vascular smooth muscles in afferent arteriole

↓

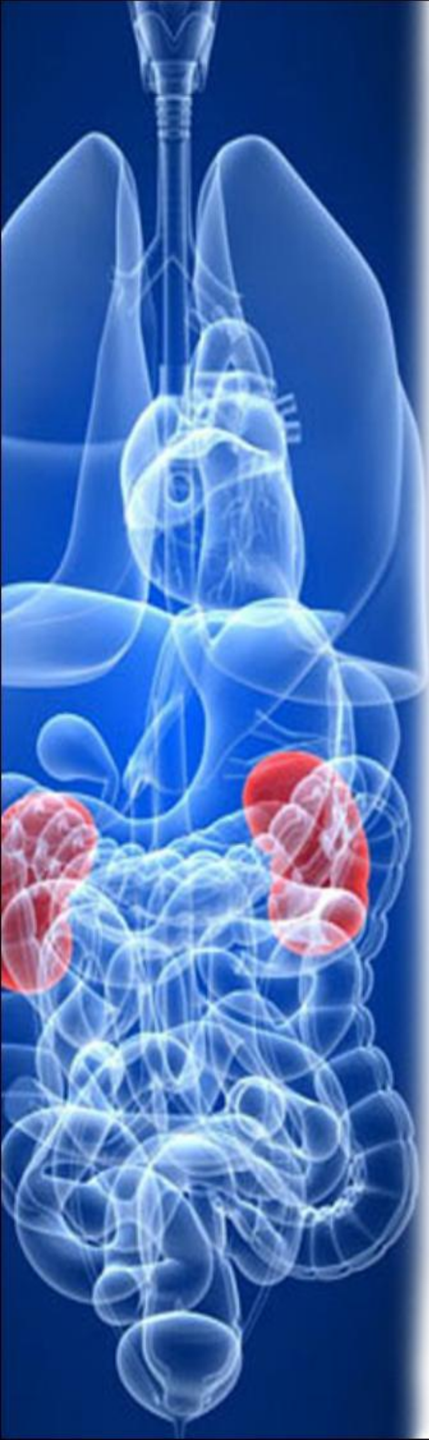
send a signal that cause
VC of afferent arterioles

Decrease GFR



Q) What happen when the ABP rises from 100 to 180 mmHg?

In this condition, constriction (narrowing) of afferent arterioles occurs, so both the RBF & GFR are kept relatively constant (or increase slightly) in spite of the increased ABP. The afferent arteriolar V.C is produced by either the myogenic mechanism or the tubuloglomerular feedback mechanism.

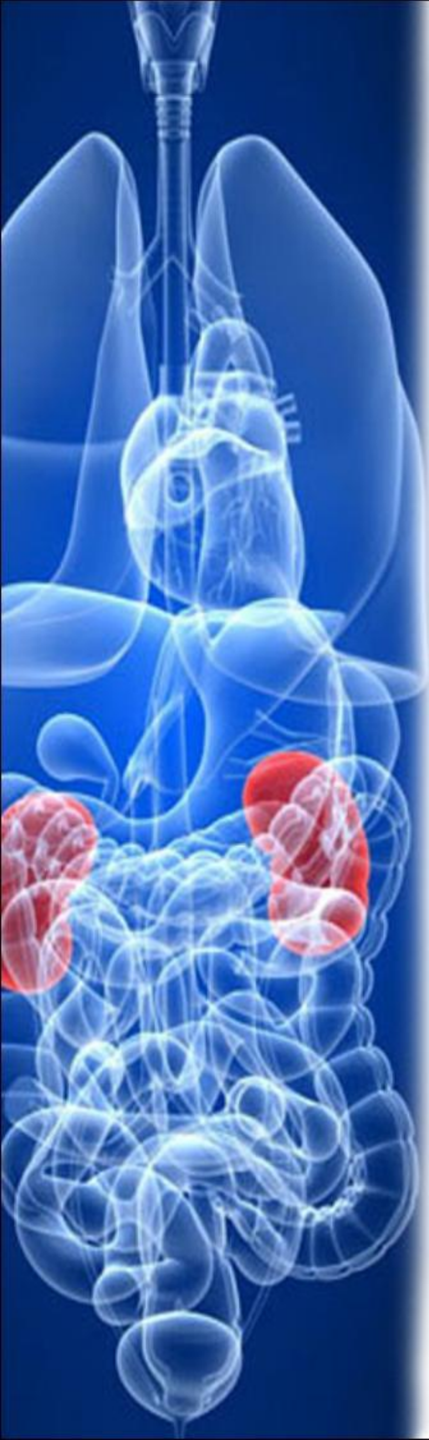


Q) What happens when the ABP falls from 100 to 70 mmHg?

A. V.D of the afferent arterioles by releasing a prostaglandin (PGI2).

B. V.C of the efferent arterioles by secreting renin which increase the formation of angiotensin II.

- The former increases the RBF while the later increases the renal vascular resistance , and both increase the glomerular capillary pressure , so the GFR is kept relatively constant(or decrease slightly) in spite of the decreased ABP.



Decrease ABP→ decease glomerular hydrostatic pressure→ decrease GFR → decrease in NaCL load and Decrease flow in DCT→ Stimulation of macula densa cells

↓

+ Juxtaglomerular cells

To secret Renin

↓

Increase Angiotensin II

↓

V.C of efferent arteriole That increase efferent arteriolar resistance

↘

releasing a prostaglandin (PGI2).

↓

V.D of Afferent arterioles that decrease afferent arteriolar resistance

increase GF. Pressure → restore GFR

The tubuloglomerular feedback.


THank you