Just to remember:
glomerular pressure is affected by afferent arteriolar resistance and efferent arteriolar resistance.
If the afferent arteriolar resistance increases,, renal blood flow will decrease,, and pressure in the glomerulus will decrease and GFR will decrease.
If the efferent arteriolar resistance increases,, renal blood flow will decrease,, and pressure will increase and GFR will increase “for a while because of the increase in pressure but it will go back to decrease because renal blood flow decreases”
Colloid osmotic pressure in glomerulus depends on filtration fraction and on colloid osmotic pressure of plasma,, the higher the colloid pressure in plasma the higher it will be in the glomerulus. Regarding filtration fraction if it is high it will increase the osmotic pressure in glomarulus “because there is less water”.
If filtration coefficient increases it will cause increase in GFR, if pressure in Bowman’s capsule increases GFR will decrease,, if osmotic pressure in glomerulus increases it will cause decrease in GFR,, If plasma colloid pressure increases it will decrease GFR.
If filtration fraction increases this will increase the colloid osmotic pressure,, If glomerular pressure increases this will increase GFR.
If afferent arteriolar resistance increases this will decrease the pressure in glomarulus and will decrease GFR,, if efferent arteriolar resistance increases this will increase GFR “for a while”.

Renal blood flow:
Flow = delta P/ R
so what effects the flow is pressure and resistance “from afferent and efferent arterioles and venous resistance”.
Renal flow is about ¼ the cardiac output “1200ml/min” and it’s not for oxygen supply but it’s for filtration.
Oxygen that is consumed by the kidneys is directly proportional to the tubular reabsorption of sodium “which consumes most of the energy of the kidney because it’s active and it’s through K+/Na+ pump (it’s not primary active it’s secondary active).

Control of GFR and renal blood flow:
they are mostly auto-regulated “even if the arterial pressure increased from 60 to 180 renal blood flow will remain nearly constant”.
It works through two mechanisms:
1- Tubulu-glumerular feedback “macula densa mechanism”:
it is macula densa with afferent and efferent arterioles uniting with distal tubule,, they have sensors for sodium chloride concentration, accourding to the NaCl concentration in the distal tubule they react”the reaction is through secretion of nitric oxide “vasodilator” or endothelin “vasoconstrictor””
so the control is local and neurohormonal “almost all tissues control their blood flow locally”.
Neurohormones that affect the kidneys are sympathetic and parasympathetic.
Hormones that affect the kidneys are nitric oxide, endothelin and angiotensin 2 and prostaglandin.
Sympathetic control “epinephrine and nore-epinephrine” this will increase the resistance so blood flow and GFR decreases “especially in severe hemorrhage GFR will decrease and eventually this will lead to renal failure (signs for it are: creatinine increase in the blood and urea increases, acidosis may happen”.
Angiotensin 2 control: “it’s secreted due to increase in rennin” it increases the efferent arteriolar resistance partially so blood flow will decrease but GFR will increase at the beginning then it will decrease “so overall it will not change”.
Nitric oxide “used to be called endothelial cell derived relaxing factor” it causes vasodilation so blood flow will increase and GFR will increase, this protects against high constriction in cases of shock “endothelial cells miss-function”.
Prostaglandin: causes decrease in the resistance of kidney’s arterioles and this will lead to increase in GFR and in renal blood flow.
Prostaglandins can be blocked “GFR will decrease” using drugs like cyclo-oxygenase inhibitors ,nonsteroidal anti-inflammatory drugs “aspirin”.
,prostaglandins come from arachidonic acid “20carbons and 4 double bonds” by the effect of cyclo-oxygenase.
endothelin: it increases the arteriolar resistance in the kidneys causes decrease in renal blood flow and GFR, patients with problem in this hormone can be given “endothelin antagonist”.

If there is an increase in GFR, this will increase filtration so there will be more sodium in the distal tubule so the macula densa cells will sense that there is more sodium, so it sends more vasoconstrictors to the afferent arterioles “endothelin” so renal blood flow will decrease and GFR will return to normal.
If the GFR decreases macula densa will sense less Na+ so it will send vasodilators to the arterioles “nitric oxide” and this will increase renal blood flow and GFR will return to normal.

2- Myogenic mechanism:
when there is increase in pressure “increase in blood flow and GFR” this will stretch the wall of the vessel and this will lead to opening of calcium channels, so calcium will enter the cell “calcium influx” this will cause the cells to contract and this will increase the resistance “and decrease the flow and GFR”.

\*\*macula densa cells are in the distal tubule.
\*\*change in the reabsorption will help the auto regulation in conserving constant urine volume.

Angiotensin 2 is so important for auto-regulation.
Macula densa cells have three hormones to control the blood flow with “endothelin, nitric oxide, and angiotensin”

After age of 40 every 10 years there will be decrease by 10% in GFR.
Dietary proteins causes increase in the GFR through two mechanisms:
1- they increase formation of urea.
2- Through increasing proximal tubule reabsorption, so macula densa will sense decrease in NaCl in distal tubule, then it secrets vasodilators to the arterioles causing increase in blood flow and GFR.