

Physiology of renal circulation

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Overview

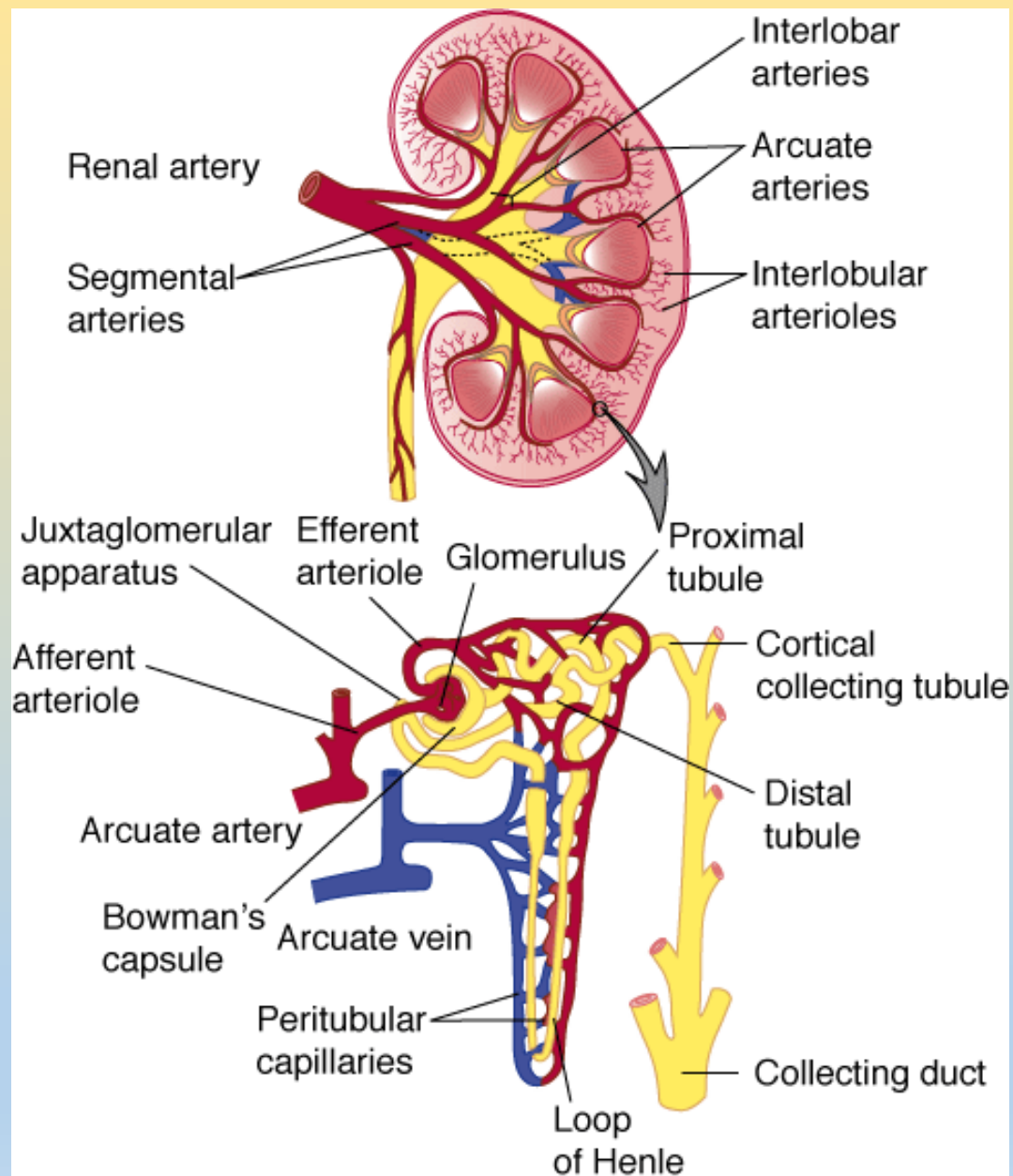
- Anatomy of renal circulation
- Physiological control of renal blood flow
- Renal autoregulation -Mechanisms

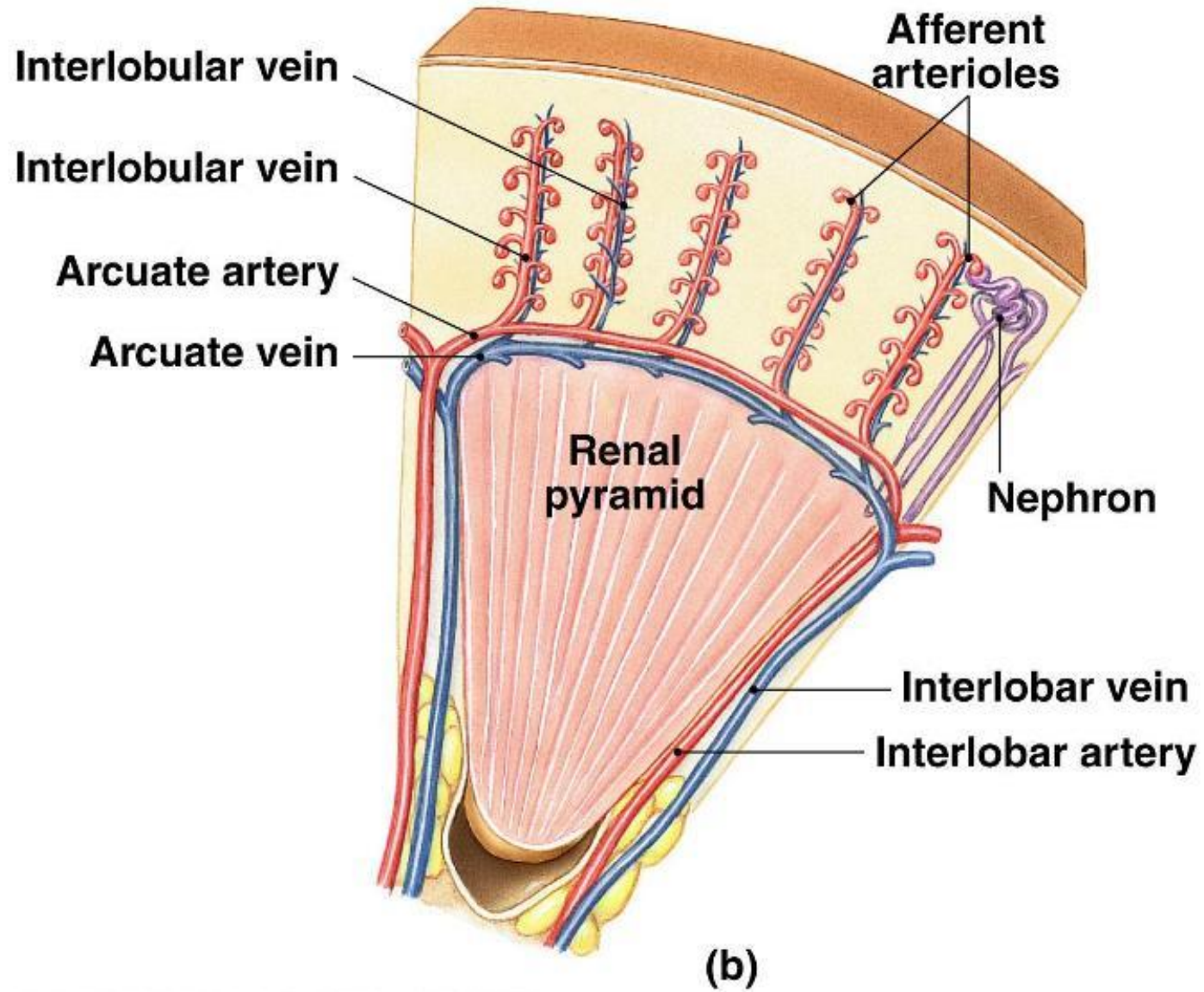
Renal blood flow

- About 22 % of the cardiac output, or 1100 ml/min
- The renal artery enters the kidney through the hilum and then branches to
 - interlobar arteries
 - arcuate arteries
 - interlobular arteries (also called radial arteries)
 - afferent arterioles, which lead to the glomerular capillaries

Renal blood flow ctd..

- The distal ends of the capillaries of each glomerulus coalesce to form the efferent arteriole
- second capillary network, the peritubular capillaries, that surrounds the renal tubules.





Determinants of Renal Blood Flow

(Renal artery pressure) - (Renal vein pressure)

Total renal vascular resistance

- Most of the renal vascular resistance resides in three major segments:
 - interlobular arteries
 - afferent arterioles
 - efferent arterioles
- Resistance of these vessels is controlled by the sympathetic nervous system, various hormones, and local internal renal control mechanisms

Physiologic control of renal circulation

- **sympathetic nervous system**
- **hormones**
- **autacoids (vasoactive substances that are released in the kidneys and act locally)**

Sympathetic nervous system

- all the blood vessels of the kidneys(including the afferent and the efferent arterioles) are richly innervated by sympathetic nerve fibres.
- Strong activation of the renal sympathetic nerves can constrict the renal arterioles and decrease renal blood flow and GFR
- Eg-during severe, acute disturbances lasting for a few minutes to a few hours, such as those elicited by the defense reaction, brain ischemia, or severe hemorrhage

hormonal and autacoid response

Substance	Vascular response
Noradrenaline	Vasoconstriction
Endothelin	Renal vasoconstriction
Angiotensin II	Vasoconstriction Efferent >Afferent
PG	Renal vasodilation in the cortex
NO	vasodilation
Bradykinins/dopamine	Vasodilation

hormonal and autacoid control

Hormones and Autacoids That Influence Glomerular Filtration Rate (GFR)

Hormone or Autacoid	Effect on GFR
Norepinephrine	↓
Epinephrine	↓
Endothelin	↓
Angiotensin II	↔ (prevents ↓)
Endothelial-derived nitric oxide	↑
Prostaglandins	↑

Renal autoregulation

- Feedback mechanisms intrinsic to the kidneys normally keep the renal blood flow and GFR relatively constant, despite marked changes in arterial blood pressure.
- This relative constancy of GFR and renal blood flow is referred to as autoregulation
- Renal blood flow and GFR relatively constant over an arterial pressure range between 80-170 mmhg

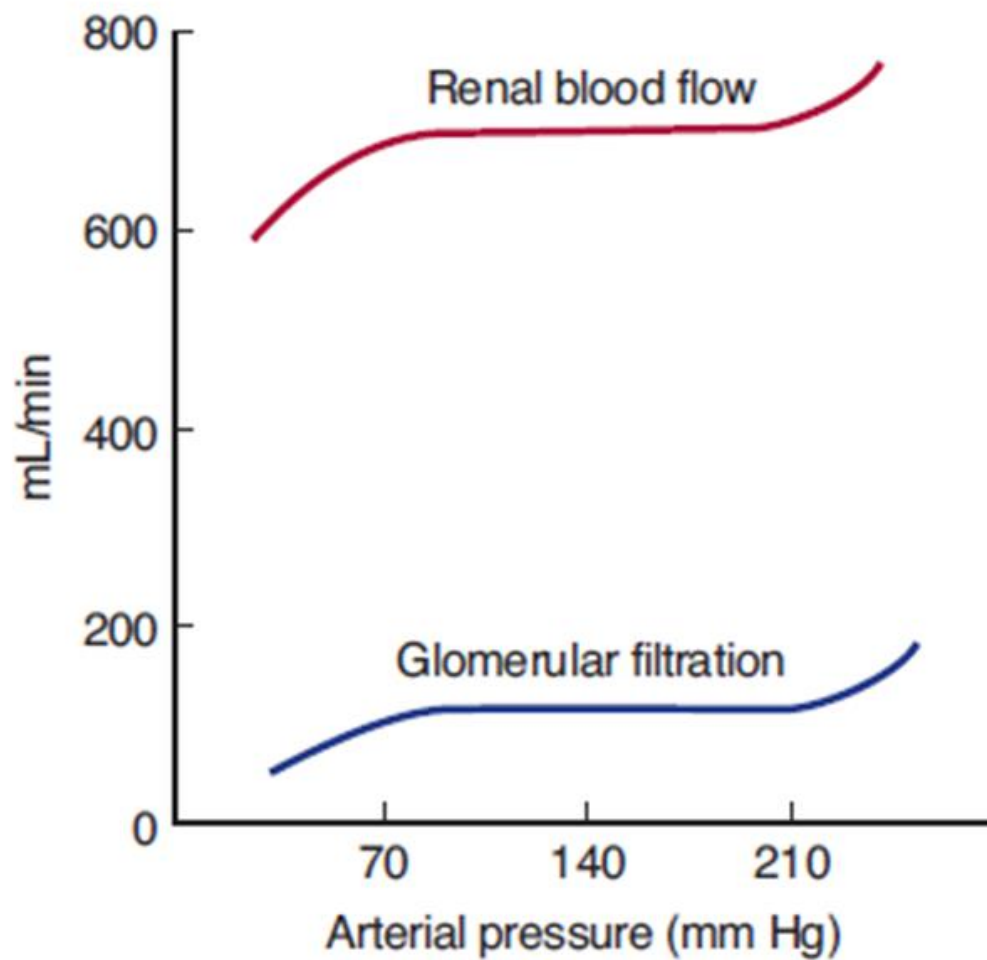


FIGURE 37-4 Autoregulation in the kidneys.

Renal autoregulation

- The major function of autoregulation in the kidneys is to
 - maintain a relatively constant GFR
 - to allow precise control of renal excretion of water and solutes

Mechanisms involved in autoregulation

- Tubular glomerular feed back
- Myogenic response

Myogenic response

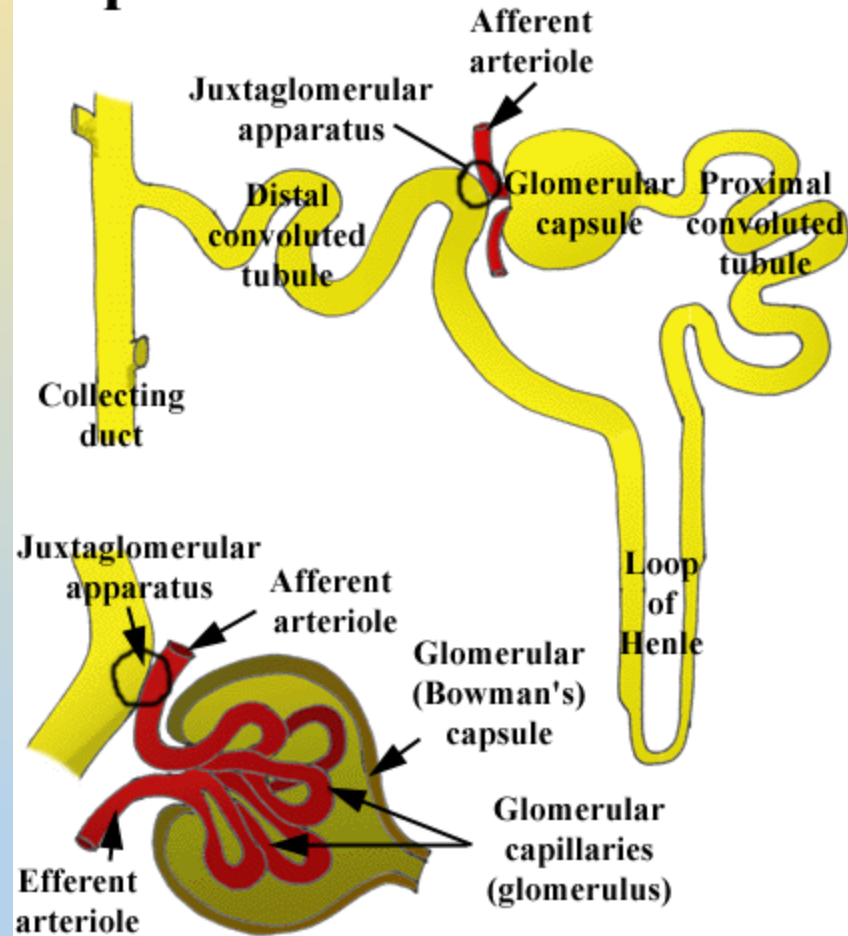
- The ability of individual arteries to resist stretching in response to increased arterial pressure.
- Stretch of the vascular wall allows increased movement of calcium ions from the extracellular fluid into the cells, causing them to contract

Tubular glomerular feed back

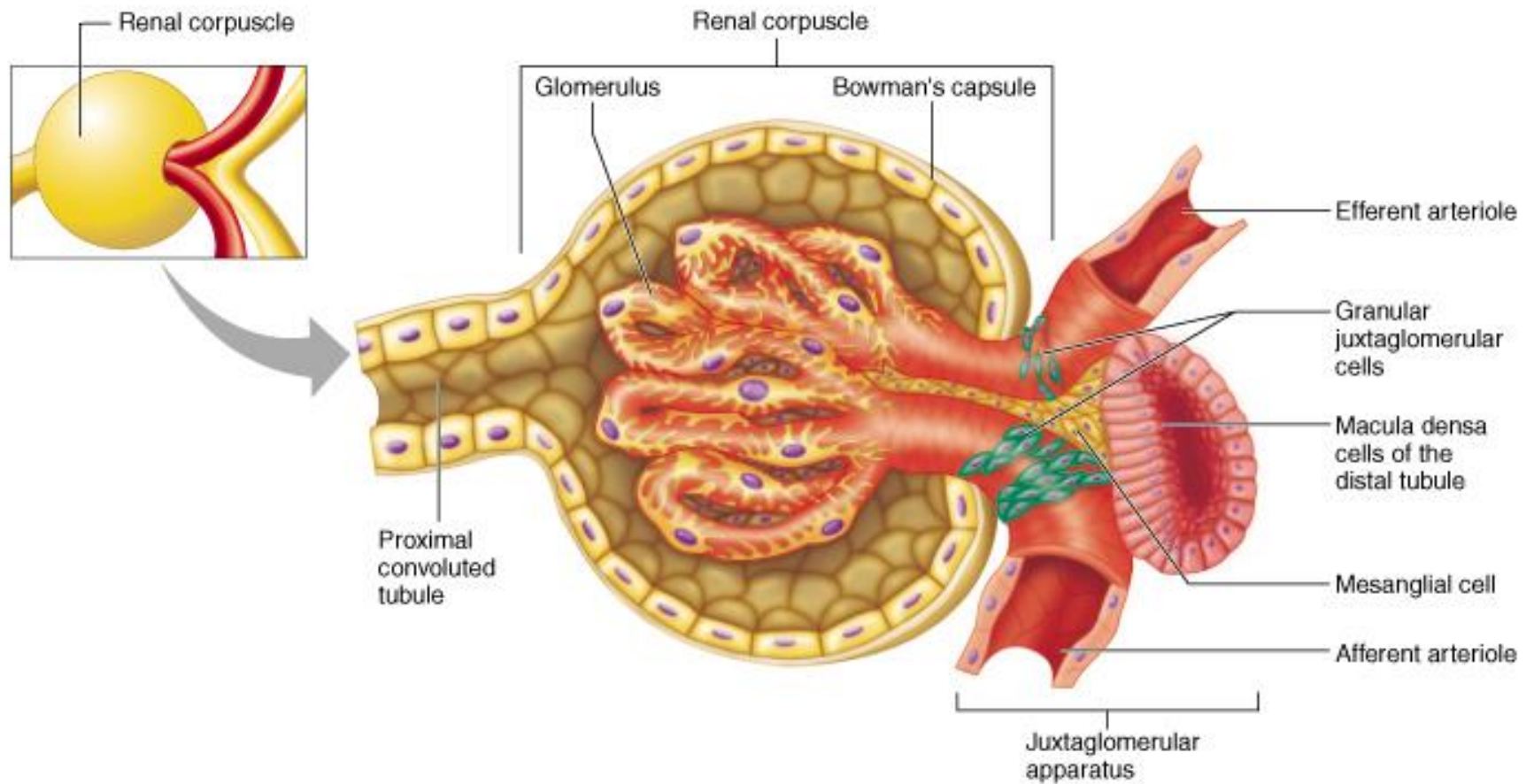
Changes in the GFR detected by the tubules that sends feed back to the glomerulus to bring about correction of GFR to appropriate levels.

- 1) afferent arteriolar feedback
- 2) efferent arteriolar feedback

Nephron



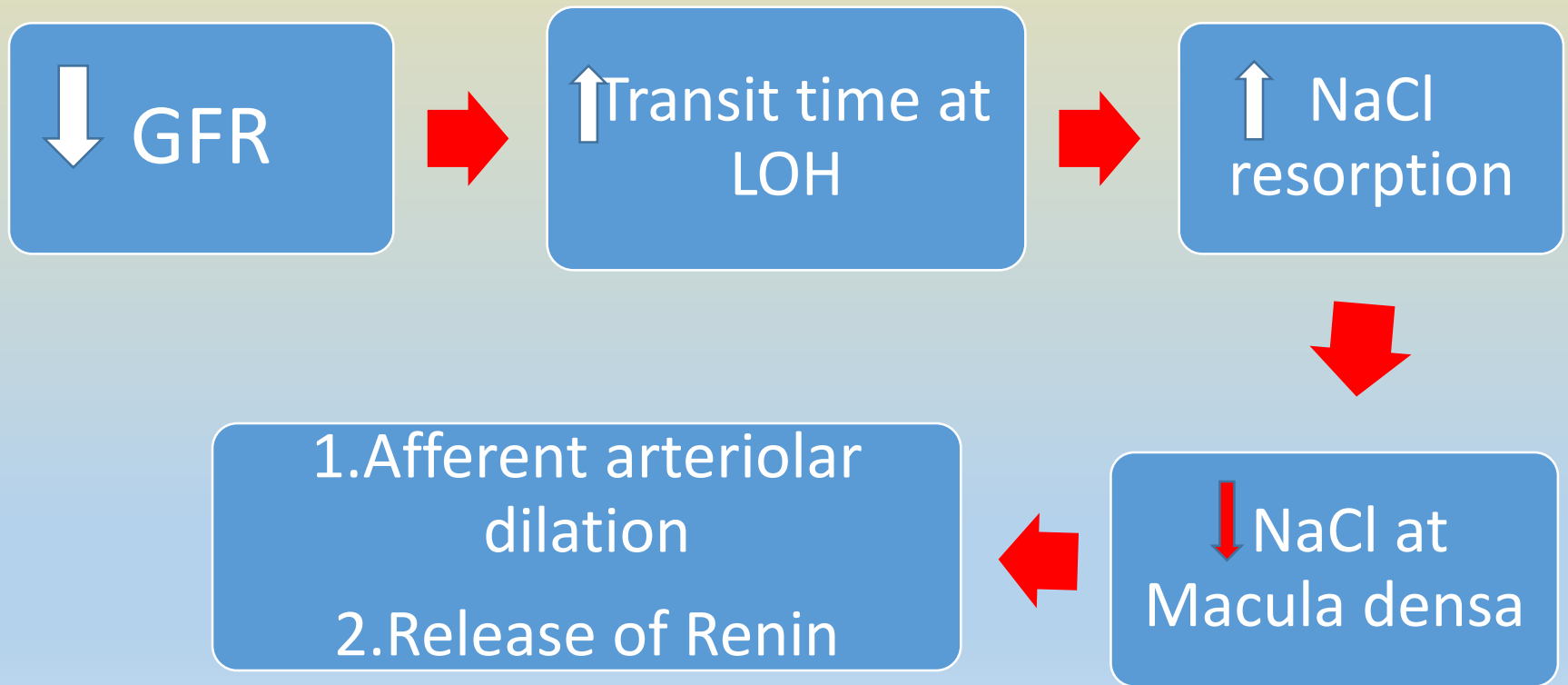
Juxtaglomerular apparatus



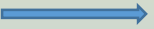
Macula densa

- Tuft of epithelial cells at the junction of thick ascending limb and DCT
- Macula densa rest between afferent and efferent arterioles of its own glomerulus
- It uses the composition of the tubular fluid as an indicator of GFR

Tubular glomerular feed back



Tubuloglomerular feedback

- Increase in Renin causes a rise in Angiotensin II.
- Angiotensin II  Efferent arteriolar constriction



Rise in hydrostatic
pressure at glomerular capillaries

Adaptations of the circulation to optimize renal function

hydrostatic pressures

- hydrostatic pressure in the glomerular capillaries are high (66 0mmhg)
- Afferent arterioles are short, straight branches of the interlobular arteries.
- Efferent arterioles are high resistant vessels
- Peritubular capillary hydrostatic pressure is low (13mmhg)

- This allows for increase in filtration of plasma at the glomeruli and reabsorption at the peritubular capillary plexus.

MCQ

- Regarding renal circulation
- A) The kidney receives about 10% of CO
- B) Renal blood flow changes with the changes in arterial blood pressure
- C) Increase in sympathetic stimulation increases renal blood flow
- D) Renal blood flow is a main determinant of the GFR
- E) Angiotensin II only affect efferent arterioles
- F) Angiotensin is produced by the afferent arterioles

home work

- Why does high protein intake increase renal blood flow?