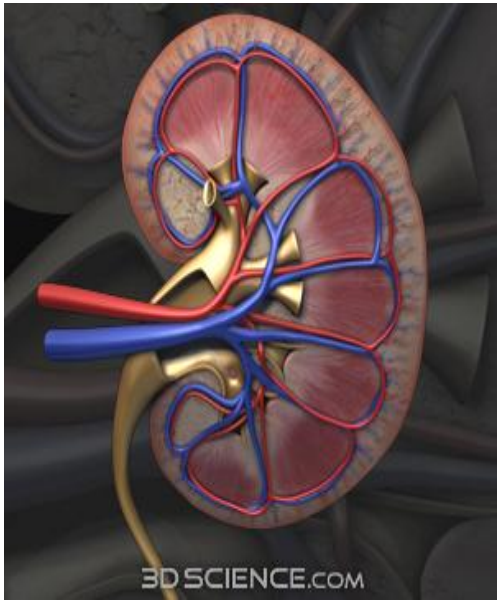
Topic 4: Renal System

Chapters 25 and 26



4.1 Describe the anatomy of the male & female urinary systems

4.1.1 describe the gross anatomy of the kidneys, including the blood & nerve supplies

- bean-shaped; **retroperitoneal**
- superior lumbar region: from ____ thoracic vertebra to _____ lumbar vertebra
- some protection from _____; rt kidney pushed lower by liver
- ~150 g; 12 x 6 x 3 cm
- adrenal glands sit on top of kidneys

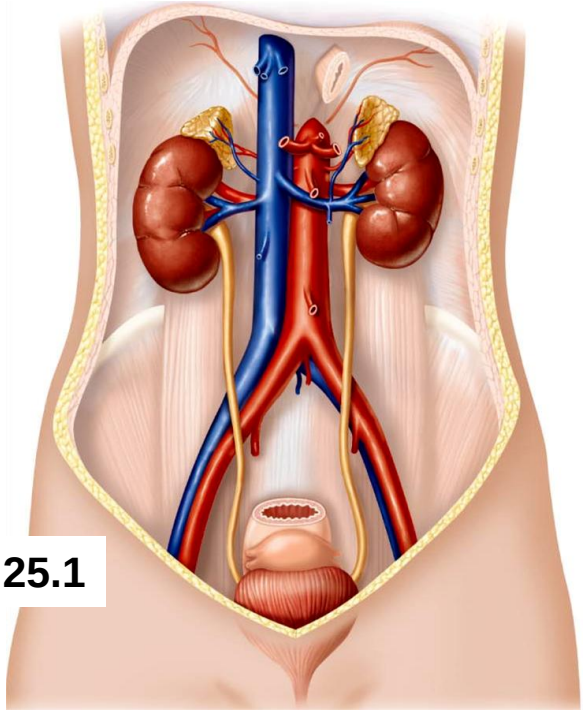


Fig. 25.1

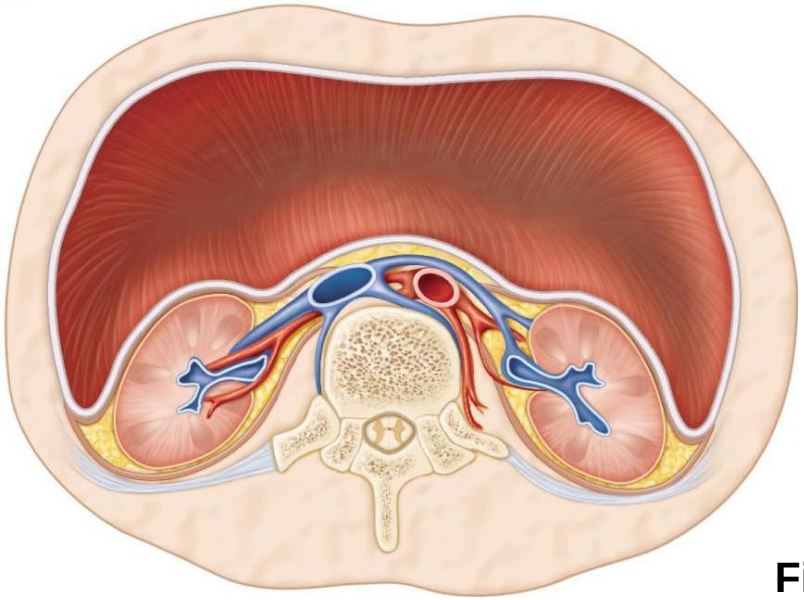
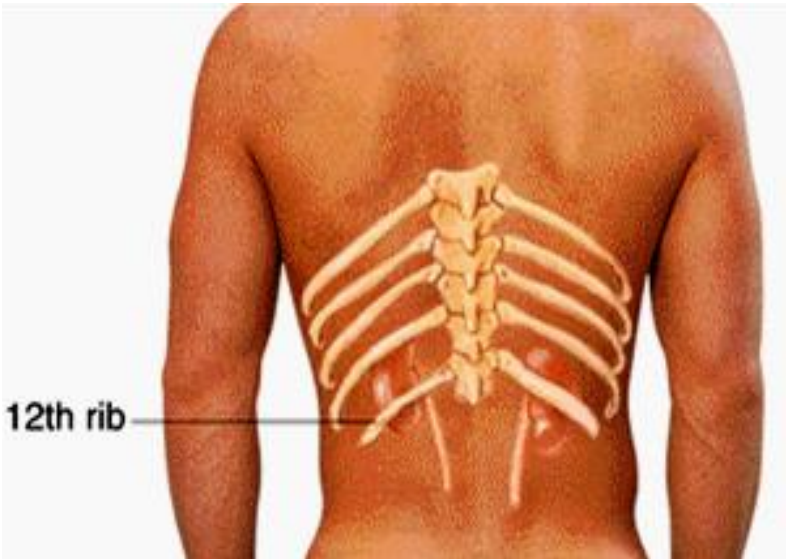


Fig. 25.2



concave medially: renal hilus to renal sinus

3 layers of supportive tissue

renal capsule: fibrous, adheres directly to kidney surface, strong barrier to ?

adipose capsule: cushions, helps hold kidney in place

What is renal ptosis?

renal fascia: dense CT surrounds adrenal gland & kidney; anchoring role

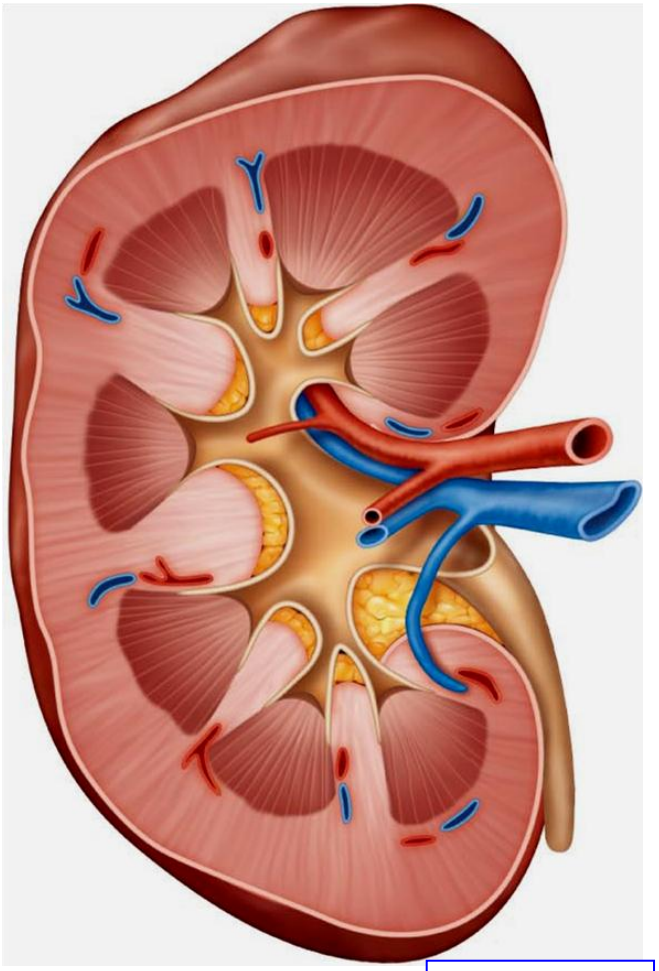
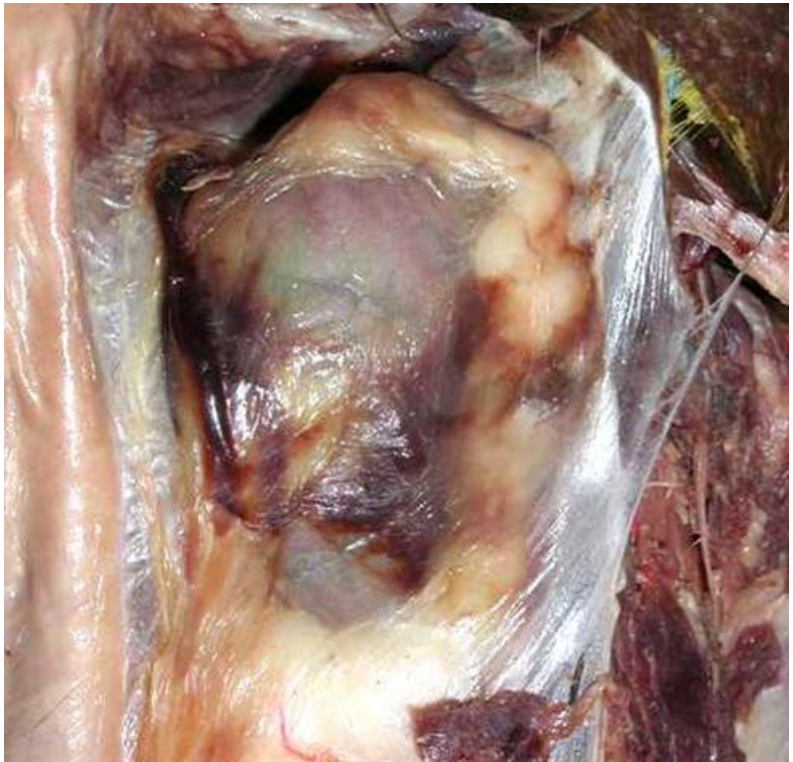


Fig. 25.3

J. Carnegie, UofO



http://biology.clc.uc.edu/fankhauser/Labs/Anatomy_&_Physiology/A&P203/Urinary_Tract_Histology/Anatomy/urinary_anatomy.html

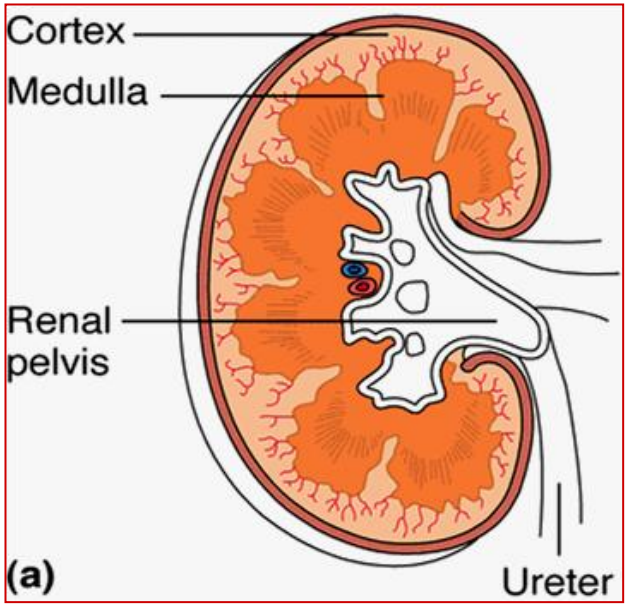
INTERNAL ANATOMY

(i) **cortex:** *What process occurs here?*

(ii) **medulla:** darker colour; **medullary** or **renal pyramids**; *Why do they appear striped ?* separated by **renal columns** (cortical tissue); each medullary unit = ~1/8 of kidney

(iii) **pelvis:** flat, funnel-shaped tube continuous with ureter; major & minor calices; minor calices enclose papillae of pyramids; calices collect urine

walls of calyces, pelvis, ureter contain smooth muscle; propel urine by peristalsis



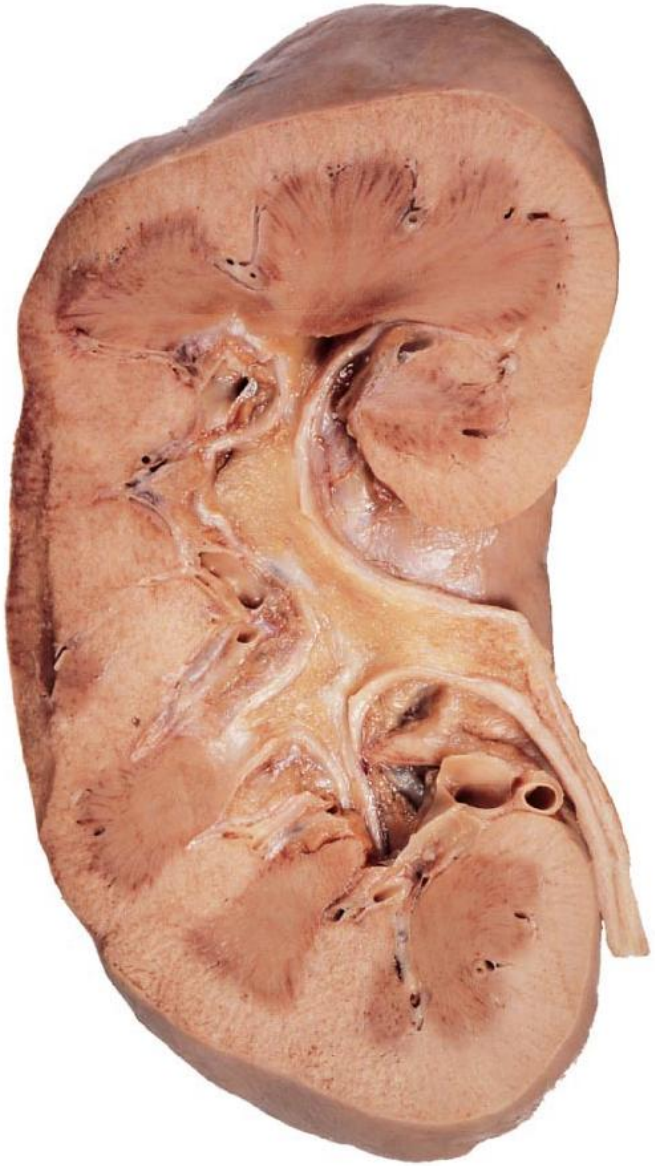
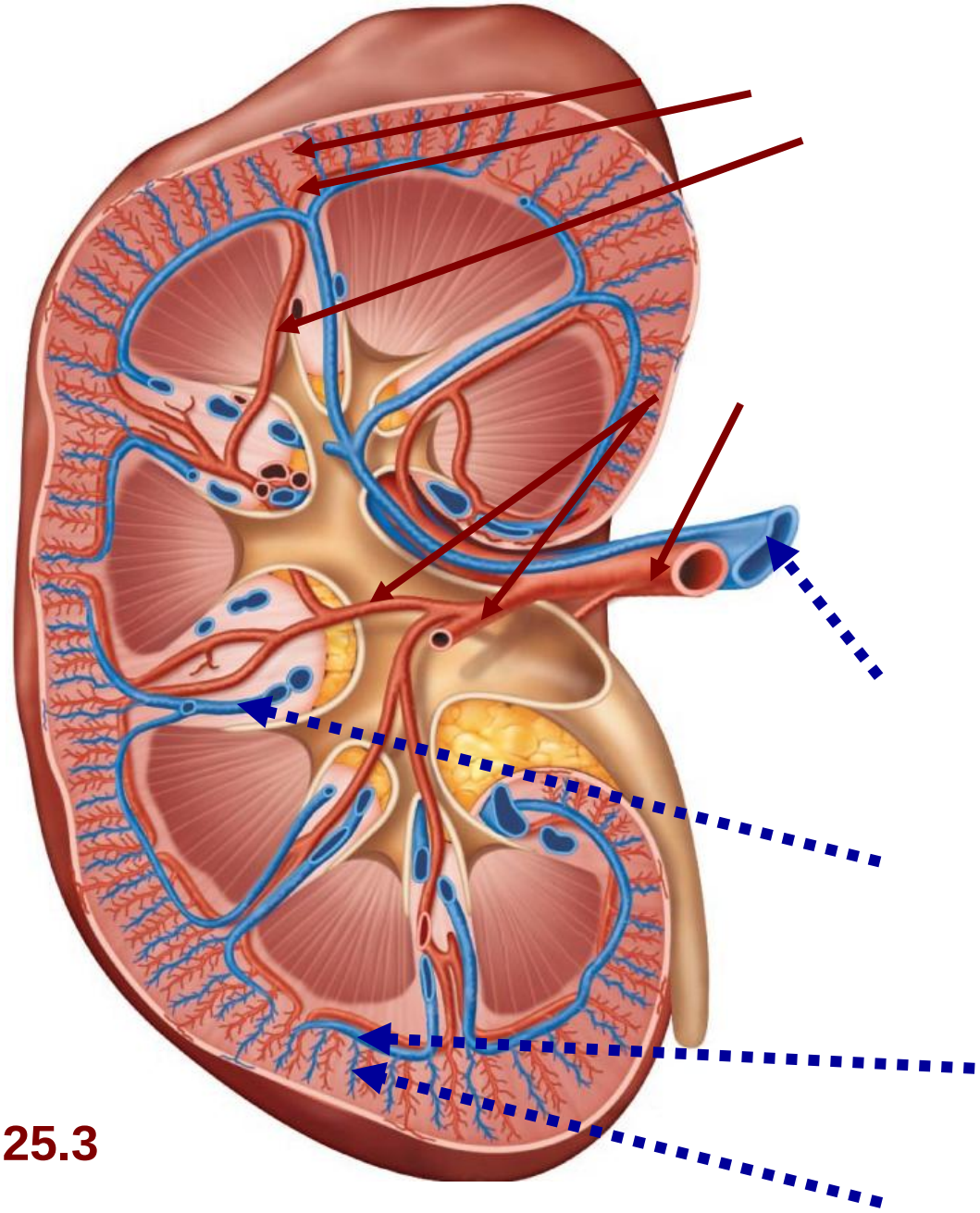


Fig. 25.3

What is pyelitis? What is pyelonephritis?

Circulatory Pathway through Kidney



renal artery

segmental artery

interlobar artery

arcuate artery

cortical radiate artery

afferent arteriole

efferent arteriole

cortical radiate vein

arcuate vein

interlobar vein

renal vein

Fig. 25.3

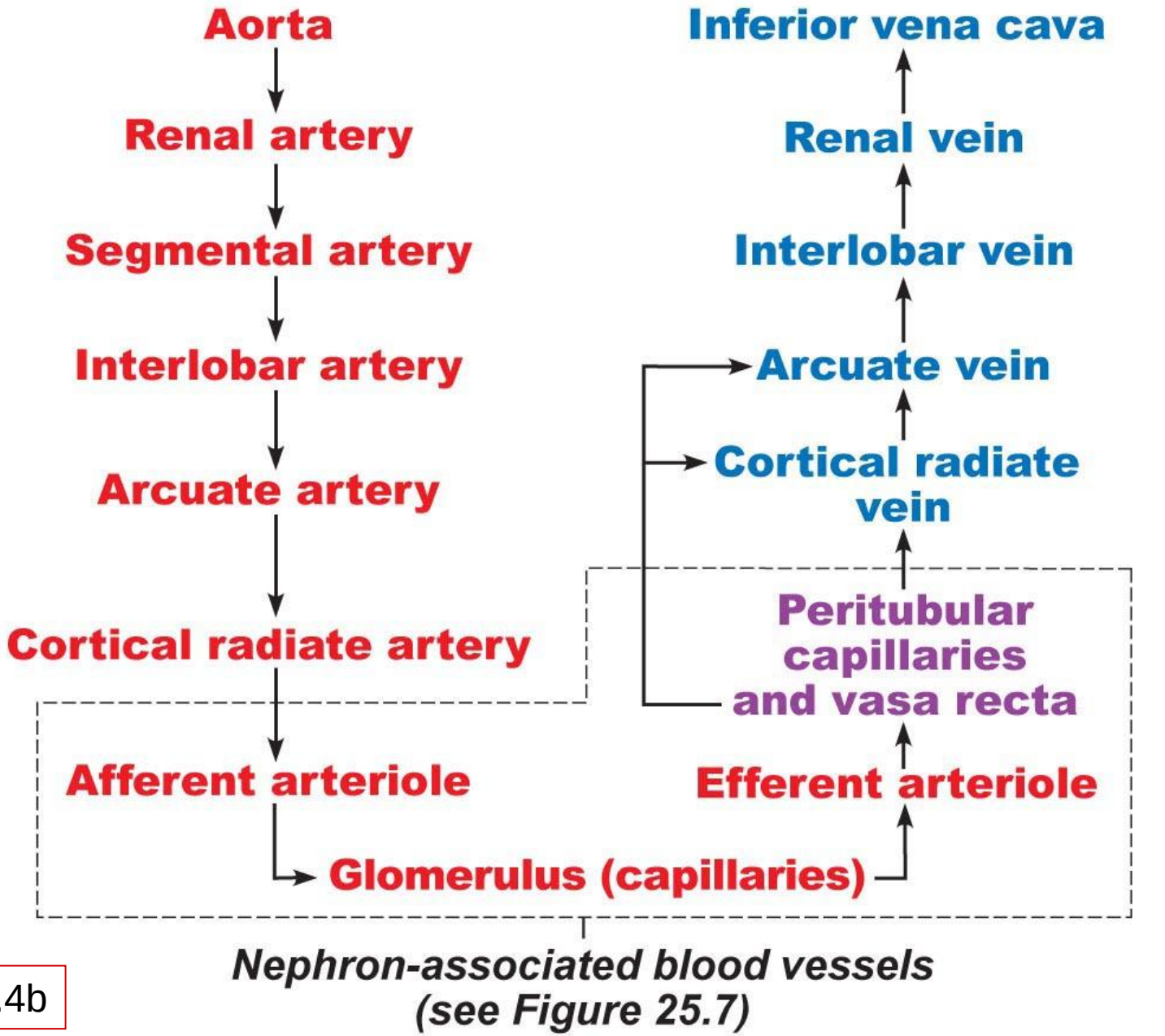
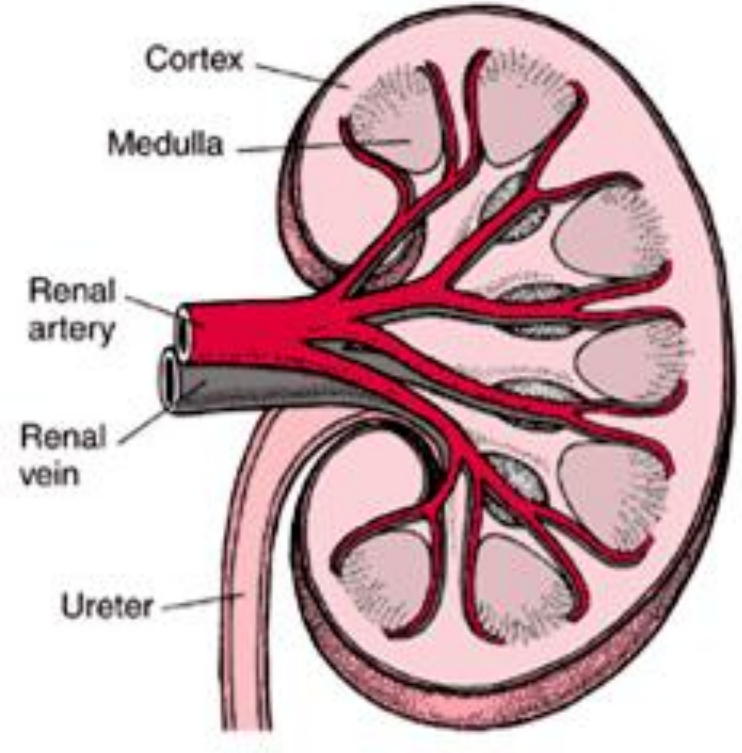


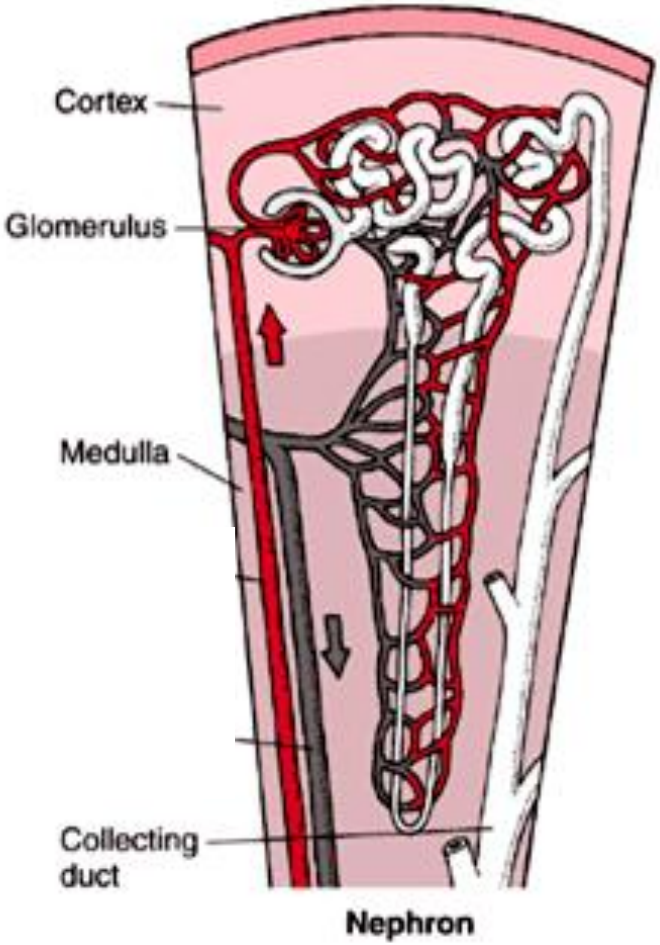
Fig. 25.4b

(b) Path of blood flow through renal blood vessels

BLOOD & NERVE SUPPLY

- renal arteries: ~1/4 total systemic CO (~1.2 L) to kidneys/min
- arterial branches pass up between medullary pyramids to reach cortex; venous branches drain back via same route
- nerve supply provided by **renal plexus** of primarily **sympathetic** fibers ➔ regulate renal blood flow by adjusting diameters of renal arterioles



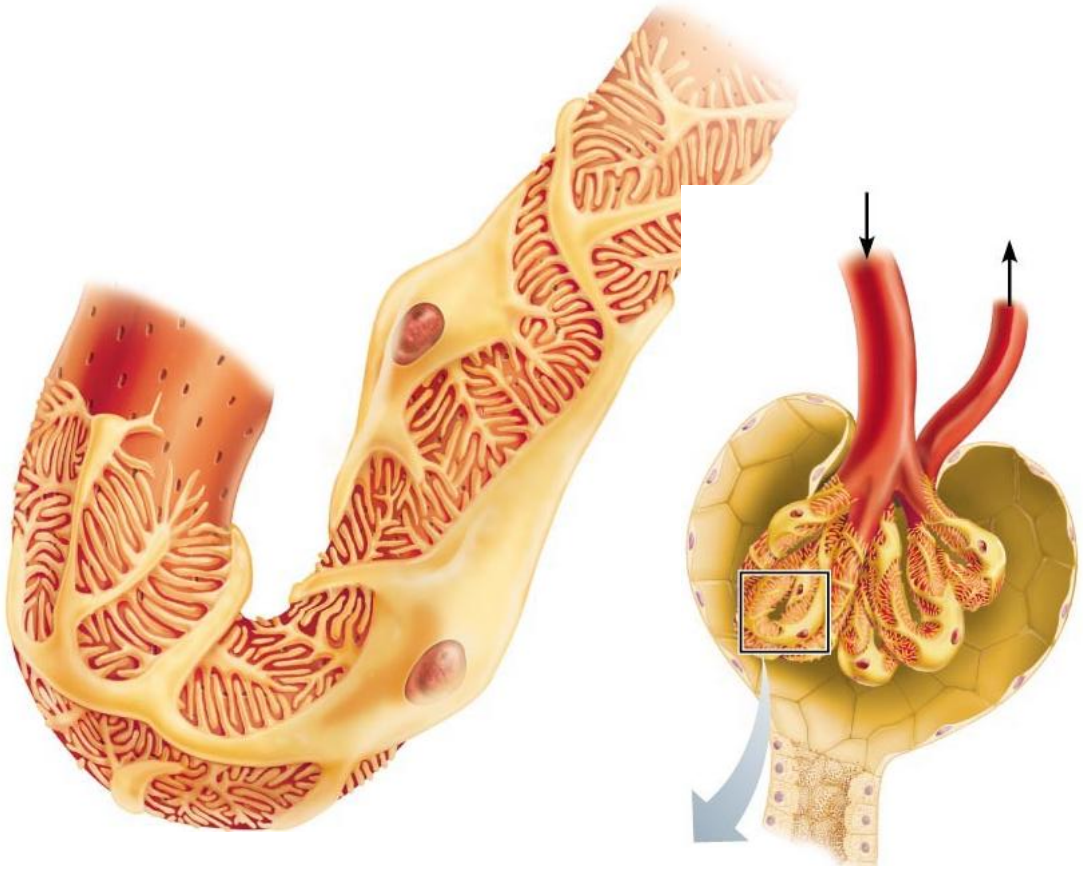


http://www.merck.com/media/mmhe2/figures/fg145_1.gif

4.1.2 describe the structure of a nephron; differentiate between cortical & juxtamedullary nephrons

~10^6 nephrons & thousands of collecting ducts/kidney

glomerulus
Bowman's capsule
renal corpuscle
fenestrated glomerular endothelium
podocytes, pedicels
filtration slits



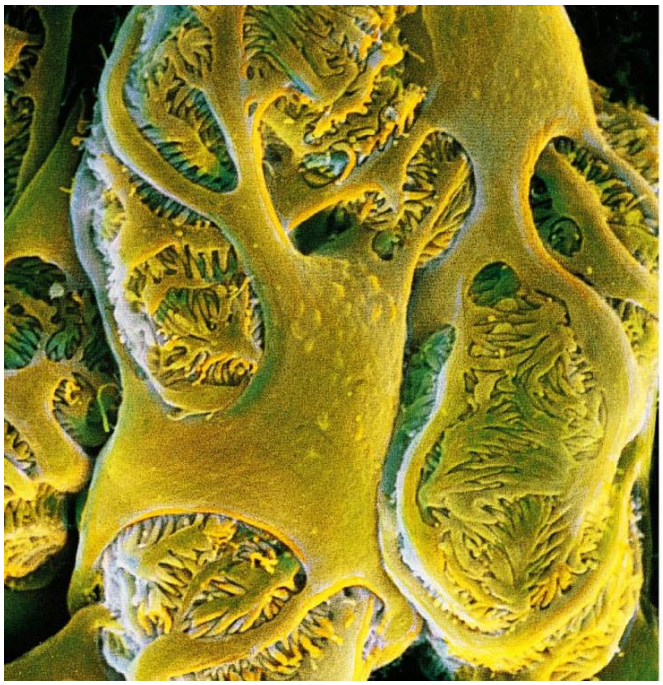
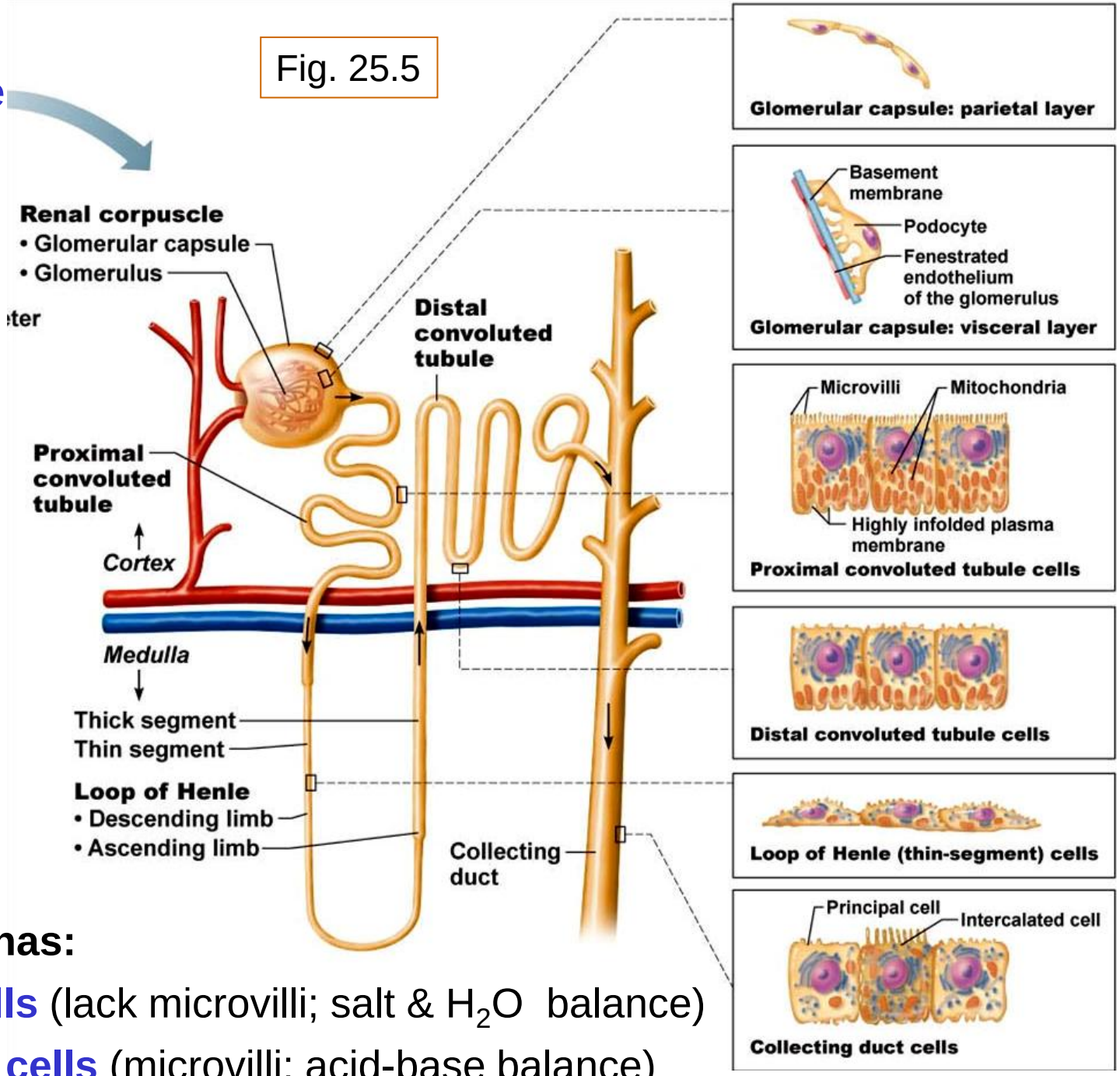


Fig. 25.9

J. Carnegie, UofO

proximal convoluted tubule
↓
loop of Henle
↓
distal convoluted tubule
↓
collecting duct
↓
papillary duct
↓
minor calyx

Fig. 25.5



collecting duct has:

1) **principal cells** (lack microvilli; salt & H_2O balance)
2) **intercalated cells** (microvilli; acid-base balance)

2 types of nephrons:

(i) cortical (85%)

(ii) juxtamedullary (15%)

Roles??

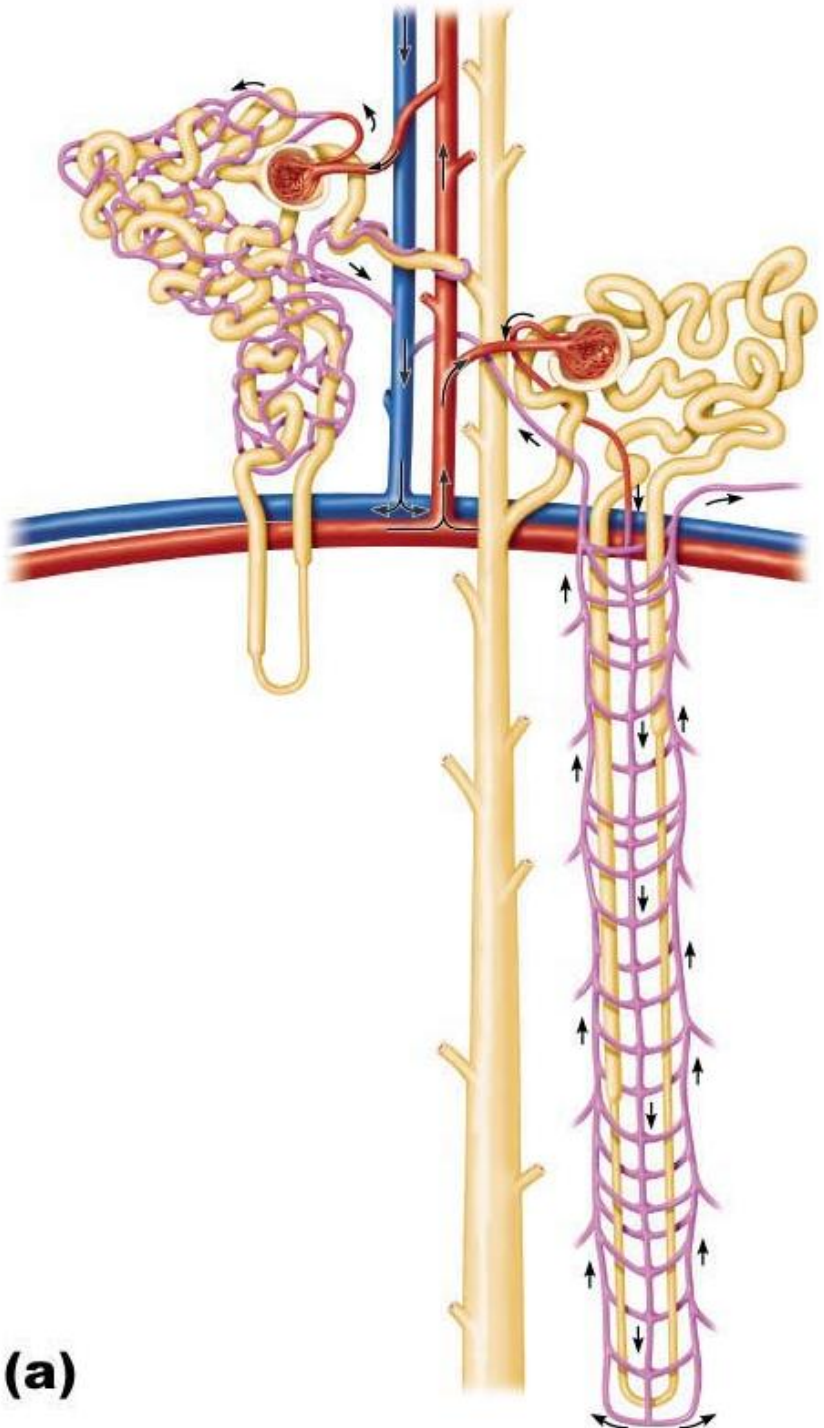


Fig. 25.7

A. Microcirculation of the Nephron

glomerulus: specialized for filtration ➢ both fed & drained by arterioles

√ arterioles are high resistance vessels

√ afferent arteriole has larger diameter

Peritubular Capillaries:

arise from efferent arterioles – these drain into renal venules

What is the vasa recta and where is it found??

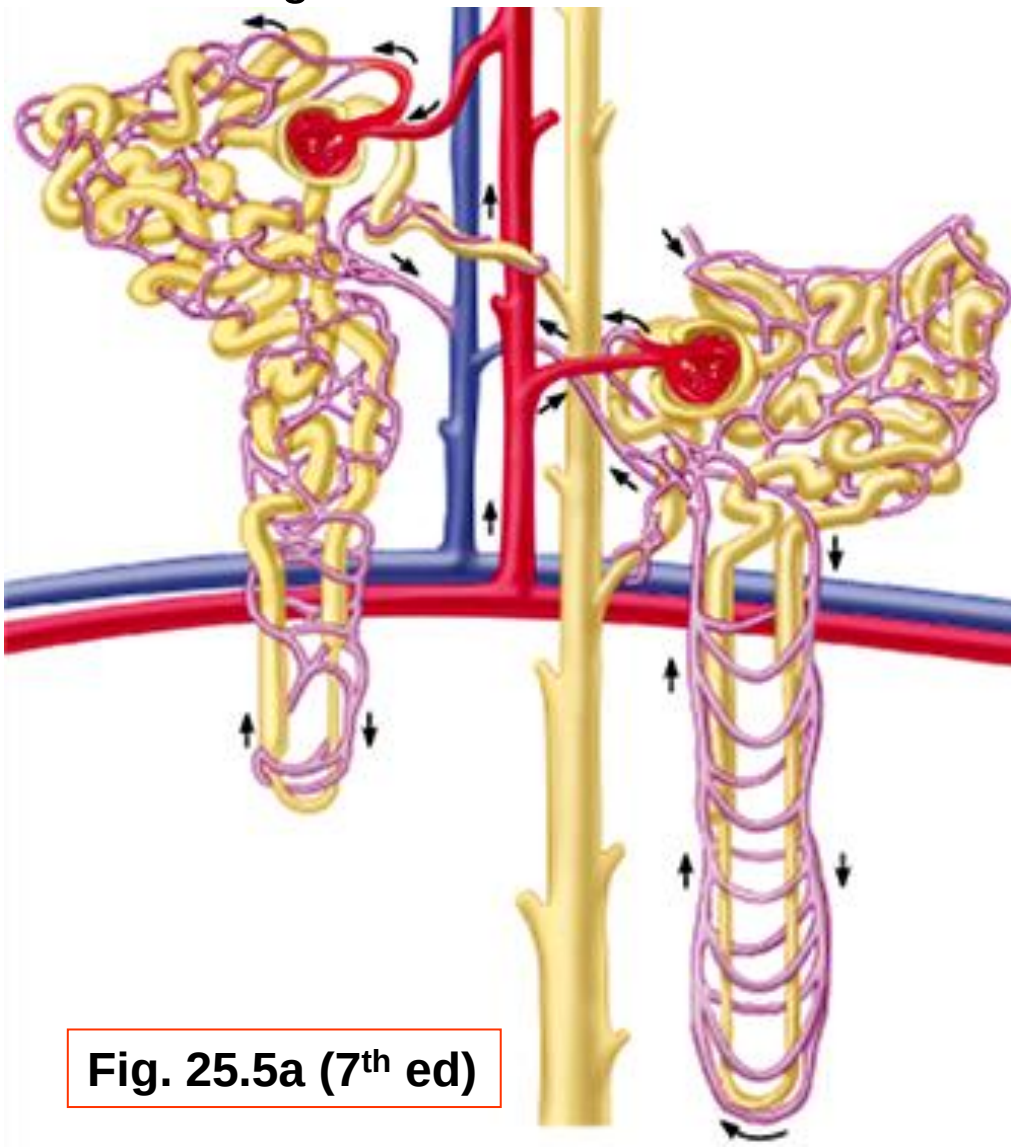


Fig. 25.5a (7th ed)

4.1.3 describe the structural organization of the juxtaglomerular apparatus

at junction of early DCT and afferent/efferent arterioles; regulate renal function:

(i) arteriole walls: JG cells (granular cells) ➢ enlarged smooth muscle cells – mechanoreceptors; secrete renin

(ii) tubule wall: macula densa cells ➢ chemo- or osmoreceptors – monitor filtrate & adjust GFR accordingly

JGA regulates:

a) filtrate formation

b) systemic bp

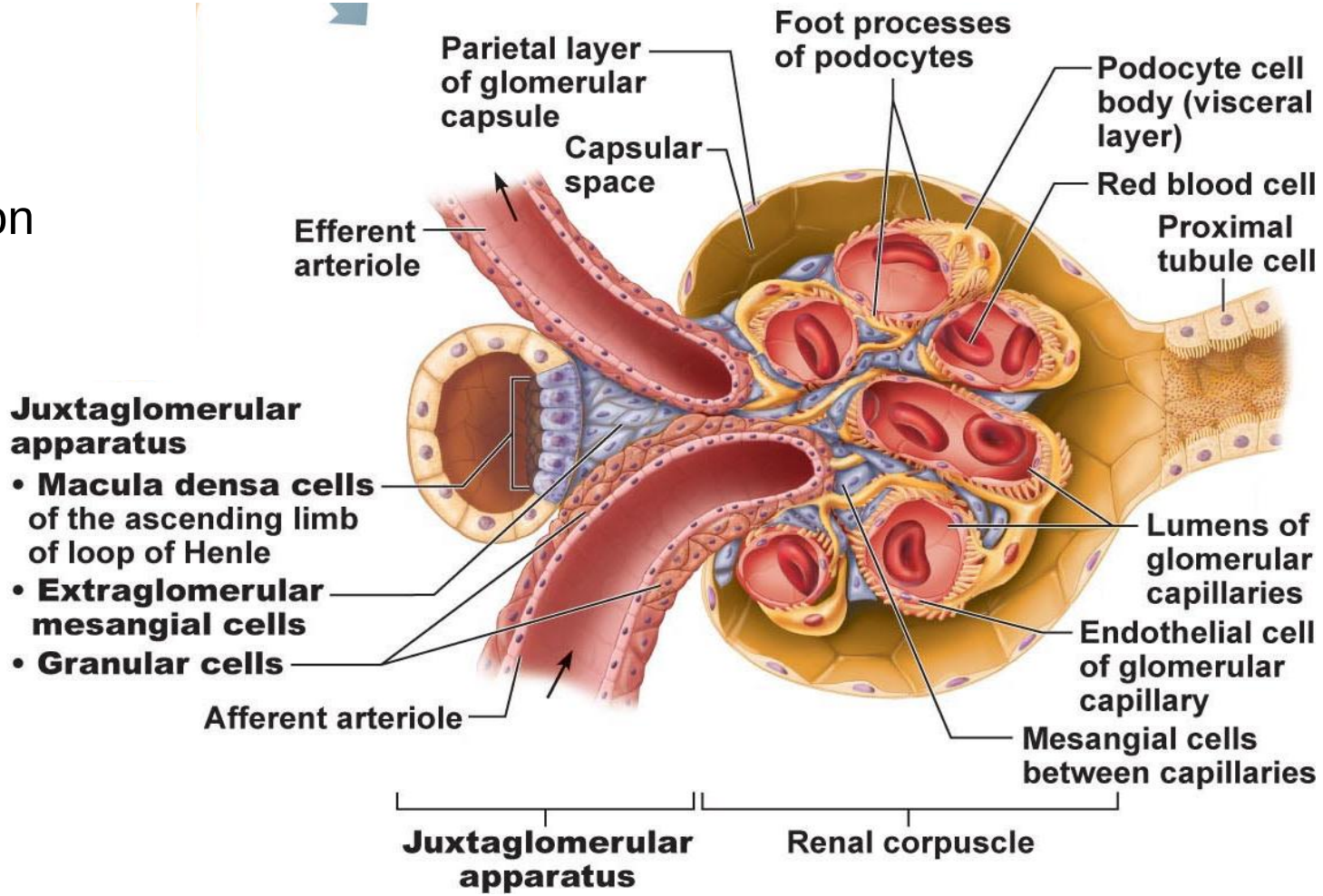


Fig. 25.8

4.1.4 differentiate between micturition pathway in males versus females

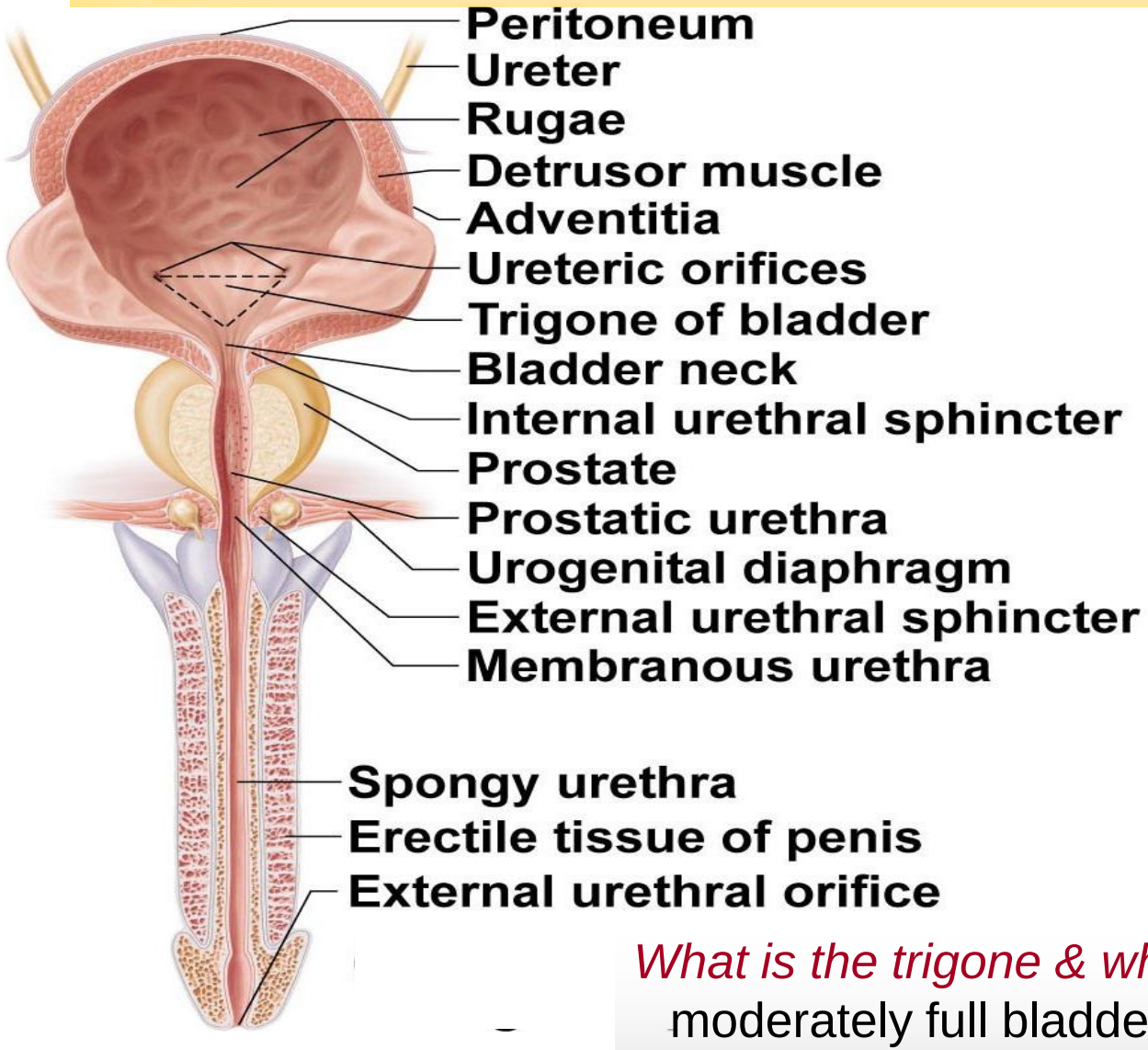


Fig. 25.21a

What is the trigone & why is it important clinically?

moderately full bladder (500 ml) ~12.5 cm long

can hold up to about double that volume

What forms the internal urethral sphincter?

What forms the external urethral sphincter?

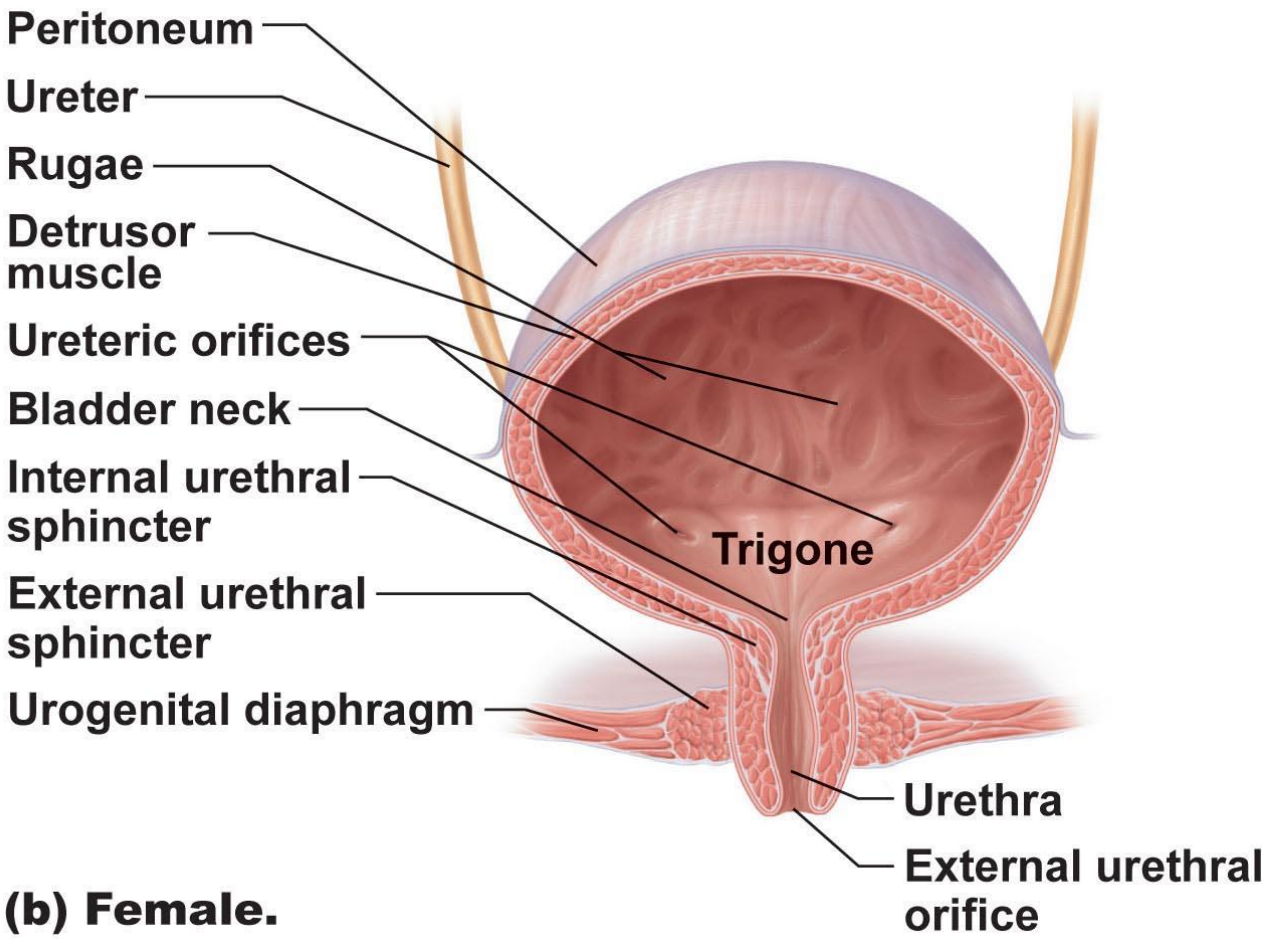
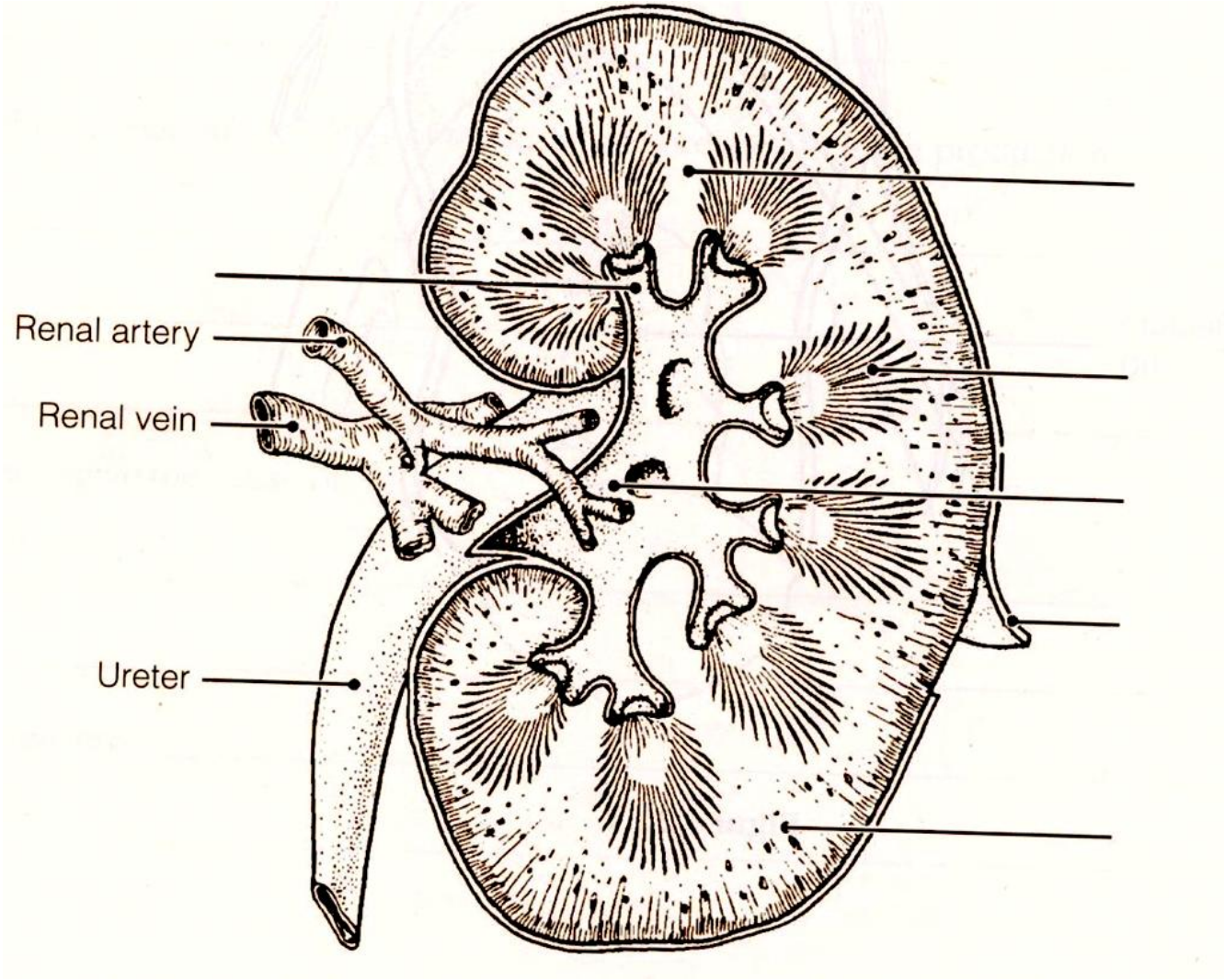

Peritoneum
Ureter
Rugae
Detrusor muscle
Ureteric orifices
Bladder neck
Internal urethral sphincter
External urethral sphincter
Urogenital diaphragm
Trigone
Urethra
External urethral orifice
(b) Female.

Fig. 25.21b



J. Carnegie, UofO

RENAL PHYSIOLOGY

4.2 Describe the nephron as the functional unit of the kidney

4.2.1 list the 2 primary & 4 additional functions carried out by the kidneys

- major excretory organs: perfect examples of homeostatic organs

Main function: **filter** several litres of fluid from bloodstream daily ➢
(i) toxins, metabolic wastes, excess ions leave body in urine
(ii) materials still needed by body returned to bloodstream

Additional functions:
(i) regulate blood **volume** & **composition** (eg ……& ……)
(ii) produce enzyme **renin** ➢ helps regulate **bp** & kidney function
(iii) produce hormone **erythropoietin** ➢ _____
(iv) metabolize **vitamin D** to its active form

4.2.2 define glomerular filtration, tubular reabsorption, tubular secretion; differentiate between filtrate & urine

1000-1200 ml of blood pass through gomeruli each minute; (~650 ml plasma; of this 120-125 ml plasma forced into renal tubules every minute!)

equiv to filtering entire plasma volume >60 times/day

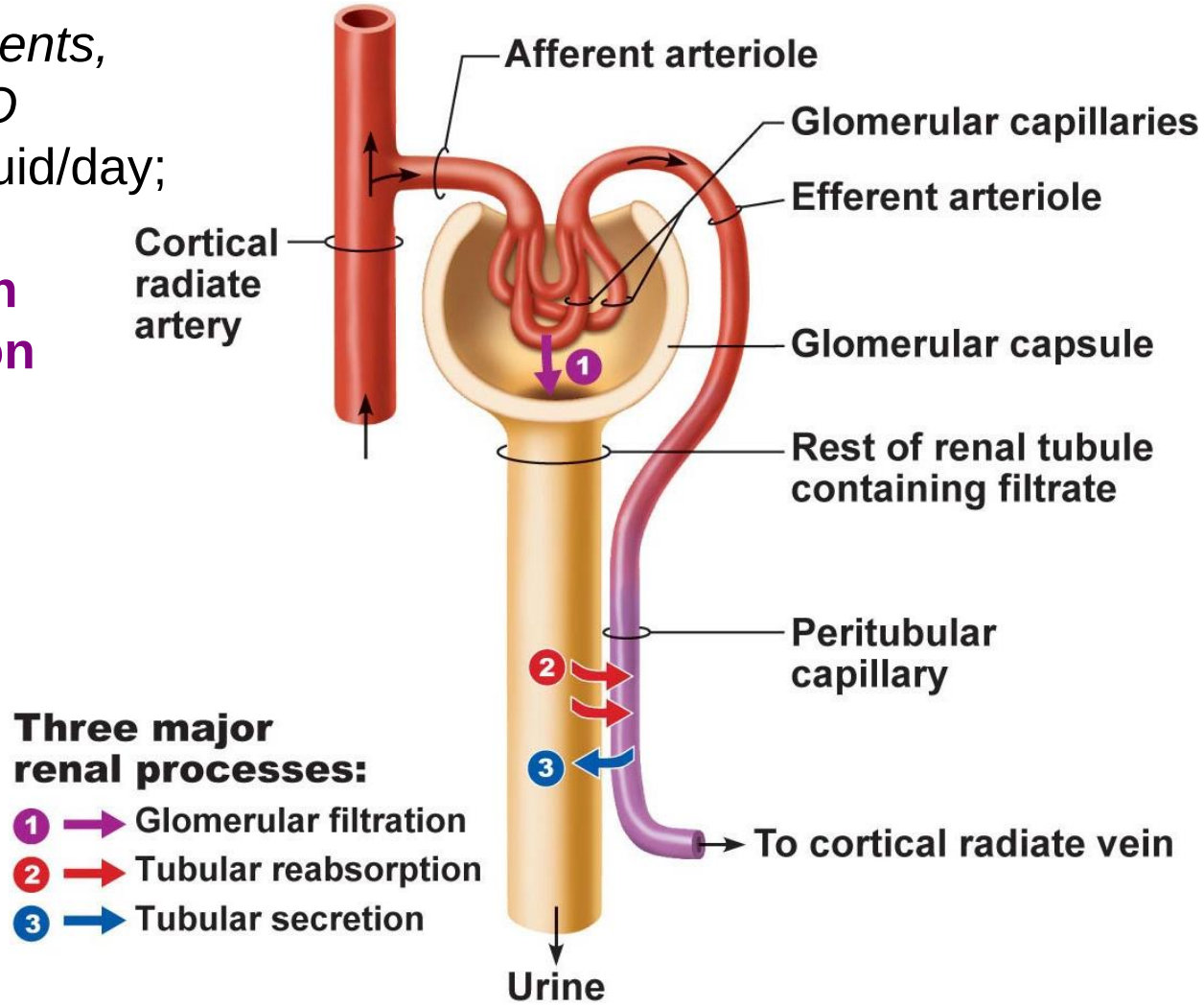
filtrate = *plasma minus proteins*

urine = *filtrate minus nutrients, essential ions, most H_2O*

kidneys process ~180 L fluid/day; ~1% is urine (~1.5 L)

(i) glomerular filtration
(ii) tubular reabsorption
(iii) tubular secretion

Fig. 25.10



Glomerular Filtration

- passive, nonselective ➔ fluids & solutes forced thru by hydrostatic pressure
- glomerulus very efficient filter because:

(i) filtration membrane 1000sX more **permeable** than other cap membranes

(ii) glomerular bp **higher** than in other cap beds (55 mm Hg vs ≤ 18 mm Hg) – *what allows this bp to be maintained at such a high level??*

180 L filtrate formed by kidney caps vs 3-4 L by all other cap beds combined!

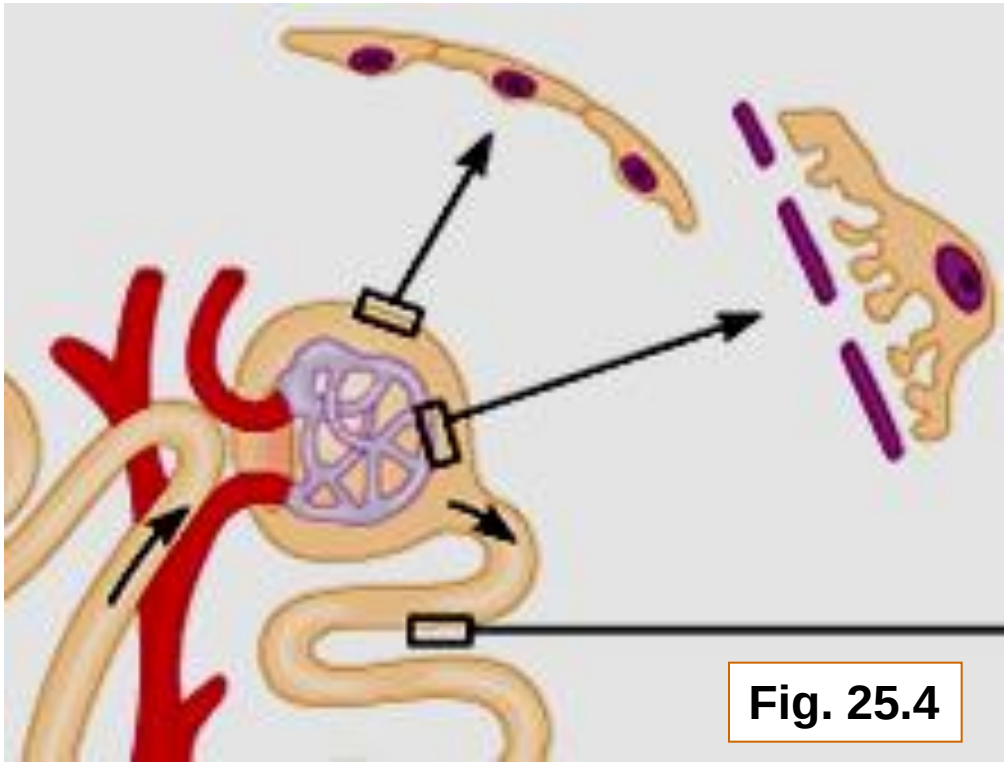


Fig. 25.4

4.2.3 describe the functional anatomy of the filtration membrane

- between blood & interior of glomerular capsule - 3 layers
  - *(i) fenestrated capillary endothelium*
  - *(ii) basement membrane*
  - *(iii) visceral membrane of glomerular capsule = podocytes*

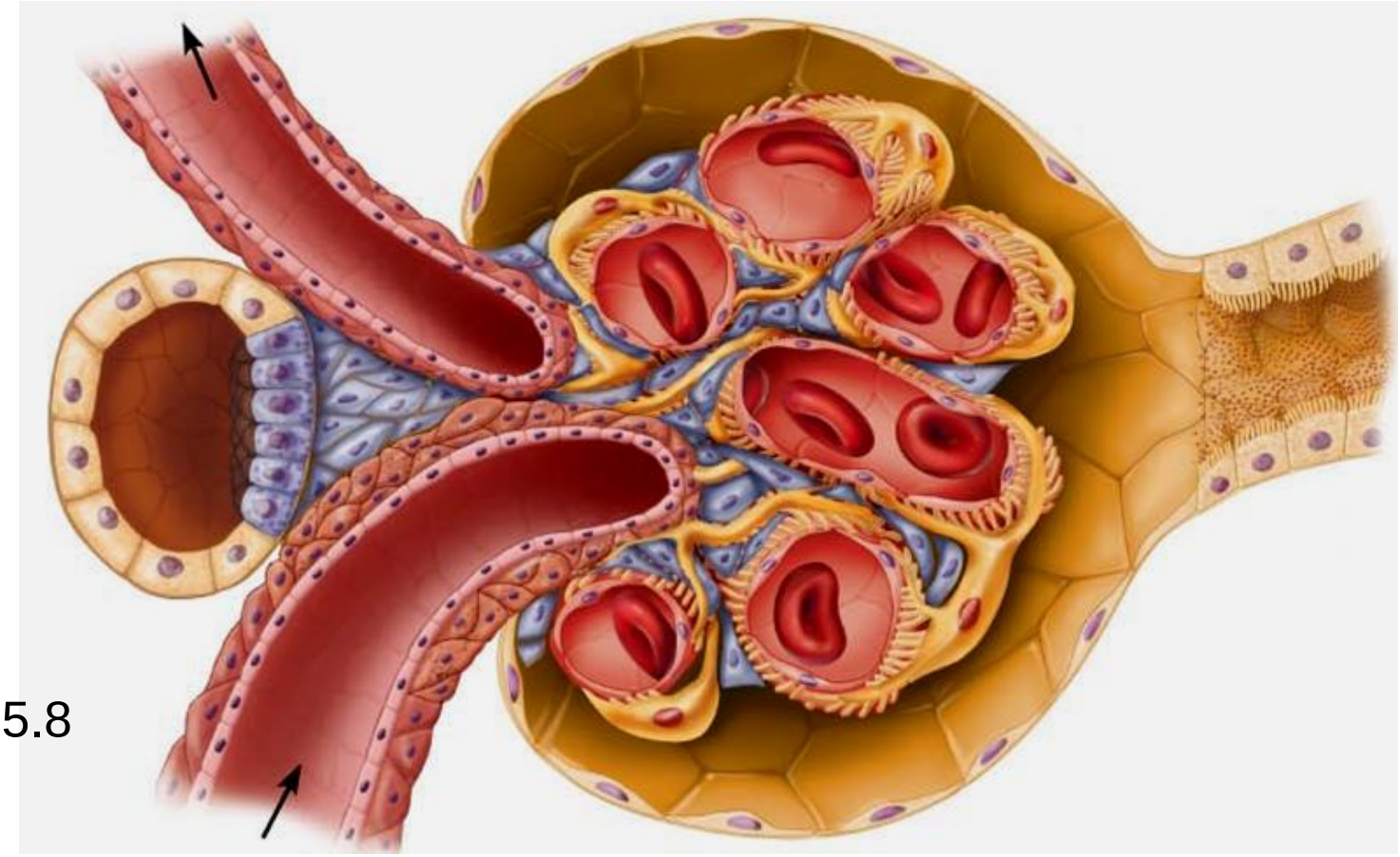


Fig. 25.8

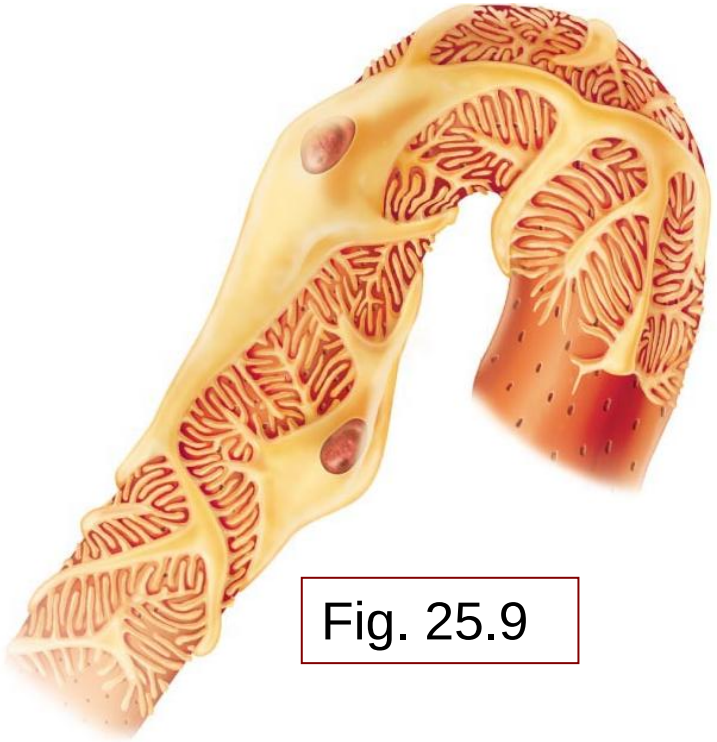
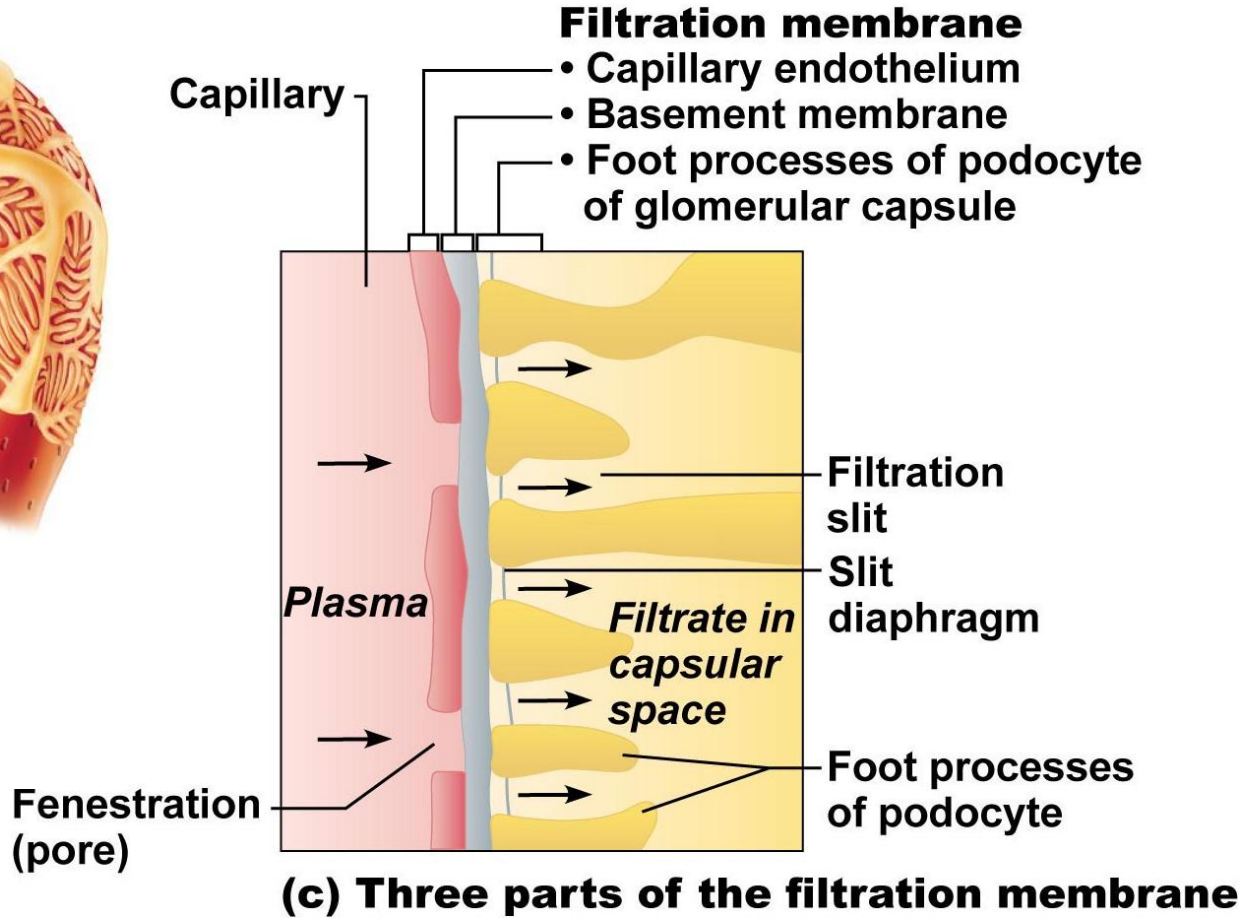


Fig. 25.9



In summary:

(i) molecules ≤ 3 nm (water, glucose, amino acids, N-wastes) pass easily
(ii) molecules 3-6 nm pass, but with greater difficulty (meaning??)
(iii) molecules ≥ 7 nm are not filtered

- bm restricts passage of most larger ptns; aided by negative charges on most bm proteins *(how does this help??)*
- retention of plasma ptns maintains colloid osmotic pressure, blood flow
- presence of proteins or RBCs in urine suggests filtration membrane damage

4.2.4 define net filtration pressure

pressure responsible for filtrate formation

NFP = HPg – (OPg + HPc)

NFP = 55 – (30 + 15)

NFP = 10 mm Hg

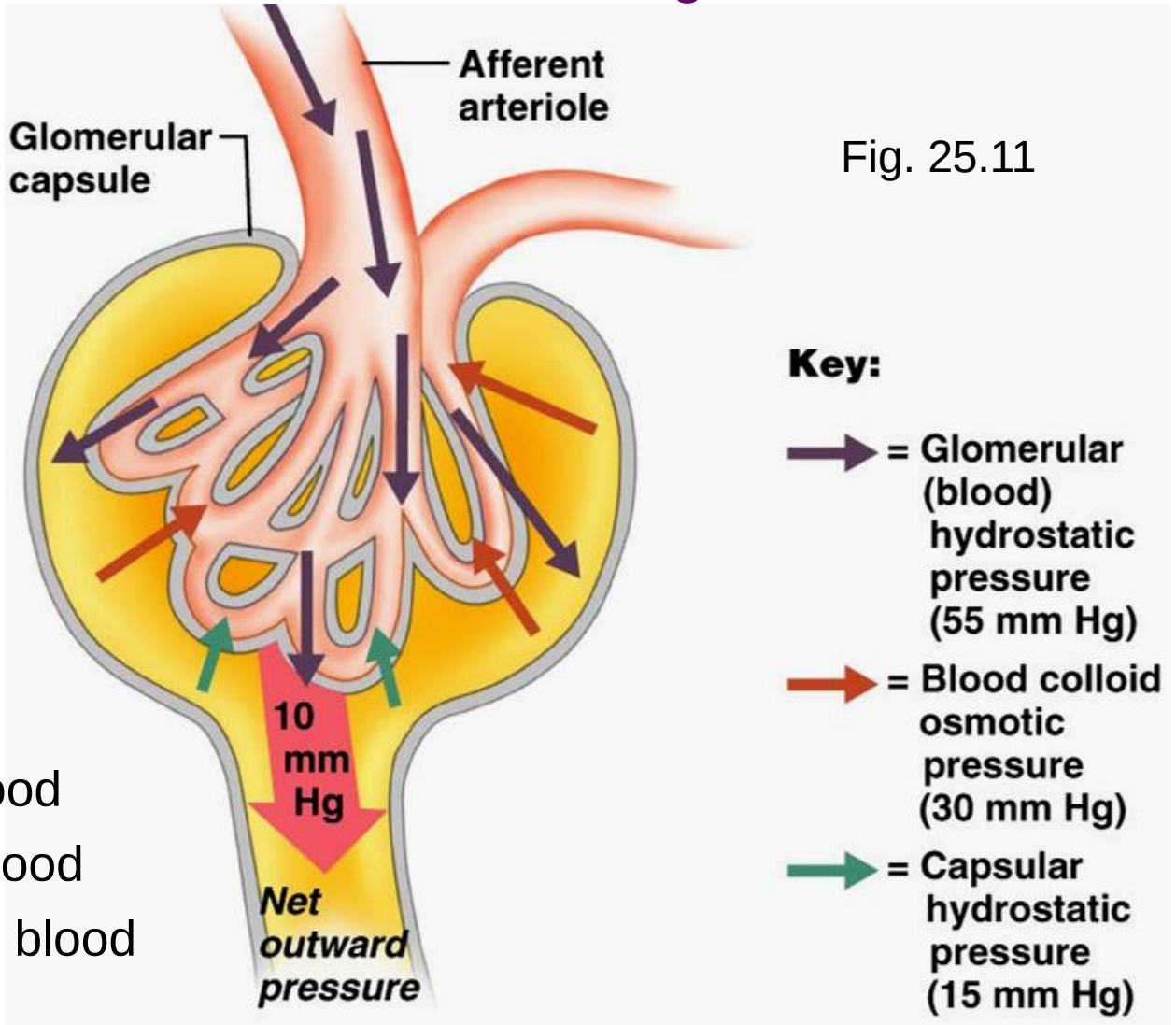


Fig. 25.11

HPg – pushes **out** of blood

OPg – pulls back **into** blood

HPc – pushes back **into** blood

4.2.5 define glomerular filtration rate; be aware of normal value

= total amount of filtrate formed per minute (120-125 ml/min)

- depends on: *(i) total SA for filtration*
  (ii) filtration membrane permeability
  (iii) net filtration pressure (usu ?? mm Hg)
- glomerular caps have huge SA (= SA of skin); even though NFP only 10 mm Hg, get large amts of filtrate
- decrease in glomerular bp of only ~15% completely stops filtration!
- GFR is directly proportional to NFP

What happens to GFR with an increase in bp? In case of dehydration?

4.2.6 list & describe the 3 regulatory influences on GFR

Why is this important?

- 3 regulatory influences allow for an **optimal rate of flow**:

(i) renal autoregulation (intrinsic)

(ii) neural controls

(iii) renin-angiotensin system

1. Renal Autoregulation:

kidney keeps GFR ~constant by determining own rate of flow & adjusting nephron blood flow

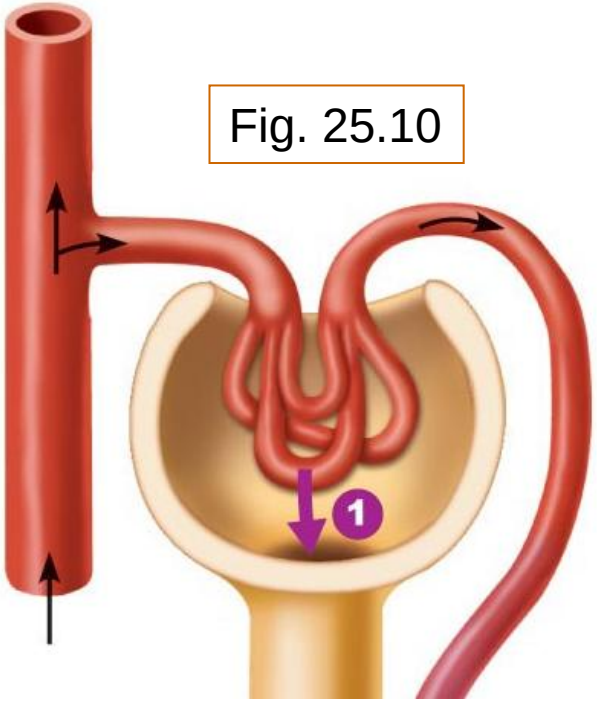
regulates **diameter** of **afferent** (primarily) and efferent arterioles

2 types of controls:

(i) myogenic mechanism

(ii) tubuloglomerular feedback mechanism

Fig. 25.10



1a) myogenic mechanism:

- responds to △ bp in renal blood vessels
- vascular smooth muscle tends to contract when stretched – so what happens when systemic bp **increases**?? **decreases**??

1b) tubuloglomerular feedback mechanism:

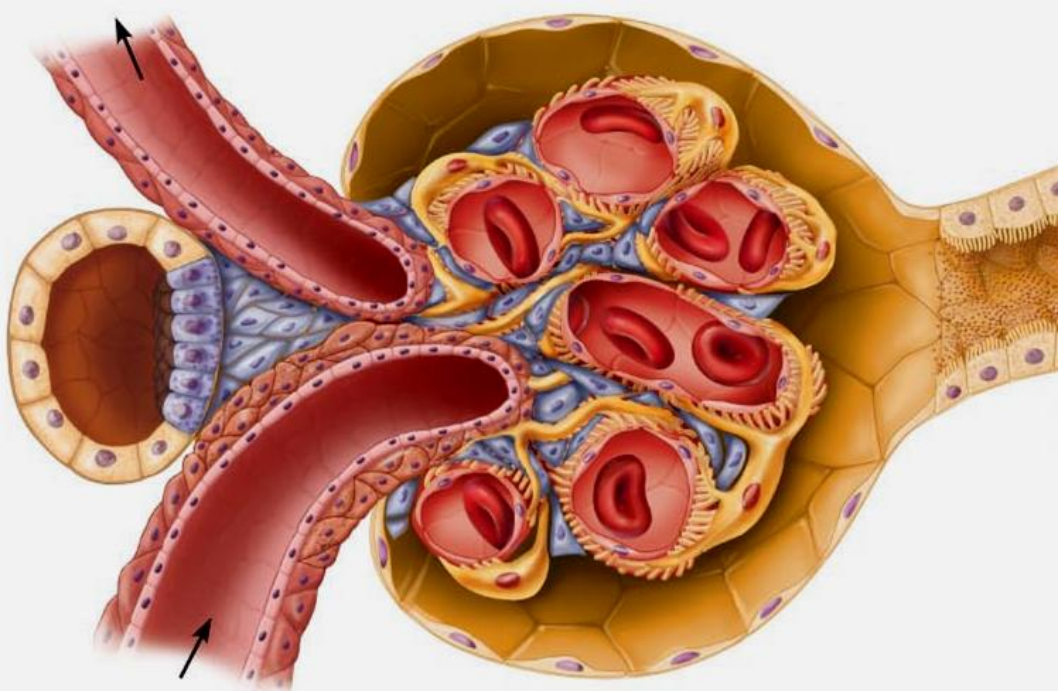
- directed by macula densa cells of JGA ➢ located in walls of distal tubules

(i) slow filtrate/low osmolarity: vasodilation of afferent arteriole ➢ ??

(ii) fast filtrate/high osmolarity: vasoconstriction of afferent arteriole (via JG cells) ➢ ??

renal autoregulation can maintain ~constant GFR with 80-180 mm Hg systemic bp

systemic bp< 70mm Hg (hypovolemic shock) renal autoregulation & filtrate formation shut down



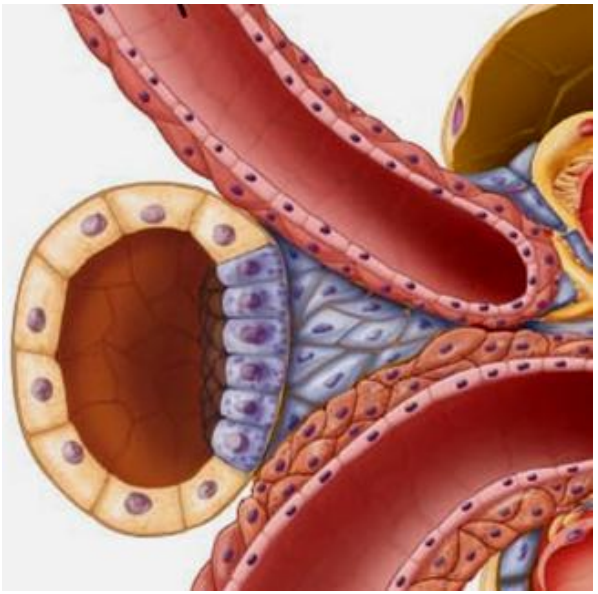
2. Extrinsic Mechanisms:

2a. Neural Controls

- **sympathetic ns** comes into play during times of **extreme** stress ➢ overrides renal autoregulation & shunts blood to heart, brain, skeletal muscles at expense of kidneys

(i) direct sympathetic-induced vasoconstriction of afferent arterioles

(ii) activation of renin-angiotensin system *(how??)*



angiotensinogen (from liver)
↓ (renin)
angiotensin I
↓ (ACE)
angiotensin II

JG cells: smooth muscle cells in afferent arterioles = ***mechanoreceptors***

macula densa cells: tubule cells = ***chemoreceptors***

2b. Renin-angiotensin mechanism:

- angiotensin II: potent **vasoconstrictor** ➢ ……..

Other effects:

(i) Angiotensin II stimulates release of aldosterone (by _____); stimulates reabsorption of Na^+, water follows ➢ *end effect on bp??*

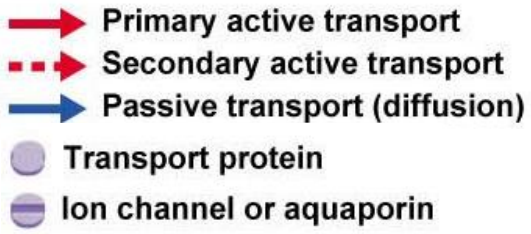
(ii) Afferent arterioles have fewer angiotensin receptors than efferent – *effect on glomerular bp, filtration rate, etc???*

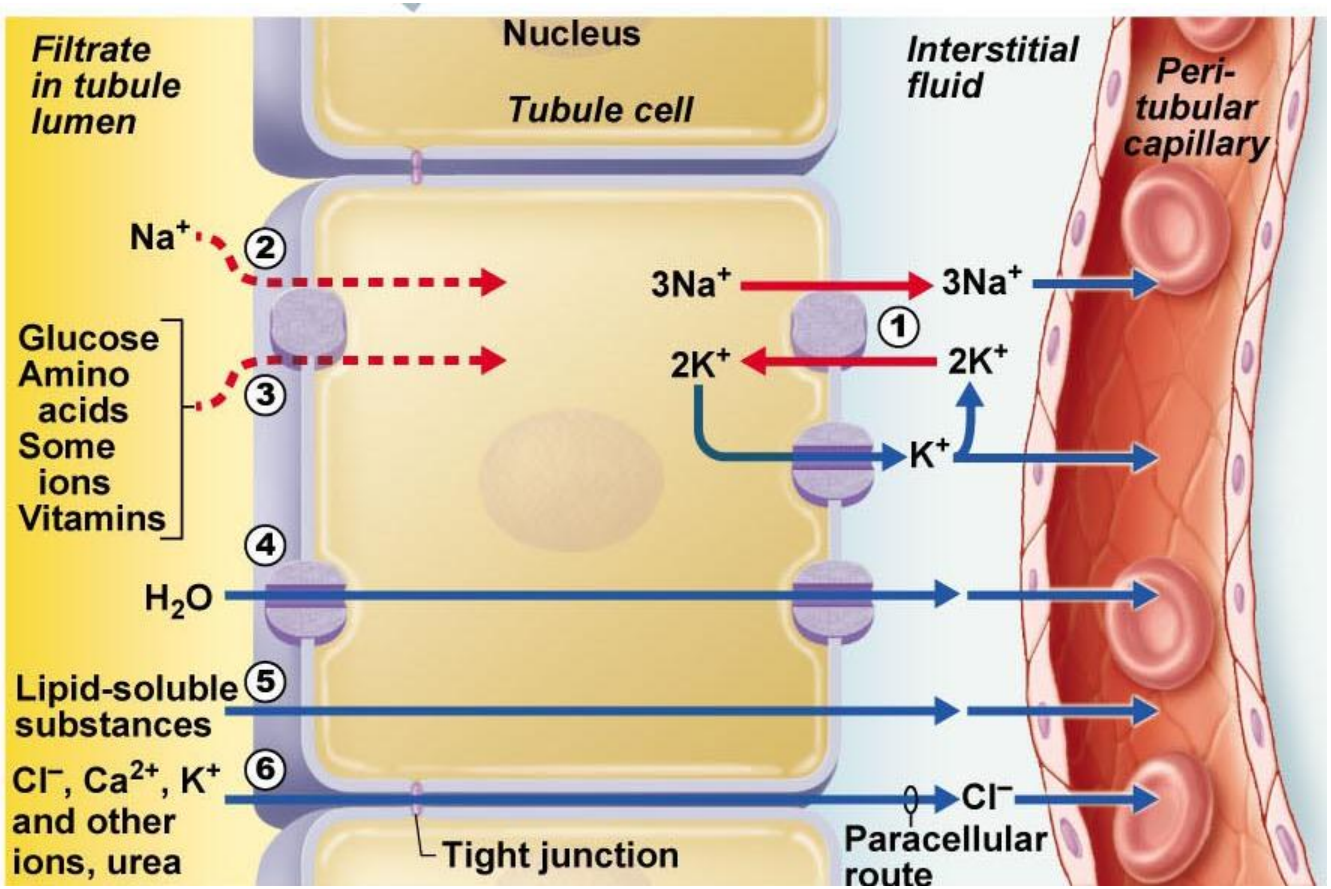
What can trigger release of renin?

(i) bp < 80 mm Hg ➢ decreased stretch of JG cells
(ii) direct stimulation of JG cells by macula densa cells
(iii) direct stimulation of JG cells by renal symp. nerves
(iv) direct stimulation of JG cells by angiotensin II

Renin-angiotensin system is involved in renal regulation, but main purpose is to stabilize systemic bp & ECF volume

Fig. 25.14





4.2.7 indicate the importance of tubular reabsorption

- 45 min: total blood volume filtered by kidney; most does **not** become urine
- most material moves through rather than between cells – Why?

Reabsorption: general principles

- organic nutrients (glucose, amino acids) ~100% reabsorbed
- reabsorption can be **active** or **passive**
- rate/degree of reabsorption of H_2O /ions hormonally adjusted

4.2.7 (cont) distinguish between active and passive tubular reabsorption; list 3 substances that are not reabsorbed & indicate why

Active tubular reabsorption

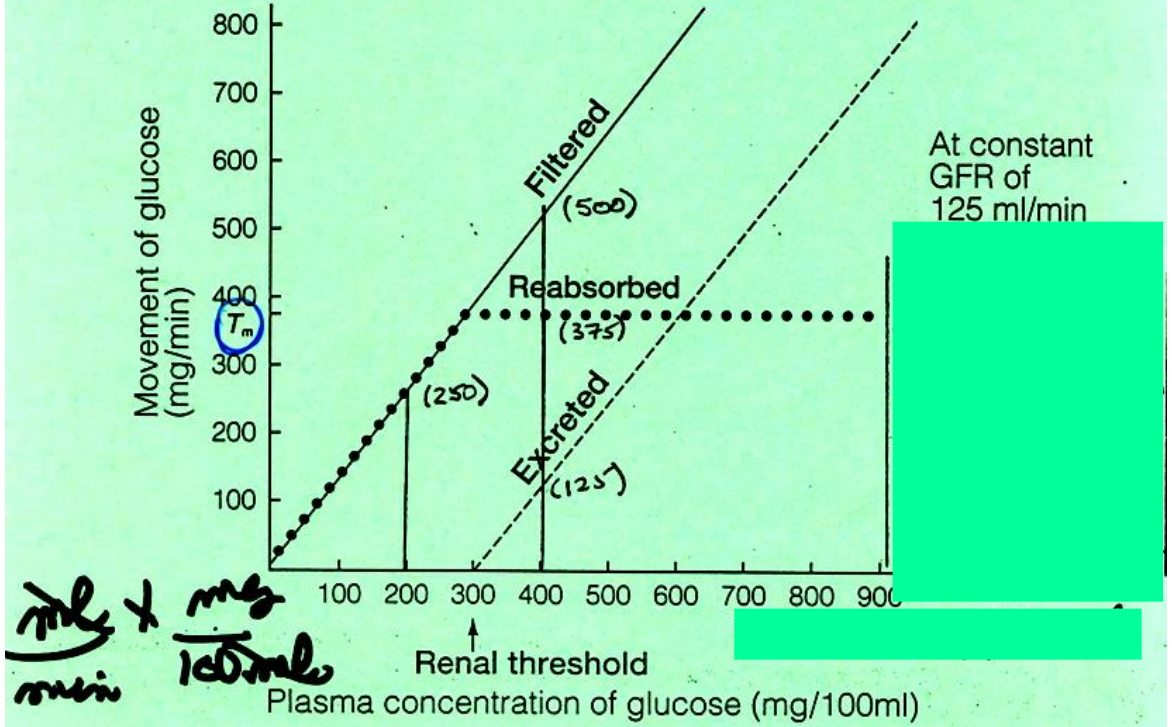
- usu substances being moved **against** electrical and/or chemical gradient
- active process at level of **basolateral membrane**: substance moves passively into tubule cell, but must be actively moved into interstitial space

How does material move into adjacent peritubular capillaries?

- **actively reabsorbed:** glucose, amino acids, lactate, vitamins, most ions
- transport usu linked to that of **Na^+**
- transport systems (carriers) fairly **specific** ➢ transport maximum (**T_m**)

  ➔ when carriers saturated, excess of substance appears in urine (eg:???)
- plasma ptns: if squeeze through, taken up by tubule cells; a.a. to blood

Renal Handling of Glucose as Function of Plasma Glucose Concentration



T_m = tubular maximum

Plasma concentration of substance × GFR = Amount of substance filtered

For example, 100 mg glucose/100 ml plasma × 125 ml plasma filtered/min = 125 mg glucose filtered/min

Plasma Concentration (mg/100ml)	GFR (ml/min)	Amount Filtered (mg/min)	T_m (mg/min)	Amount Reabsorbed (mg/min)	Amount Excreted (mg/min)
a. 80	125	100	375	100	0
b. 100	125	125	375	125	0
* c. 200	125	250	375	250	0
d. 300	125	375	375	375	0
* e. 400	125	500	375	375	125
f. 500	125	625	375	375	250

J. Carnegie, UofO

Passive tubular reabsorption

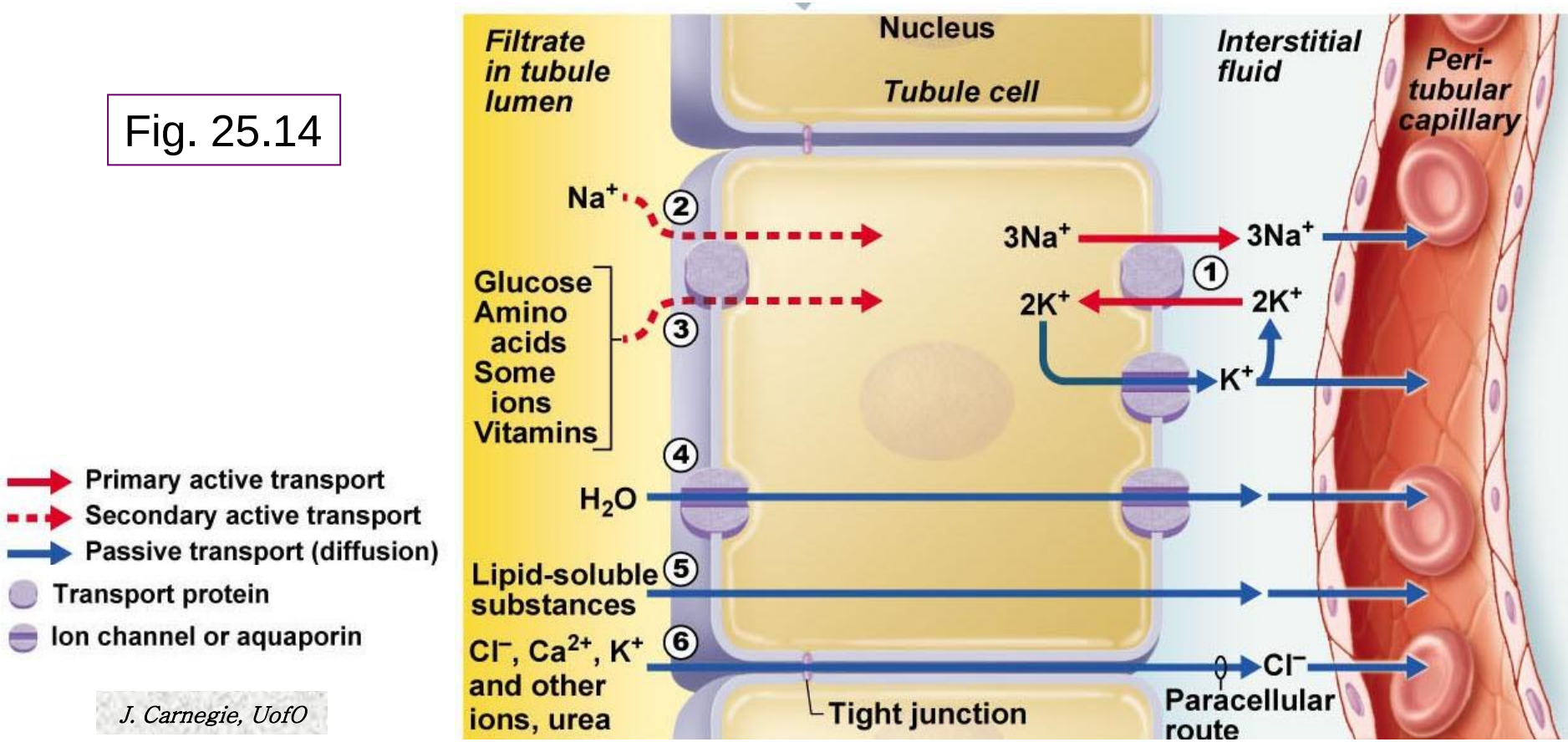
- diffusion, facilitated diffusion, osmosis
- along electrochemical gradient ➢ no ATP required

(i) active reabsorption of Na^+ pulls anions

(ii) obligatory water reabsorption due to Na^+ transport

(iii) solvent drag creates gradient for solute reabsorption

Fig. 25.14



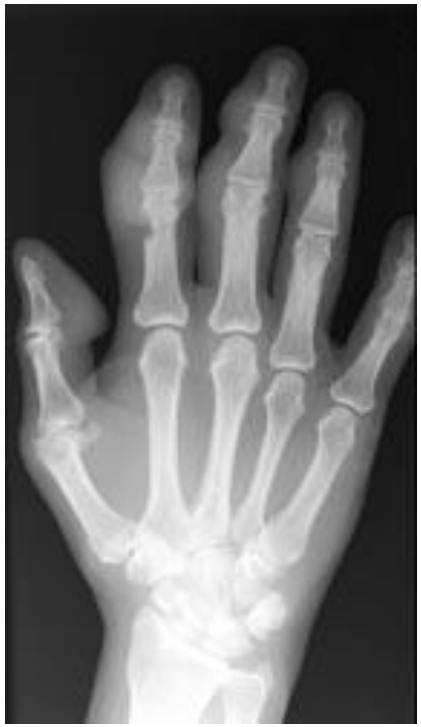
Non-reabsorbed substances

- either no carriers, not lipid soluble or too large; primarily nitrogenous end products of protein & nucleic acid metabolism

(i) urea: main nitrogen-containing end product of metabolism; amount to be excreted depends on: _____; small enough to diffuse through membrane pores; 50-60% reclaimed

(ii) creatinine: large, lipid-insoluble nitrogenous waste molecule; not reabsorbed by kidney ➢ useful for measuring GFR

(iii) uric acid: end product of purine metabolism; some excreted & some reabsorbed; too much uric acid can lead to _____



http://www.gentili.net/Hand/gout.htm

4.2.8 link tubular reabsorption with different regions of the renal tubules

(1)PCT: *all glucose & amino acids*

65% of Na^+

65% of H_2O

90% of bicarbonate

50% of Cl^- and K^+

most selective active reabsorption of specific ions (eg: ____,_____,_____)

(2)Loop of Henle:

ascending & descending limbs different

- water (not NaCl) leaves descending limb
- NaCl (not water) leaves ascending limb

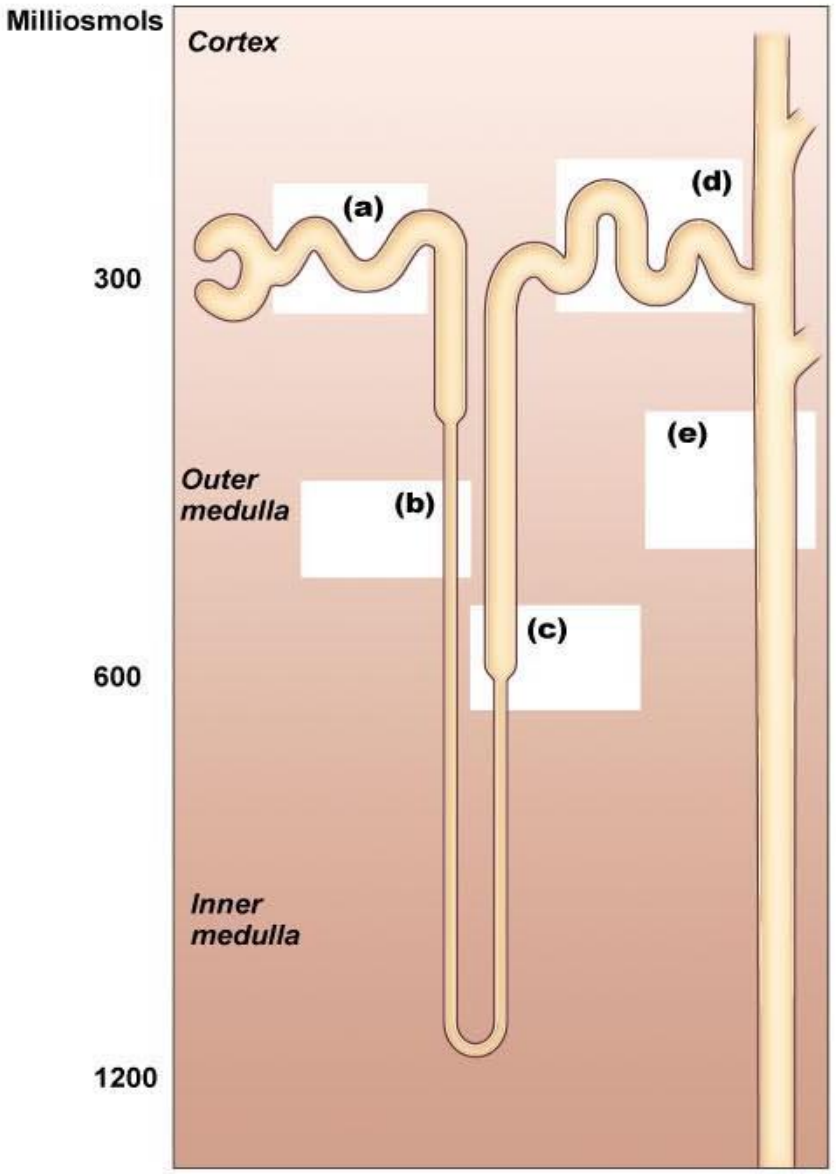
(3) DCT + collecting duct:

- here only 10% NaCl & 20% of water remain (still too much)
- from here on hormonally regulated

ADH:

aldosterone & renin-angiotensin:

ANF:



4.2.9 list (giving examples) 4 functions associated with tubular secretion

- kidneys get rid of unwanted substances by either:
  - *(i) not reabsorbing them*
  - *(ii) secreting them into the urine*

H^+, K^+, creatinine, NH_4^+, uric acid, urea

- late DCT & early collecting ducts
- *4 important functions:*

(i) dispose of substances not in original filtrate (eg: _____)

(ii) dispose of substances that underwent passive reabsorption (eg: _____)

(iii) dispose of excess K^+ ions

(iv) maintenance of blood pH

Some examples:

(i) K^+:

(ii) acidic blood pH:

(iii) alkaline blood pH:

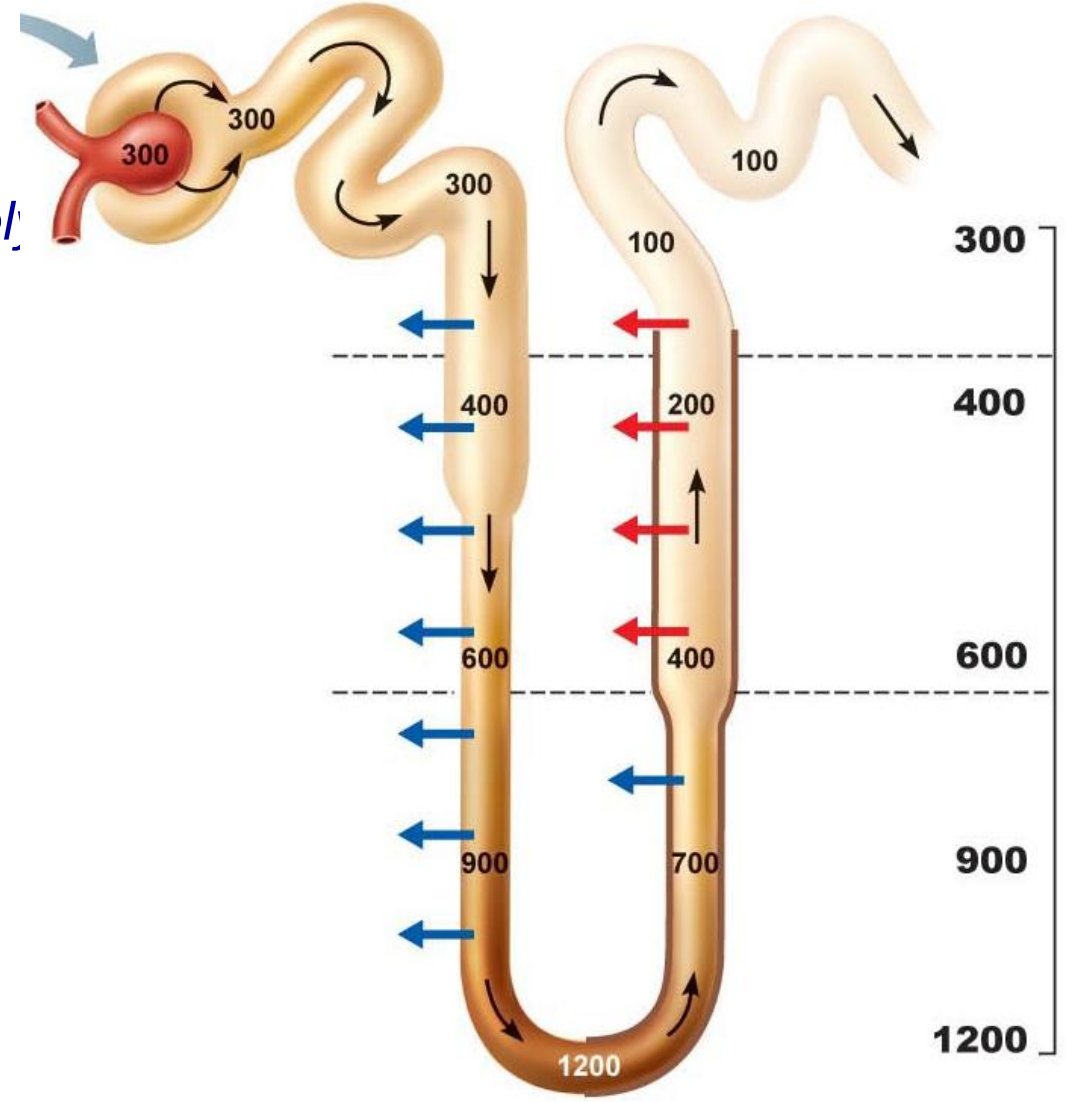
4.2.10 describe the role of the countercurrent mechanism in the regulation of urine concentration & volume; define osmolality; define osmosis

Countercurrent mechanism & medullary osmotic gradient

- much absorption of water & salt in PCT;filtrate at top of loop still at 300 mOsm
- differing permeabilities of loops allow water, then salt to be reabsorbed

(1) Descending limb is relatively impermeable to solutes & freely permeable to H_2O:

- as filtrate moves down **descending limb**, water moves **freely out** by osmosis – *Why?*
- filtrate osmolarity can reach **1200 mOsm** by "elbow" of loop of Henle *(in which nephrons??)*



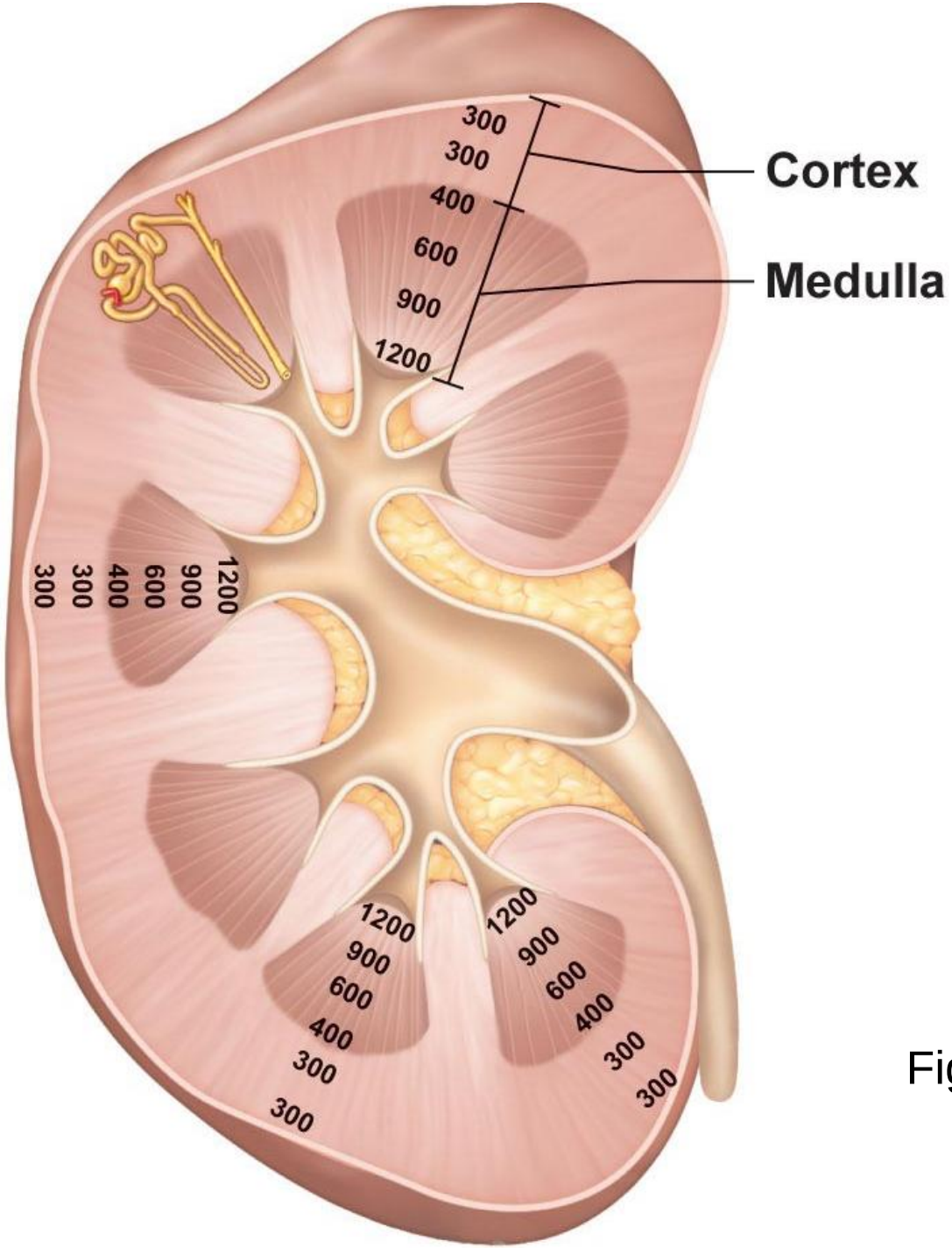
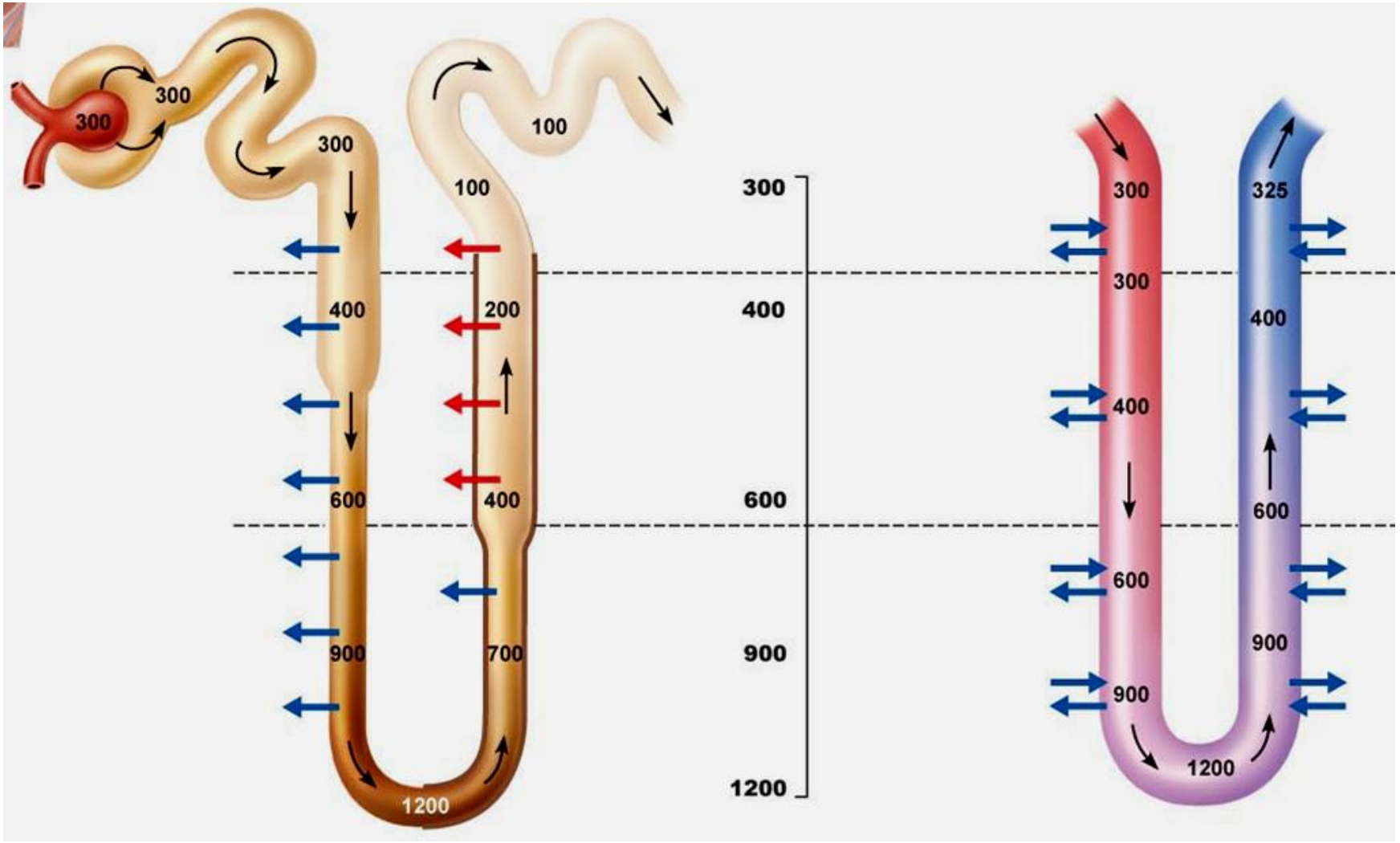

300
300
400
600
900
1200
Cortex
Medulla
1200
900
600
400
300
300
1200
900
600
400
300
300
1200
900
600
400
300
300

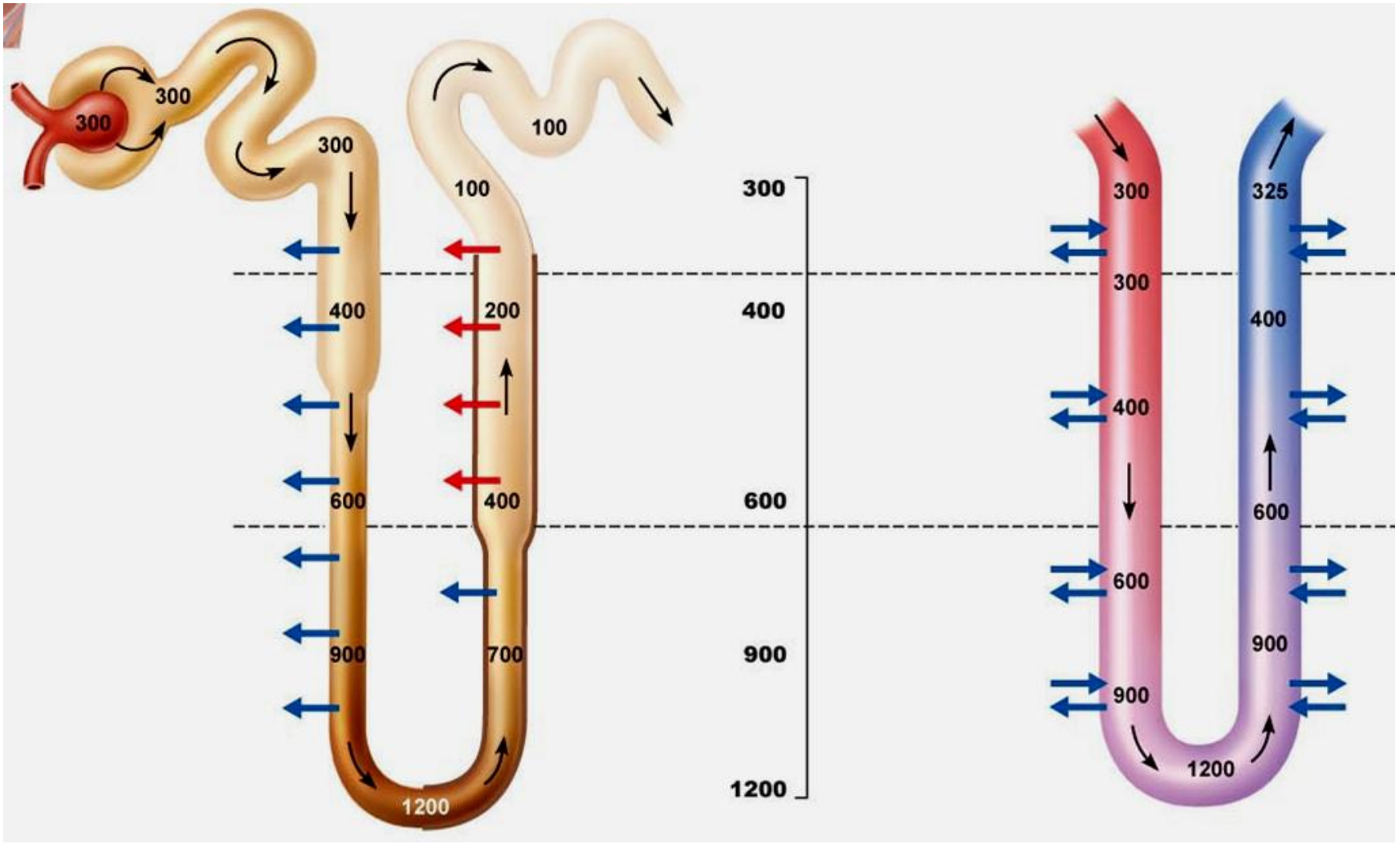
Fig. 25.15



(2) Ascending limb impermeable to water & actively moves NaCl out

- Na^+-K^+-$2Cl^-$ cotransporter moves solute ions out; water can't follow – why?
- positive feedback ➢ filtrate ~200 mOsm more concentrated at each level of descending limb
- net effect is to: ……..

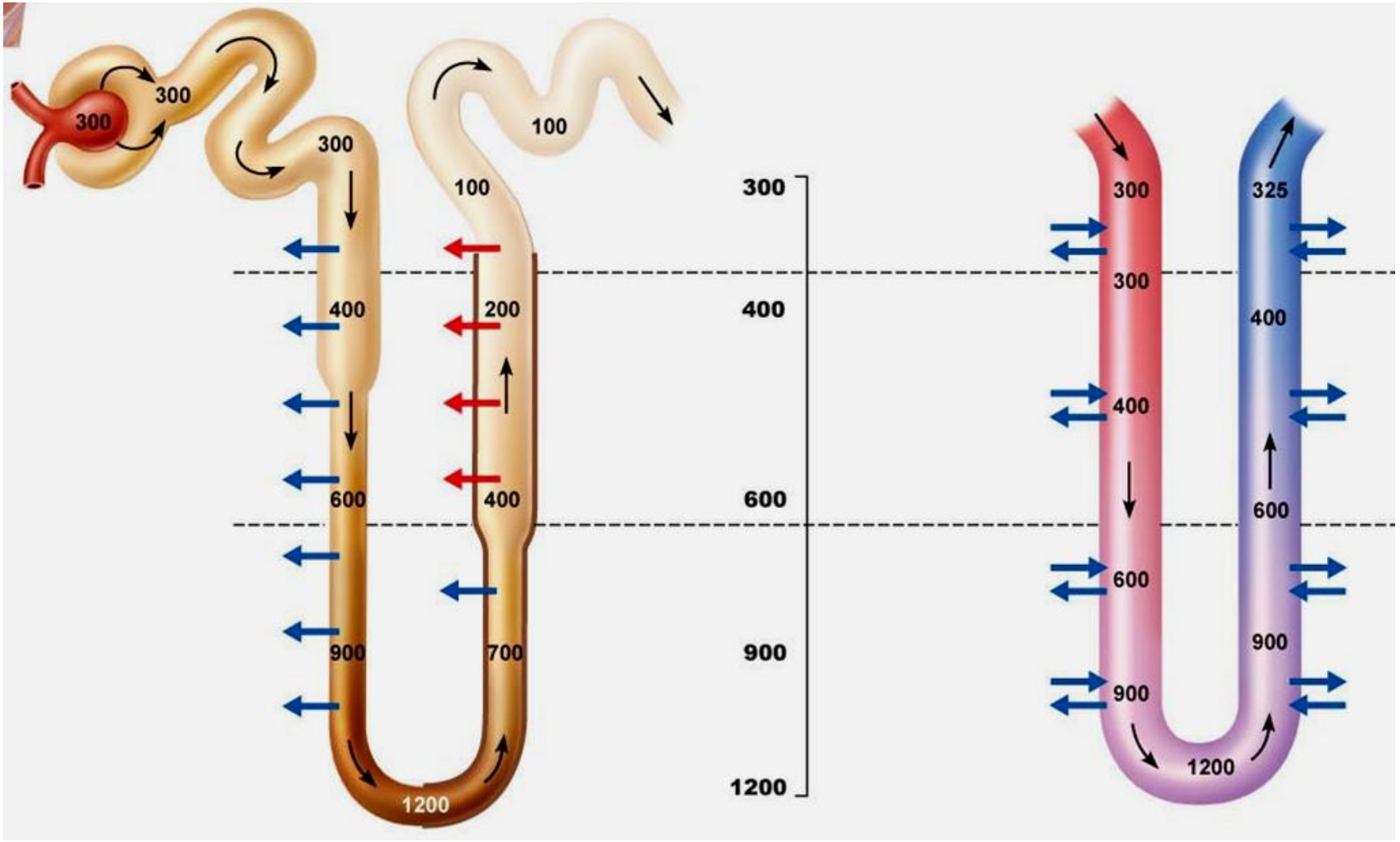
J. Carnegie, UofO



(3) Collecting ducts in deep medullary regions permeable to urea

cortical region of CD **impermeable** to urea ➢ urea stays in filtrate

medullary region: highly permeable to urea ➢ *what happens??*



(4) Vasa recta acts as countercurrent exchanger; maintains osmotic gradient

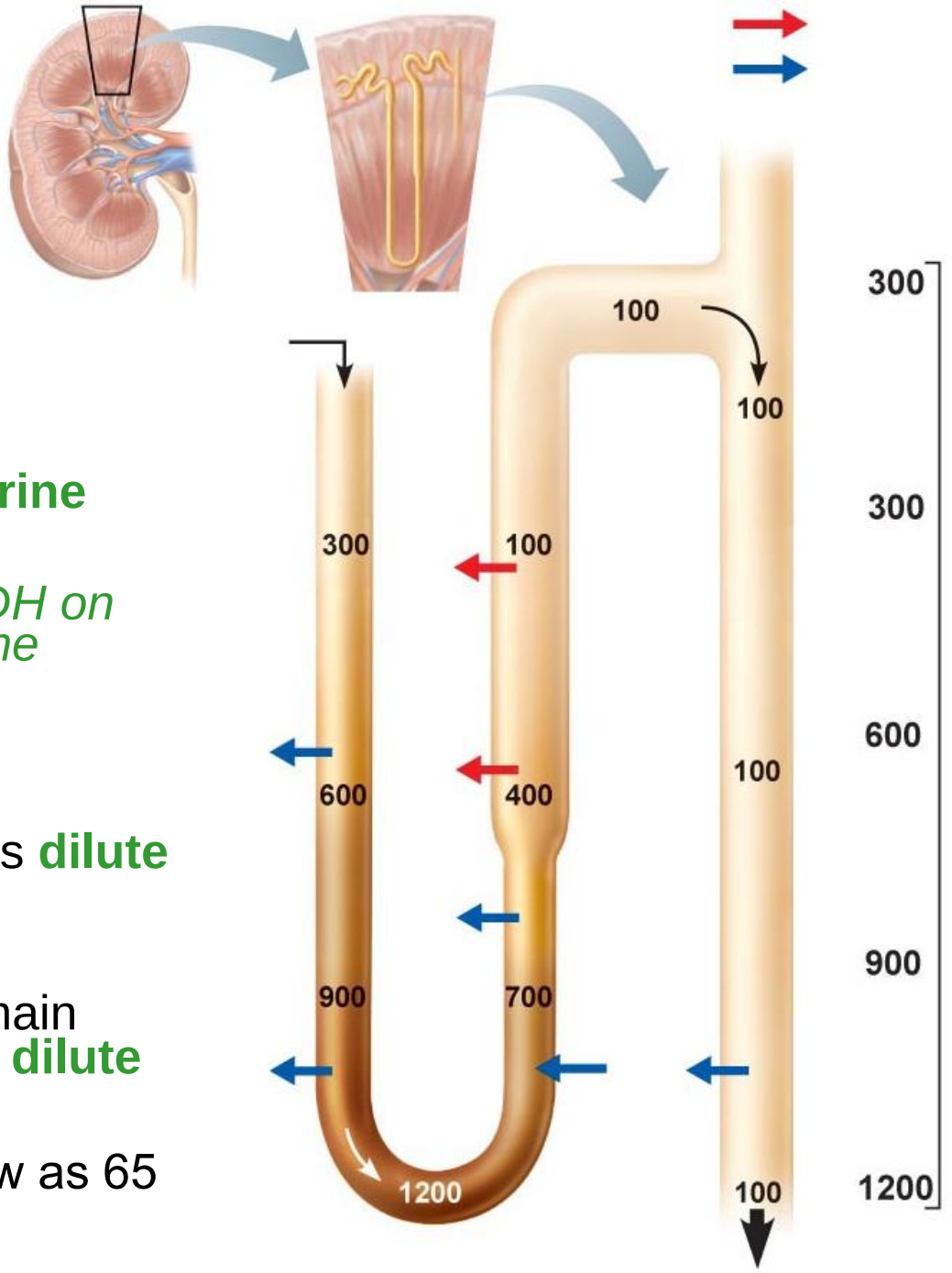
- 15% are **medullary** nephrons
- blood flow is sluggish; vessel walls freely permeable to salt & water
- blood acts as **exchanger** --- doesn't ……..gradient, but…….. it

4.3 Describe the regulation of urine concentration & volume

4.3.1 explain the influence of ADH on urine concentration and volume

Formation of dilute urine

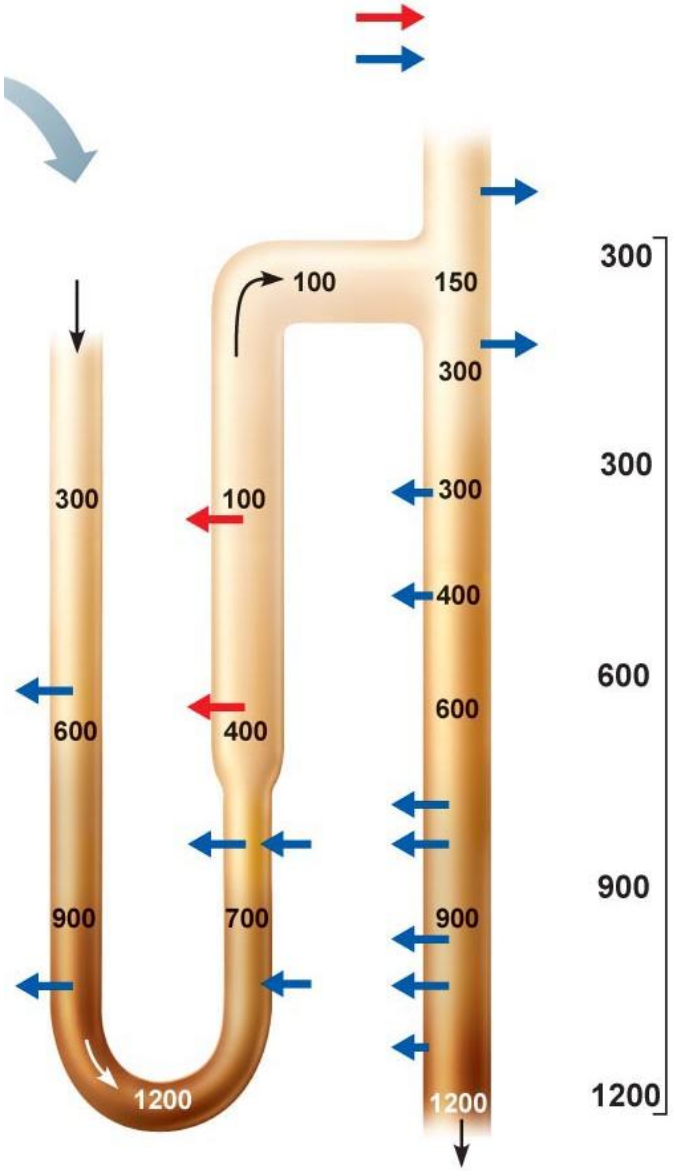
- *when would this happen??*
- filtrate at top of ascending limb is **dilute** due to salt removal
- in absence of **ADH** (_____ hormone) collecting ducts remain **impermeable** to water & very **dilute** urine produced
- osmolarity of urine can be as low as 65 mOsm



Formation of Concentrated Urine

- **ADH** from _____; amount secreted depends on body hydration
- ADH acts at **collecting ducts** ➢ increases number of water channels in **principal cells**
- collecting ducts extend into medullary area ➢ filtrate can again attain an osmolarity up to 1200 mOsm

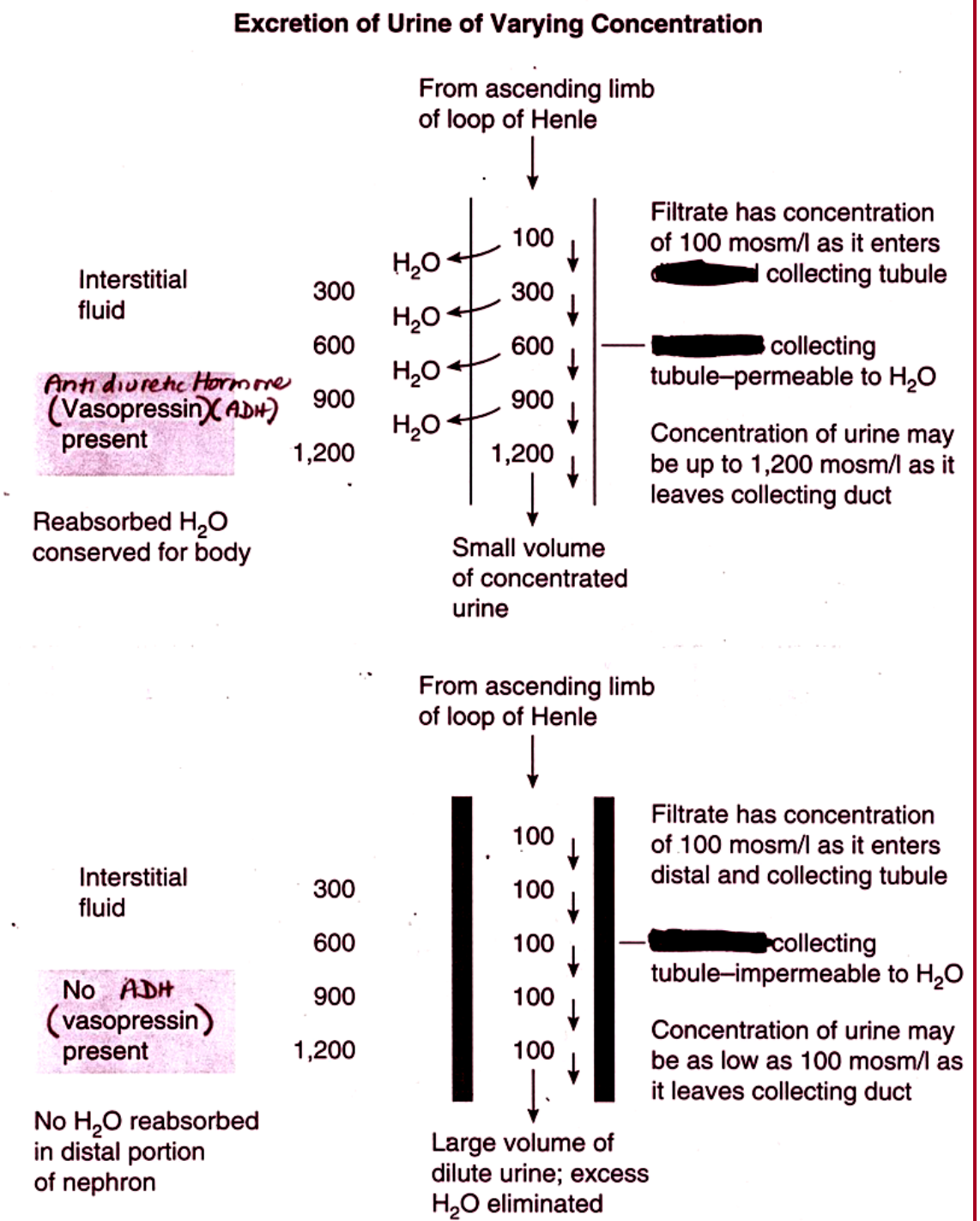
When would this ability be essential to survival??



Facultative Water Reabsorption (ADH)

constant low level of ADH secretion ➢ ***increased by any event which raises plasma osmolarity above 300 mOsm (eg: _____ _____)

ADH important for formation of concentrated urine, but ADH action depends on medullary gradient & presence of urea also critical (consequences for severely malnourished individuals??)



4.3.2 define diuretic & provide some examples

- enhances urinary output
- any substance that is:
  - ➢ not reabsorbed
  - ➢ exceeds renal reabsorption ability eg:

_____

_____

What is effect of alcohol on urine formation and why??

- caffeine and most prescribed diuretic drugs **inhibit** Na^+ reabsorption ➢ *what is the net effect of this??*

4.3.3 define renal clearance & express this parameter in terms of U, V & P

= **volume** of plasma from which a substance is 100% **cleared** per unit time

RC = UV/P

U: [substance(mg/ml)] in urine

V: flow rate of urine formation (ml/min)

P: [substance(mg/ml)] in plasma

Sample calculation:

- inulin: high MW polysaccharide used as a standard because not stored, reabsorbed or secreted by kidneys
- RC (inulin) = GFR

U = 125 mg/ml *P = 1 mg/ml* *V = 1 ml/min*

RC = (125)(1) / 1 = 125 ml/min

How do you interpret this result???

(1) What if RC (substance) < RC (inulin)? eg: urea ⇒ RC = 70 ml/min

(2) What if RC (substance) = 0? [name 3]

(3) What if RC (substance) > RC (inulin)? eg: creatinine ⇒ RC = 140 ml/min; also drug metabolites

$$RC = \frac{U\ (mg/ml) \times V\ (ml/min)}{P\ (mg/ml)}$$

RC = ml/min

4.3.4 describe the physical characteristics of urine & summarize its chemical composition; be aware of key body constituents that shouldn't be in urine

A. Physical Characteristics:

Colour & transparency:

- clear/pale to deep yellow (**urochrome** = pigment from _____)
- deepness of yellow indicates ???
- some drugs/vitamins can alter colour of urine; cloudiness can indicate ???

Odour:

- will develop ammonia odor if left to stand due to bacterial metabolism of urea
- odor can be altered by some drugs & vegetables, also diseases (prime example is:_____)

pH:

- usually ~ 6, but can vary (~4.5-~8)

(i) what is an acid-ash diet?[acidic urine]
(ii) what is an alkaline-ash diet? [alkaline urine] – 2 other causes of alkaline urine?

specific gravity:

- usually 1.001 to 1.035; *depends on???*

B. Chemical Composition:

- 95% water, 5% solutes
- solute in highest concentration is: ____
- also **uric acid** (from _____) & **creatinine** (from _____)
- in decreasing order: _____, Na^+, K^+, phosphate, sulfate, creatinine, uric acid
- much less, but more variable levels of: Ca^{++}, Mg^{++}, HCO_3^-
- v high concentrations of any constituent may indicate pathology

TABLE 25.2 Abnormal Urinary Constituents

SUBSTANCE	NAME OF CONDITION	POSSIBLE CAUSES
Glucose	Glycosuria	Diabetes mellitus
Proteins	Proteinuria, or albuminuria	Nonpathological: excessive physical exertion, pregnancy, high-protein diet Pathological (over 250 mg/day): heart failure, severe hypertension, glomerulonephritis, often initial sign of asymptomatic renal disease
Ketone bodies	Ketonuria	Excessive formation and accumulation of ketone bodies, as in starvation and untreated diabetes mellitus
Hemoglobin	Hemoglobinuria	Various: transfusion reaction, hemolytic anemia, severe burns, etc.
Bile pigments	Bilirubinuria	Liver disease (hepatitis, cirrhosis) or obstruction of bile ducts from liver or galbladder
Erythrocytes	Hematuria	Bleeding urinary tract (due to trauma, kidney stones, infection, or neoplasm)
Leukocytes (pus)	Pyuria	Urinary tract infection

4.3.5 describe the regulation of micturition

~ 200 ml urine in bladder

↓

stretch receptors trigger visceral reflex arc

↓

afferent to sacral region of spinal cord & efferent to bladder via parasympathetic pelvic nerves

↓

detrusor muscle contracts & internal sphincter relaxes ➔ urine through internal sphincter into upper part of urethra

↓

afferent also to brain; one feels urge to void

↓

external sphincter under voluntary control

↙ ↘

relax external sphincter & empty bladder

reflex subsides in ~1 min; occurs again as urine accumulates

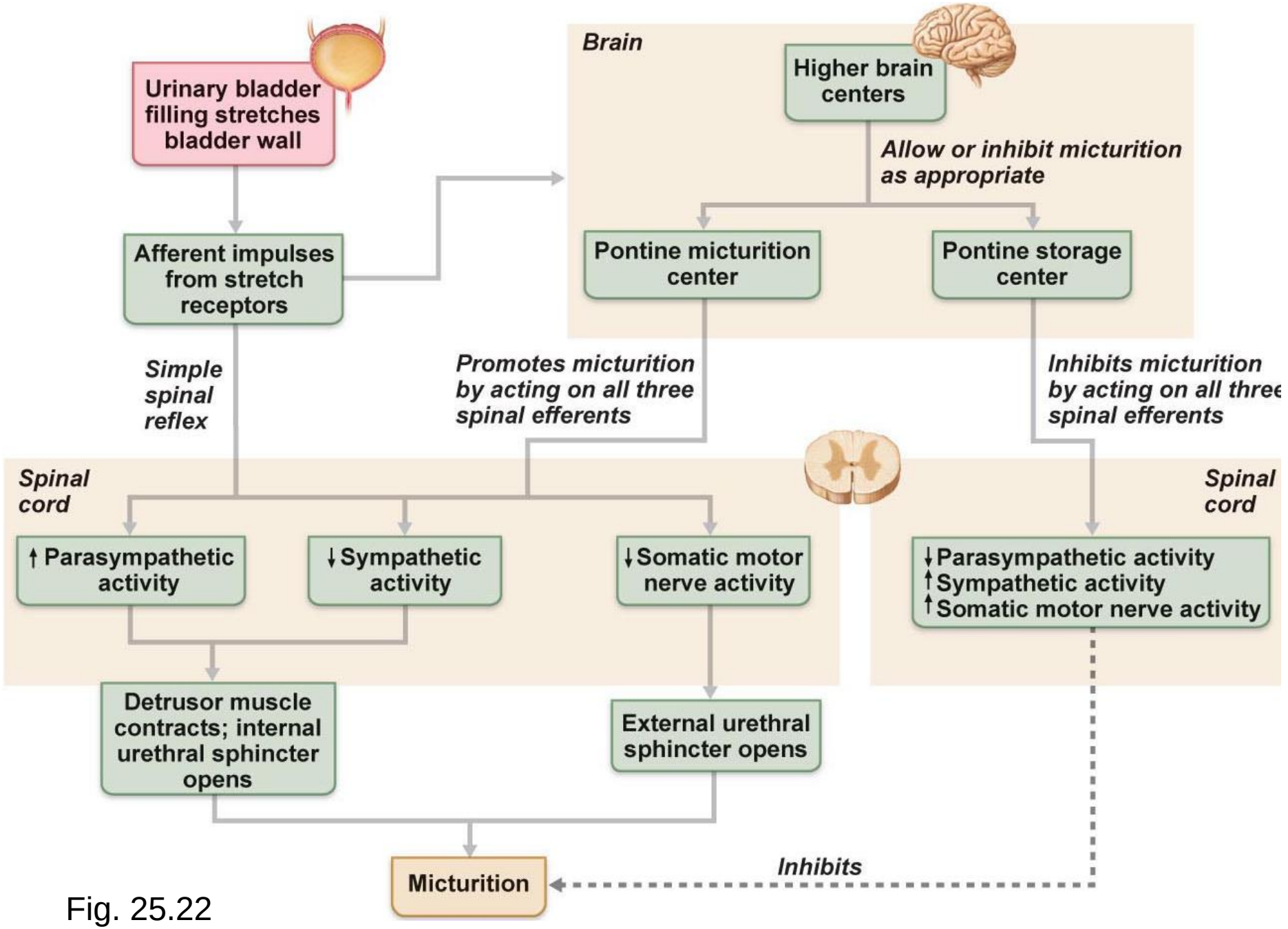
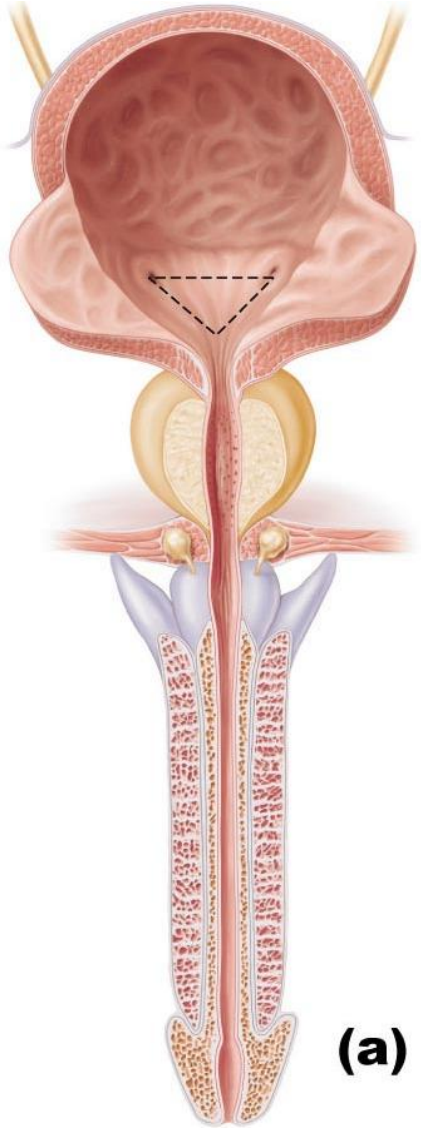
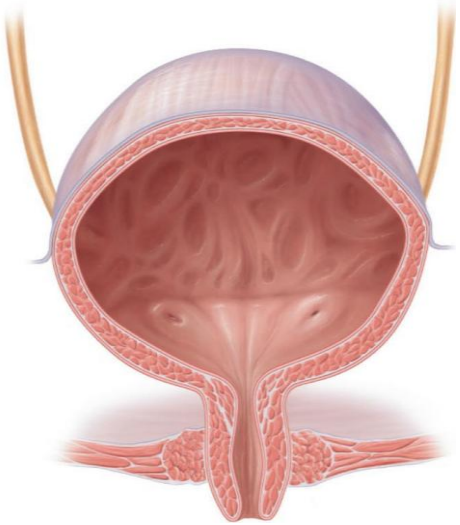

Urinary bladder filling stretches bladder wall
Afferent impulses from stretch receptors
Simple spinal reflex
Brain
Higher brain centers
Allow or inhibit micturition as appropriate
Pontine micturition center
Pontine storage center
Promotes micturition by acting on all three spinal efferents
Inhibits micturition by acting on all three spinal efferents
Spinal cord
↑ Parasympathetic activity
↓ Sympathetic activity
↓ Somatic motor nerve activity
Spinal cord
↓ Parasympathetic activity
↑ Sympathetic activity
↑ Somatic motor nerve activity
Detrusor muscle contracts; internal urethral sphincter opens
External urethral sphincter opens
Micturition
Inhibits

Fig. 25.22





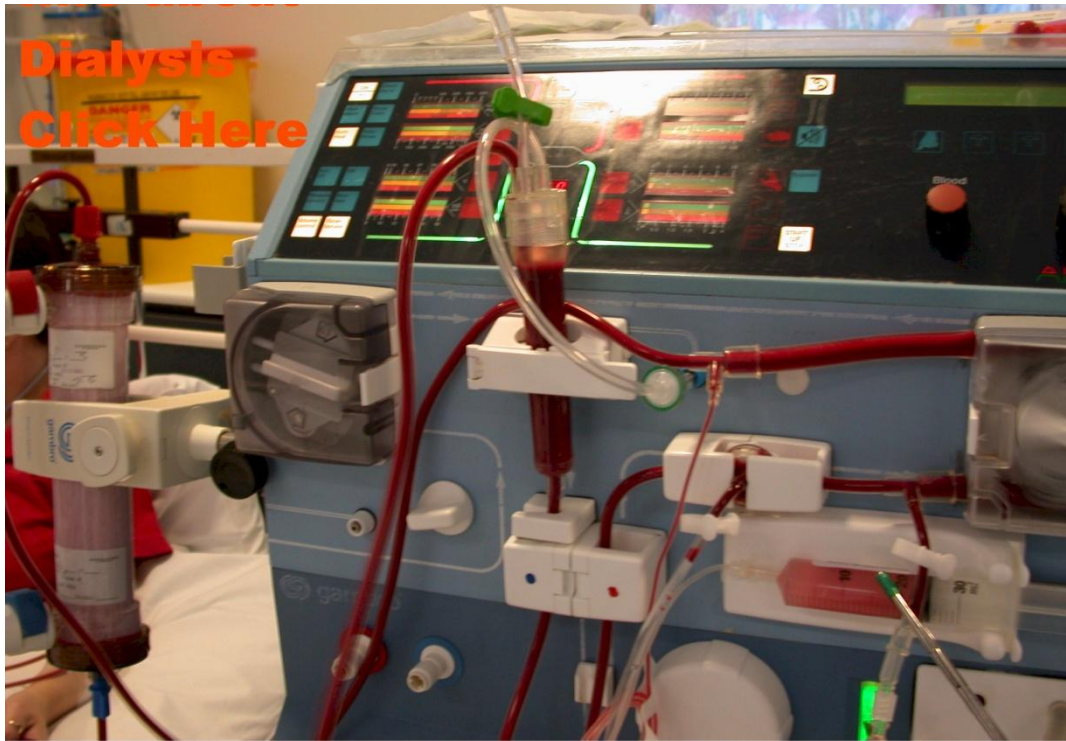
Incontinence: inability to control micturition voluntarily ➢ emotional problems, pressure of pregnancy, nervous system problems

*What is **stress** incontinence??*

Urinary retention: bladder unable to expel urine; e.g. after general anaesthetic; can also result from prostate hypertrophy

4.3.6 define renal failure; indicate potential causes & options for treatment

- not enough functioning nephrons ➢ filtrate formation reduced or stopped:
  - √ repeated damaging kidney infections
  - √ physical injuries to kidneys
  - √ prolonged pressure on skeletal muscle
  - √ inadequate blood delivery to tubules
- nitrogenous wastes accumulate & blood becomes acidic ☞ diarrhea, vomiting, edema, labored breathing, cardiac irregularities, convulsions, coma, death
- usu 3 times/week; 4-8 h session
- symptoms apparent when ~75% of renal function lost – why??

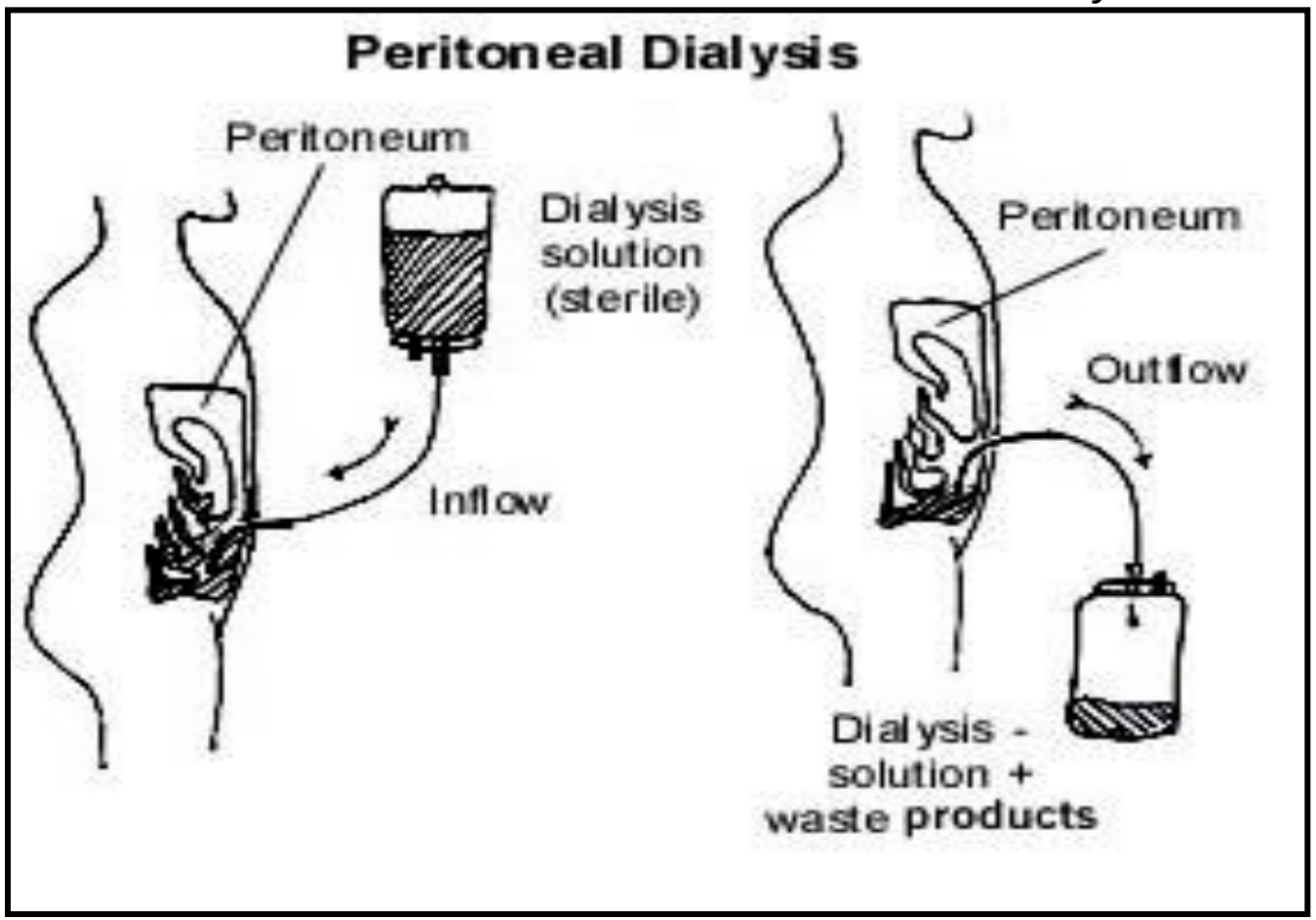


J. Carnegie, UofO

http://www.dundee.ac.uk/medther/renal

Peritoneal Dialysis:

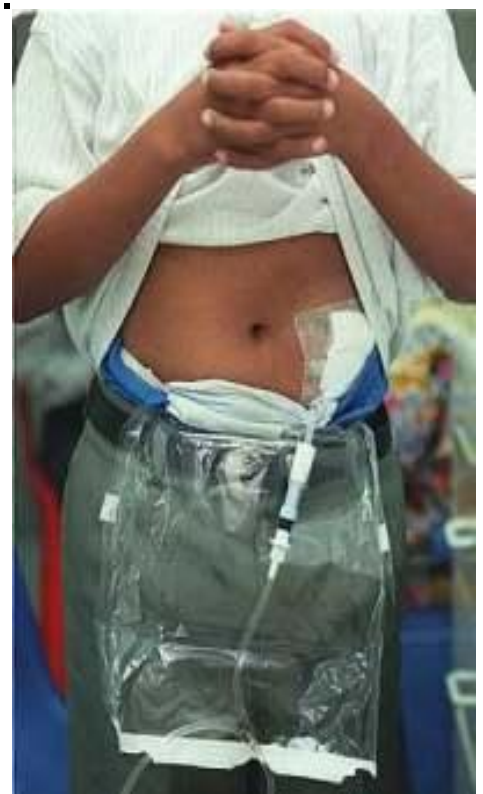
- Works by using the **peritoneal membrane**; can be done at home or at work
- **does not** require weekly hospital visits
- **dialysate** is infused into the peritoneal cavity through a catheter; dialysate made of made up mostly of salts and sugar, encourages filtration through the peritoneum
- Extra **fluid** and **wastes** is drawn from blood into dialysate



Two types:

Continuous Ambulatory Peritoneal Dialysis (CAPD): usu 4-5 times/day. Pt puts dialysate (about two litres) into peritoneal cavity through catheter - dialysate stays there for 4-5 hours before it is drained back into the bag and thrown away. There is a new bag of dialysate for each exchange.

Continuous Cycling Peritoneal Dialysis (CCPD): usu is done at home using a special machine called a cycler. This is similar to CAPD except that a number of cycles (exchanges) occur. Each cycle usually lasts 1-1/2 hours and exchanges are done during the night while pt sleeps.



Continuous Ambulatory Peritoneal Dialysis (CAPD):

http://202.186.86.35/...=health