

**RENAL PHYSIOLOGY**  
**EXCRETING A CONCENTRATED URINE**  
**COUNTER CURRENT MECHANISM**



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## COUNTER CURRENT MECHANISM

- KIDNEYS HAVE
  - MECHANISMS FOR EXCRETING EXCESS WATER
  - MECHANISMS FOR EXCRETING EXCESS SOLUTES

### Obligatory Urine Volume

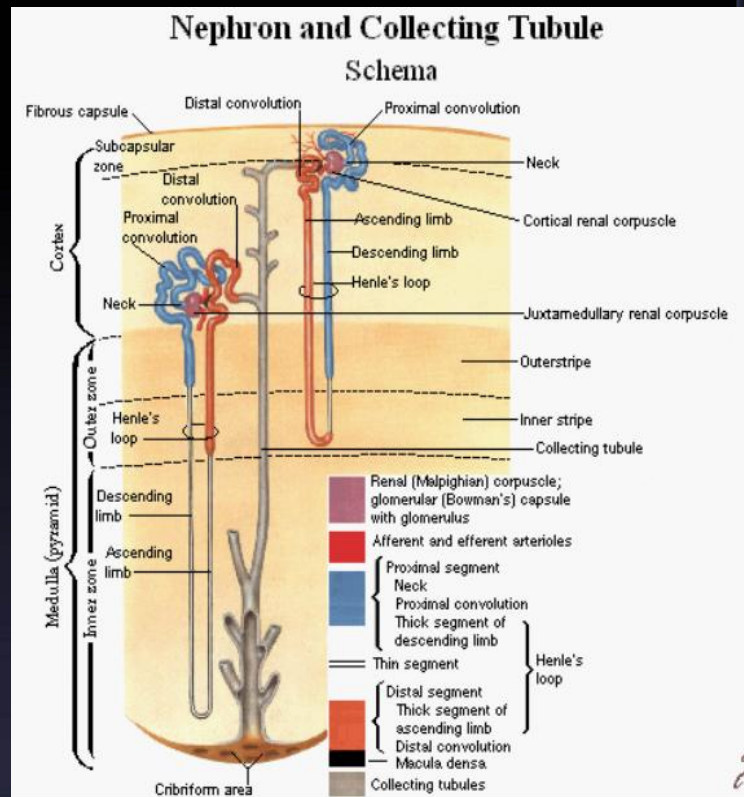
$$\frac{600 \text{ mOsm/day}}{1200 \text{ mOsm/L}} = 0.5 \text{ L/day}$$

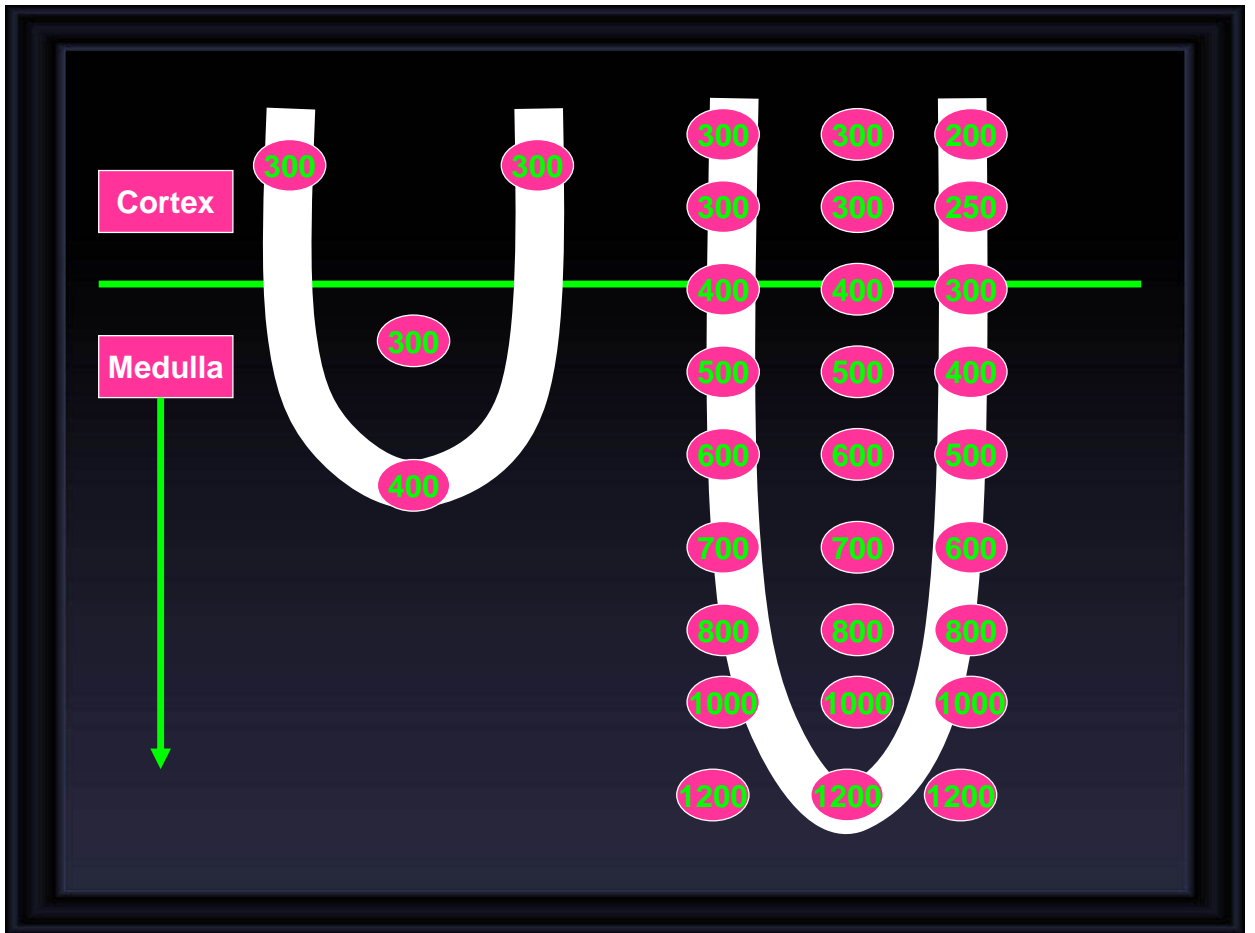
## NEPHRON TYPES

- **Superficial (cortical) [85 %]**
  - Capable of forming dilute urine
- **Juxtamedullary [15 %]**
  - Capable of forming concentrated (> 300 mOsm/kg) urine

## NEPHRON TYPES Cortical and Juxtamedullary Nephrons

1-2 % Blood  
Flows  
Through  
Juxta Medullary  
Nephrons





## NEPHRON TYPES

- **Cortical Nephrons have**
  - **Peritubular Capillaries**
- **Juxtamedullary Nephron have**
  - **Vasa Recta**

## EXCRETION LIMITS

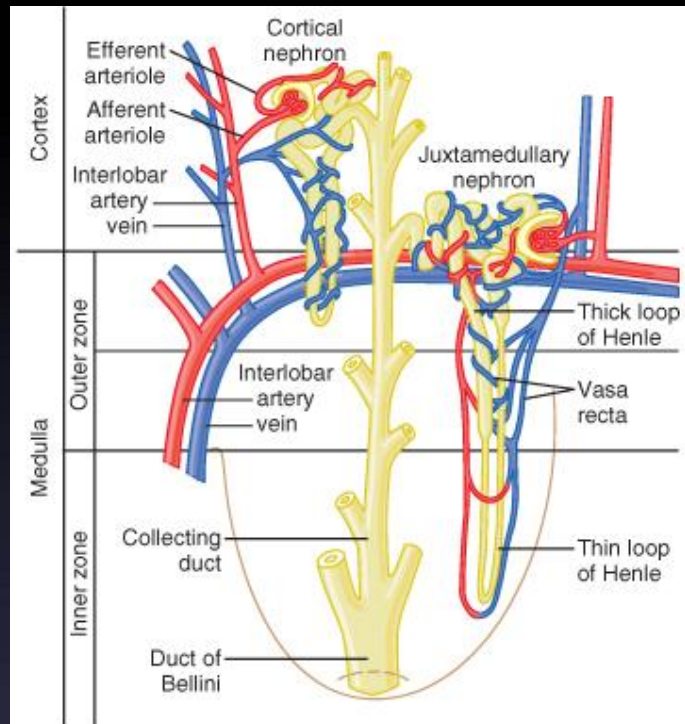
### Obligatory Urinary Output

- **At least 600 mmol must be excreted each day**
  - minimum volume =  $600/1200 = 0.5$  L
  - maximum volume = 20 L

## NEPHRON TYPES

### Cortical and Juxtamedullary Nephrons

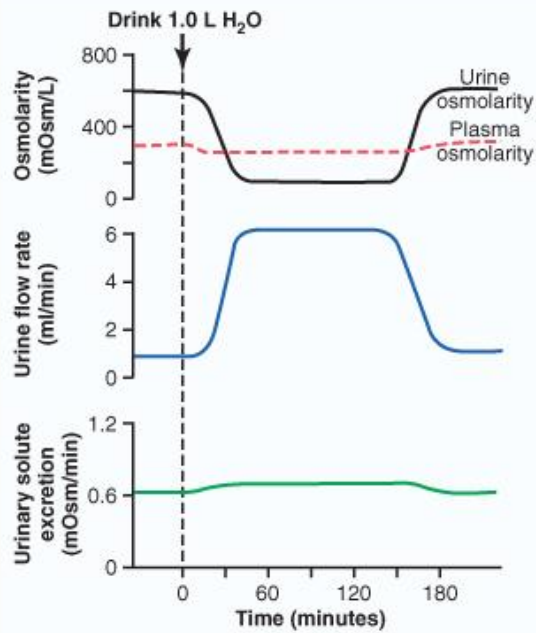
1-2 % Blood Flows Through Juxta Medullary Nephrons





## COUNTER CURRENT MECHANISM

- **LOOPS OF HENLE OF JUXTA MEDULLARY NEPHRONS** establish hyperosmolality of interstitium of medulla. They are called **COUNTER CURRENT MULTIPLIERS**
- **VASA RECTA** maintain hyperosmolality established by counter current multipliers. They are called **COUNTER CURRENT EXCHANGERS**



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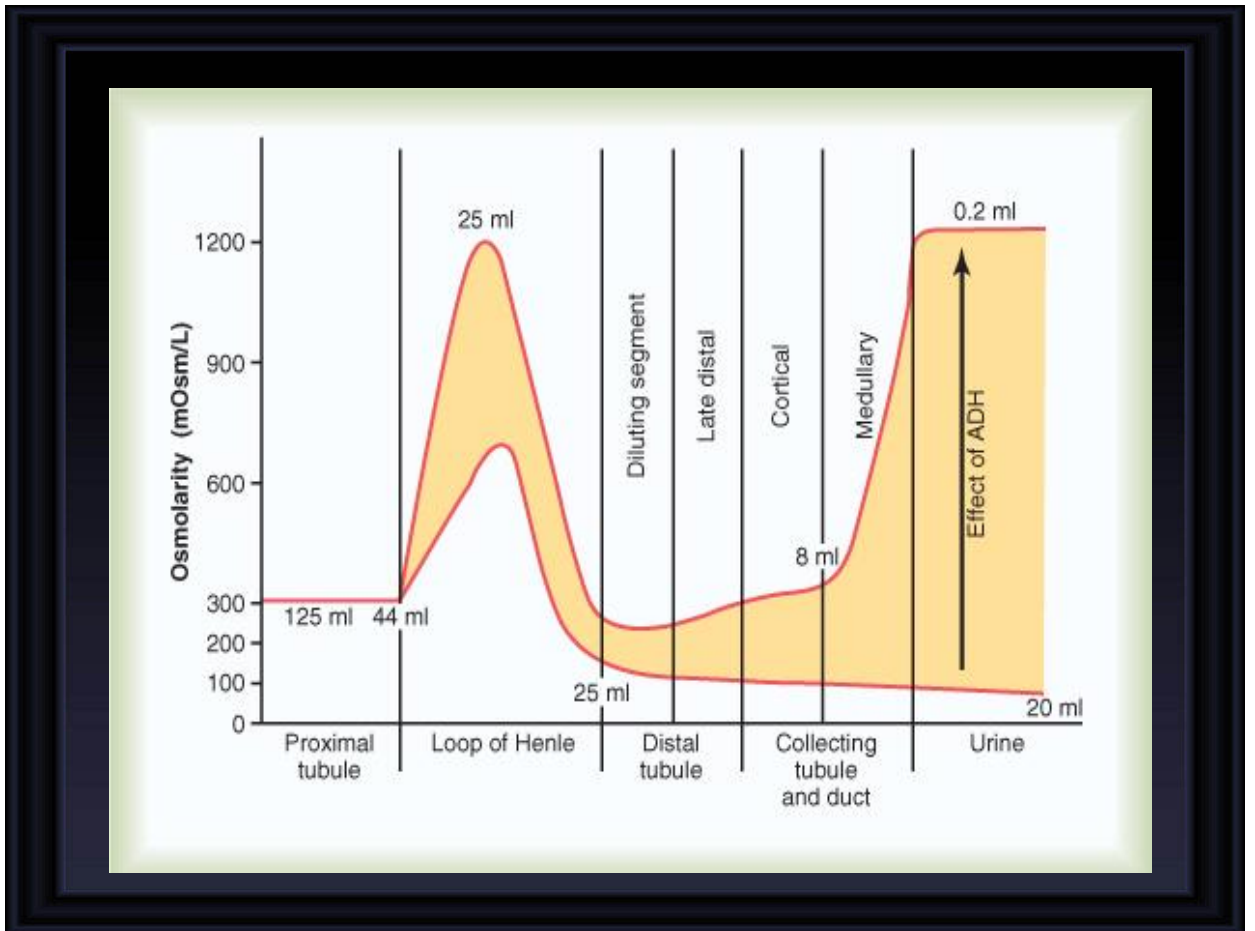
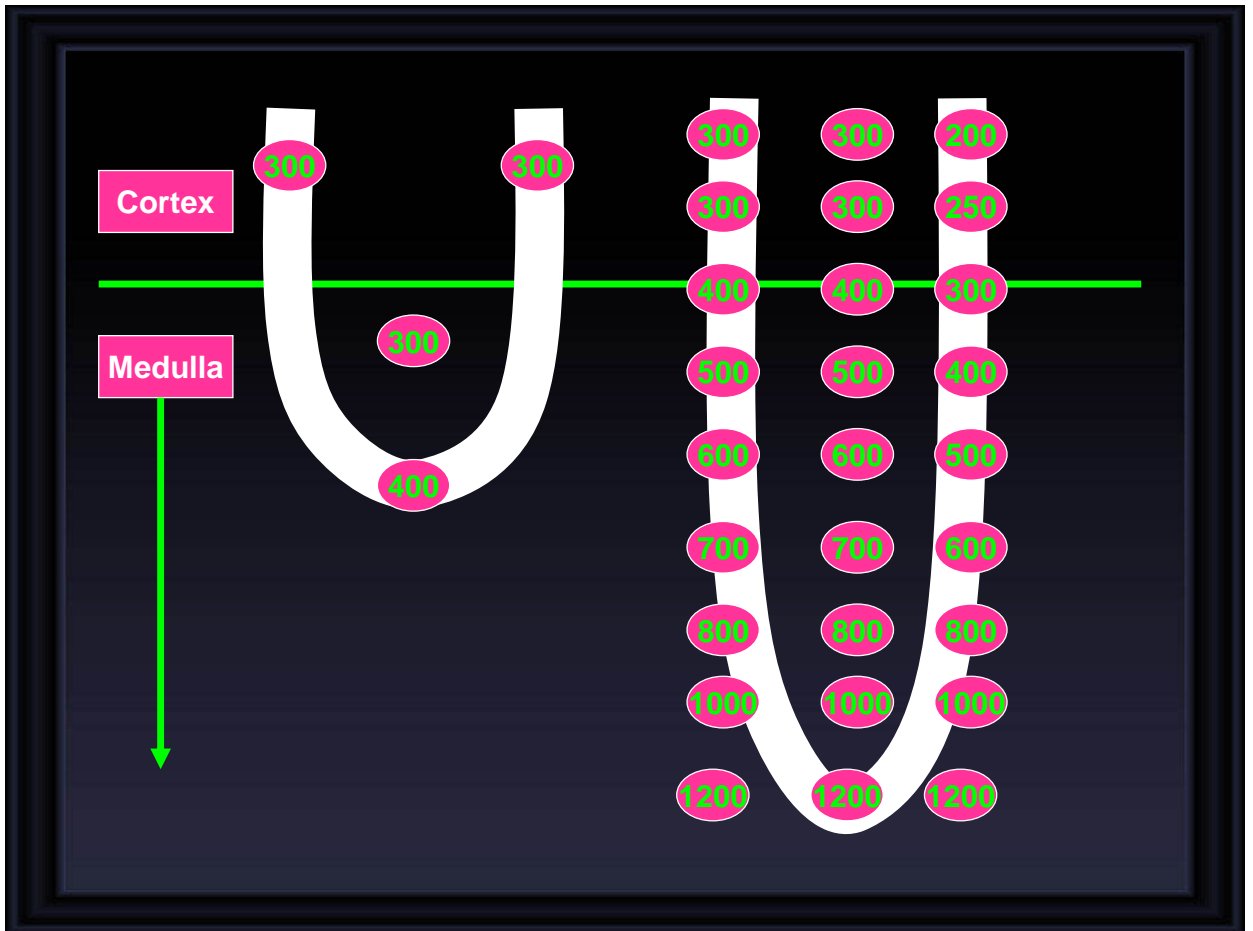


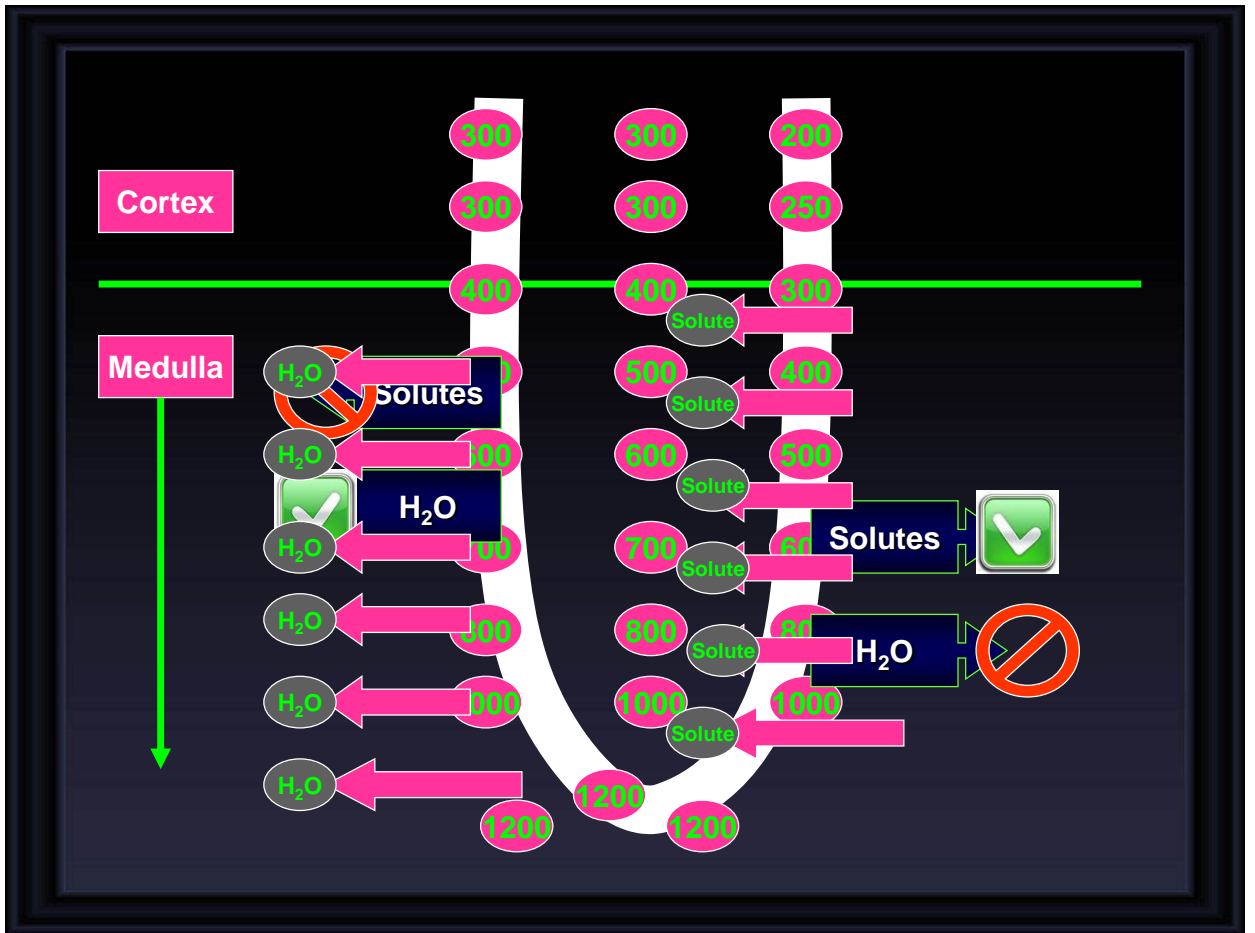
Table 28-1

## Summary of Tubule Characteristics—Urine Concentration

	Active NaCl Transport	Permeability		
		<i>H<sub>2</sub>O</i>	<i>NaCl</i>	<i>Urea</i>
Proximal tubule	++	++	+	+
Thin descending limb	0	++	+	+
Thin ascending limb	0	0	+	+
Thick ascending limb	++	0	0	0
Distal tubule	+	+ADH	0	0
Cortical collecting tubule	+	+ADH	0	0
Inner medullary collecting duct	+	+ADH	0	++ADH

0, minimal level of active transport or permeability; +, moderate level of active transport or permeability; ++, high level of active transport or permeability; +ADH, permeability to water or urea is increased by ADH.





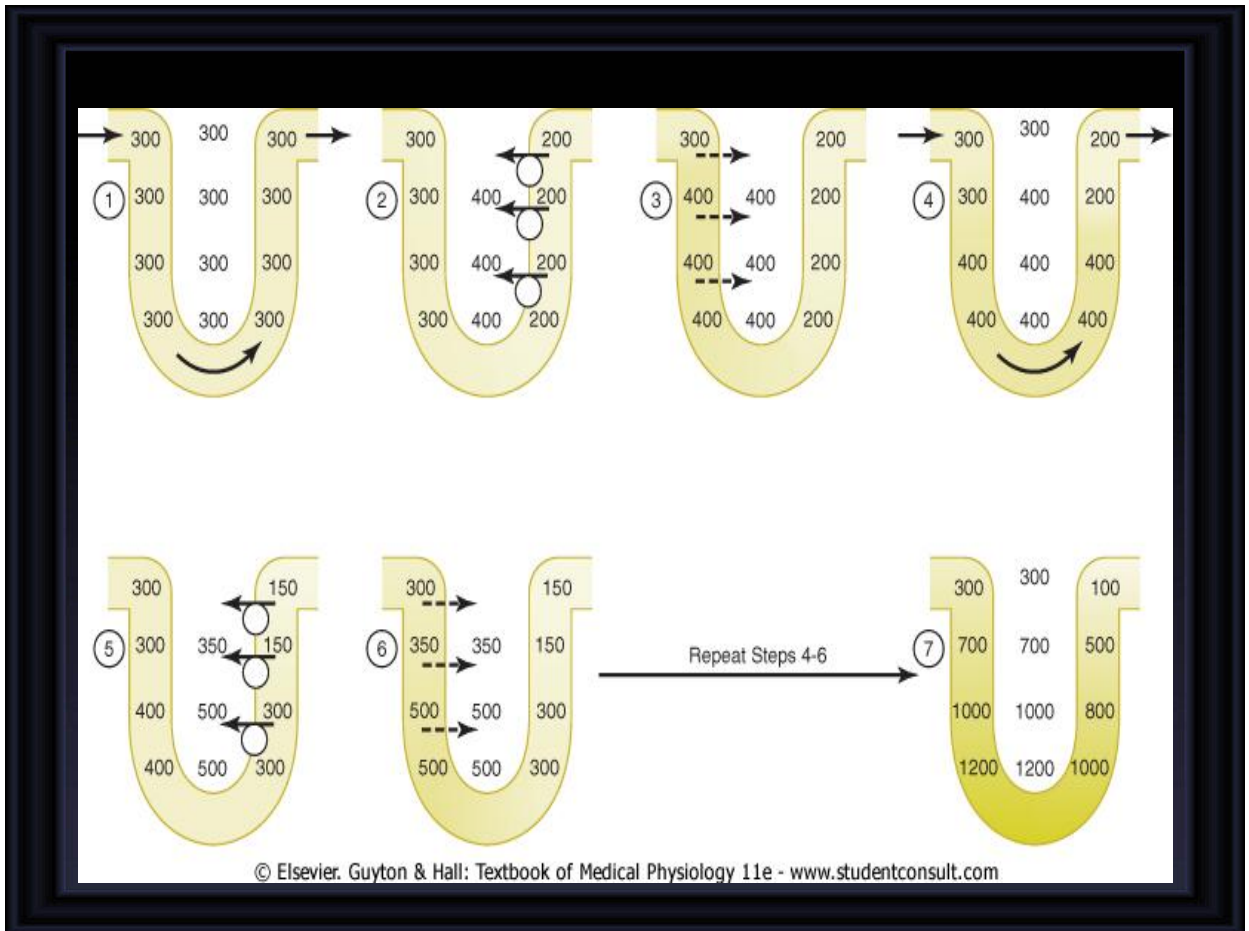
## LOOP OF HENLE

<u>Descending Loop</u>	<u>Ascending Loop</u>
highly permeable to water	•impermeable to water
•impermeable to $\text{Na}^+$	•permeable to $\text{Na}^+$ (mediated by $\text{Na}^+/\text{K}^+/\text{2Cl}^-$ apical carrier - inhibited by furosemide (Lasix))
•water exit promoted	• $\text{Na}^+/\text{K}^+$ -ATPase actively pumps out sodium of cell into interstitium

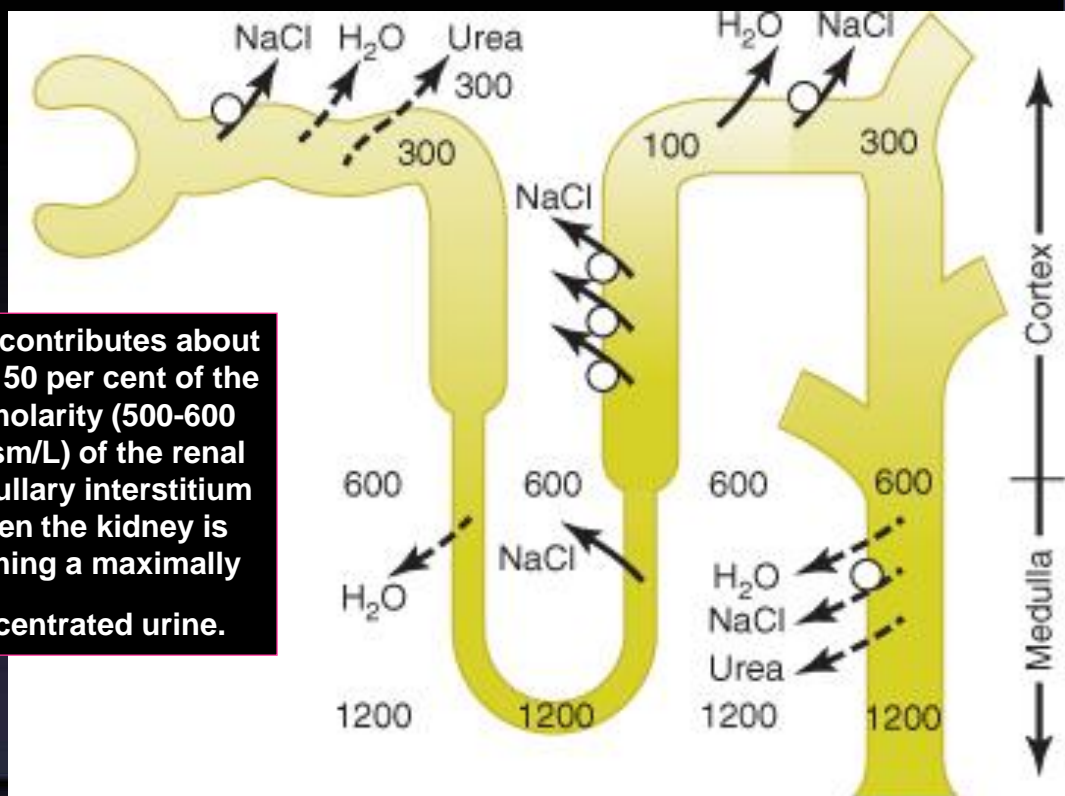
## Buildup of solute concentration into the renal medulla

1. **Active transport of sodium ions and co-transport of potassium, chloride, and other ions** out of the thick portion of the ascending limb of the loop of Henle into the medullary interstitium
2. **Active transport of ions** from the collecting ducts into the medullary interstitium
3. **Facilitated diffusion of large amounts of urea** from the inner medullary collecting ducts into the medullary interstitium
4. **Diffusion of only small amounts of water** from the medullary tubules into the medullary interstitium, far less than the reabsorption of solutes into the medullary interstitium





## Role of DCT & CT in Excreting a Concentrated Urine

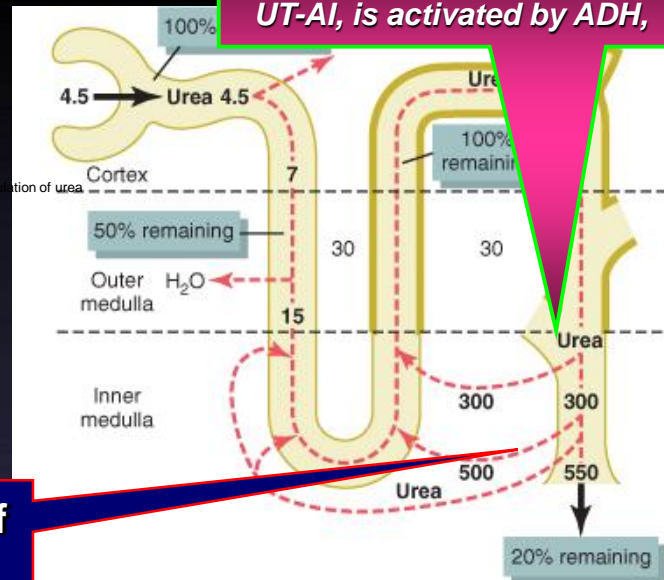


Urea contributes about 40 to 50 per cent of the osmolarity (500-600 mOsm/L) of the renal medullary interstitium when the kidney is forming a maximally concentrated urine.

## ROLE OF UREA IN MAKING A HYPEROSMOTIC RENAL MEDULLARY INTERSTITIUM AND CONCENTRATED URINE

About 40 to 50 % of the osmolarity (500-600 mOsm/L) of the renal medullary interstitium when the kidney is forming a maximally concentrated urine.

*A specific urea transporter UT-A1, is activated by ADH,*



**Recirculation of  
UREA**

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## **Two special features of the renal medullary blood flow**

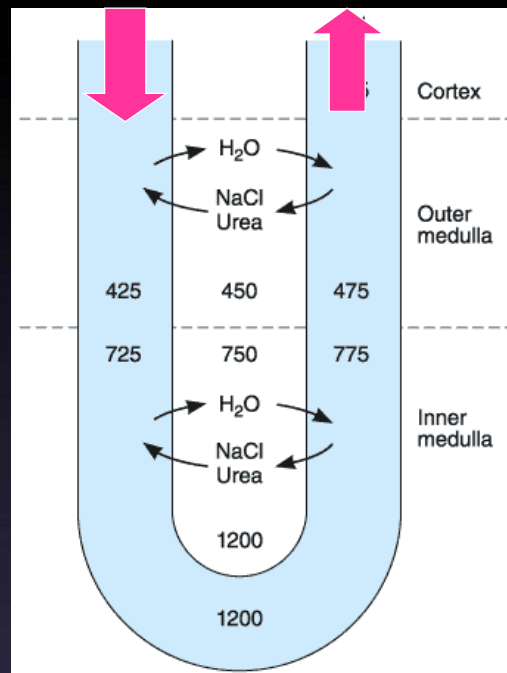
- **The medullary blood flow is low, accounting for less than 5 per cent of the total renal blood flow. This sluggish blood flow is sufficient to supply the metabolic needs of the tissues but helps to minimize solute loss from the medullary interstitium.**
- **The vasa recta serve as countercurrent exchangers, minimizing washout of solutes from the medullary interstitium.**

## COUNTER CURRENT EXCHANGERS

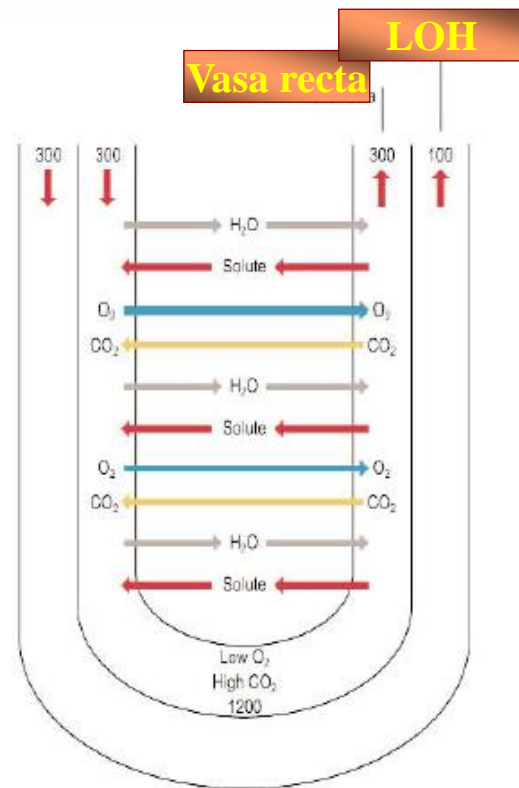
- Sluggish blood flow (1-2 %)**
- Close proximity**
- High permeability**

# COUNTER CURRENT EXCHANGERS

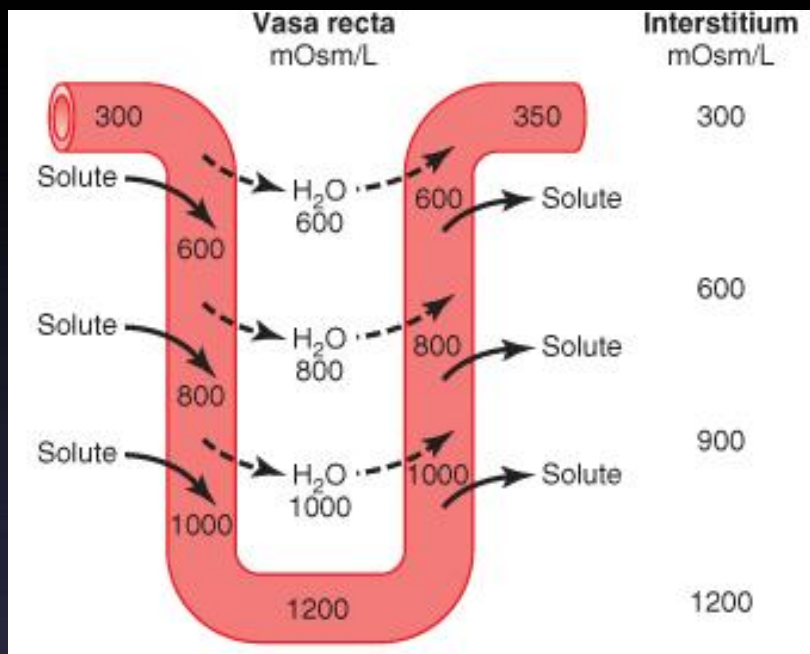
A PASSIVE  
PROCESS



**COUNTER  
CURRENT  
EXCHANGERS  
COUNTER  
CURRENT  
MULTIPLIERS**



# COUNTER CURRENT EXCHANGERS





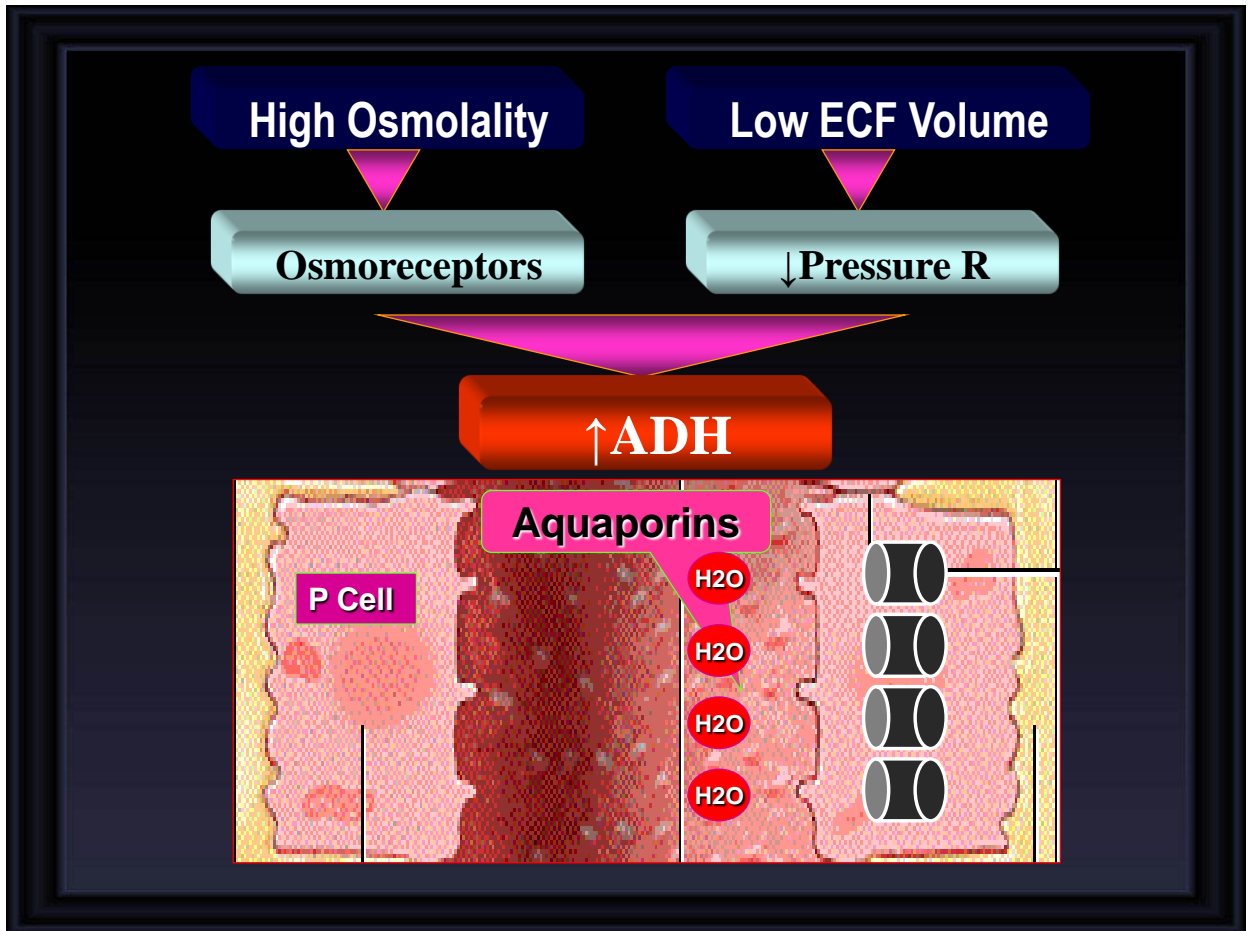


Table 28-2

### Regulation of ADH Secretion

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#### Increase ADH

↑ Plasma osmolarity  
 ↓ Blood volume  
 ↓ Blood pressure

Nausea  
 Hypoxia

#### Drugs:

Morphine  
 Nicotine  
 Cyclophosphamide

#### Decrease ADH

↓ Plasma osmolarity  
 ↑ Blood volume  
 ↑ Blood pressure

#### Drugs:

Alcohol  
 Clonidine (antihypertensive drug)  
 Haloperidol (dopamine blocker)

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## DISORDERS OF URINARY CONCENTRATING ABILITY

- Failure to Produce ADH: "**Central**"  
Diabetes Insipidus.
- Inability of the Kidneys to Respond to  
ADH: "**Nephrogenic**"  
Diabetes Insipidus.

## DISORDERS OF URINARY CONCENTRATING ABILITY

- Inappropriate secretion of ADH (SIADH)