



Sodium disorders in ICU patients

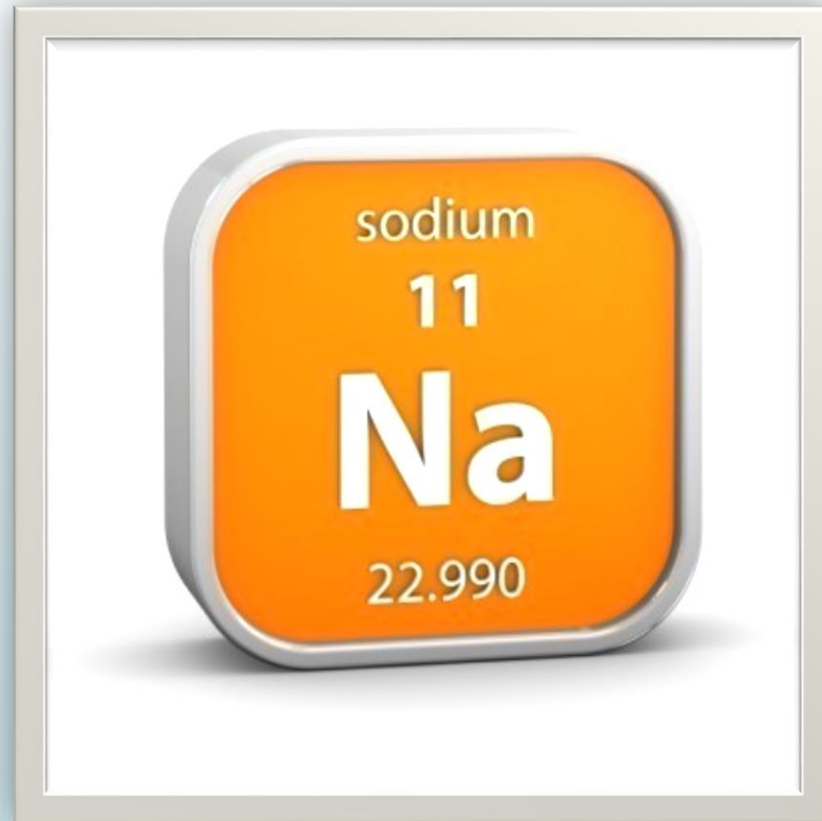
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Sodium Disorders



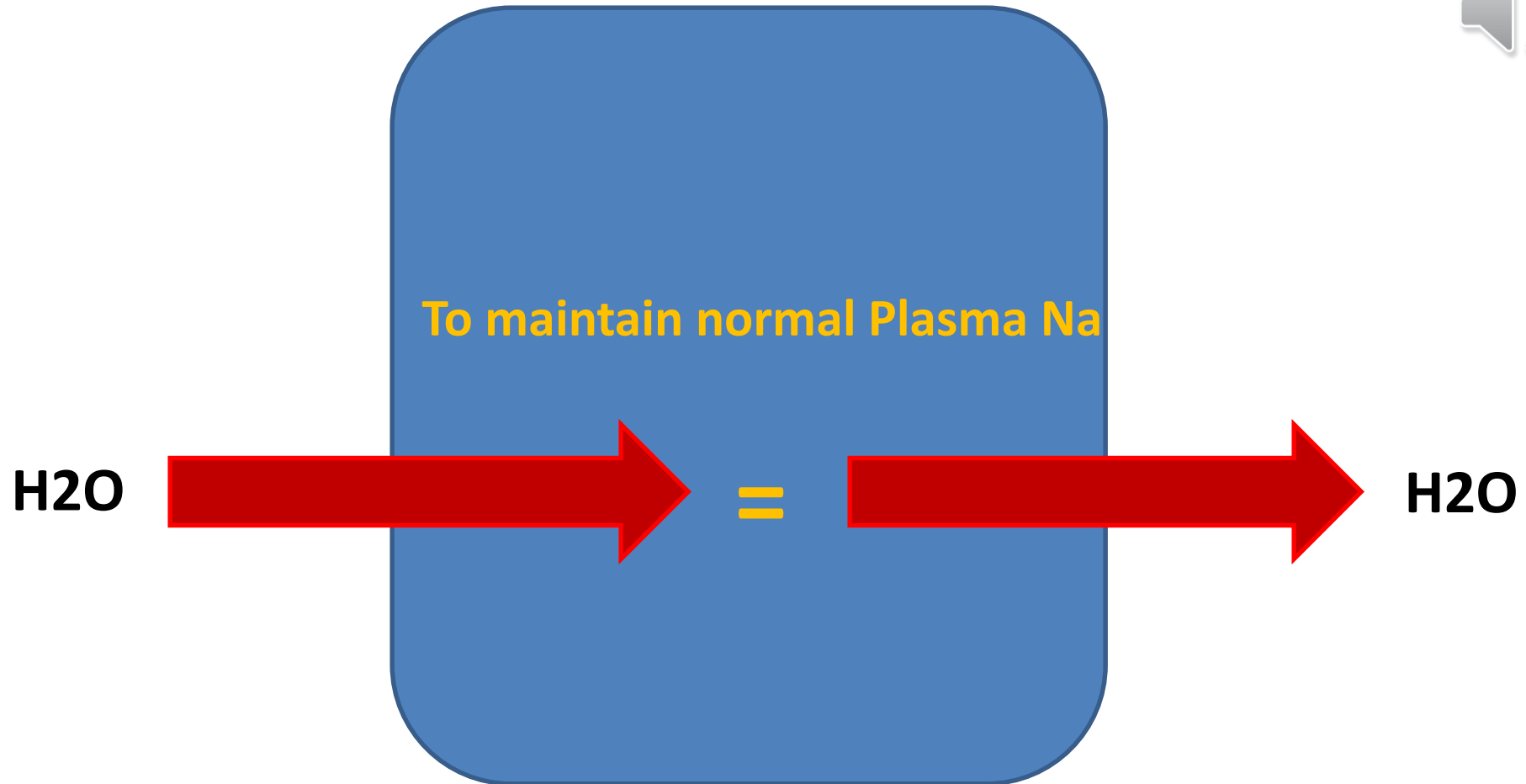
Sodium Disorders



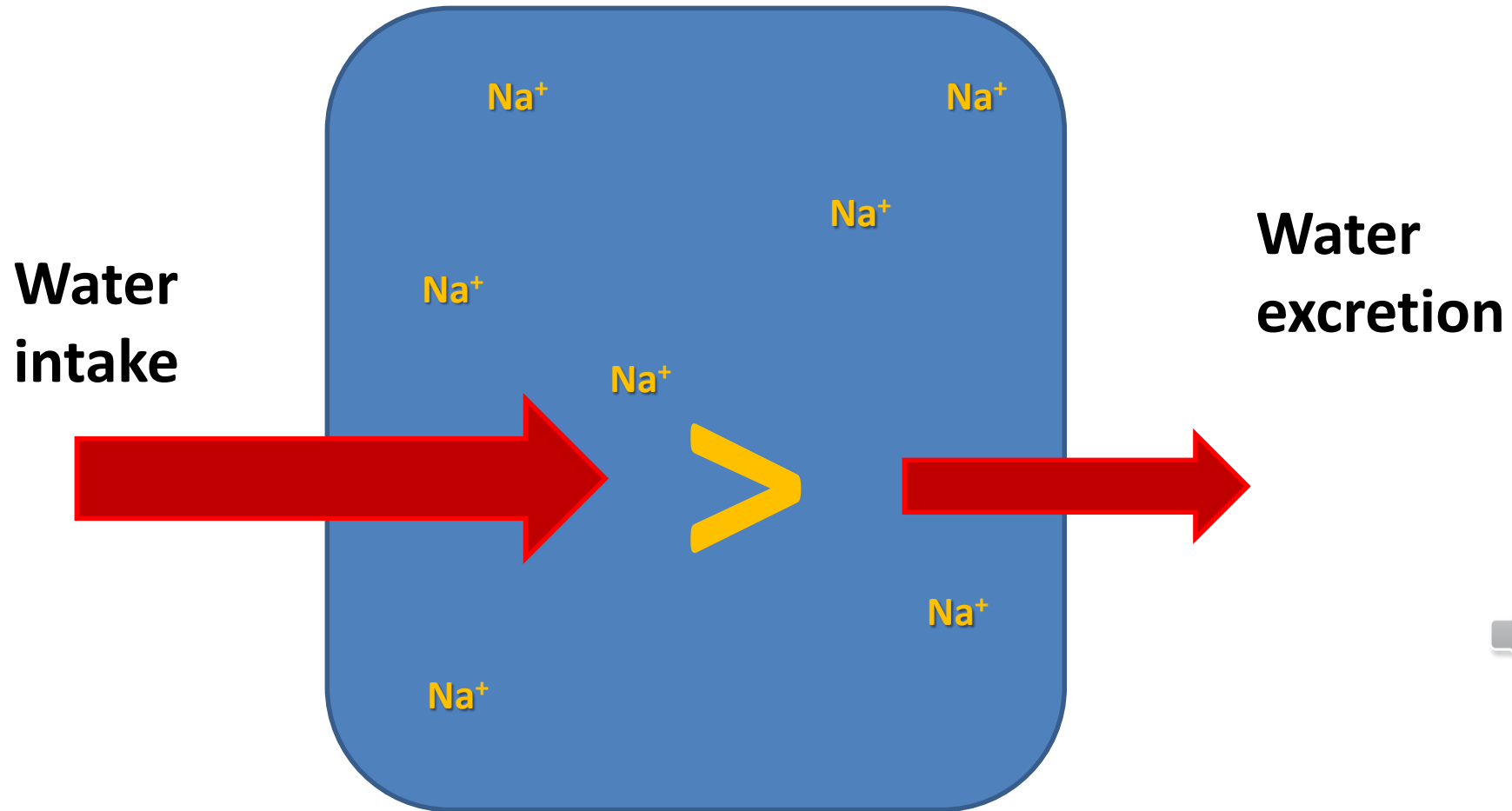
- Disorders of serum Na^+ concentration are caused by abnormalities in water homeostasis that lead to **changes in the relative ratio of Na^+ to body water.**

Na^+/TBW

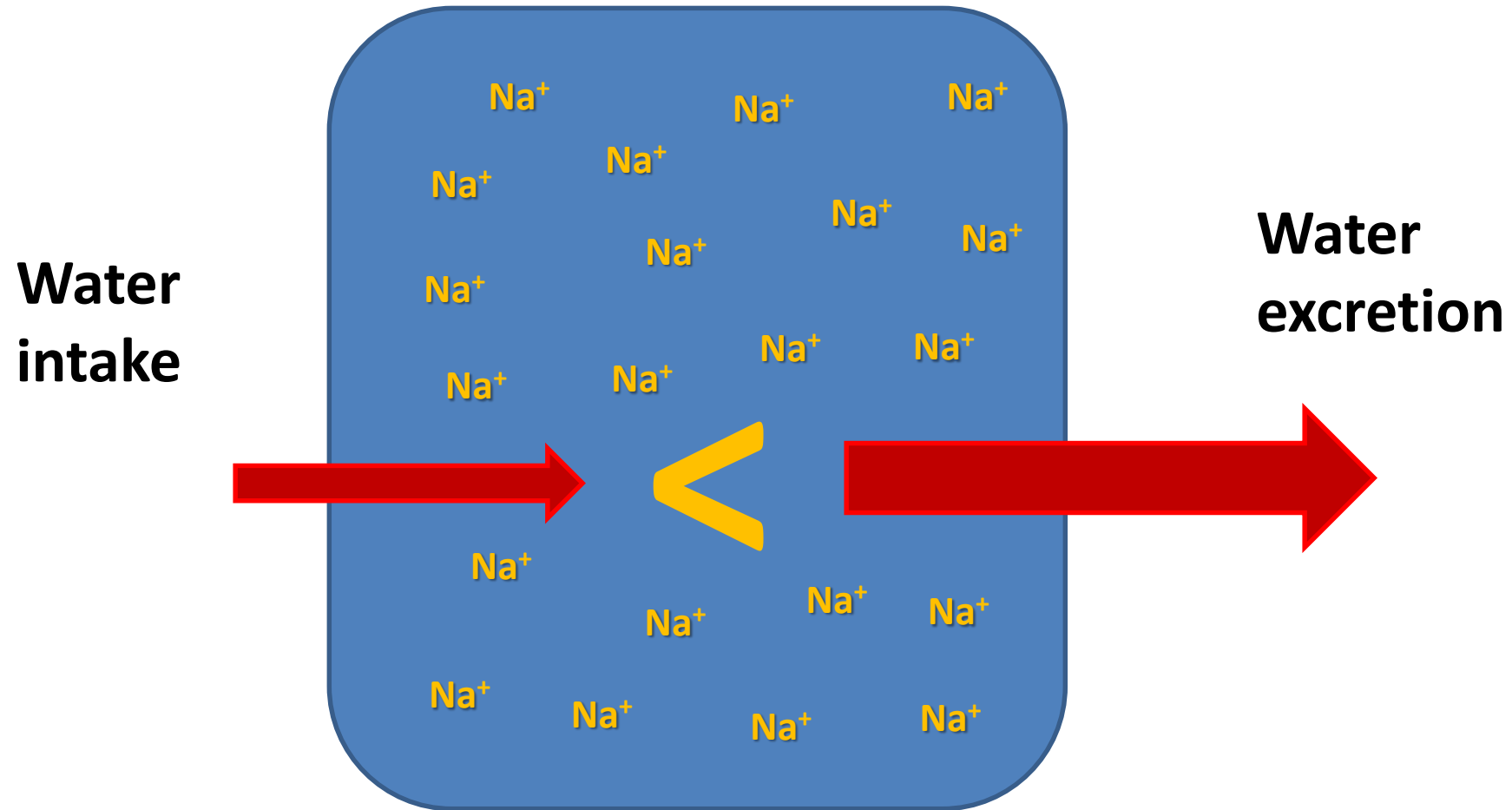
WATER BALANCE



HYPONATREMIA



HYPERNATREMIA



Water intake

AVP

Abnormalities in water homeostasis

Disorders of serum Na⁺ concentration





Hyponatremia

Hyponatremia

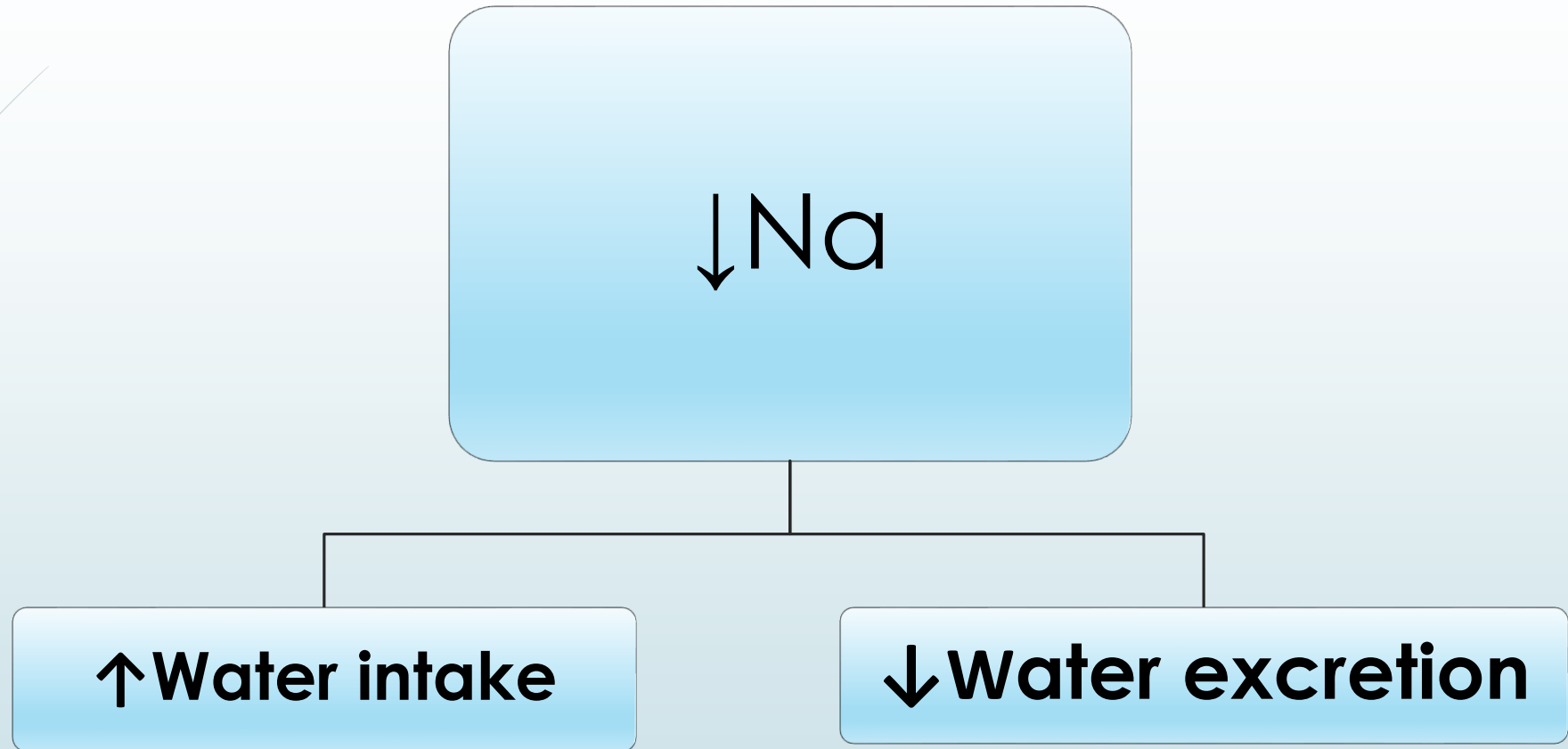


- Plasma Na^+ concentration <135 mmol/L usually reflects a hypotonic state.
- Most causes of hyponatremia are associated with a low plasma osmolality.

$$\text{Plasma osmolality} = 2 \times \text{Na} + \frac{\text{Glucose}}{18} + \frac{\text{BUN}}{2.8}$$

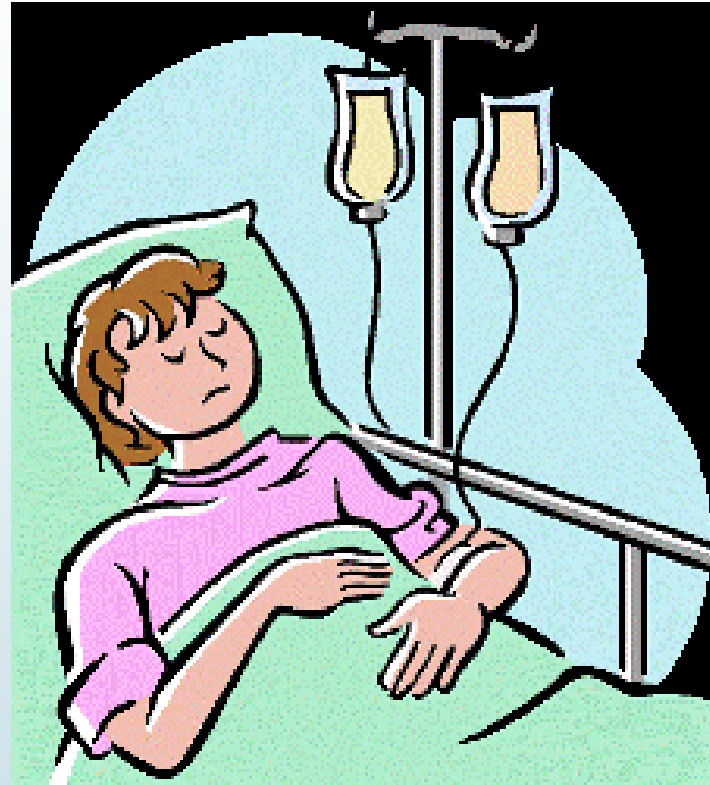


Causes of hyponatremia



Hypotonic intravenous fluids

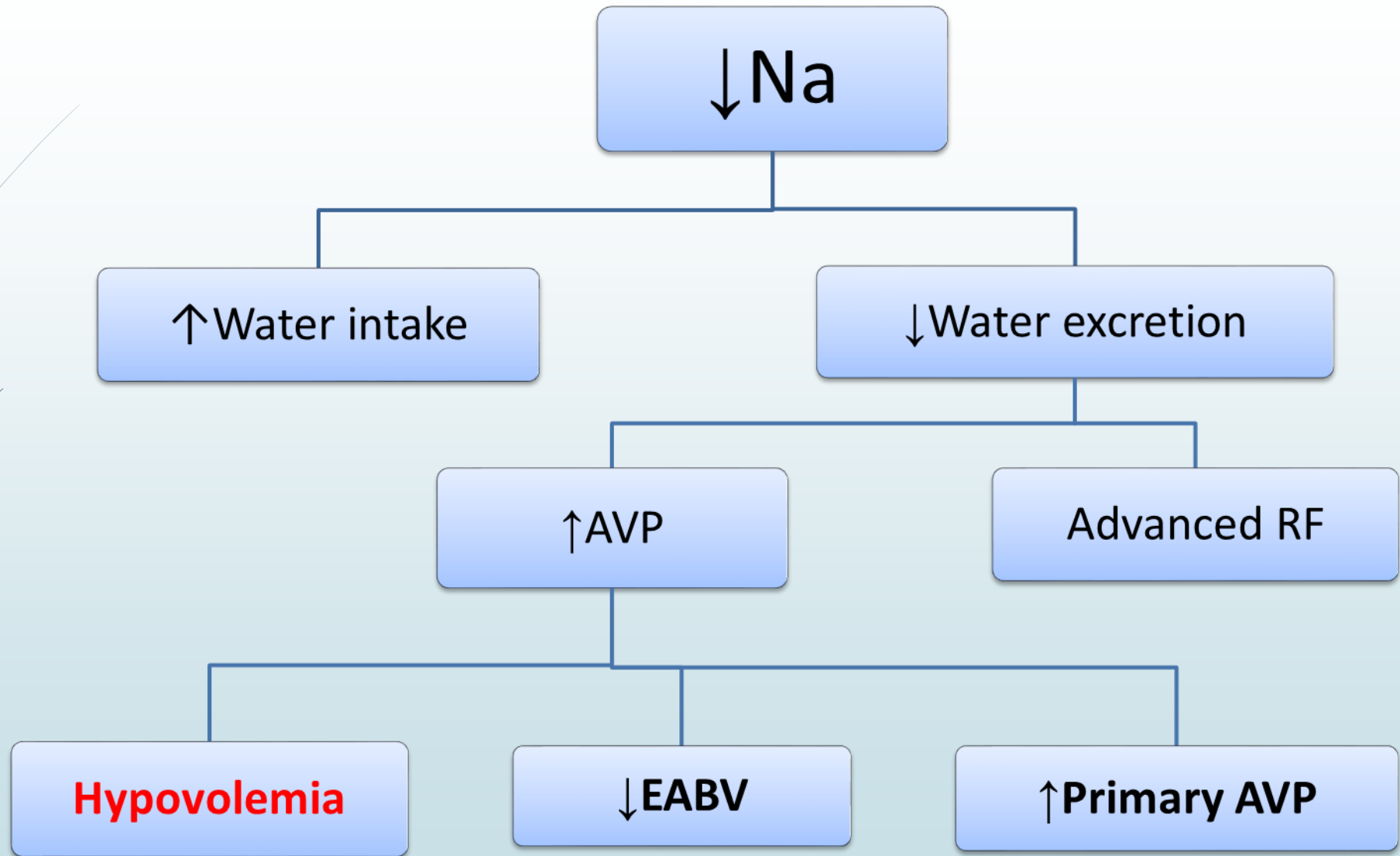
↑Water intake



↓AVP



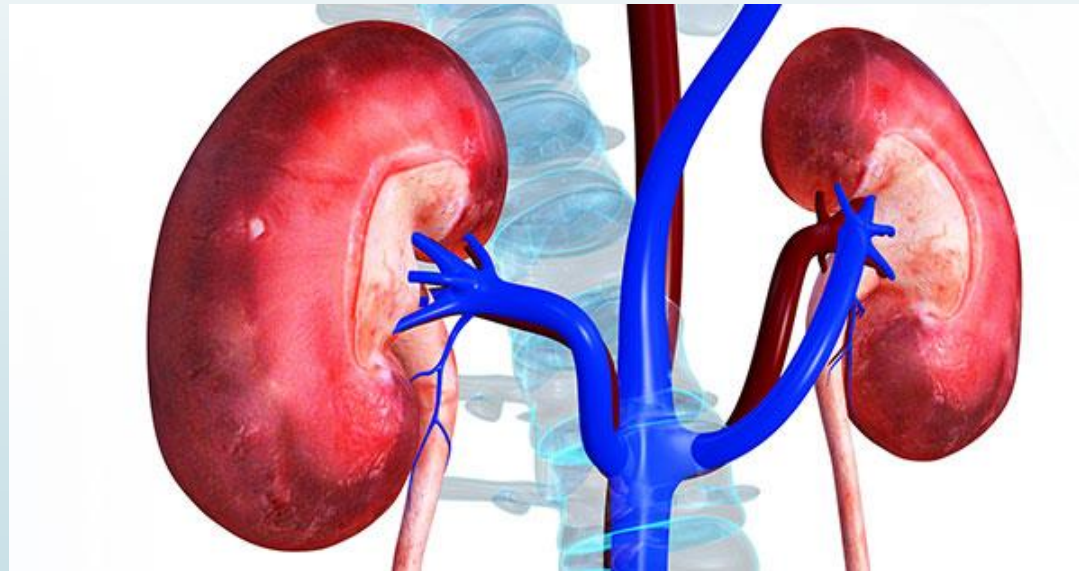
↓U osmolality < 100



Advanced Renal Failure

↓ Water excretion

- ▶ The impairment in free water excretion in advanced renal failure can lead to the retention of ingested water and the development of hyponatremia.





Increased secretion of AVP

↓Water excretion

- ▶ Hypovolemia
- ▶ ↓Tissue perfusion(↓EABV)
- ▶ A primary ↑ADH

Hypovolemia

↑AVP

↑water
reabsorption

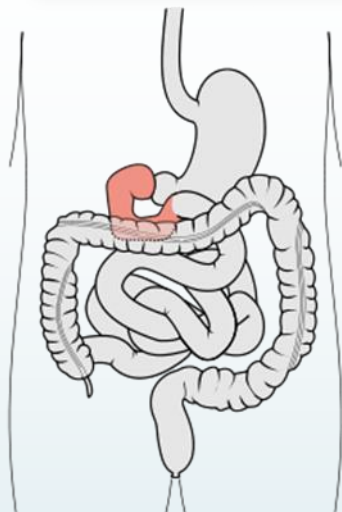
If ↑free
water intake

Hyponatremia



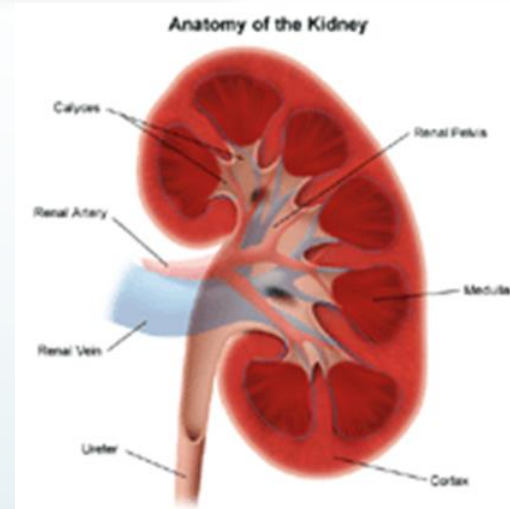
↓Na due to Hypovolemia

18



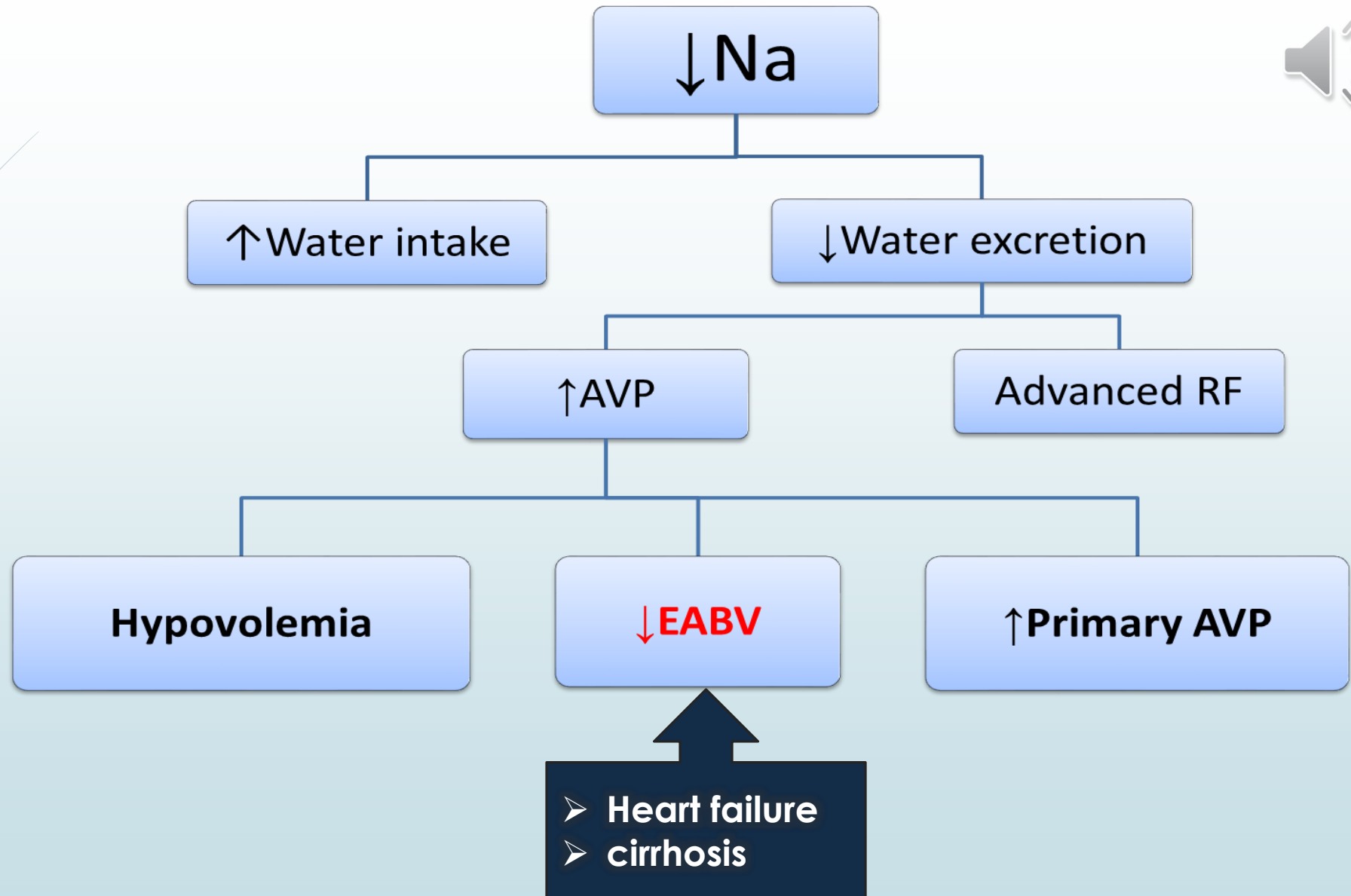
- Vomiting
- Diarrhea

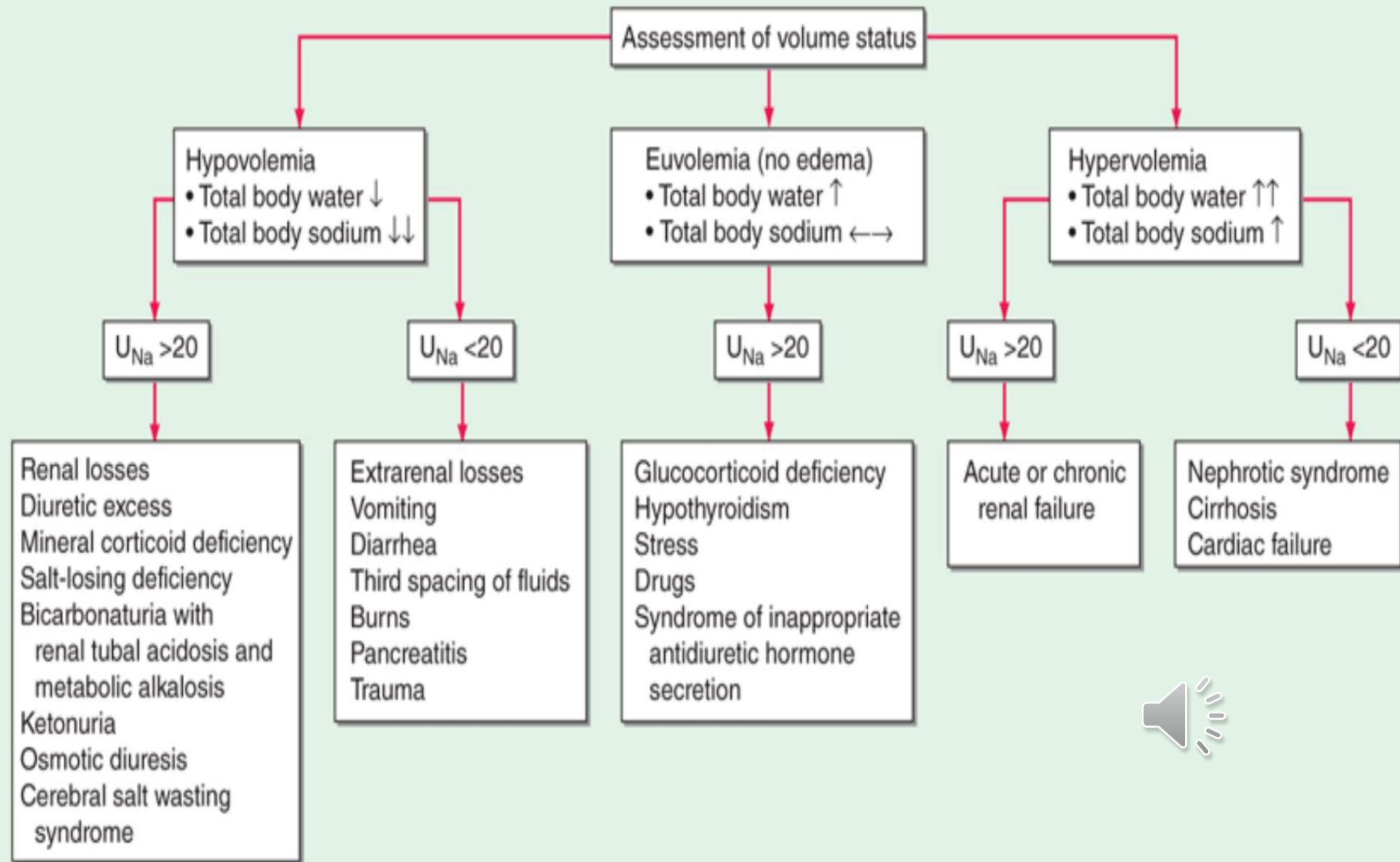
Urine Na < 20



- ↓Aldosterone
- Thiazide diuretics

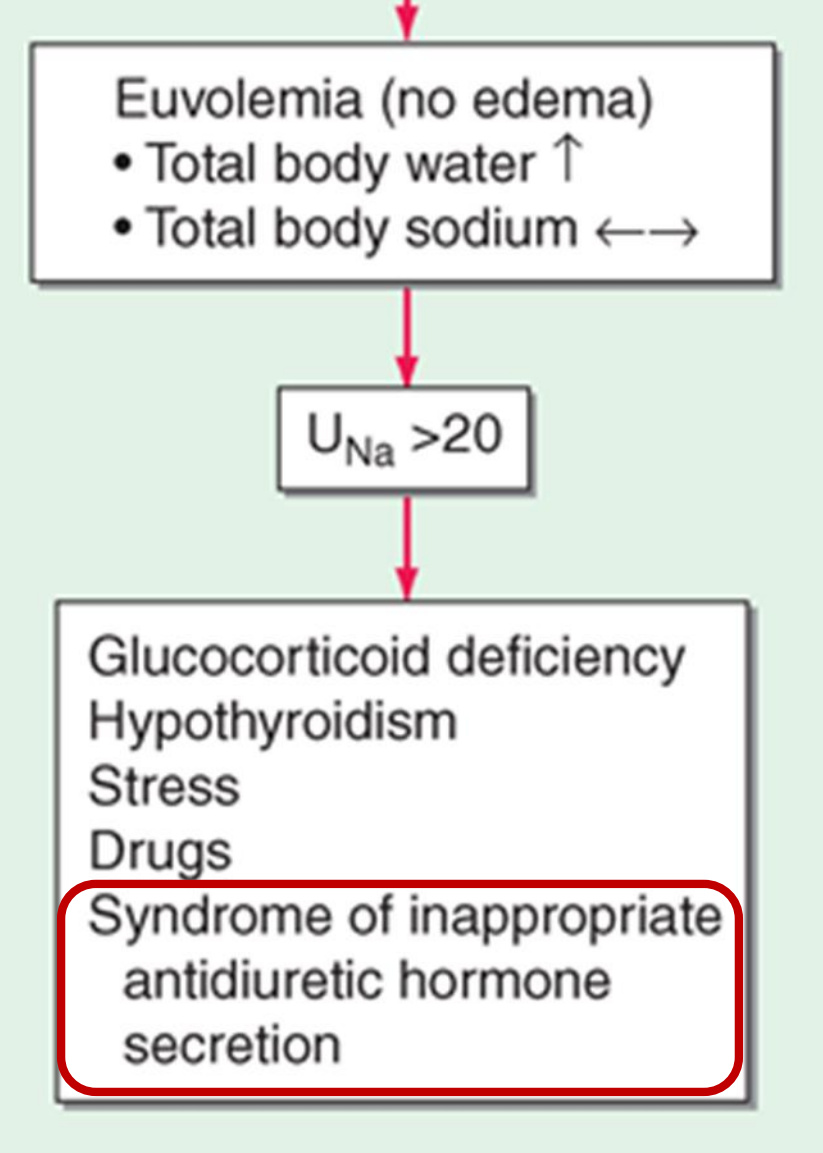
Urine Na > 20





Source: D. L. Kasper, A. S. Fauci, S. L. Hauser, D. L. Longo, J. L. Jameson, J. Loscalzo: Harrison's Principles of Internal Medicine, 19th Edition
www.accessmedicine.com

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```
graph TD; A["Euvoemia (no edema)  
• Total body water ↑  
• Total body sodium ↔"] --> B["UNa >20"]; B --> C["Glucocorticoid deficiency  
Hypothyroidism  
Stress  
Drugs  
Syndrome of inappropriate antidiuretic hormone secretion"]; style C stroke:#f00,stroke-width:2px
```

Euvoemia (no edema)

- Total body water ↑

- Total body sodium ↔

$U_{Na} > 20$

Glucocorticoid deficiency

Hypothyroidism

Stress

Drugs

Syndrome of inappropriate antidiuretic hormone secretion



SIADH


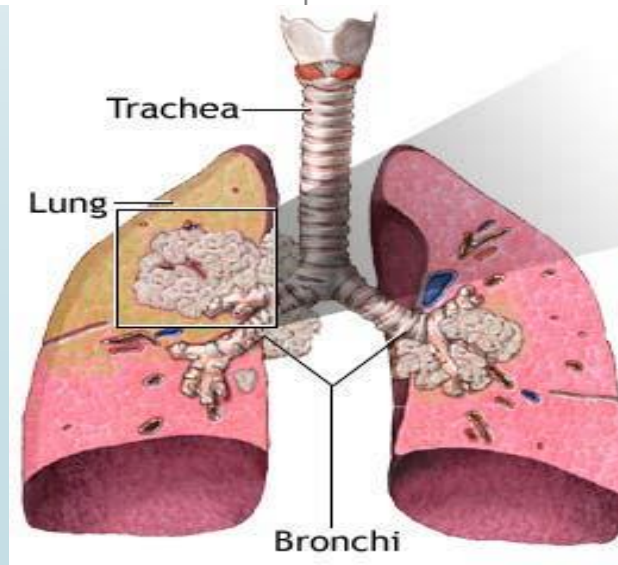
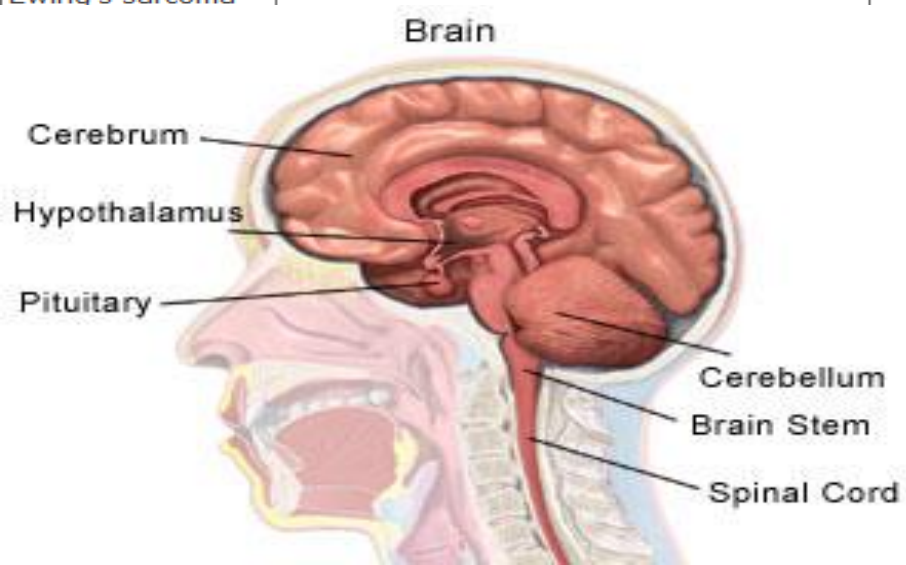
- The osmotic threshold and osmotic response curves for the sensation of thirst are shifted downward in patients with SIADH. 
- Hyponatremia in patients with SIADH is primarily due to the intake of water that is not excreted.

Table 45-1 Causes of the Syndrome of Inappropriate Antidiuresis

Malignant Diseases	Pulmonary Disorders	Disorders of the Central Nervous System	Drugs	Other Causes
Carcinoma	Infections	Infection	Drugs that stimulate release of AVP or enhance its action	Hereditary (gain-of-function mutations in the vasopressin V ₂ receptor)
Lung	Bacterial pneumonia	Encephalitis	Chlorpropamide	Idiopathic
Small cell	Viral pneumonia	Meningitis	SSRIs	Transient
Mesothelioma	Pulmonary abscess	Brain abscess	Tricyclic antidepressants	Endurance exercise
Oropharynx	Tuberculosis	Rocky Mountain spotted fever	Clofibrate	General anesthesia
Gastrointestinal tract	Aspergillosis	AIDS	Carbamazepine	Nausea
Stomach	Asthma	Bleeding and masses	Vincristine	Pain
Duodenum	Cystic fibrosis	Subdural hematoma	Nicotine	Stress
Pancreas	Respiratory failure associated with positive-pressure breathing	Subarachnoid hemorrhage	Narcotics	
Genitourinary tract		Cerebrovascular accident	Antipsychotic drugs	
Ureter		Brain tumors	Ifosfamide	
Bladder		Head trauma	Cyclophosphamide	
Prostate		Hydrocephalus	Nonsteroidal anti-inflammatory drugs	
Endometrium		Cavernous sinus thrombosis	MDMA (ecstasy)	
Endocrine		Other	AVP analogues	
thymoma		Multiple sclerosis	Desmopressin	
Lymphomas		Guillain-Barré syndrome	Oxytocin	
Sarcomas		Shy-Drager syndrome	Vasopressin	
Ewing's sarcoma				



Small cell carcinoma

Manifestations of hyponatremia



- ▶ The symptoms of hyponatremia are primarily **neurologic**, reflecting the development of cerebral edema within a rigid skull.
- ▶ Nausea and malaise, serum sodium concentration falls below **125 to 130** meq/L.
- ▶ Headache, lethargy, obtundation and eventually seizures, coma, and respiratory arrest can occur if the serum sodium concentration falls below **115 to 120** meq/L. Noncardiogenic pulmonary edema has also been described.

Manifestations of hyponatremia (continue)

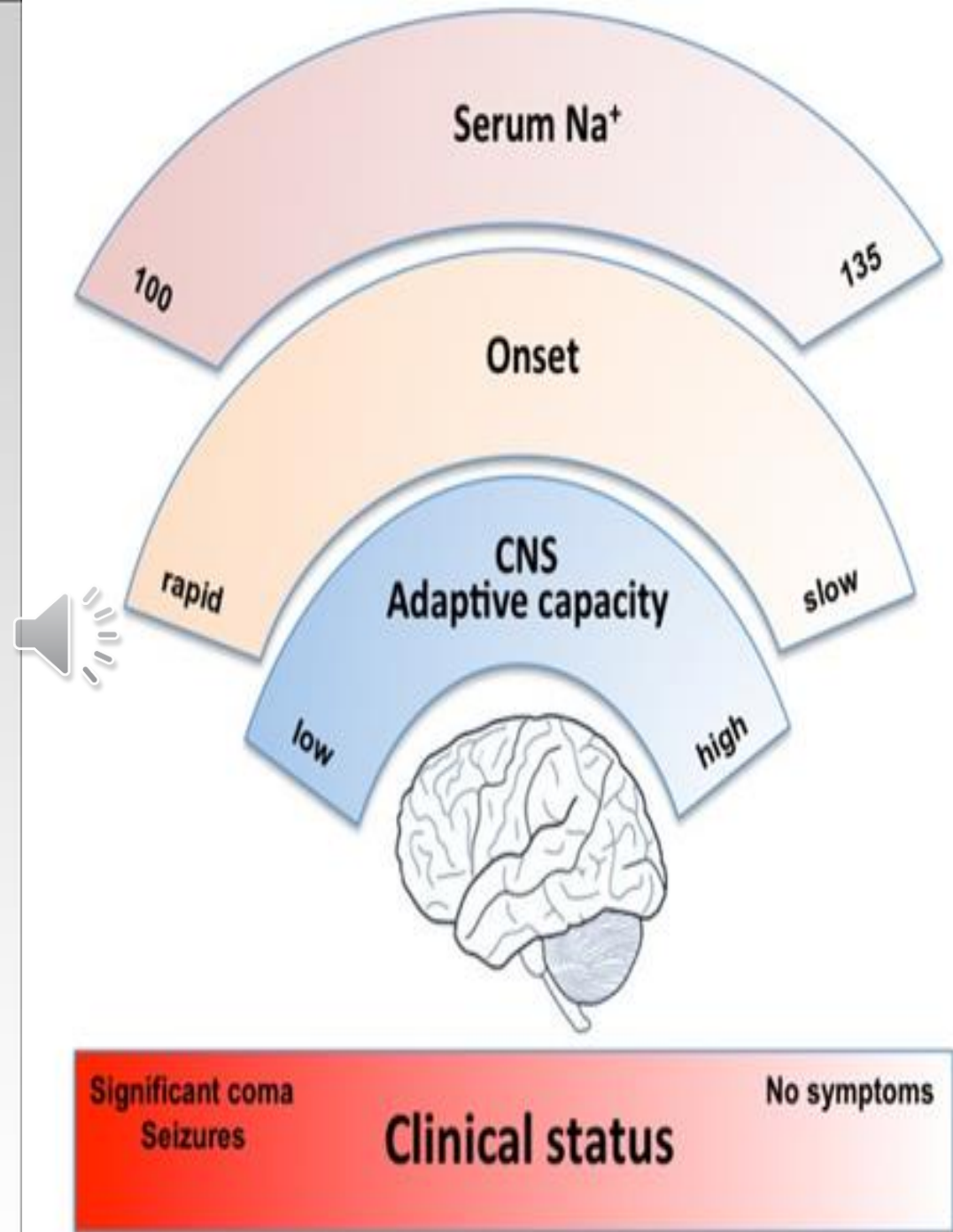
- ▶ The cerebral adaptation permits patients with chronic hyponatremia to appear to be asymptomatic.
- ▶ This reduction in intracellular osmolytes is largely complete within **48 hrs**, the time period that clinically defines chronic hyponatremia.
- ▶ Mild to moderate hyponatremia may contribute to fractures in elderly patients. Patients with hyponatremia are more likely to have osteoporosis than patients without hyponatremia.



Acute vs Chronic Hyponatremia

- Symptomatic but less impaired (usually chronic)
- Life-threatening (usually acute)

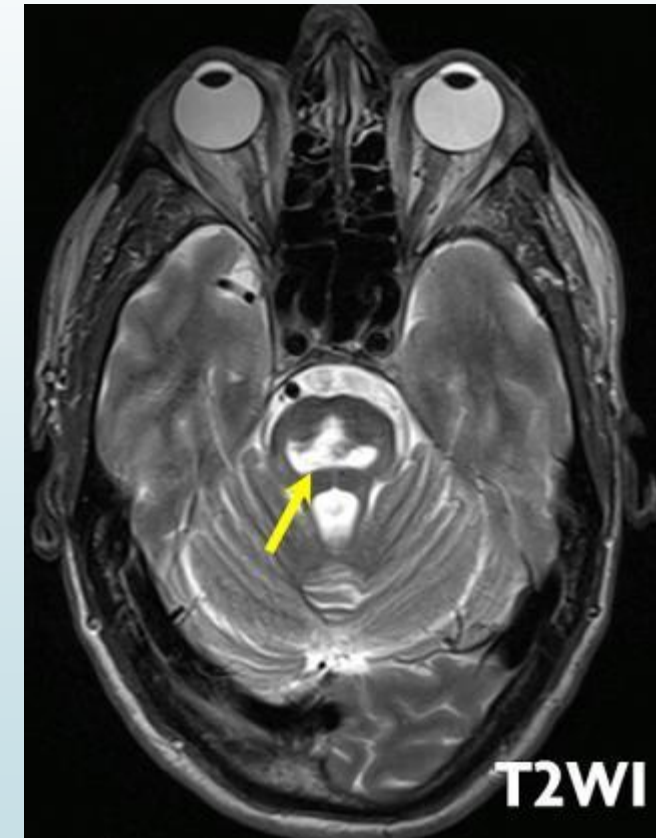
Acute (≤ 48 h)	Chronic (>48 h)
Symptoms include: <ul style="list-style-type: none"> • Cerebral edema • Seizures • Delirium • Increased mortality risk 	Symptoms include: <ul style="list-style-type: none"> • Nausea/vomiting • Confusion or personality changes • Fatigue • Headache • Neurological dysfunction • Gait disturbances • Seizures (with very low serum sodium levels)
Rapid correction reverses cerebral edema without sequelae	Rapid correction may cause brain dehydration and osmotic demyelination syndrome



Osmotic Demyelination Syndrome(ODS)



- **Osmotic Demyelination Syndrome (ODS)** is associated with rapid correction of hyponatremia or fluid shifts, and is characterized by neurological involvement related to **pons**, brainstem or other areas of the brain. All possible measures should be taken to prevent this serious disorder.





Treatment

Emergency therapy

- ▶ Severe symptoms
 - ▶ seizures or obtundation
- ▶ Hyperacute
 - ▶ over just a few hours.

- ▶ a 4 to 6 meq/L increase in the Na should be achieved as soon as possible

100 mL of 3 percent saline given IV bolus

- ▶ If severe neurologic symptoms persist or worsen, or if the serum sodium is not improving, a 100 mL bolus of 3 percent saline can be repeated one or two more times at 10-minute intervals.

Non-emergency therapy

- ▶ **Severe hyponatremia (≤ 120 meq/L):**
 - ▶ A slow IV hypertonic saline at 15 to 30 mL/hour,
 - ▶ Correction rate of 6 meq/L per day.
- ▶ **Asymptomatic patients with acute* hyponatremia:**
 - ▶ IV hypertonic saline (50 mL over 10 minutes).
 - ▶ Two or three additional boluses of 50 to 100 mL of hypertonic saline can be given if symptoms develop and/or the serum sodium does not improve.
- ▶ **Should generally receive hypertonic saline.**
- ▶ **the total elevation in serum sodium should be 4 to 6 meq/L**

**Hyponatremia developed within the previous 24 hours.*

Calculation of Na⁺ deficit

- Na⁺ deficit = $0.6 \times \text{Wt.} \times (\text{target-plasma Na}^+)$

Hypertonic saline

Indications	3 % saline	Rate
Severe symptoms	100 mL IV bolus	4 to 6 meq/L increase as soon as possible
acute* hyponatremia	50 mL over 10 minutes	total elevation 4 to 6 meq/L
≤120 meq/L	A slow IV hypertonic saline at 15 to 30 mL/hour	Correction rate of 6 meq/L per day

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Rate of Correction

- ▶ Treatment of acute symptomatic hyponatremia should include hypertonic 3% saline (513 mM) to acutely increase plasma Na⁺ concentration by 1–2 mM/h to a total of 4–6 mM.
- ▶ Every effort should be made to keep the rise in serum sodium less than 9 meq/L in any 24-hour period.



Goal of Therapy

- ▶ The goal of therapy should not be a predefined serum sodium level, as this will lead to overcorrection of hyponatremia when the serum sodium concentration is extremely low.
- ▶ Small (4 to 6 meq/L) increases in the serum sodium concentration are sufficient; larger increases offer no therapeutic advantage and only increase the risk of osmotic demyelination.

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Fluid restriction

- ▶ For the treatment of symptomatic or severe hyponatremia in edematous states (such as heart failure and cirrhosis), SIADH, and advanced renal impairment.
- ▶ In general, fluid intake should be less than 800 mL/day.

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Hypernatremia

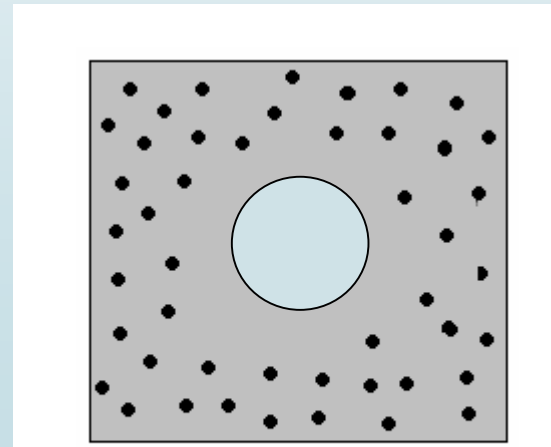
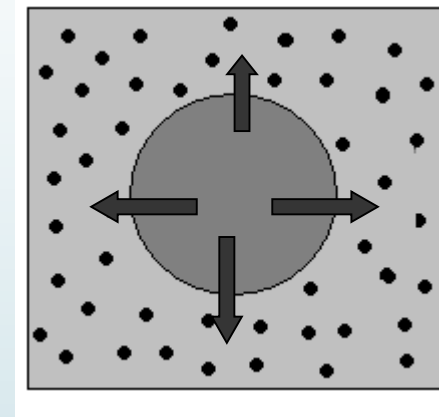
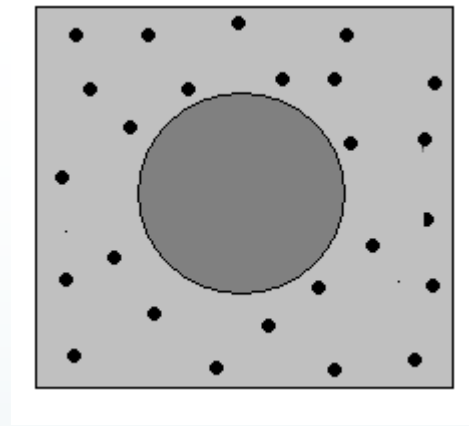
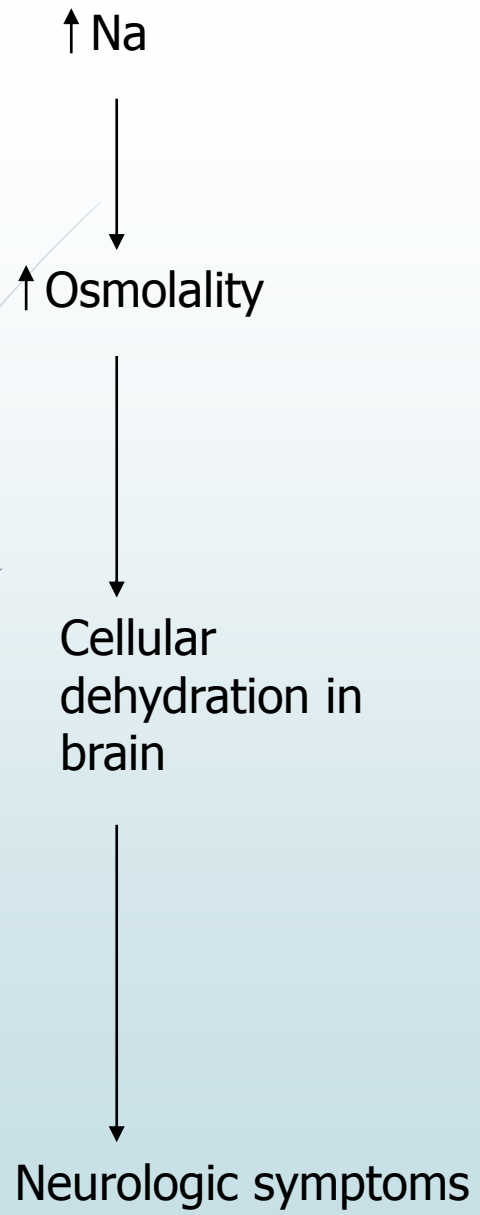
Ηλβενατρεμία

Hypernatremia



- ▶ Plasma Na^+ concentration >145 mmol/L
- ▶ Hypernatremia = hyperosmolality

Fixed number of ICF particles \rightarrow \downarrow ICF Volume



Response to \uparrow Na:



➤ Thirst

➤ AVP

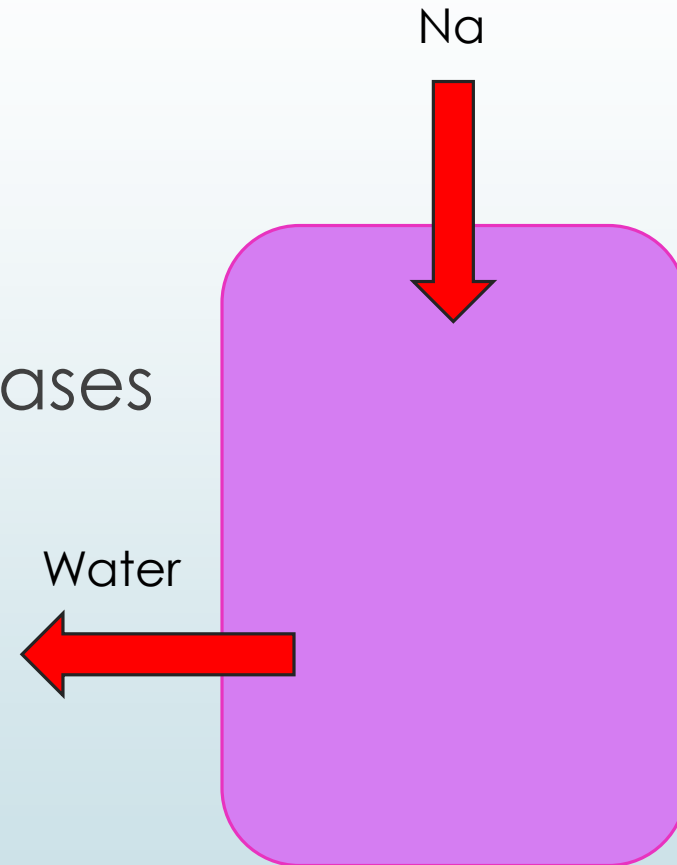
↳ \downarrow U vol + \uparrow U osm

Etiology

► Water loss:

→ The majority of cases

► Sodium gain



Etiology

Sodium gain:

- Hypertonic NaCl
- NaHCO₃
- Ingestion of sodium

Na



Water loss:

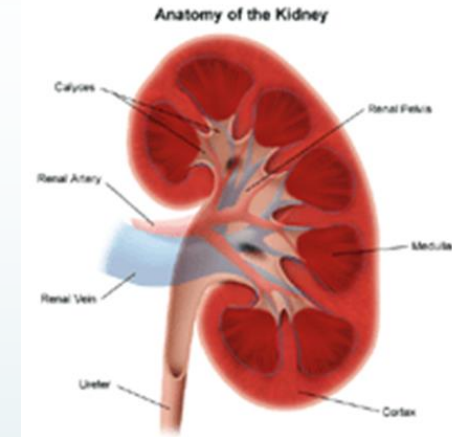
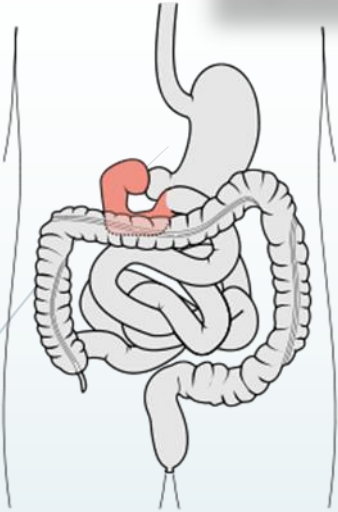
- Insensible losses
- GI tract
- Renal water loss

Water



↑Na due to Water loss

43



Osmotic diarrheas :

- Lactulose, sorbitol
- Malabsorption of carbohydrate

- Fever
- Exercise
- Heat exposure

- DI
- Osmotic diuresis:
 - poorly controlled DM

↑ Urine Osmolality

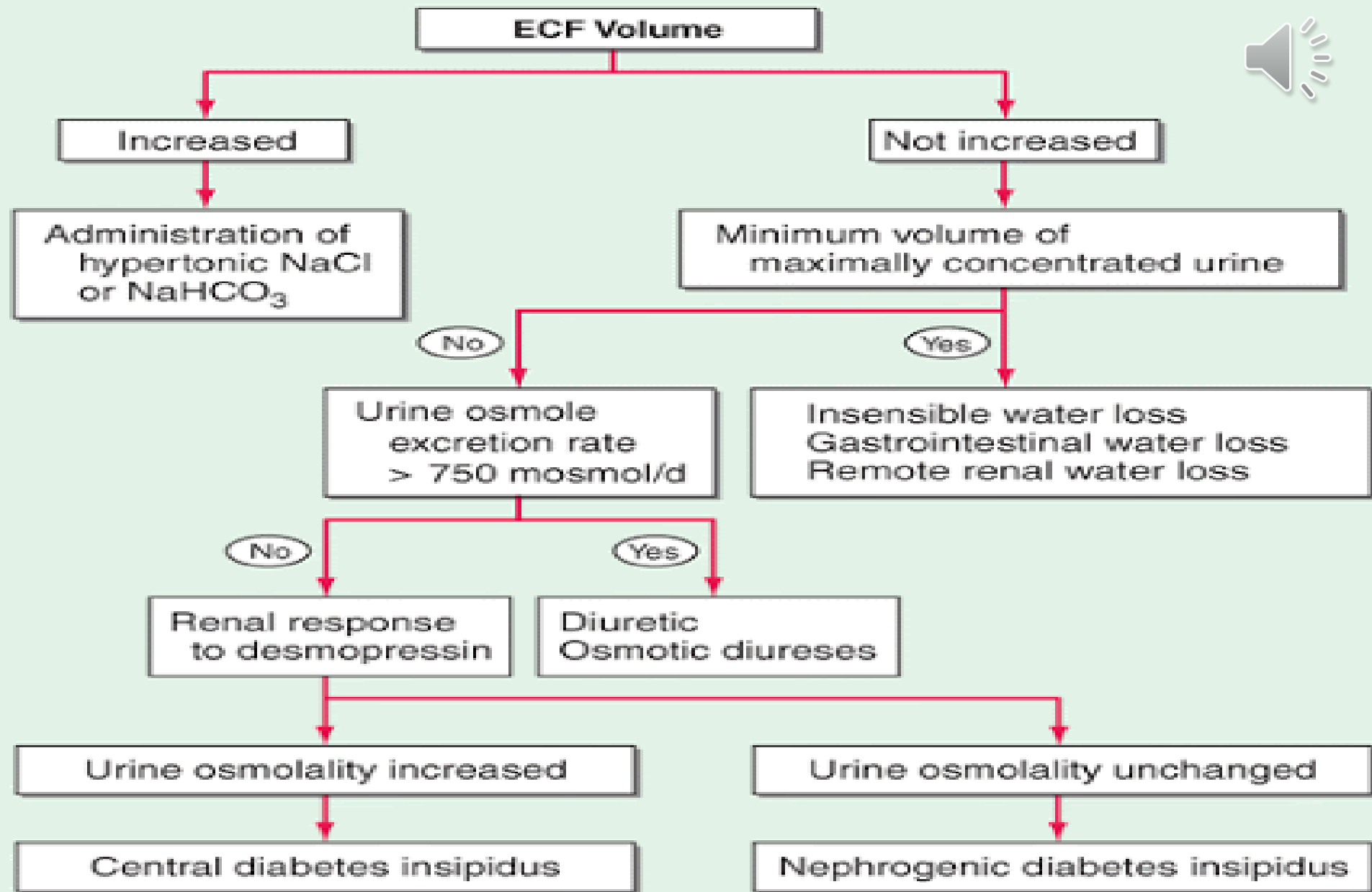
↓ Urine Osmolality



Approach To Hybernatriemia

Approach To Hybernatriemia

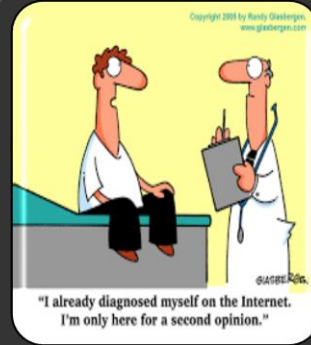
Approach to hypernatremia



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

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Approach to hypernatremia



- Diarrhea & vomiting
- Polyuria
- Fever
- Salt intake



- Hyper & hypovolemia



- $U/A \Rightarrow SG = 1.010$
($U_{osm} = 10 * 35 = 350$)

CLINICAL FEATURES



The major symptoms of hypernatremia are neurologic and include altered mental status, weakness, neuromuscular irritability, focal neurologic deficits, and occasionally coma or seizures.

A decreased brain cell volume is associated with an increased risk of subarachnoid or intracerebral hemorrhage.

Osmotic damage to muscle membranes can lead to hypernatremic rhabdomyolysis.

Remember

*Persistent hypernatremia should not occur
in patients who are alert, have an intact
thirst mechanism, and have access to*

water.





Treatment Of Hybernatriemia

Treatment Of Hybernatriemia



Treatment of Hypernatremia

Correct hypernatremia slowly to avoid cerebral edema

Typically replacing the calculated free water deficit over 48 h

The plasma Na^+ concentration should be corrected by no more than 10 mM/d



TABLE 45-3 Management of Hyponatremia

Water Deficit

1. Estimate total-body water (TBW): 50% of body weight in women and 60% in men
2. Calculate free-water deficit: $\{([Na^+] - 140)/140\} \times TBW$
3. Administer deficit over 48–72 h, without increasing the plasma Na^+ concentration by >10 mM/24 h

Ongoing Water Losses

4. Calculate electrolyte-free water clearance, $C_e H_2O$:

$$C_e H_2O = \frac{V(1 - U_{Na} + U_K)}{P_{Na}}$$

where V is urinary volume, U_{Na} is urinary $[Na^+]$, U_K is urinary $[K^+]$, and P_{Na} is plasma $[Na^+]$

Insensible Losses

5. ~ 10 mL/kg per day: less if ventilated, more if febrile

Total

6. Add components to determine water deficit and ongoing water loss; correct the water deficit over 48–72 h and replace daily water loss. Avoid correction of plasma $[Na^+]$ by >10 mM/d

Calculation of Water Deficit

In case of sodium and water deficit

$$\text{Water Deficit} = \frac{(\text{Plasma Sodium} - 140)}{140} \times \text{TBW}$$

- Ongoing losses (insensible, renal) need to be added.



Treatment of Hypernatremia

- ▶ Safest route of administration of water is by mouth or via a nasogastric tube.
- ▶ 5% dextrose in water or half-isotonic saline can be given intravenously.

Treatment of Hypernatremia

► **CDI:**

- **Desmopressin**
- **low-salt diet + low-dose thiazide diuretic**
- **Stimulate AVP secretion or enhance its action:**
 - Chlorpropamide, clofibrate, carbamazepine, NSAIDs

► **NDI:**

- **Low-salt diet + low-dose thiazide diuretic**
- **NSAIDs**
- **Amiloride:**
 - NDI who need to be on lithium
 - The nephrotoxicity of lithium requires the drug to be taken up into collecting duct cells via the amiloride-sensitive Na⁺ channel.