

ELECTROLYTE DISTURBANCES IN MEDICINE

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OSMOLALITY AND VOLUME STATUS

* hyper osmolar or hyper tonic > 284.
* hypo osmolar or hypo tonic < 280.

Normal serum ^{tonicity} **osmolality** is usually 282 ± 2 mOsm/ kg H₂O. If no osmolal gap exists from an acid alcohol ingestion, then measured osmolality should be equal to calculated osmolality, which is (roughly):

if it in mg/dl we will ÷ 18 / but if it in osmol 2.8

$$\text{Osm}_{\text{calc}} = 2[\text{Na}^+] + (\text{glucose}/18) + (\text{BUN}/2.8)$$

$\text{BUN} = \frac{\text{urea}}{2.14}$

If glucose and **BUN** are normal, use $2 \times (\text{Na}^+)$ to quickly see if the patient's osmolality is 270.

4

Antidiuretic hormone (ADH) is the common term for arginine vasopressin (AVP). ADH is the primary regulator of renal water excretion; it is a neurohypophysial hormone that acts on the late distal tubule and cortical collecting duct to increase water permeability and mediate the urine concentration.

primary regulator of H₂O in the body.

ADH levels are regulated by several mechanisms. The 2 most important are:

1) **Osmoreceptors** in the hypothalamus

2) Volume (**stretch**) ^{Baroreceptor} receptors in the left atrium (and possibly in the pulmonary veins) and blood vessels ^{arch of aorta}

The strongest stimulant for ADH release is significant volume loss resulting in hypotension; e.g., hemorrhage. This stimulates both the **stretch receptors and baroreceptors**.

High plasma osmolality is a weaker but more sensitive stimulus for ADH release.

↑ osmolality, ↑ Hypotension → release ADH

لن يفرج أكثر

Baroreceptors Can Be Divided Into Two

a) High pressure Arterial Baroreceptors

Baroreceptors Found Within Aortic Arch

↓
Enable The Examination of Blood Being

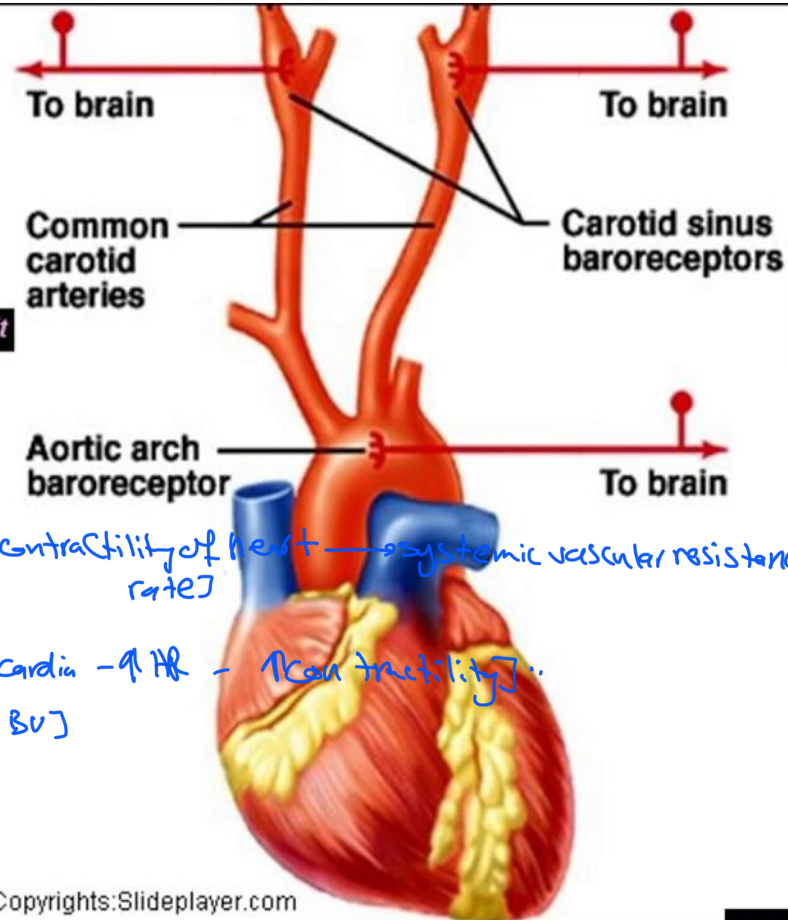
↓
Delivered to All Blood Vessels Via Systemic Circuit

Baroreceptors Within The Carotid Artries

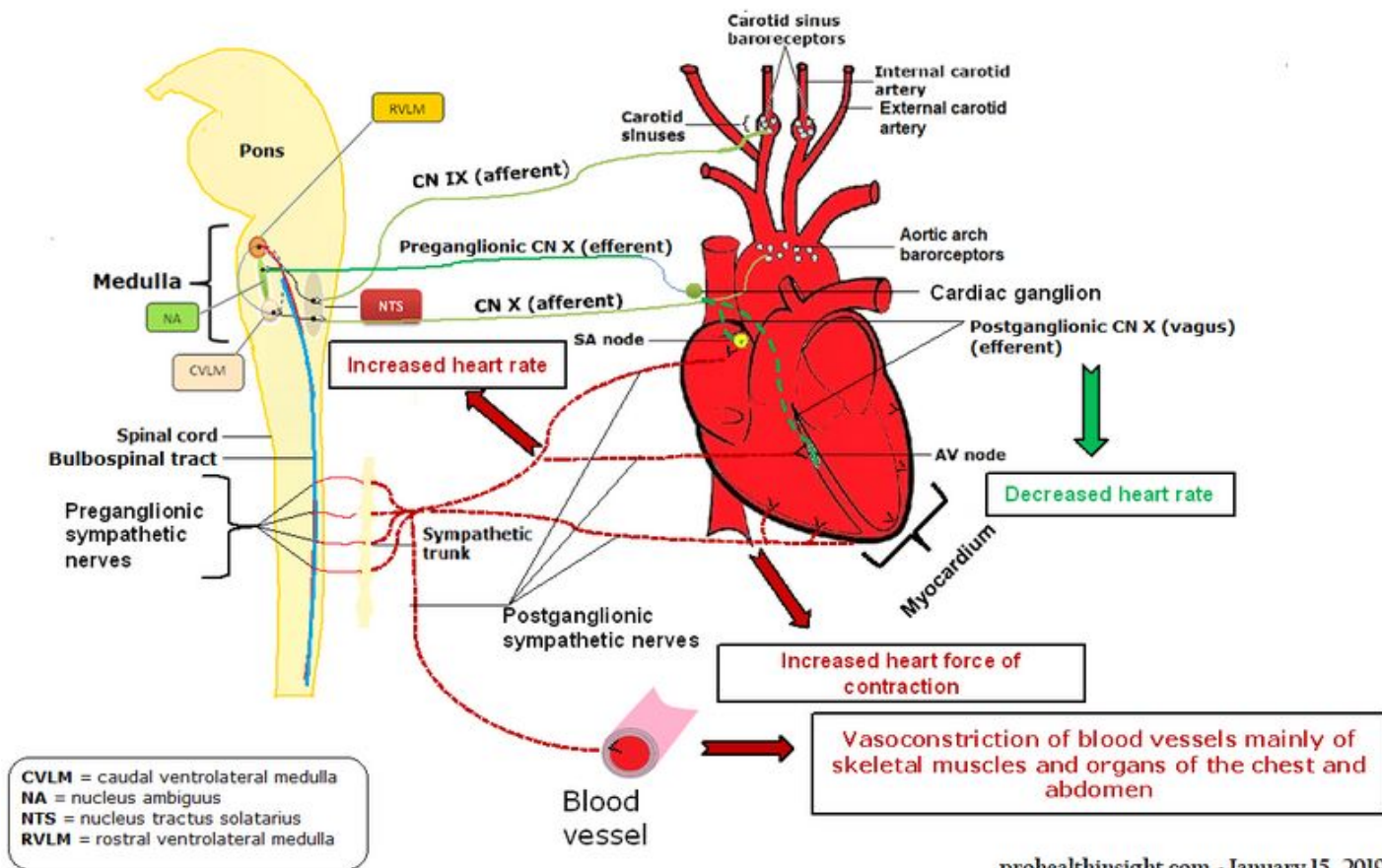
↓
Monitor Blood Pressure of Blood Being

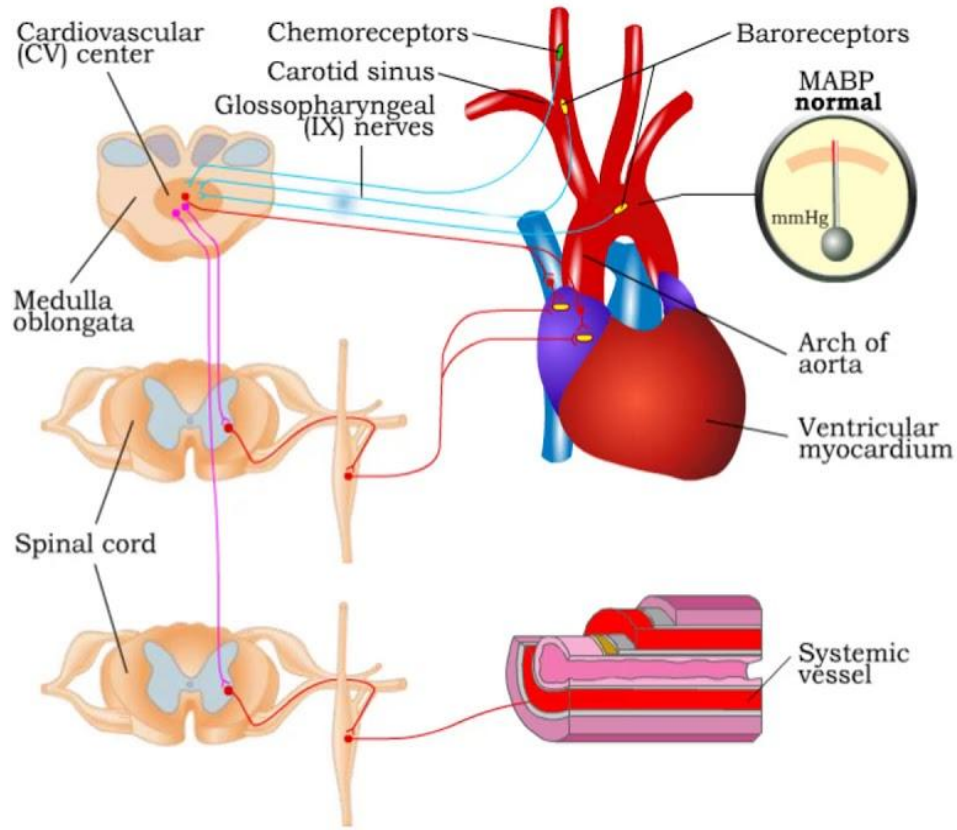
stimulation of Sympathetic: $\rightarrow \uparrow \text{Cap} + \text{VC} + [\uparrow \text{Contractility of heart} + \text{systemic vascular resistance rate}]$

Inhibition of paraSympathetic: $\rightarrow \uparrow \text{Cap} [\text{tachycardia} - \uparrow \text{HR} - \uparrow \text{Contractility}]$
[no effect in BV]



Arterial Baroreceptor Reflex Pathways





Neural regulation of blood pressure - baroreceptor reflexes

- Carotid sinus reflex helps maintain normal blood pressure in brain.
- Baroreceptors are stimulated by the changes in the stretch of the carotid sinus wall; nerve impulses propagate from the baroreceptors over glossopharyngeal nerves to the CV center in the medulla oblongata.
- Aortic reflex maintains general systolic blood pressure.

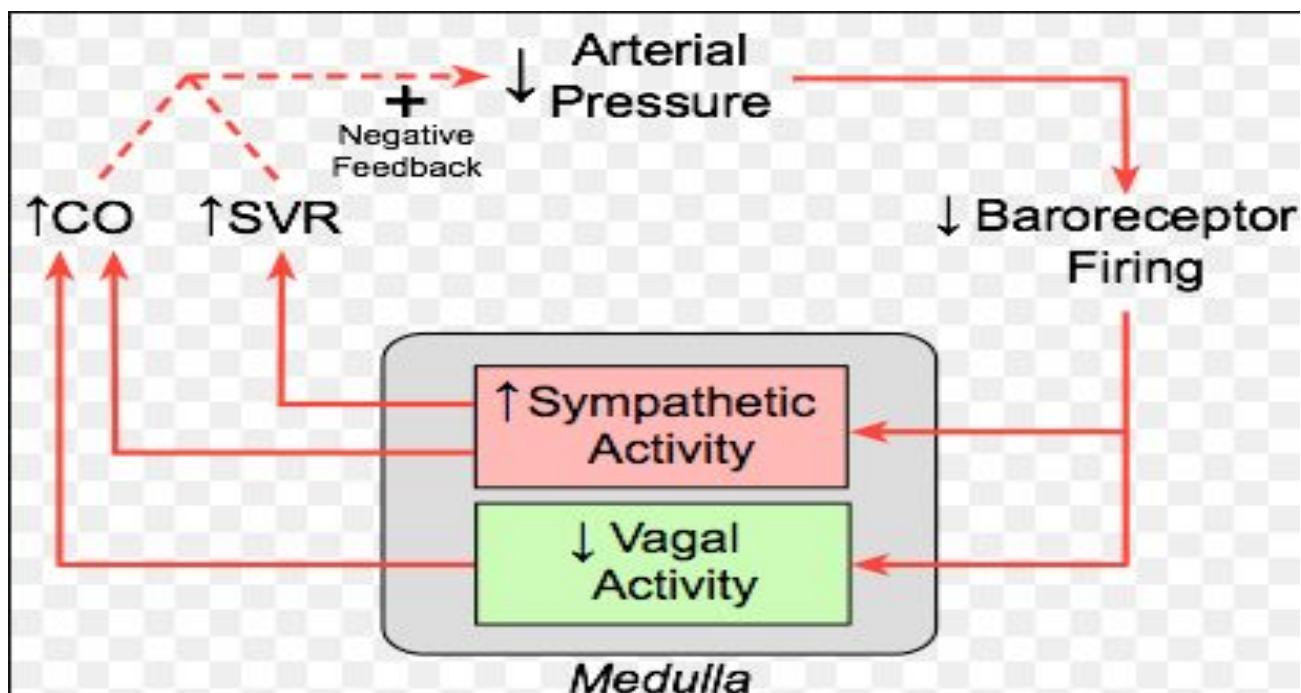
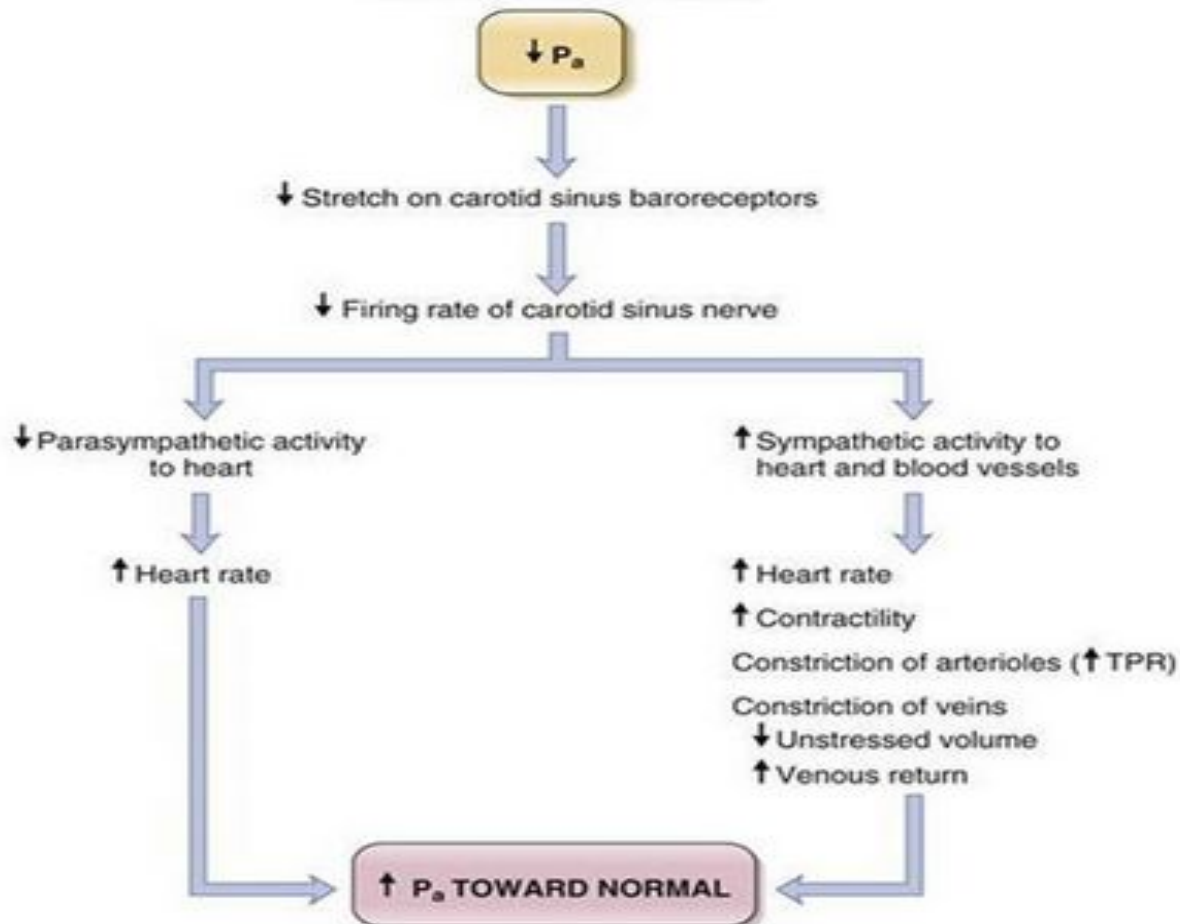


Figure 3. A sudden decrease in arterial pressure decreases baroreceptor firing, which activates sympathetic neurons and inactivates vagal neurons in the medulla. The resulting increases in CO and SVR act as a negative feedback mechanism to attenuate the fall in arterial pressure.

BARORECEPTOR REFLEX



input

Imposed decrease in
arterial blood pressure



Sensed by arterial baroreceptors
as decreased stretch



Decreased baroreceptor
afferent nerve activity



processing

Signal processed by medullary
cardiovascular control pathways



output

Increased sympathetic activity

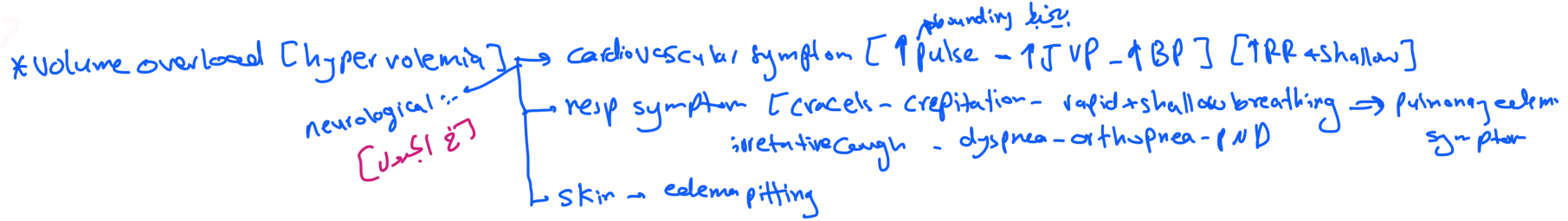
- Increased heart rate
- Increased force of heart contraction
- Arteriolar vasoconstriction

Decreased parasympathetic activity

- Increased heart rate



Increased arterial blood pressure



Volume status of the patient with a sodium abnormality is critical in determining the treatment. In general, if the patient is edematous, there is volume overload. If the patient has the clinical signs of volume loss, there is a volume deficit. If there is neither of these, the patient is considered euvolemic.

Remember these clinical clues to hypovolemia: tachycardia, narrowed pulse pressure, orthostatic hypotension, and resting tachycardia with hypotension. Central venous pressure is low.

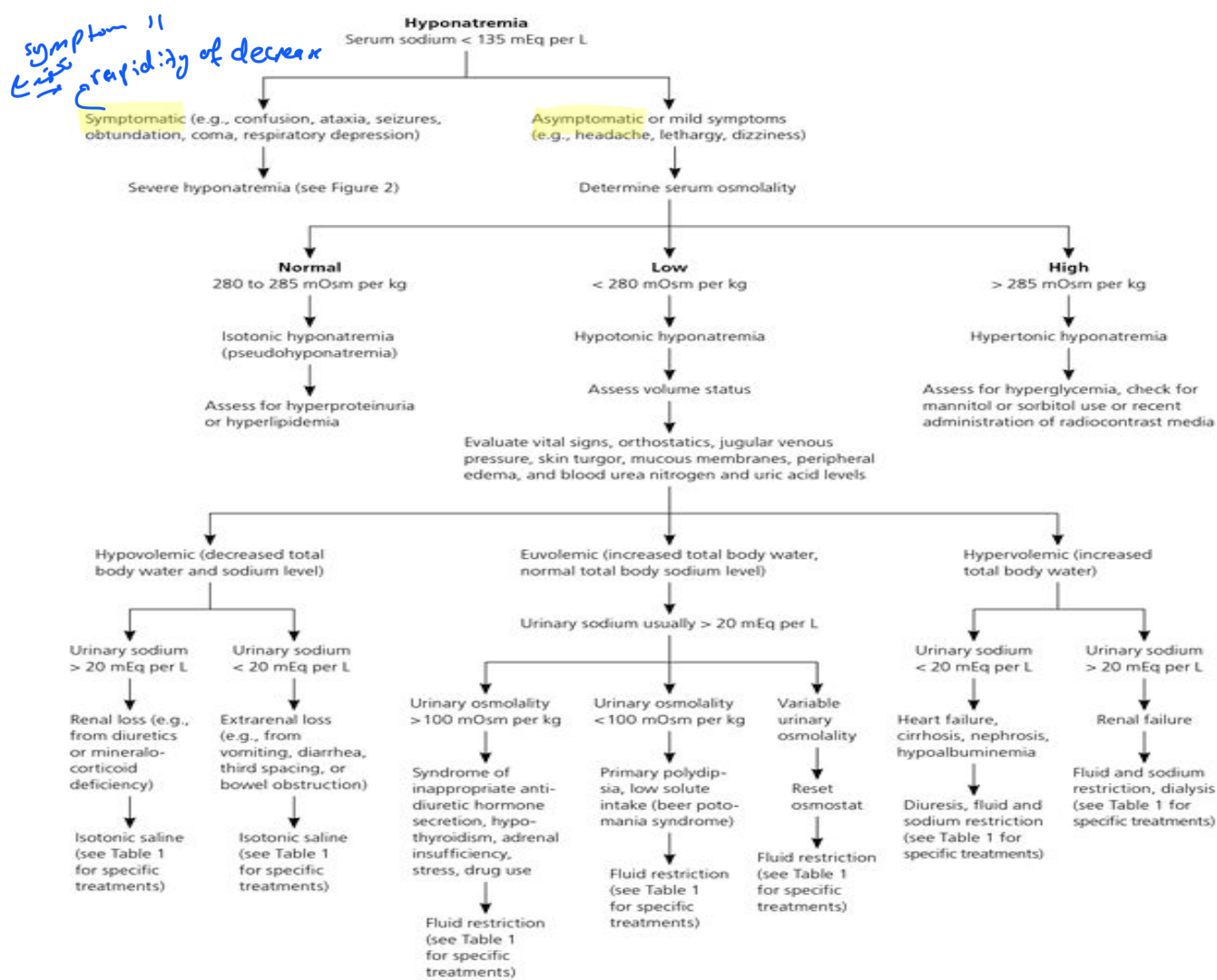
ECF Volume Excess: Hypervolemia *symptoms*

Cardiovascular Changes	Respiratory Changes	Skin Changes	Neurologic Changes	Other Changes
○ ↑ Pulse: full and bounding ○ Full peripheral pulses ○ Distended neck veins ○ ↑ BP	○ ↑ respiratory rate ○ Shallow respirations ○ ↑ dyspnea with exertion or in the supine position ○ Pulmonary congestion and pulmonary edema ○ SOB ○ Irritative cough ○ Moist crackles	○ Edematous may feel cool ○ Skin may feel taut and hard ○ Edema-eyelids, facial, dependent (sacrum), pitting, peripheral extremities 	○ Altered LOC ✓ ○ Visual ✓ disturbances ○ Skeletal muscle weakness ○ Parenthesis ✓ ○ Cerebral ✓ edema ○ Headache ○ Confusion ○ Lethargy ○ Diminished reflexes ○ Seizures, coma	○ Urine: polyuria, nocturia ○ Lab data ↓Hematocrit, BUN GI Changes ○ Increased motility ○ Enlarged liver

المريض
 TSVP
 L edema
 crepitation
 ascites
 pleural effusion

Image 4: Signs and symptoms of Hypervolemia

HYPONATREMIA



mc electrolyte disturbance
in medicine

HYPONATREMIA

① determine the osmolarity

Isoosmolar and Hyperosmolar

Low serum Na^+ is the most common electrolyte abnormality. * osmolarity:- measured [↑ protein] as in multiple myeloma.

- **Isoosmolar hyponatremia**: An artifactual decrease in the serum Na^+ associated with older lab instruments that miscalculated sodium in settings of **high protein** (e.g., myeloma) or **lipids**. The current standard in laboratory practice is to use ion-specific electrodes, thus eradicating this problem.

الارتفاع في البروتين
في الدم
hyponatremia

- **Hyperosmolar**: Both glucose and mannitol cause an osmotic shift of water out of cells, which dilutes plasma Na^+ . (elevated BUN)

+ hypernatremia.

Remember: For each 100 increase in glucose over 100 mg/dL, the sodium concentration decreases by 1.6.

Very high glucose $\rightarrow$ ↓ Na^+

كل 100 زيادة في الجلوكوز فوق 100 راجع ينزل Na^+ 1.6

Hypoosmolar

we should detect the volume -

isovolemic
hypovolemic [mc]
hypervolemic

we should detect the urine volume -

By far, the largest low- Na^+ subgroup is the hypoosmolar group. Note that the terms hypo osmolar and hypotonic are interchangeable.

The low osmolality causes water movement into cells, leading to intracellular swelling, which may result in neuromuscular excitability, seizures, and coma-usually when the Na^+ falls acutely (< 120). If the sodium level decreases slowly, the cells re-equilibrate and do not swell enough to cause these symptoms. The hypoosmolar group is further subdivided by volume status: low, high, and normal.

** 3rd space loss:- present of fluid in the compartment that not share in circulating system as:-*
pleural cavity
peritoneal cavity
pericardial cavity.
[due to burn - pancreatitis]
..etc

** extra renal loss:- HF - liver cirrhosis - hypoalbuminemia - lymphosis..*

* SIADH: Syndrome of inappropriate anti diuretic hormones...

From cells $\downarrow$ Na⁺ $\rightarrow$ $\downarrow$ Na⁺ serum $\rightarrow$ called dilutional hyponatremia $\leftarrow$ (118) $\rightarrow$ Na⁺ 1,600 $\rightarrow$ ratio between the true Na⁺ and presented H₂O
 $\rightarrow$ lead to $\downarrow$ Na⁺
 true Na⁺: - 126 $\rightarrow$ $\delta = 1.6 \times 6 \leftarrow$ Na⁺ 1,600

Always think of the serum sodium concentration as the ratio of total body sodium to water, with increased total body water content as the key disorder in most cases of hyponatremia. Therefore, hypotonic hyponatremia is a water problem. Whenever the serum sodium is low, it means the patient has more water relative to total body sodium either from loss of sodium, true water excess, or total body sodium excess that is exceeded by water excess.

In the patient with hypotonic low Na⁺, the first thing to do is assess the volume status, which is done clinically.

Volume status essentially reflects total body sodium content.

→ due to vomiting - diarrhea - renal loss - diuretic

Low Osmolality ... Low Volume (Hypovolemia) measure the urine volume.

Low-volume patients have lost both water and Na^+ , but more Na^+ than water.

This has several causes:

- **Diuretics** + intestinal obstruction [renal loss]
 - GI losses (vomiting and diarrhea)
 - Third spacing of fluid
 - Adrenal insufficiency (Addison disease) ^{+ diuretic} → renal loss [urine volume < 20]
- } extra renal loss [urine volume > 20]

In primary adrenal insufficiency, both cortisol and aldosterone are deficient.

The low aldosterone causes renal Na^+ wasting and decreased K^+ and H^+ excretion, resulting in hypovolemia (sometimes with hypotension), hyperkalemia, and metabolic acidosis. The low cortisol stimulates ADH production, resulting in hyponatremia.

measure the urine volume

Low Osmolality ... High Volume (Hypervolemia)

High-volume hyponatremic patients usually retain water and Na^+ , but more water than Na^+ . These patients have dependent edema and JVD.

Causes of low osmolality, high-volume status include:

- Edema-forming states: heart failure, cirrhosis, and nephrotic syndrome - *hypoproteinemia*
- Kidney failure: acute or chronic
 - renal loss*
 - extra renal loss*

Normal treatment is restriction of water and Na^+ .

Lithium and demeclocycline can improve hyponatremia by inducing tubular resistance to ADH, but should not be used routinely because they can lead to chronic kidney disease. Also, avoid chronic, oral thiazide diuretics because they impair urinary-diluting ability, which can worsen hyponatremia. Loop diuretics can be very effective in patients with high-volume hyponatremia.

It's easy to precipitate acute kidney injury with rapid diuresis of a high-volume cirrhotic patient. Cirrhotics tend to have a low GFR, even with a normal serum creatinine.

They have decreased muscle mass, so a decrease in their GFR is not necessarily reflected in their serum creatinine concentration.

* ↓ urine osmolarity + euovolemia ⇒ (مريض طبيعي بحال)
 psychogenic - -::: urine راد serum
 primary polydipsia..

measure urine osmolarity. $\begin{matrix} > \text{low SIADH} \\ < \text{low polydipsia} \end{matrix}$

Low Osmolality ... Normal Volume (Euovolemia)

① SIADH euovolemic hyponatremic hyponatremia + ↑ADH [concentrated urine]

Normal-volume patients usually have SIADH or are taking drugs that either mimic ADH or cause ADH release.

Know that pain and chronic nausea are potent natural stimulators of ADH release.

Normal-volume hyponatremia also can be caused by psychogenic polydipsia (but in a patient with normal renal function and solute intake, it takes over 15 L/day of water intake to produce hyponatremia), hypothyroidism, and isolated glucocorticoid deficiency.

Causes of SIADH include the following:

- ✓ CNS disease (e.g., meningitis), infection, encephalitis
- ✓ Lung disease (e.g., pneumonia) → release ADH → SIADH..
- ✓ Neoplasms (especially small cell lung cancer)
- ✓ Drugs

↪ release ADH - ACTH
SIADH ↪ causing.

* pt take (cilegiline) lead to hyponatremia
* fenytoin → endless side effect...

Most common drugs associated with SIADH:

- NSAIDs ✓ *morphine - antibiotic given*
- SSRIs ✓
- Carbamazepine and oxcarbazepine ✓ *degretol → treat the Diabetes insipidus... + partial convulsion*
- Psychotropic drugs: haloperidol, amitriptyline [tricyclic anti depressant]
- IV cyclophosphamide ✓
- Vincristine and Vinblastine ✓
- Cisplatin ✓ *for hematuria treat the Diabetes insipidus...*
- Chlorpropamide (now rarely used)
- Ecstasy (methylenedioxymethamphetamine)

Drugs (Enhance ADH release or effect)

Anti-diabetic drugs

Chlorpropamide

Anti epileptic drugs

Carbamazepine, oxcarbazepine

Sodium valproate

Selective serotonin reuptake inhibitors (eg, fluoxetine, sertraline)

Anti cancer drugs

Vincristine, vinblastine, vinorelbine

Cisplatin, thiothixene

Melphalan, ifosfamide, methotrexate

Cyclophosphamide- intravenous

Antipsychotic drugs

Thioridazine

Haloperidol

Amitriptyline

Monoamine oxidase inhibitors

Pain killers

Opiates

Nonsteroidal antiinflammatory agents

Exogenous hormone administration

Vasopressin, desmopressin (dDAVP) or oxytocin

Miscellaneous

Interferon-alpha, interferon-gamma

Bromocriptine

Amiodarone

Ciprofloxacin

High-dose imatinib

“Ecstasy” (methylenedioxymethamphetamine)^[17-20]

SIADH: Syndrome of inappropriate secretion of anti diuretic hormone

Diagnose SIADH by comparing the urine and serum osmolalities. The normal response to hyponatremia would be to excrete free water in the urine; e.g., healthy patients would have a low serum osmolality and a low urine osmolality ($< 200 \text{ mOsm/L}$) because they are making dilute urine; this is what you see in psychogenic polydipsia. In SIADH, however, the urine is inappropriately concentrated ($> 250 \text{ mOsm/L}$) in the setting of a low serum osmolality. The patients are not excreting free water, so the serum osmolality is low, but the urine osmolality is high.

The **mechanism for ADH release** in moderate-to-severe hypothyroidism is decreased cardiac output, which stimulates the carotid baroreceptors. Rule out hypothyroidism and glucocorticoid deficiency in all patients with hyponatremia **before making a diagnosis of SIADH** because both causes can have low serum osmolalities and high urine osmolalities-more commonly seen in SIADH. Sometimes you cannot tell the difference using these tests, so look for the hormone deficiencies in everyone first, before diagnosing SIADH.

Schwartz diagnostic criteria for SIADH

Decreased measured serum osmolality ($<275 \text{ mOsm/kg H}_2\text{O}$)

Clinical euvolemia

Urinary osmolality $>100 \text{ mOsm/kg H}_2\text{O}$

Urinary $[\text{Na}^+] >40 \text{ mmol/L}$ with normal dietary sodium intake

Normal thyroid and adrenal function.

Normal renal functions

Exclude use of diuretic agents within the week prior to evaluation

No hypokalemia, no acid base disorders

Supporting diagnostic criteria for SIADH

Serum uric acid $<4 \text{ mg/dL}$

Blood urea nitrogen $<10 \text{ mg/dL}$

Fractional sodium excretion $>1\%$; fractional urea excretion $>55\%$

Failure to improve or worsening of hyponatremia after 0.9% saline infusion

Improvement of hyponatremia with fluid restriction

SIADH: Syndrome of inappropriate secretion of anti diuretic hormone

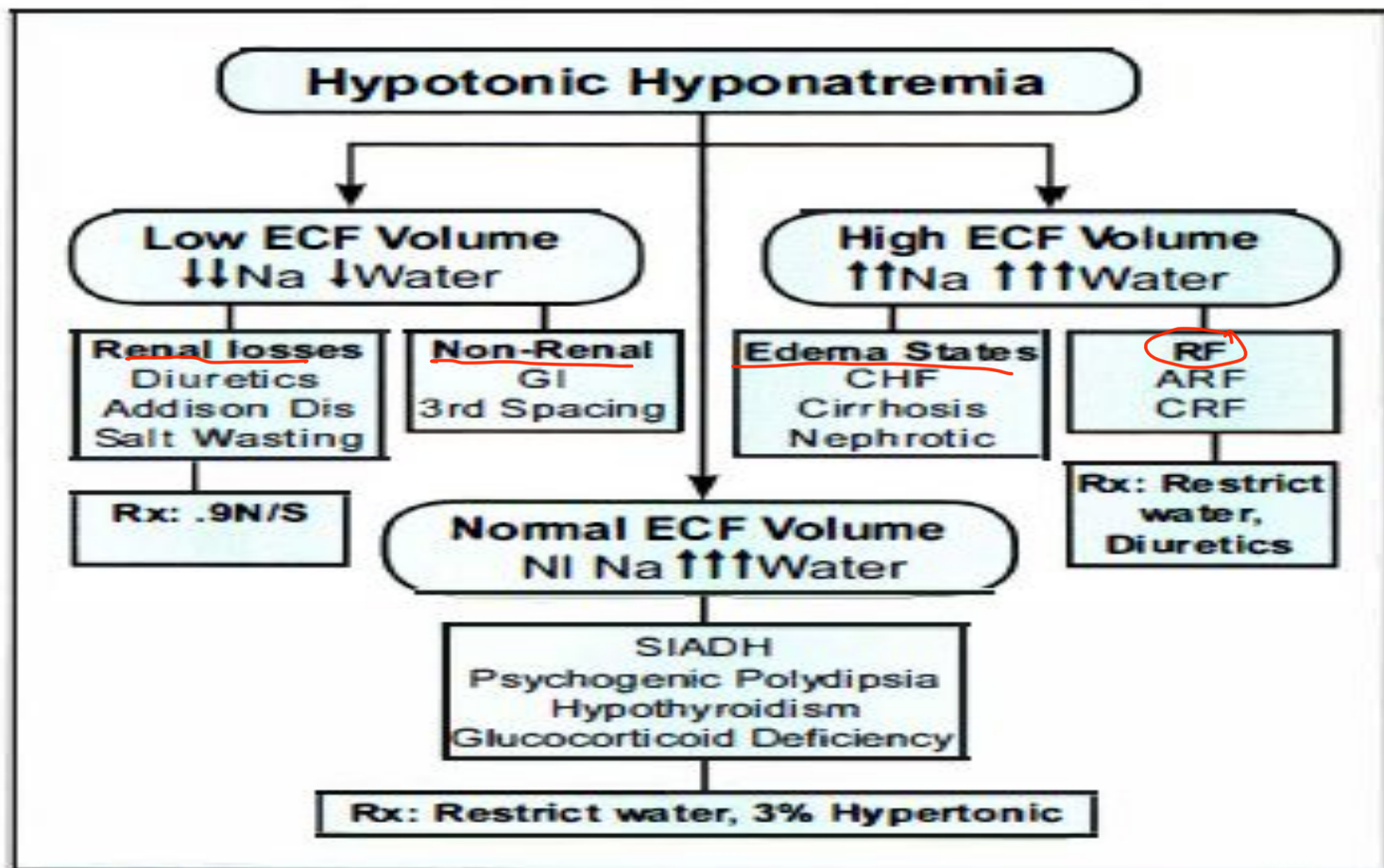


Figure 4-2: Causes of Hypotonic Hyponatremia

Thiazide Diuretics

Thiazides also can cause euvolemic low osmolality hyponatremia via several possible mechanisms, both ADH-related (by inducing mild volume depletion and directly stimulating ADH release) and non-ADH-related (by impairing urinary dilution in the early distal tubule). Elderly patients are highly susceptible to this effect of thiazides.

Treatment of Hyponatremia

↳ depend on the cause..

Treat asymptomatic hyponatremia (no CNS changes) based on etiology:

- Normal serum osmolality hyponatremia: This is artifact and does not require treatment.
- High osmolality hyponatremia: sometimes attributable to glucose or mannitol. Treating the underlying disease process (e.g., DKA) normalizes the sodium with time.
- Low osmolality and:
 - o Hypovolemic: normal saline to replenish deficit (watch for over-rapid correction!).

* isotonic - hypertonic - isovolemic $\Rightarrow$ treat the cause...

o **Hypervolemic**: fluid restriction (- 800 cc/day)
+/- loop diuretics.

$\nearrow$ HF - liver cirrhosis .. etc

o **Euvoletic**: SIADH is treated with fluid restriction (- 800 cc/day). Refractory cases can be treated with an ADH receptor antagonist, such as conivaptan or tolvaptan. Treat hypothyroidism or glucocorticoid deficiency with appropriate replacement.

$\nearrow$ if he un response to the fluid restriction...

$\nearrow$ treat the cause

Remember: Isotonic saline worsens hyponatremia in patients with SIADH.

{ hypertonic saline + water restriction $\rightarrow$ normal saline $\rightarrow$ SIADH $\rightarrow$ pt $\rightarrow$ * منع ر Pt $\rightarrow$ SIADH خطير بالخصوص في حالة نقص الماء + hypertonic saline + water restriction
if he symptomatic نقل الماء 1000cc per day.. }

[note] [تعالجنا hypernatremia بطريقة منع يه قد الحريفة : cerebral edema]

If the symptoms of hyponatremia are severe (e.g., neurologic symptoms such as lethargy, confusion, coma, seizures) and the patient is not hypovolemic, treat with 3% saline. Correction of hyponatremia should never exceed 9 mEq/L over 24 hours due to the risk of osmotic demyelination syndrome (see next slide).

or called central pontine myolysis

← حتی لازم نزخ الصدویہ بشکل سریع عنه محدود المانی حتی 6 یه دخل المريض بـ osmotic demyelination syndrome.

✗ لمكان اذ Na^+ الیوم 135 و بعد یوم كان 120 ف افض له و یفضل 135 خارج و یفضل 135 ف الصودیة یل یزید بدرجة یدی نزخه بسرعة.

و لكن اذ Chronic Pt یستكون عنده osmole anti enologenous فی لا Brain یخفف نزخ الصدویة ف منه ایست بشکل سریع.

[8 meq. per day] (یخفف نزخه 8 من 24 ساعة)

← مريض عنده Na^+ : 120 یقفه قلبه آتتھا 128 فلال 24 ساعة ..

رتبه - یوحده 135 : the pt presented with Na^+ : 120 andue treatthim , next day the Na^+ became:- 135
 ولكن لاصفح یصح لا تقدر علیه ایضا [comm. paralysis - dysarthria] حتی یقل المانی راج یبكره ولكن انقل
 ↳ irreversible quadriplegia..

100cc hypertonic saline response در نطفه 10 دله به 100cc hypertonic saline 3% coma - seizure ← severe hyponatremia ← H₂O
 100cc hypertonic saline response در نطفه 10 دله به 100cc hypertonic saline 3% coma - seizure ← severe hyponatremia ← H₂O
 100cc hypertonic saline response در نطفه 10 دله به 100cc hypertonic saline 3% coma - seizure ← severe hyponatremia ← H₂O
 100cc hypertonic saline response در نطفه 10 دله به 100cc hypertonic saline 3% coma - seizure ← severe hyponatremia ← H₂O

Standard therapy: Give enough 3% saline to increase the serum sodium by 6-8 mEq/L over 24 hours.

For **severe symptoms** (seizure, coma), a **100 mL bolus of 3% saline** is recommended to **quickly** raise the serum sodium by **2-3 mEq/L**. If **symptoms persist**, **2 more boluses** can be given in **10-minute intervals**.

For **moderate symptoms** (confusion, lethargy) and suspected **SIADH**, hypertonic saline can also be used. A reasonable initial rate is **0.5-1.0 mL/kg lean body weight/hour**. The serum sodium should be checked frequently (every 2-4 hours) and the rate adjusted to achieve the target correction of 6-8 mEq/L over 24 hours. Watch carefully for the abrupt onset of a water diuresis, which can cause the serum sodium concentration to rise too fast, especially in patients with psychogenic polydipsia, hypovolemic hyponatremia, and hyponatremia due to thiazide diuretics.

Osmotic demyelination syndrome was previously referred to as central pontine myelinolysis because it is most prominent in the pons. If the sodium concentration is raised too rapidly, the cells can shrink (water rushes out of cells into the blood stream, where the osmolality has risen), potentially causing this demyelination syndrome. This rare effect is more likely to occur in the patient with chronic, severe hyponatremia ($\text{Na}^+ < 115 \text{ mEq/L}$ for > 2 days) whose sodium is corrected rapidly ($> 10 \text{ mEq/L}$ over 24 hours). Symptoms are delayed by about a week, compared to the rise in the sodium concentration, and are usually not reversible. Presentation includes speech and swallowing difficulties, weakness or paralysis, cognitive deficits, and coma. Seizures occasionally occur.

A related cause of severe and sometimes symptomatic hyponatremia is exercise-induced hyponatremia, reported in marathon and other endurance exercise participants and caused by both nonosmotic production of ADH and excessive intake of hypotonic fluids.

3% hypertonic saline should be promptly administered when diagnosed.

HYPERNATREMIA

?Can You Drink Too Much Water



Overview

Severe hypernatremia is fairly rare but always represents a water deficit. It does not occur unless the patient is unable to get to water (debilitated or H₂O unavailable) or the thirst mechanism is defective. Unlike hyponatremia, these patients are always hyperosmolar, so the 1st step is **determining volume status**. Low volume implies water and total body Na⁺ loss (water deficit exceeding Na⁺ deficit).

Treat severe hypovolemic hypernatremia with **normal saline first** to correct the volume deficit, and only then with hypotonic fluids to further replace the water deficit.

Remember: Even normal saline has a lower osmolality than serum in a patient with hypernatremia.

Cellular swelling can occur from too rapid correction of any severe hyperosmolar state, such as hypernatremia, nonketotic hyperglycemic coma, and severe uremia. (These are just the variables in the osmolality equation.) Cellular swelling can cause cerebral edema, seizures, and coma. So be careful with the fluid rate and measure serum sodium frequently

High-volume hypernatremia:

This is unusual and typically not serious. It most often occurs with mineralocorticoid excess, such as primary hyperaldosteronism. Usually, the only time a serious result occurs is after giving large amounts of sodium bicarbonate or hypertonic saline during advanced cardiac life support. Treat with loop diuretics and free water.

Normal-volume hypernatremia is most often seen in patients with diabetes insipidus (01) and reduced access to water (and thus become hypernatremic) but have not yet developed frank volume depletion.

Typical DI patients with normal access to water have normal or borderline-high serum Na^+ levels because they are constantly drinking water (hyperosmolar, and therefore always thirsty). Their primary complaint is usually polyuria and polydipsia.

Think of central DI in the patient with high Na^+ and high urine volume who also has a history of recent neurosurgery, head trauma, or brain cancer/metastases.

Otherwise, DI is typically nephrogenic DI (also called "ADH resistant").

Nephrogenic DI can be hereditary, with most cases presenting in childhood (mutations in the vasopressin 2 receptor or aquaporin 2 genes) or due to:

1. hypercalcemia (serum $\text{Ca}^{2+} > 11 \text{ mg/dL}$),
2. chronic hypokalemia (serum $\text{K} < 3 \text{ mEq/L}$),
3. intrinsic renal disease (especially Sjogren syndrome),
4. drugs (especially lithium).

? Water Restriction Test

The water restriction test not only diagnoses DI but also differentiates between central and nephrogenic types.

In a healthy person, when plasma osmolality increases to 295, ADH level is high and urine is maximally concentrated (> 700 mOsm/L).

In central DI, even with water restriction, the ADH stays low and the urine dilute.

Treat **mild cases** of central DI with **thiazides and salt restriction**.

Chlorpropamide and carbamazepine can be used in cases of partial central DI, when desmopressin might be too potent or limited in supply. These drugs stimulate further ADH production if the patient has a partial defect.

Treat more resistant central DI with **oral or intranasal desmopressin** (synthetic vasopressin analog). Because the kidneys immediately respond to ADH, giving desmopressin results in a quick increase of the urine concentration and reduction in urine output. The intranasal preparation is the most potent. Be careful to titrate the dose properly and counsel the patient to drink only when thirsty, because volume overload and hyponatremia can easily occur (i.e., drug-induced SIADH).

In nephrogenic DI, the ADH is appropriately high, but the urine is dilute: The collecting duct is resistant to the effect of ADH. Giving extra ADH (desmopressin) does not increase the concentration of the urine. **Treat nephrogenic DI with thiazide diuretics or amiloride.** NSAIDs are used to treat rare hereditary forms.

Remember: SIADH (high ADH) usually presents as hyponatremia with normal volume. DI typically presents as polyuria/polydipsia, with or without hypernatremia, also with normal volume!.

Urine Osmolality

Urine osmolality can range from 50- 1,200 mOsm/L. To make sense of the osmolality, you must also know the urine output (L/d). Multiply the osmolality x output (1 kg = 1 L) to get total osmoles output per day. Normal is about 500. This is important in the case of a patient with high Na^+ and high urine output. If the 24-hour solute output is > 900 mOsm, think of an osmotic cause of the hypernatremia (e.g., hyperglycemia); whereas in DI, the 24-hour osmoles are normal, so the urine is very dilute.

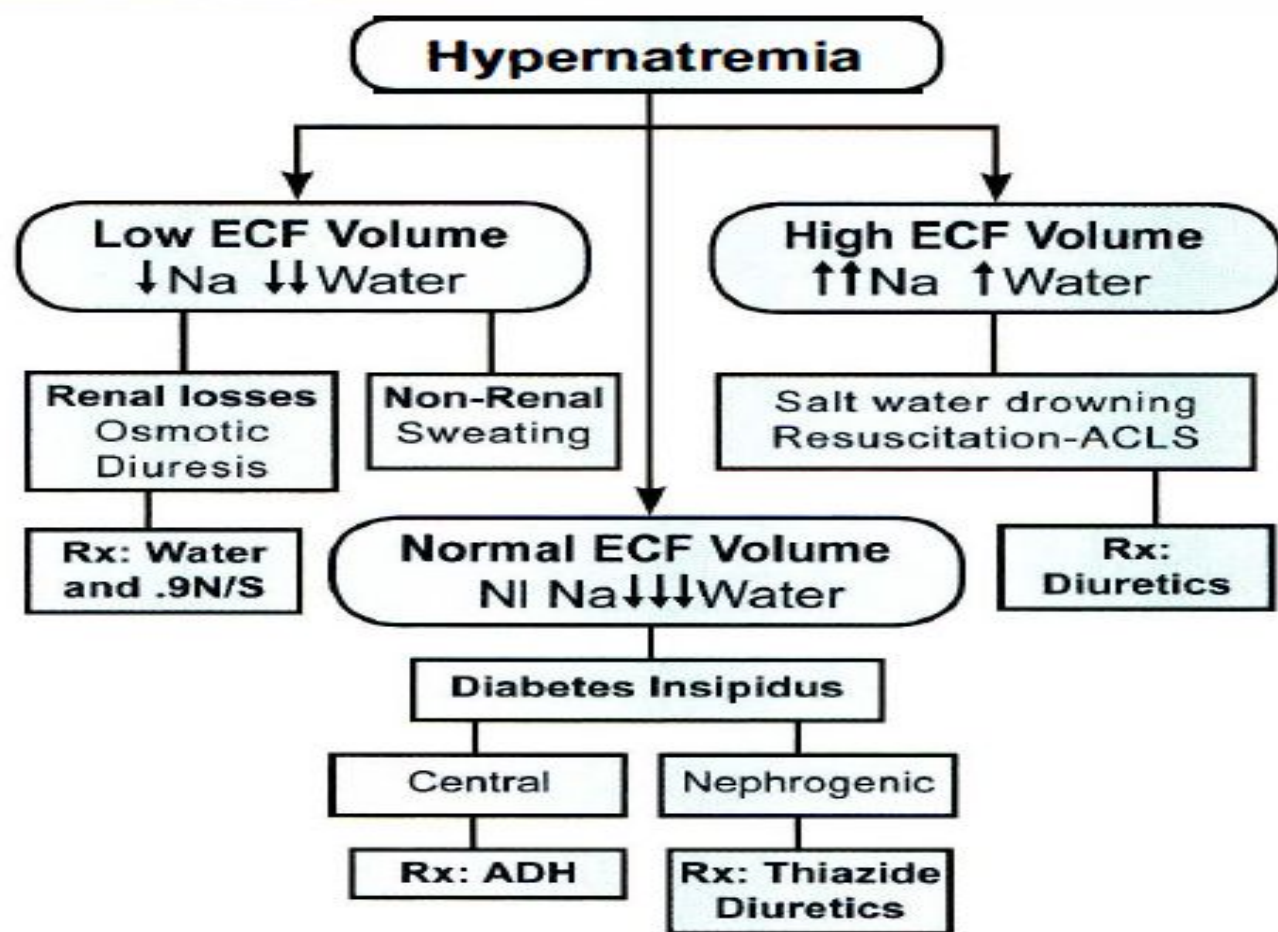


Figure 4-3: Causes of Hypertremia



A Dreamy World

A man's dreams are an index to his greatness

* Polyemia nervosa: - بتأكل كل شئ دنتغرفه مثل الحورق

$HCO_3 : 40 \Leftarrow$ metabolic alkalosis منجيب مع

due to vomiting $\leftarrow$ + hypo kalemia
+ hypo natremia.

POTASSIUM

* SLE has

- LL vasculitis
- pericarditis
- MI
- renal failure
- death
 - early [sepsis] ^{due to}
 - late [renal failure or MI]...

TOP Potassium

Rich Foods

