Hyponatremia

Amandeep Khurana
Southwest Kidney Institute

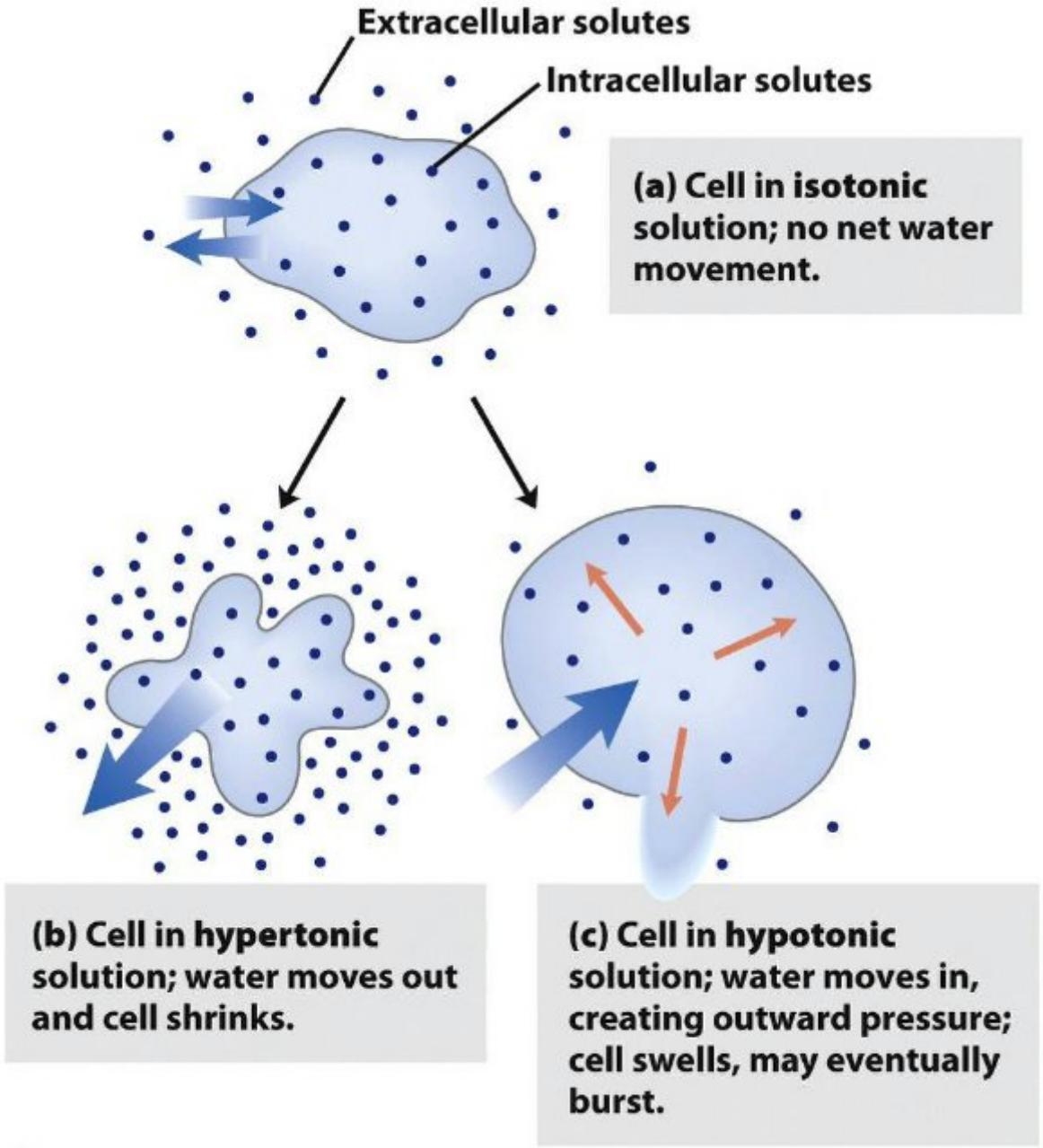


Figure 2-12
Lehninger Principles of Biochemistry, Fifth Edition

Why are we interested in the sodium level?

- Surrogate measure of osmolarity/infection
- Better measures available, but most convenient

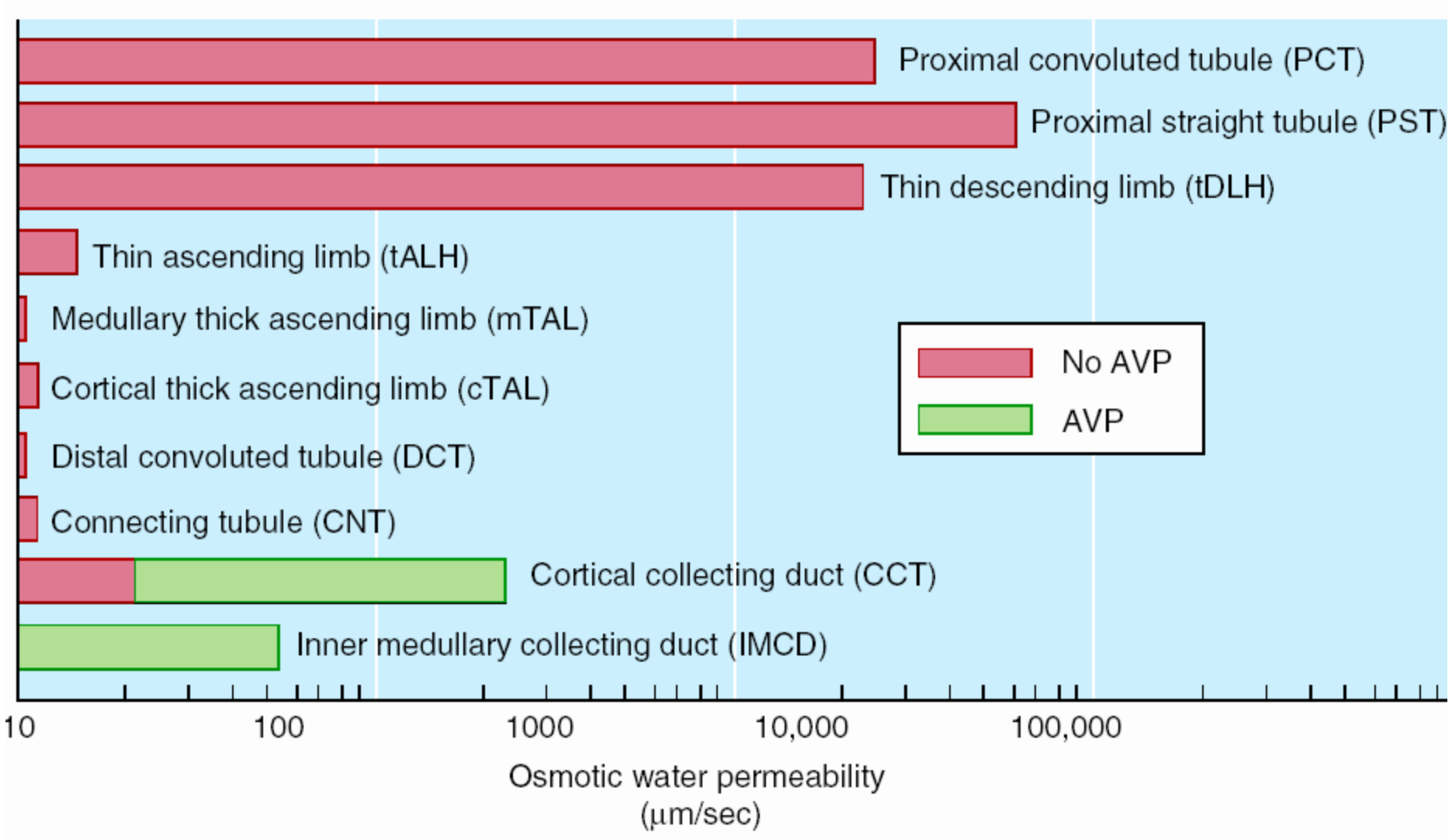
Is hyponatremia an issue of water or Na regulation?

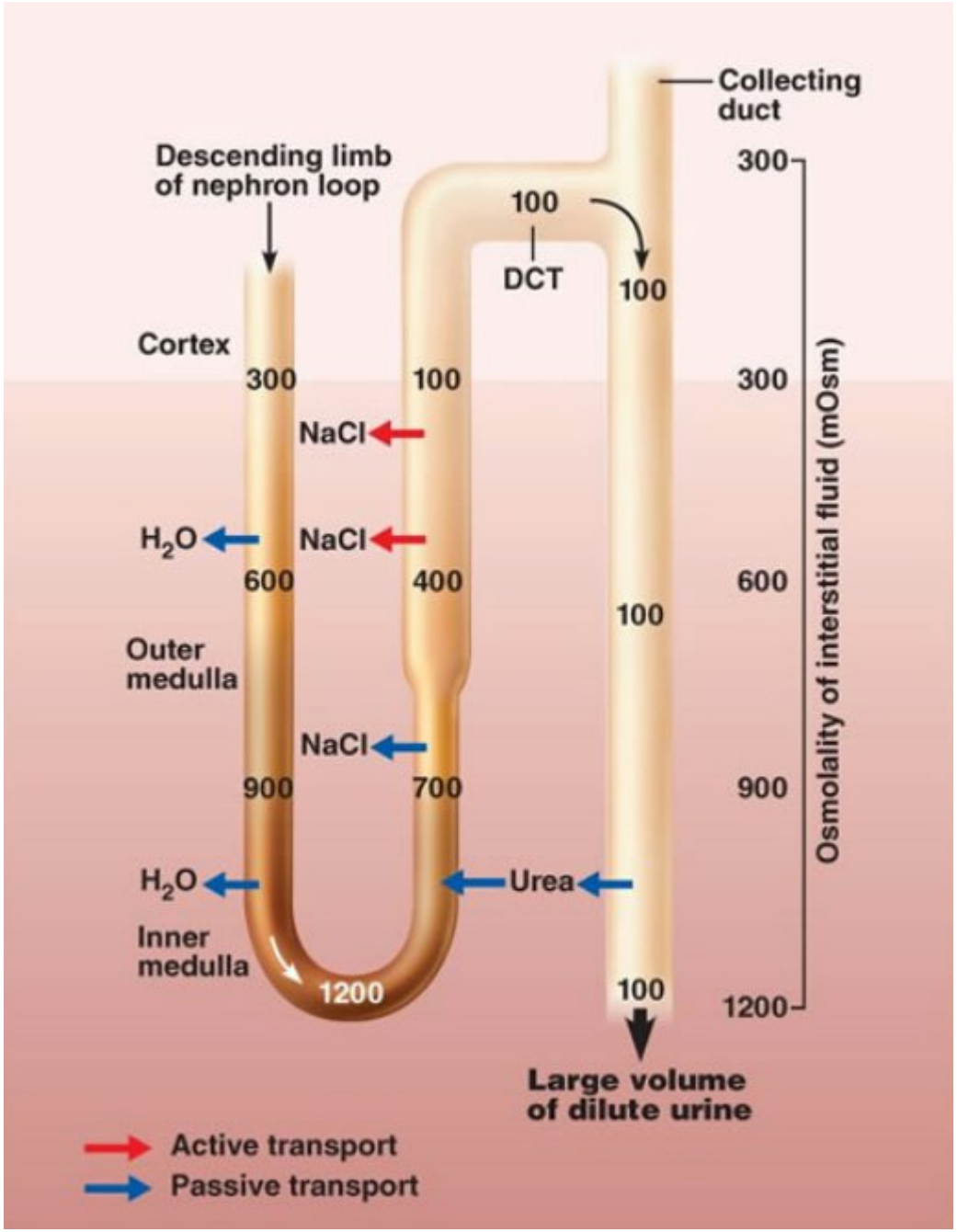
Purely water regulation!

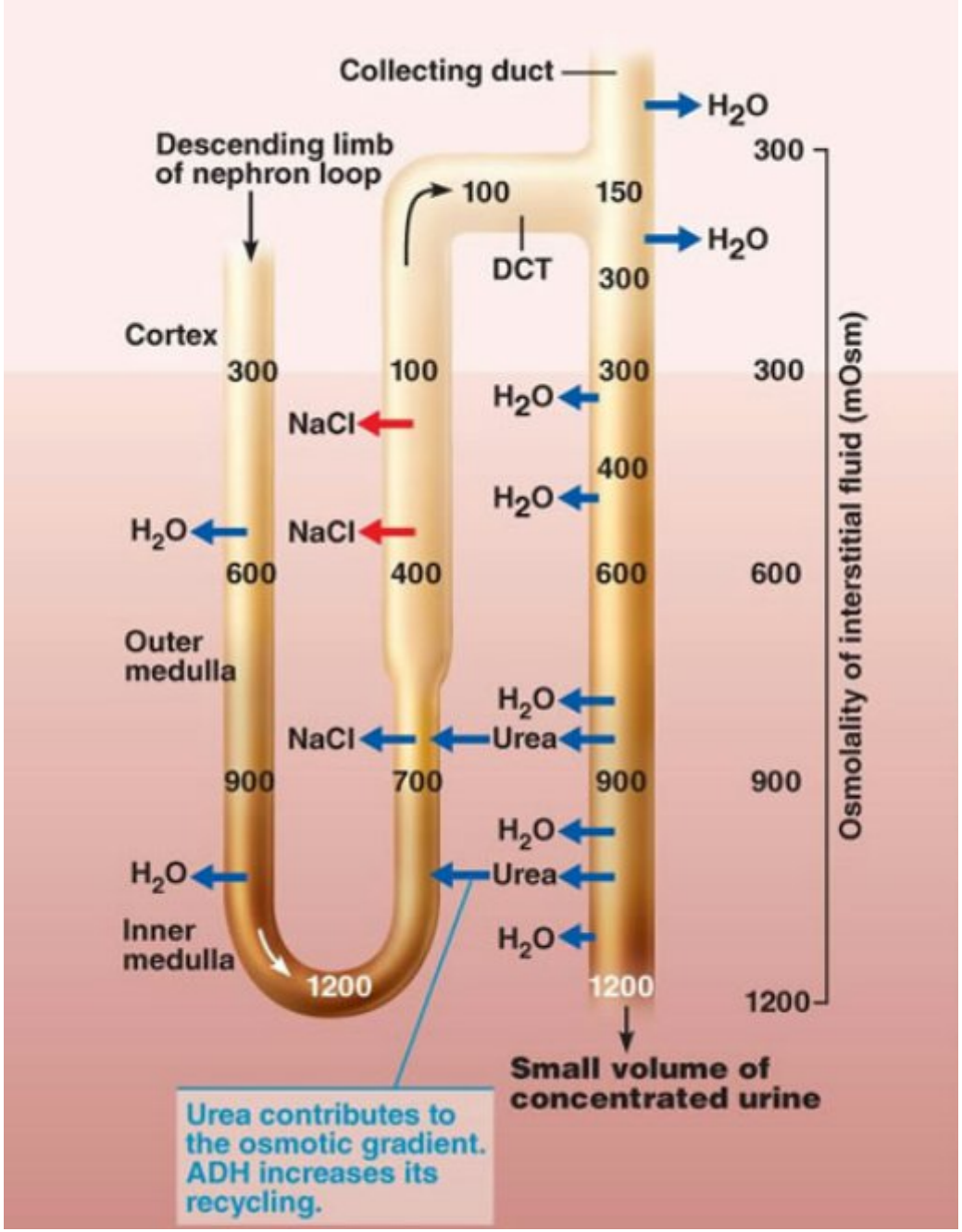
Single most important concept in the entire talk

Excess water = hyponatremia

Excess Na = Edema


Proximal convoluted tubule (PCT)
Proximal straight tubule (PST)
Thin descending limb (tDLH)
Thin ascending limb (tALH)
Medullary thick ascending limb (mTAL)
Cortical thick ascending limb (cTAL)
Distal convoluted tubule (DCT)
Connecting tubule (CNT)
Cortical collecting duct (CCT)
Inner medullary collecting duct (IMCD)
No AVP
AVP
10
100
1000
10,000
100,000
Osmotic water permeability
(μm/sec)


Collecting duct
Descending limb of nephron loop
100
DCT
100
300
Cortex
300
100
300
NaCl
H_2O
NaCl
600
400
600
100
Outer medulla
NaCl
900
700
900
H_2O
Urea
Inner medulla
1200
100
1200
Osmolality of interstitial fluid (mOsm)
Large volume of dilute urine
Active transport
Passive transport



ADH
U Osm > P Osm

If you spend a day in June hiking in the sun (poor water intake)

What's your ADH level going to be?

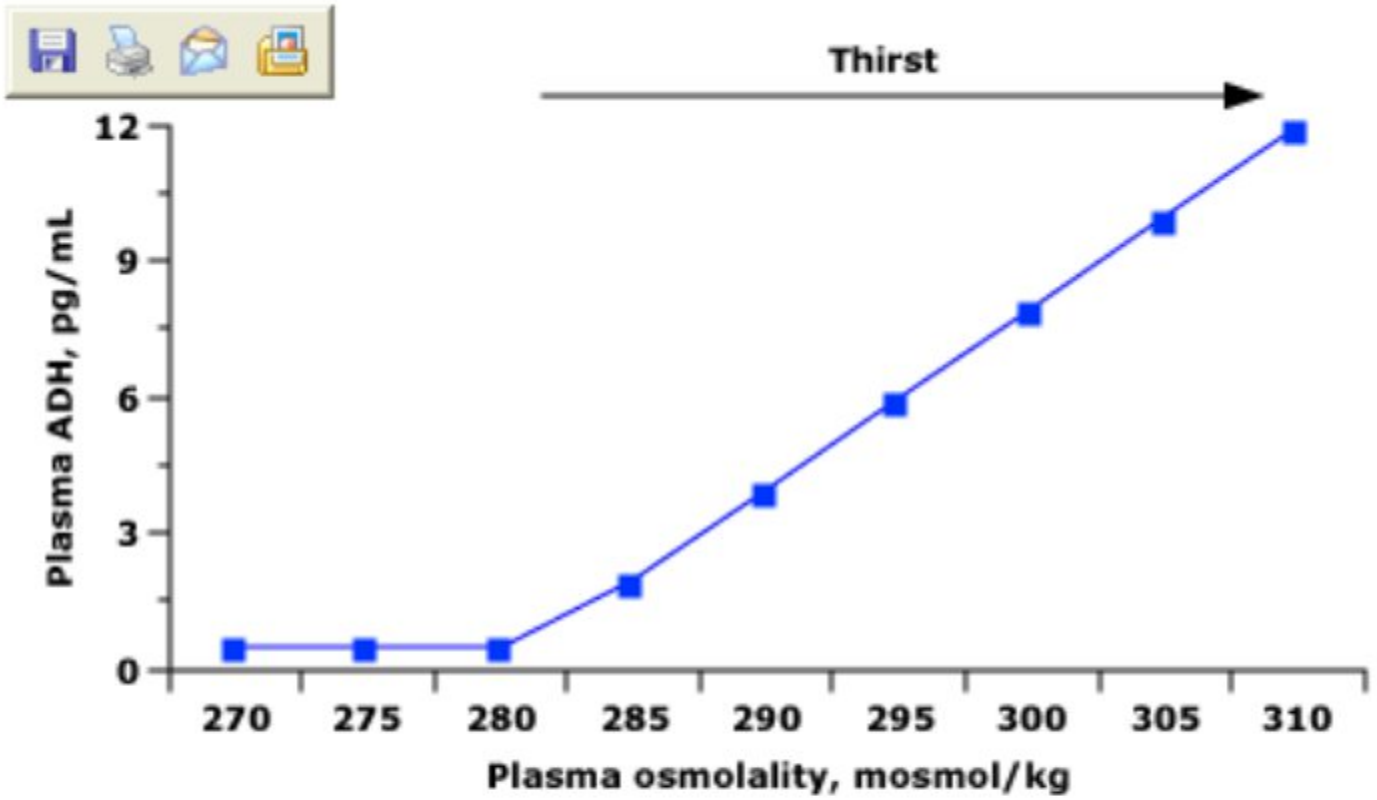
What will be your U Osm?

ADH → U Osm > P Osm

Dark yellow concentrated urine

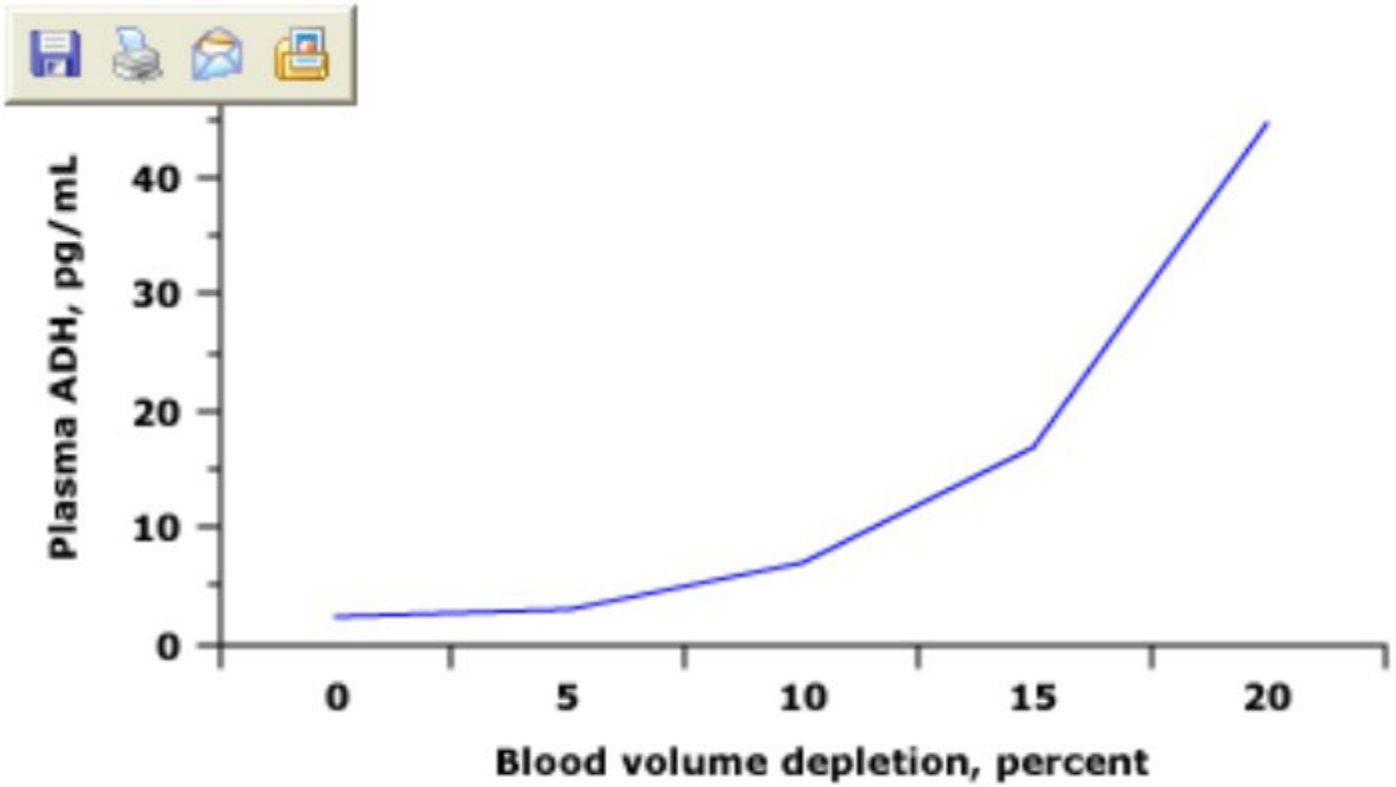
	Osmoregulation	**Volume regulation**
What is being sensed	Plasma osmolality	Effective circulating volume
Sensors	Hypothalamic osmoreceptors	Carotic sinus
		Afferent glomerular arteriole
		Atria
Effectors	Antidiuretic hormone	Sympathetic nervous system
		Renin-angiotensin- aldosteron
		Natriuretic peptides
		Pressure natriuresis
		Antidiuretic hormone
What is affected	Water excretion (via ADH)	Sodium excretion
	Water intake (via thirst)	

Osmotic regulation of ADH release and thirst

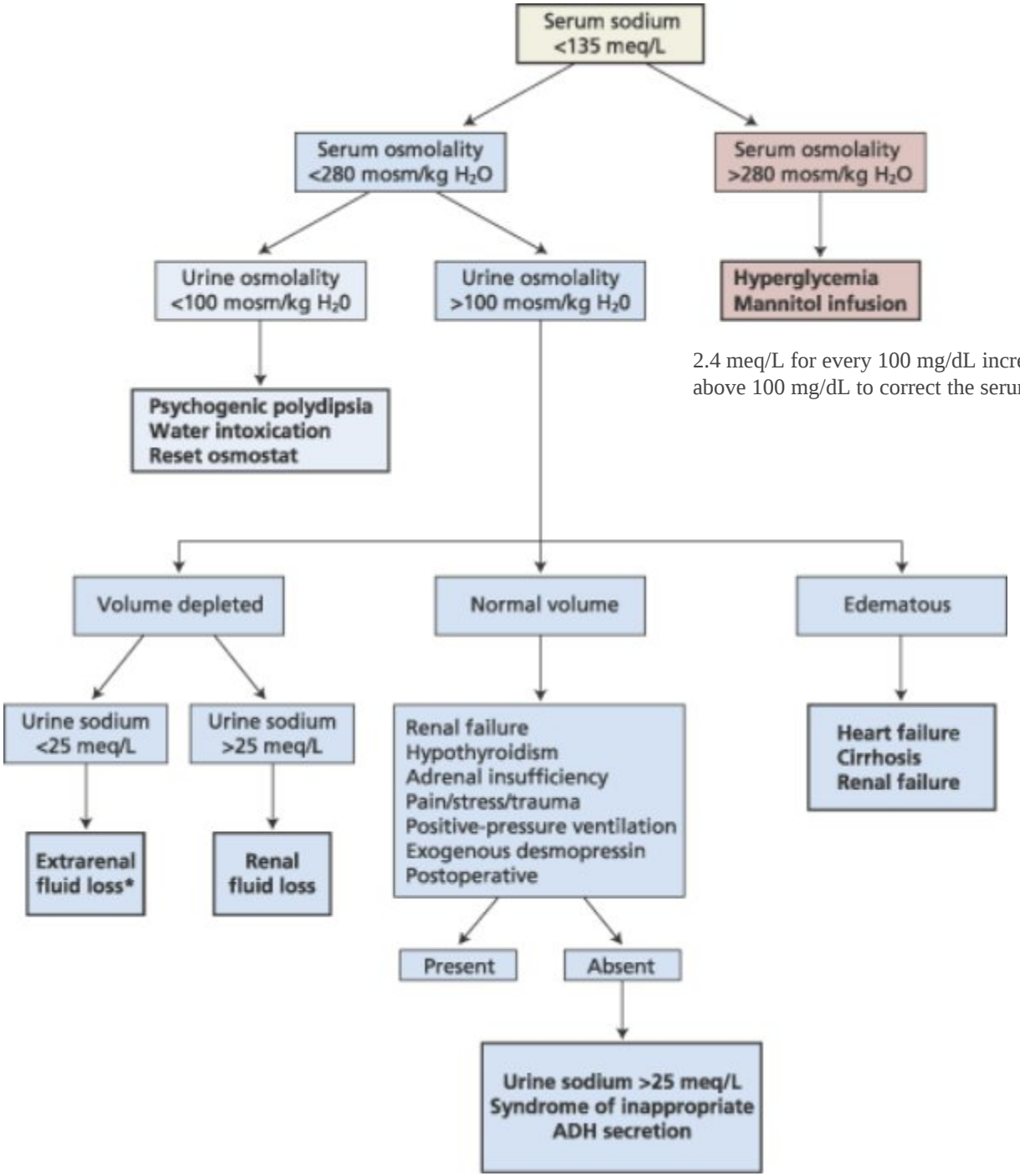


Relation between plasma antidiuretic hormone (ADH) concentration and plasma osmolality in normal humans in whom the plasma osmolality was changed by varying the state of hydration. The osmotic threshold for thirst is a few mosmol/kg higher than that for ADH. *Data from Robertson, GL, Aycinena, P, Zerbe, RL, Am J Med 1982; 72:339.*

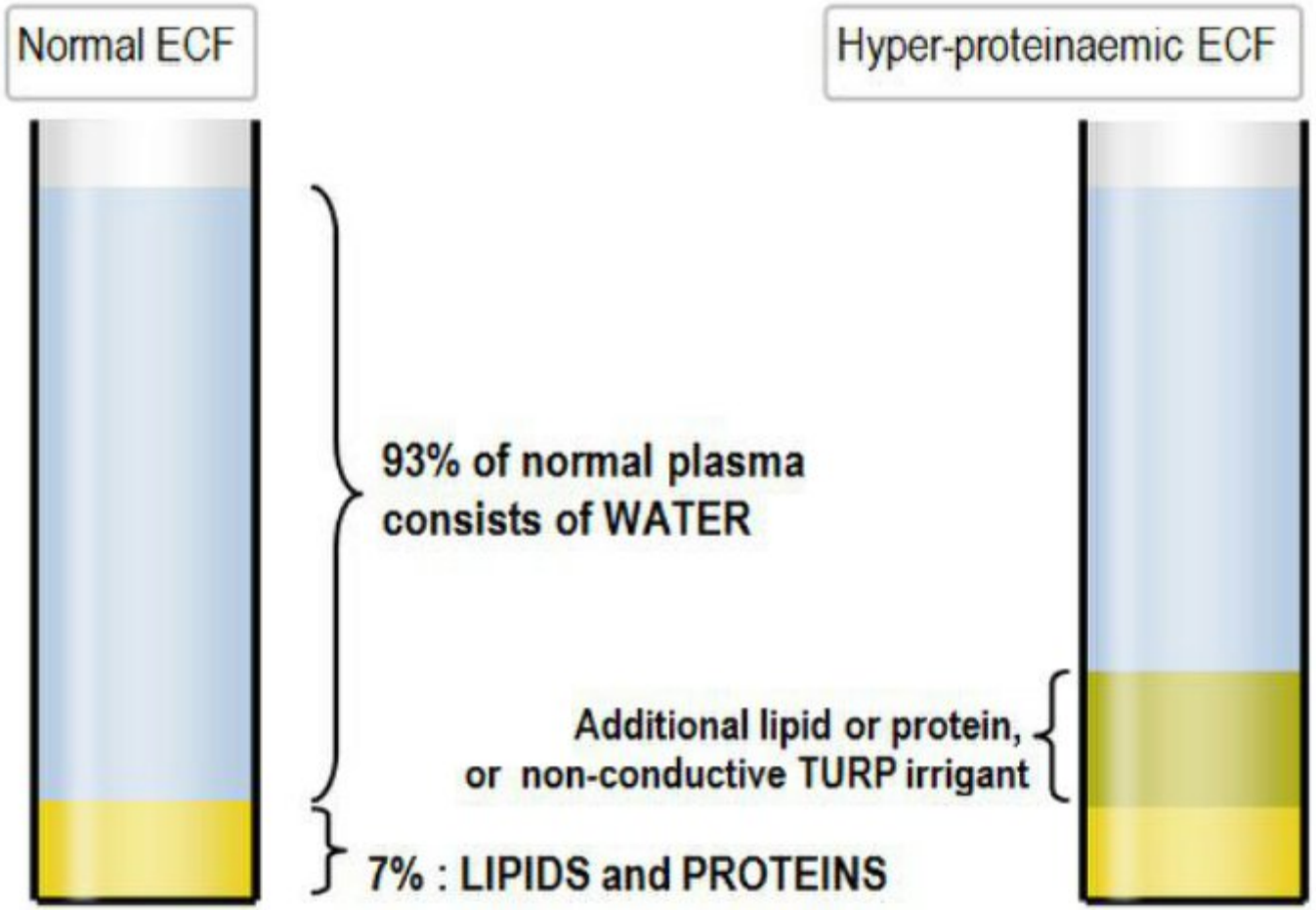
Hypovolemic stimulus to ADH release

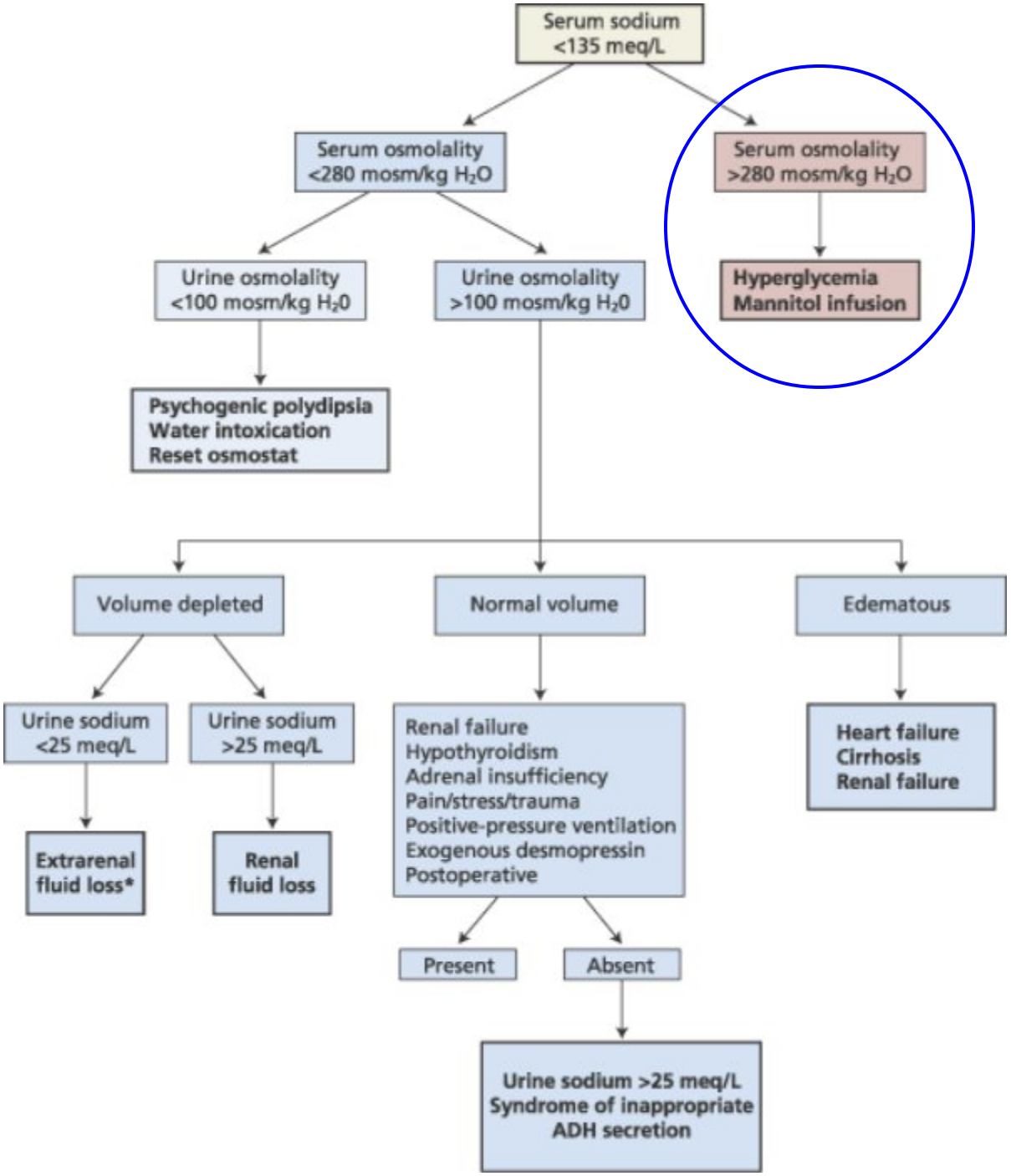


Relationship of plasma antidiuretic hormone (ADH) concentrations to isosmotic changes in blood volume in the rat. Much higher ADH levels can occur with hypovolemia than with hyperosmolality, although a relatively large fall in blood volume is required before this response is initiated. *Data from Dunn, FL, Brennan, TJ, Nelson, AE, Robertson, GL, J Clin Invest 1973; 52:3212.*



2.4 meq/L for every 100 mg/dL increment of serum glucose above 100 mg/dL to correct the serum sodium


Normal ECF
Hyper-proteinaemic ECF
93% of normal plasma
consists of WATER
Additional lipid or protein,
or non-conductive TURP irrigant
7% : LIPIDS and PROTEINS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Serum osmolality <280 mosm/kg H_2O
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Hyperglycemia
Mannitol infusion
Urine osmolality <100 mosm/kg H_2O
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Psychogenic polydipsia
Water intoxication
Reset osmostat
Volume depleted
Normal volume
Edematous
Urine sodium <25 meq/L
Urine sodium >25 meq/L
Renal failure
Hypothyroidism
Adrenal insufficiency
Pain/stress/trauma
Positive-pressure ventilation
Exogenous desmopressin
Postoperative
Heart failure
Cirrhosis
Renal failure
Extrarenal fluid loss*
Renal fluid loss
Present
Absent
Urine sodium >25 meq/L
Syndrome of inappropriate ADH secretion

Hyponatremia with normal or elevated plasma osmolality

High plasma osmolality

- Hyperglycemia
- Mannitol

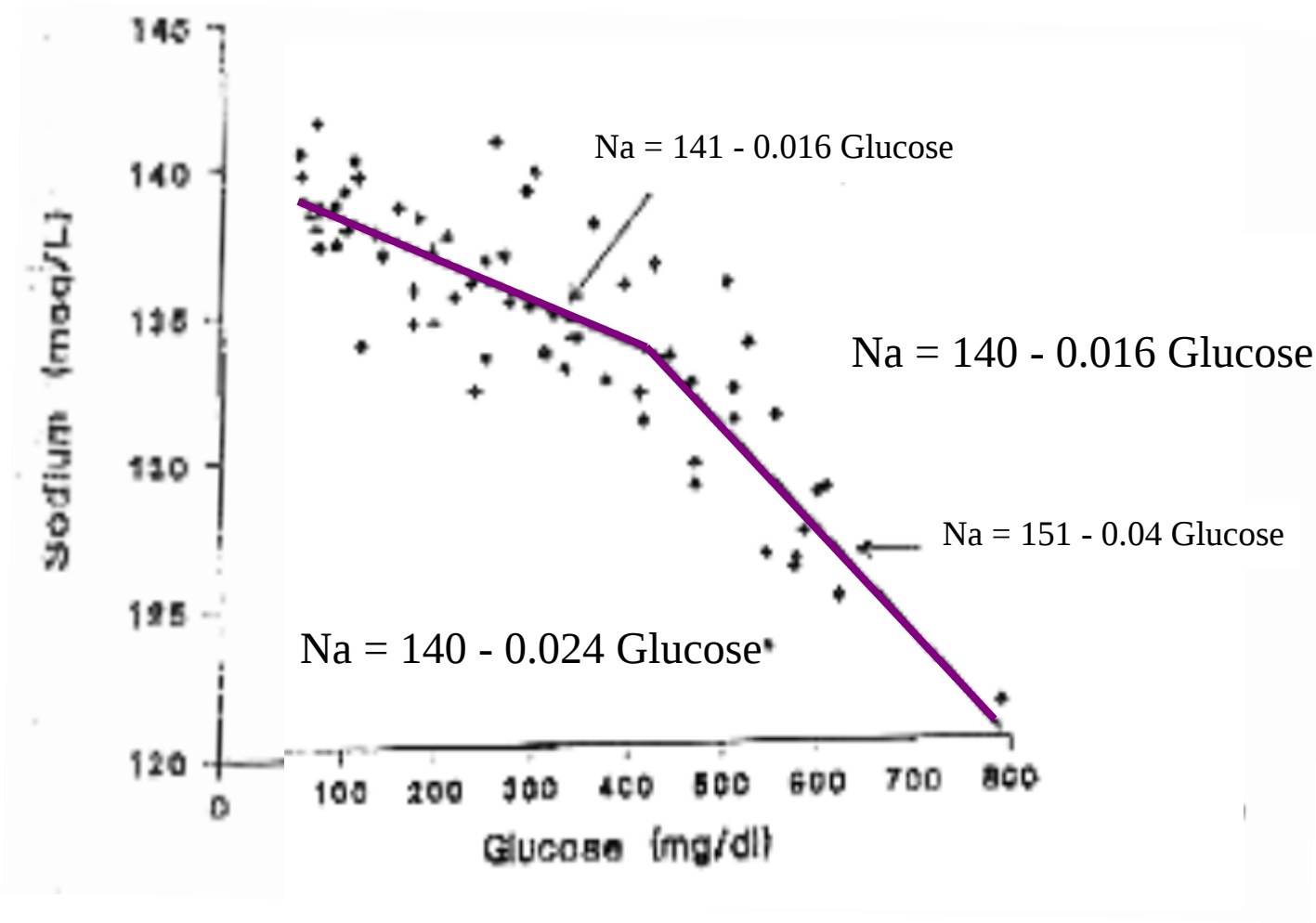
Normal plasma osmolality

- Pseudohyponatremia
  - Hyperlipidemia
  - Hyperproteinemia
- Glycine solutions (TURP-> hyposmolal + glycine metabolism)

The only study on pseudohyponatremia!

- Published 1999
- N=6
- Somatostatin infusion prevents endogenous insulin release.
- D20 0.45 NaCl infused to raise glucose > 600 mg/dL.
- An insulin drip then gradually lowers glucose to 140 mg/dL.
- Glucose and Na measured every 10 minutes.

Hillier TA, Abbott RD, Barrett EJ. Am J Med 1999; 106.



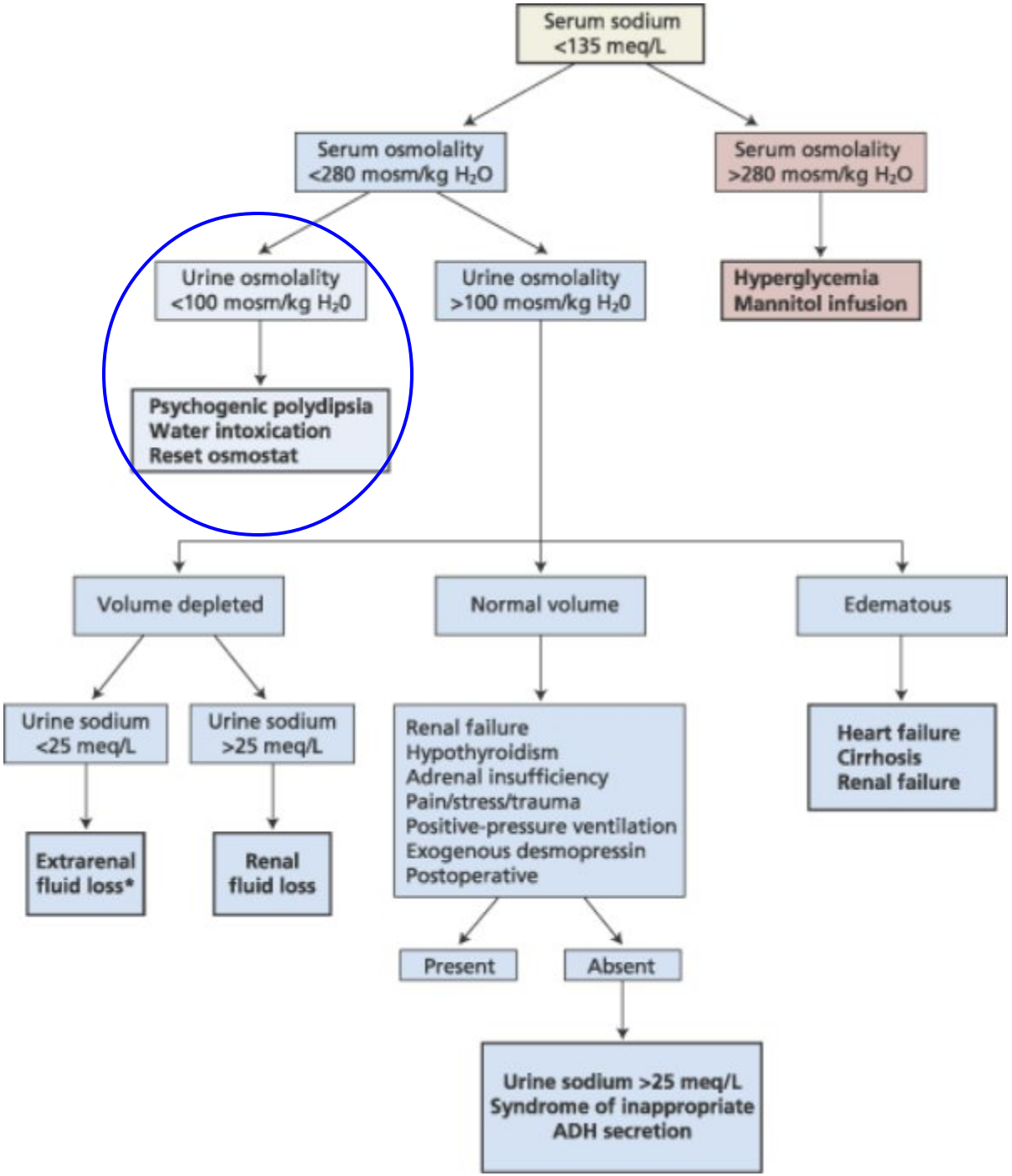
Hillier TA, Abbott RD, Barrett EJ. Am J Med 1999; 106

Sodium has a biphasic relationship to glucose.

- Glucose less than 400 use 1.6 mg per 100mg/dL
- Glucose over 400 use 4.0 per 100mg/dL

- Alternatively you can use 2.4 per 100 mg/dL.

Hillier TA, Abbott RD, Barrett EJ. Am J Med 1999; 106: 399-403.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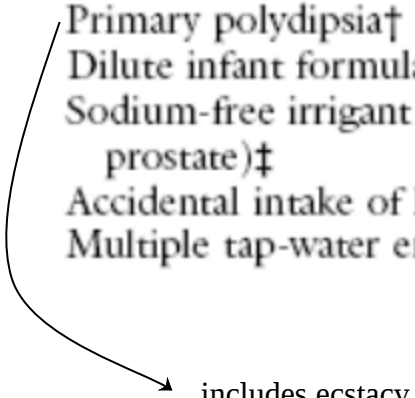
Syndrome of inappropriate ADH secretion

Excessive Water Intake

Primary polydipsia†
Dilute infant formula
Sodium-free irrigant solutions (used in hysteroscopy, laparoscopy, or transurethral resection of the prostate)‡
Accidental intake of large amounts of water (e.g., during swimming lessons)
Multiple tap-water enemas



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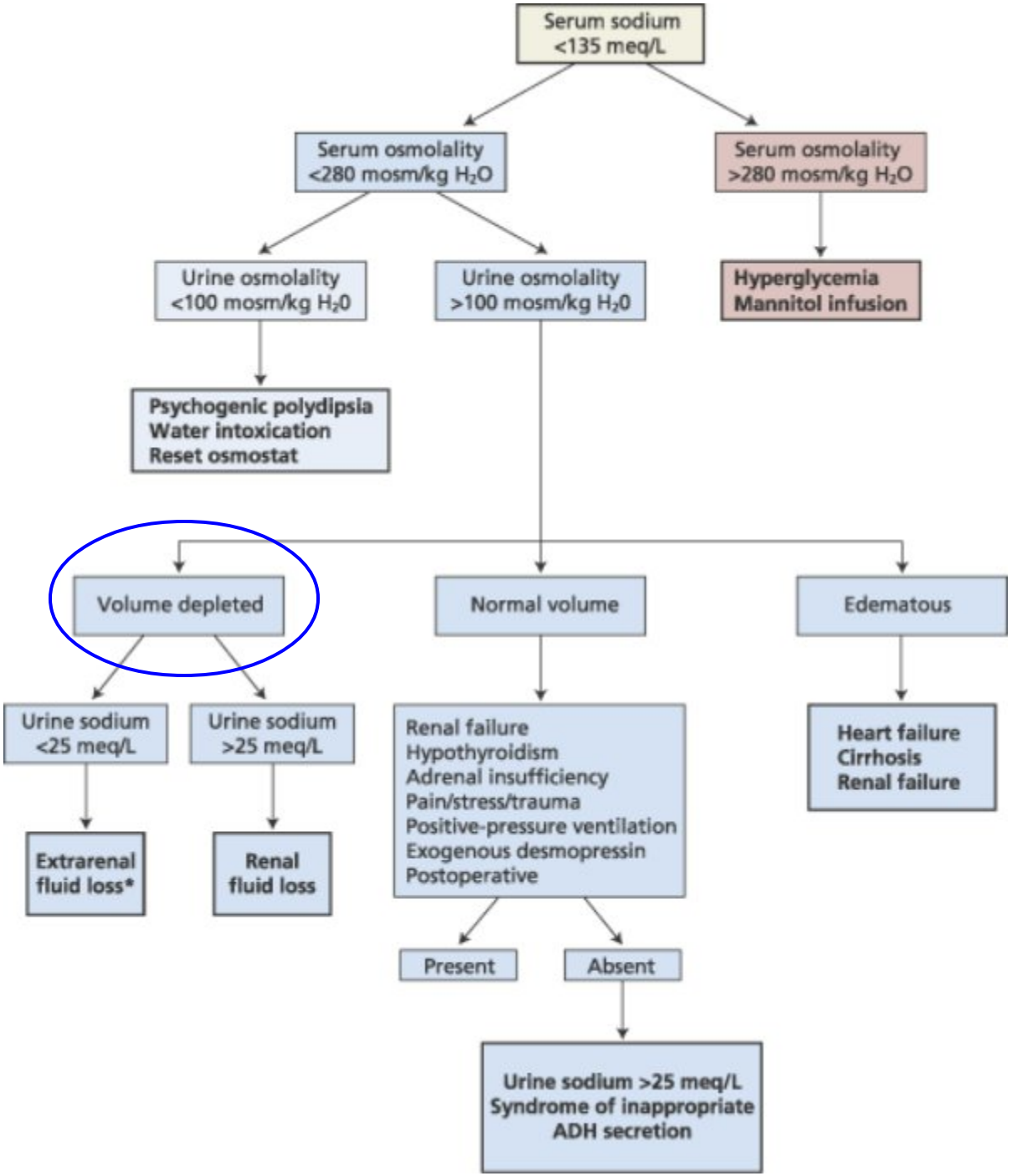
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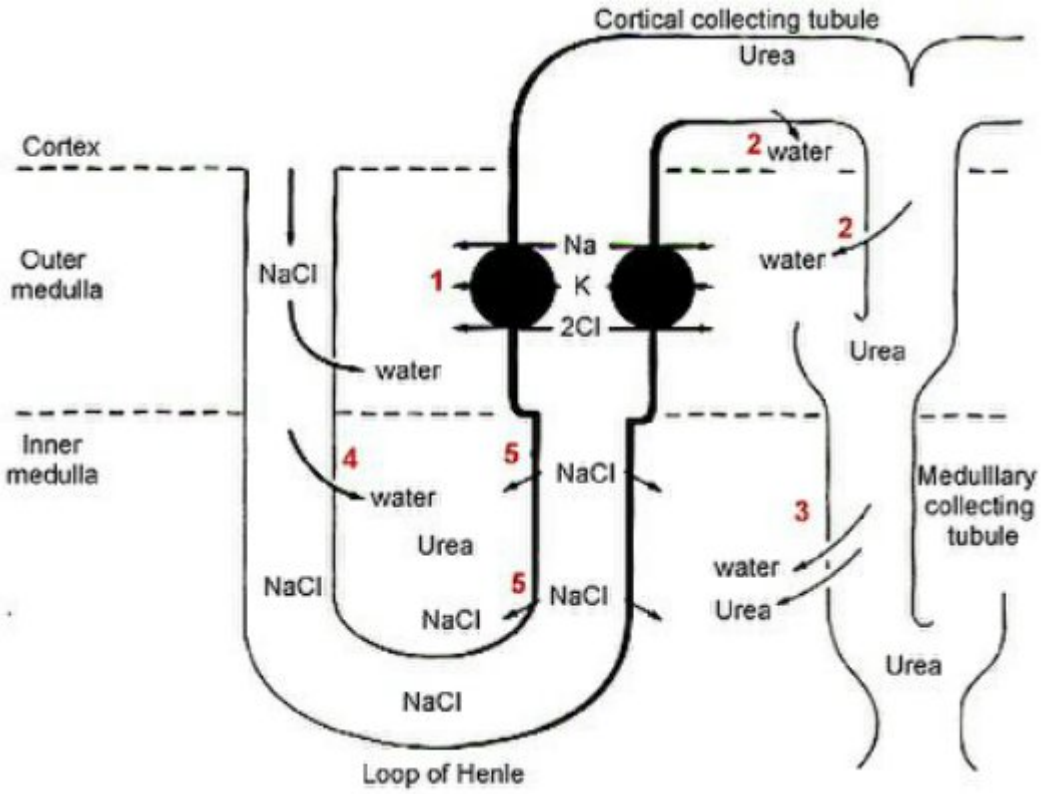
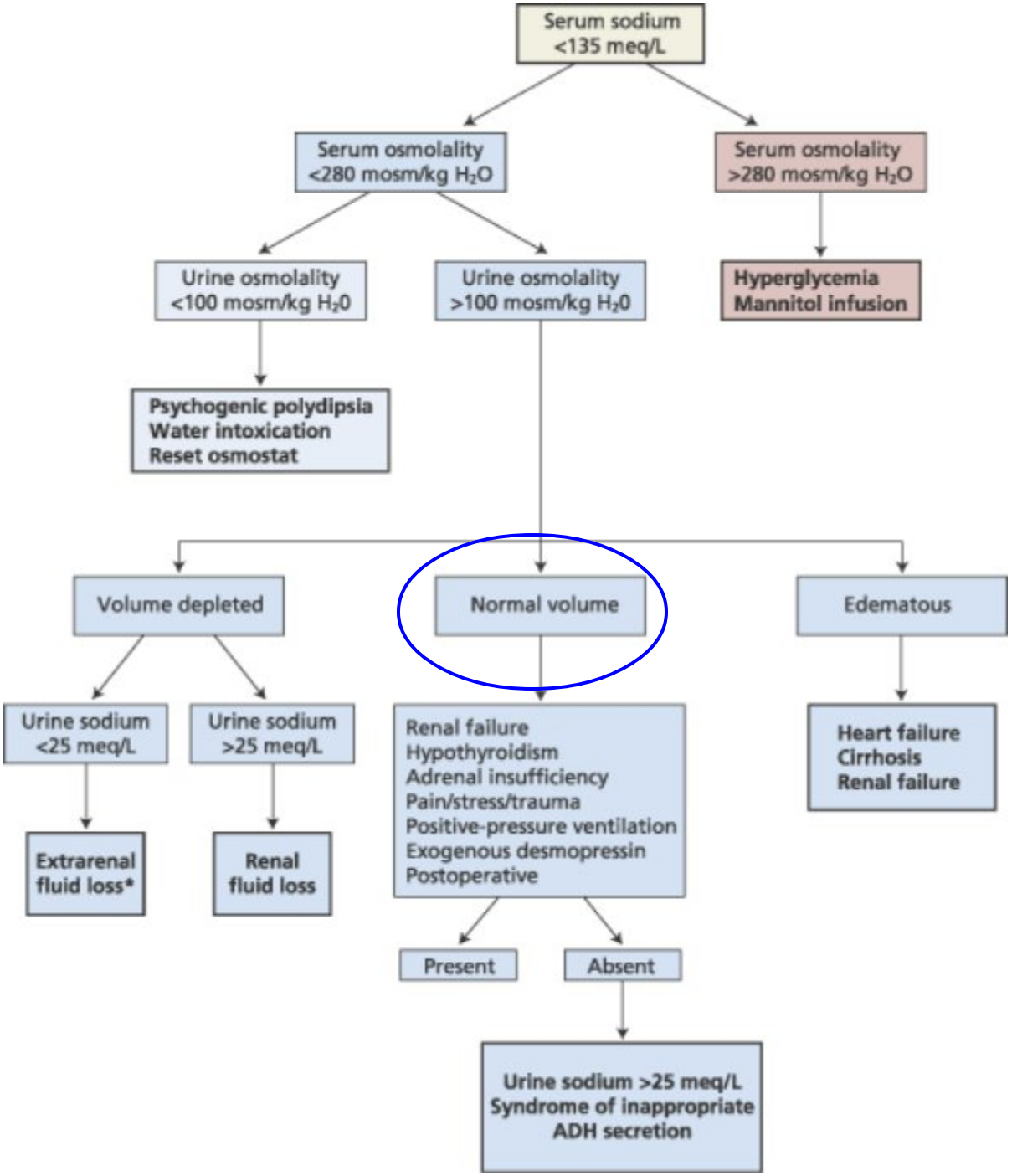
Syndrome of inappropriate ADH secretion

Hypotonic Hypovolemic Hyponatremia

- Total body water (TBW) decreases
- Total body Na (TBNa) decreases to a greater extent
- The extracellular fluid (ECF) volume is decreased
    - Effective circulating volume depletion
        - Cirrhosis
        - CHF
    - True volume depletion
        - Thiazide diuretics (Why *not* furosemide?)

Why *not* furosemide?

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Cirrhosis
Renal failure
Extrarenal fluid loss*
Renal fluid loss
Present
Absent
Urine sodium >25 meq/L
Syndrome of inappropriate ADH secretion

Hypotonic Euvolemic Hyponatremia

- TBW increases
- TBNa remains normal
- ECF is increased minimally to moderately
- Edema is not present
- Examples: SIADH, adrenal insufficiency

SIADH

- A low plasma osmolality
- An inappropriately elevated urine osmolality (above 100 mosmol/kg and usually above 300 mosmol/kg)
- A urine sodium concentration usually above 40 meq/L
- Low BUN and serum uric acid concentration
- A relatively normal plasma creatinine concentration
- Normal acid-base and potassium balance
- Normal adrenal and thyroid function

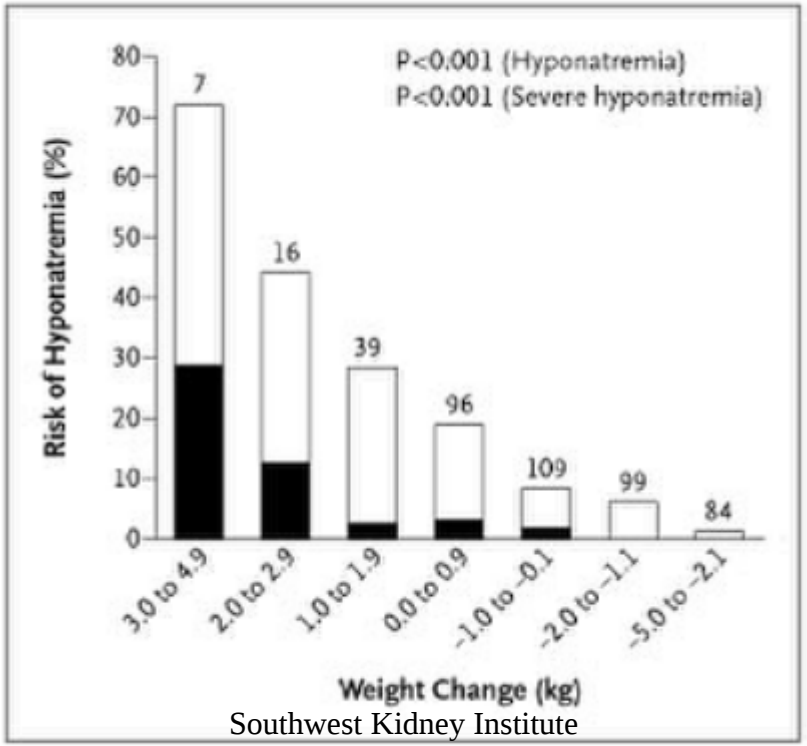
SIADH

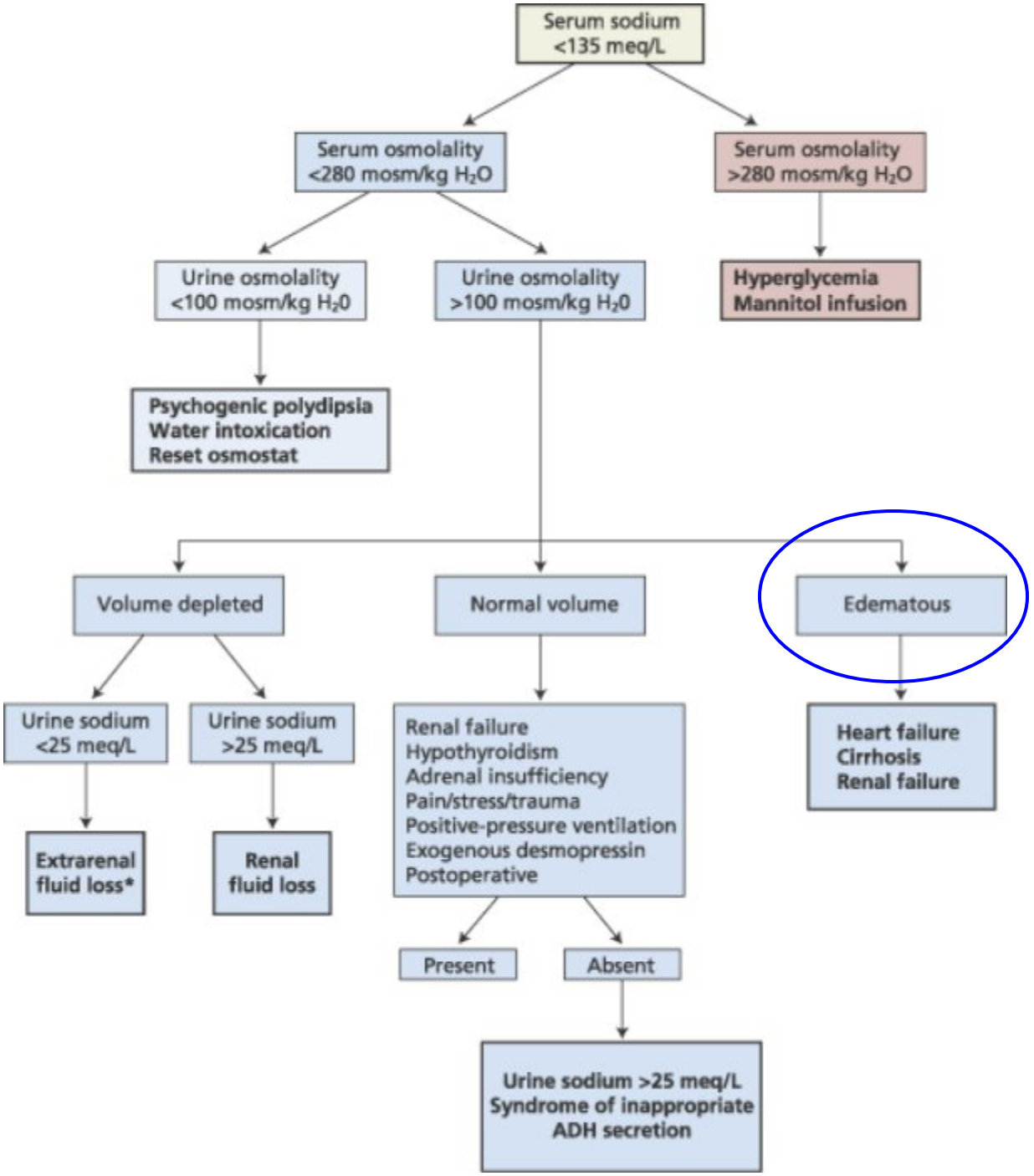
Treatment

- Fluid restriction (except subarachnoid hemorrhage d/t vasospams)
- NaCl +/- loop diuretics
- Demeclocycline
- Increased solute intake (urea)
- V2 antagonists (conivaptin IV)

Exercise-associated hyponatremia or Marathon runner's hyponatremia

- dilute, but sodium-containing sweat loss
- mild volume depletion (with elevated ADH)
- replaced with excessive amounts of hypotonic solutions
- NSAIDs

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Renal fluid loss
Present
Absent
Urine sodium >25 meq/L
Syndrome of inappropriate ADH secretion

Hypotonic Hypervolemic Hyponatremia

- Total body sodium increases
- TBW increases to a greater extent
- ECF is increased markedly
- Edema is present
- Usually, effective circulating volume depletion
- Examples: cirrhosis, CHF, Nephrotic syndrome

Cerebral salt wasting: Truths, fallacies, theories, and challenges

Sheila Singh, MD; Desmond Bohn, MB; Ana P. C. P. Carlotti; Michael Cusimano, MD; James T. Rutka; Mitchell L. Halperin, MD

Background: The reported prevalence of cerebral salt wasting has increased in the past three decades. A cerebral lesion and a large natriuresis without a known stimulus to excrete so much sodium (Na^+) constitute its essential two elements.

Objectives: To review the topic of cerebral salt wasting. There is a diagnostic problem because it is difficult to confirm that a stimulus for the renal excretion of Na^+ is absent.

Design: Review article.

Intervention: None.

Main Results: Three fallacies concerning cerebral salt wasting are stressed: first, cerebral salt wasting is a common disorder; second, hyponatremia should be one of its diagnostic features; and third, most patients have a negative balance for Na^+ when the diagnosis of cerebral salt wasting is made. Three causes for the large natriuresis were considered: first, a severe degree of extracellular fluid volume expansion could down-regulate transporters involved in renal Na^+ resorption; second, an adrenergic surge could cause a pressure natriuresis; and third, natriuretic agents might become more potent when the effective extracellular fluid volume is high.

Conclusions: Cerebral salt wasting is probably much less common than the literature suggests. With optimal treatment in the intensive care unit, hyponatremia should not develop. (Crit Care Med 2002; 30:2575–2579)

KEY WORDS: antidiuretic hormone; adrenaline; hyponatremia; natriuretic hormones; syndrome of inappropriate secretion of antidiuretic hormone

Cerebral Salt Wasting?

- CNS disease + clinical evidence of hypovolemia with
  - Hyponatremia (less than 135 meq/L) with a low plasma osmolality
  - inappropriately elevated urine osmolality (above 100 mosmol/kg and usually above 300 mosmol/kg)
  - urine sodium concentration usually above 40 meq/L
- Clinical evidence of hypovolemia is crucial since all of these laboratory findings are also seen in SIADH.

Cerebral Salt Wasting?

diagnosis of CSW should require that

- volume repletion leads to a dilute urine → which would be due to the removal of the hypovolemic stimulus to ADH release.
- evidence of net negative sodium balance prior to therapy

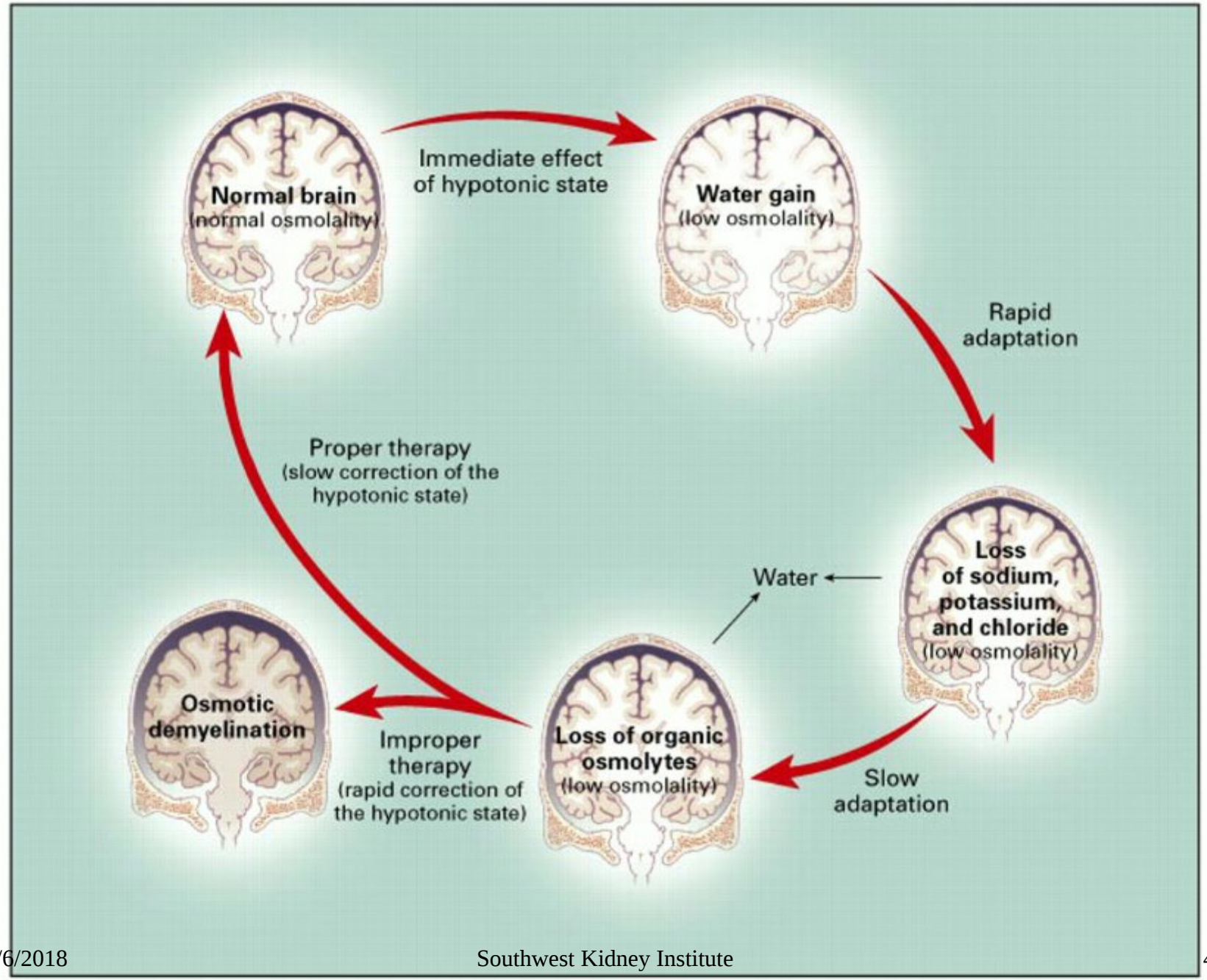
Cerebral Salt Wasting?

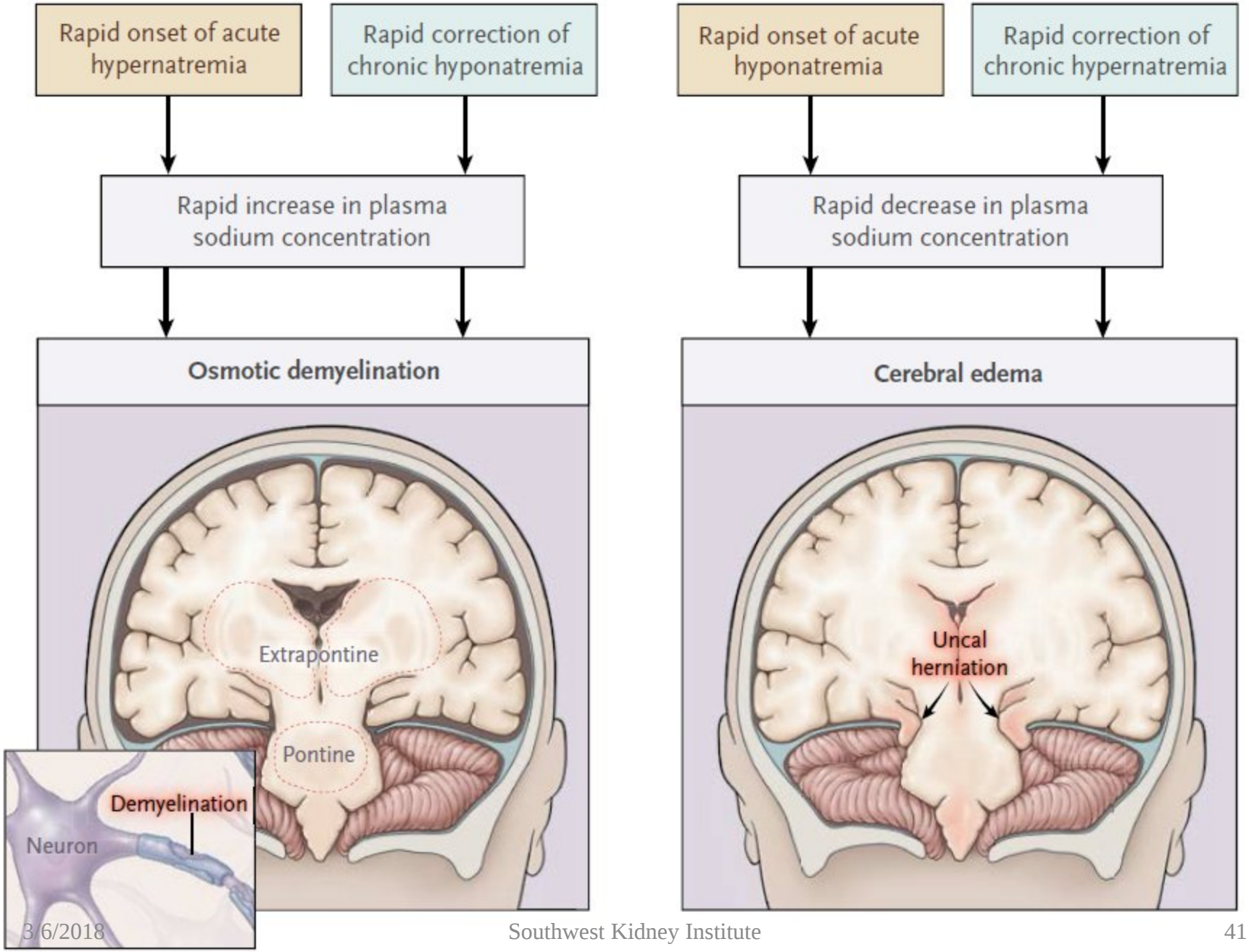
- Volume repletion with isotonic saline is the recommended therapy in CSW since it will suppress the release of ADH thereby permitting excretion of the excess water and correction of the hyponatremia.
- If CSW is the sole cause of the hyponatremia, volume repletion would result in the urine osmolality falling below 100 mosmol/kg,

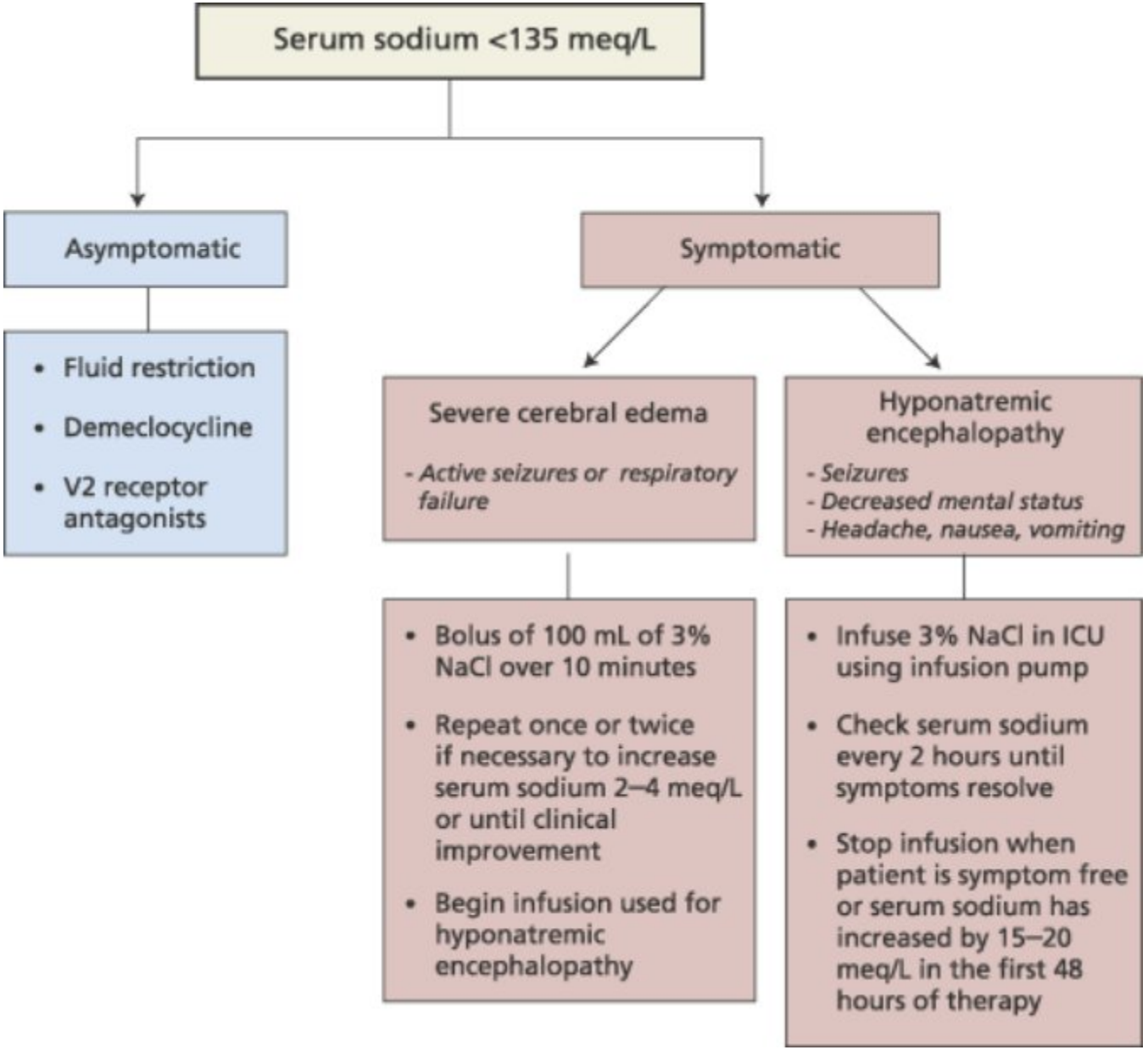
Management of Hyponatremia

Symptoms

- Anorexia
- Nausea and vomiting
- Difficulty concentrating
- Confusion
- Lethargy
- Agitation
- Headache
- Seizures
- Dysgeusia


Normal brain
(normal osmolality)
Immediate effect
of hypotonic state
Water gain
(low osmolality)
Rapid
adaptation
Loss
of sodium,
potassium,
and chloride
(low osmolality)
Water
Slow
adaptation
Loss of organic
osmolytes
(low osmolality)
Improper
therapy
(rapid correction of
the hypotonic state)
Osmotic
demyelination
Proper therapy
(slow correction of the
hypotonic state)


Rapid onset of acute hypernatremia
Rapid correction of chronic hyponatremia
Rapid increase in plasma sodium concentration
Osmotic demyelination
Extrapontine
Pontine
Demyelination
Neuron
Rapid onset of acute hyponatremia
Rapid correction of chronic hypernatremia
Rapid decrease in plasma sodium concentration
Cerebral edema
Uncal herniation


Serum sodium <135 meq/L
Asymptomatic
Symptomatic
• Fluid restriction
• Demeclocycline
• V2 receptor antagonists
Severe cerebral edema
- Active seizures or respiratory failure
Hyponatremic encephalopathy
- Seizures
- Decreased mental status
- Headache, nausea, vomiting
• Bolus of 100 mL of 3% NaCl over 10 minutes
• Repeat once or twice if necessary to increase serum sodium 2–4 meq/L or until clinical improvement
• Begin infusion used for hyponatremic encephalopathy
• Infuse 3% NaCl in ICU using infusion pump
• Check serum sodium every 2 hours until symptoms resolve
• Stop infusion when patient is symptom free or serum sodium has increased by 15–20 meq/L in the first 48 hours of therapy

Emergency treatment

- Patients with severe symptoms due to hyponatremia, such as seizures or obtundation.
- Patients with acute hyponatremia who have symptoms due to hyponatremia, **even if such symptoms are mild**.
- Patients with hyperacute hyponatremia due to self-induced water intoxication, **even if there are no symptoms** at the time of initial evaluation.
- Symptomatic patients who have either acute postoperative hyponatremia or hyponatremia associated with intracranial pathology.

Treatment of Hyponatremia

3% saline 100cc X 3

Aggressive initial correction at a rate of 1.5 to 2 meq/L per hour, is indicated for the first three to four hours or until the symptoms resolve.

Plasma sodium concentration should be raised by no more than 9 meq/L in the first 24 hours and no more than 18 meq/L in the first 48 hours

Osmotic demyelination syndrome

- Overly rapid correction of severe hyponatremia (serum sodium concentration is almost always 120 meq/L or less and usually 115 meq/L or less)

- High risk for osmotic demyelination
  - Na < 105 meq/L
  - hypokalemia, alcoholism, malnutrition, and liver disease.

- Low risk for osmotic demyelination
  - hyperacute hyponatremia (few hours) due to a marked increase in water intake, as can occur in marathon runners, patients with primary polydipsia, and users of ecstasy.

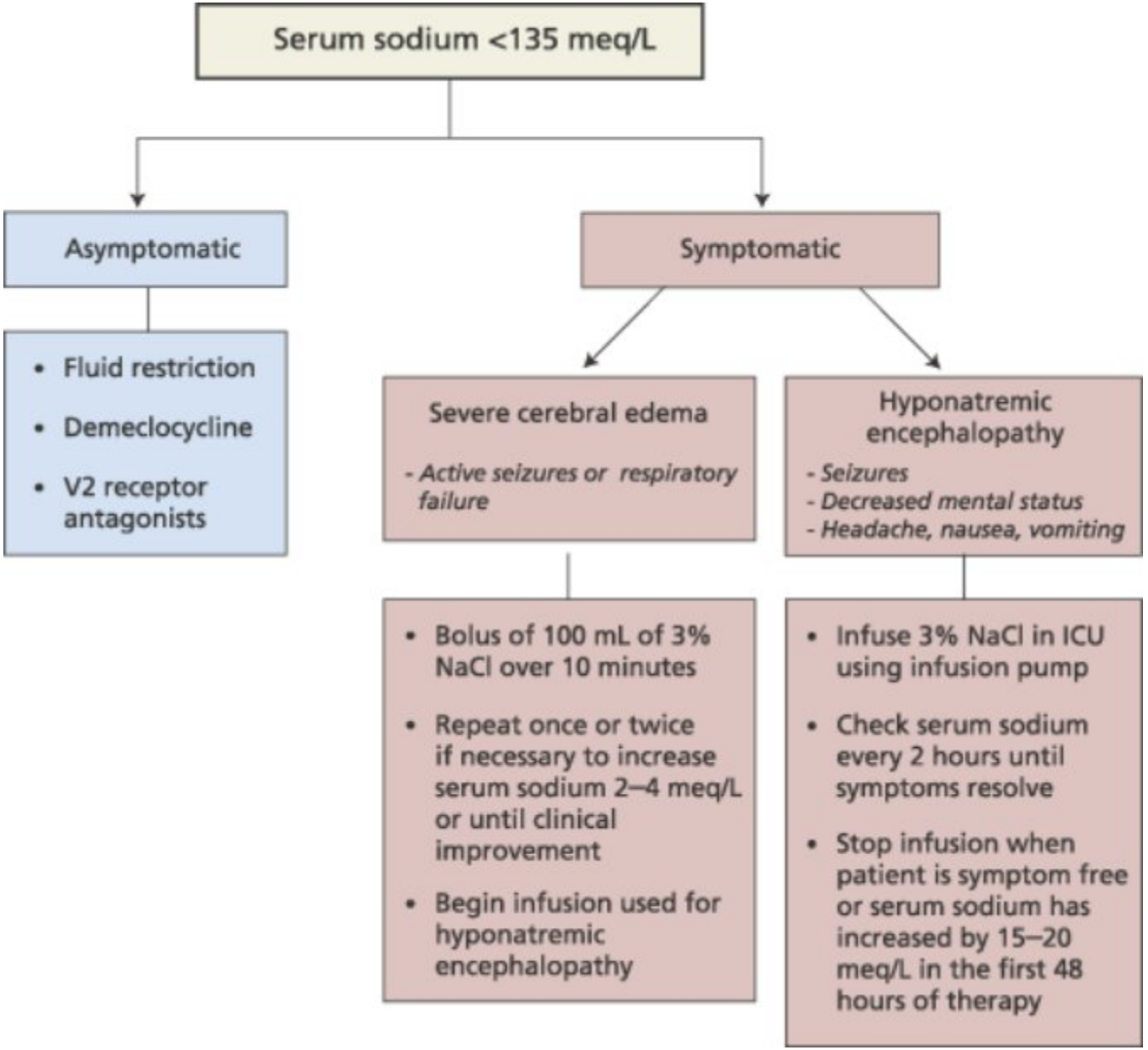
Herniation

reported almost exclusively in the following settings:

- Women and children with acute postoperative hypoNa

- Hyperacute hyponatremia caused by massive water ingestion associated with psychosis, marathon running, or use of the recreational drug, ecstasy

- Hyponatremic patients with intracranial pathology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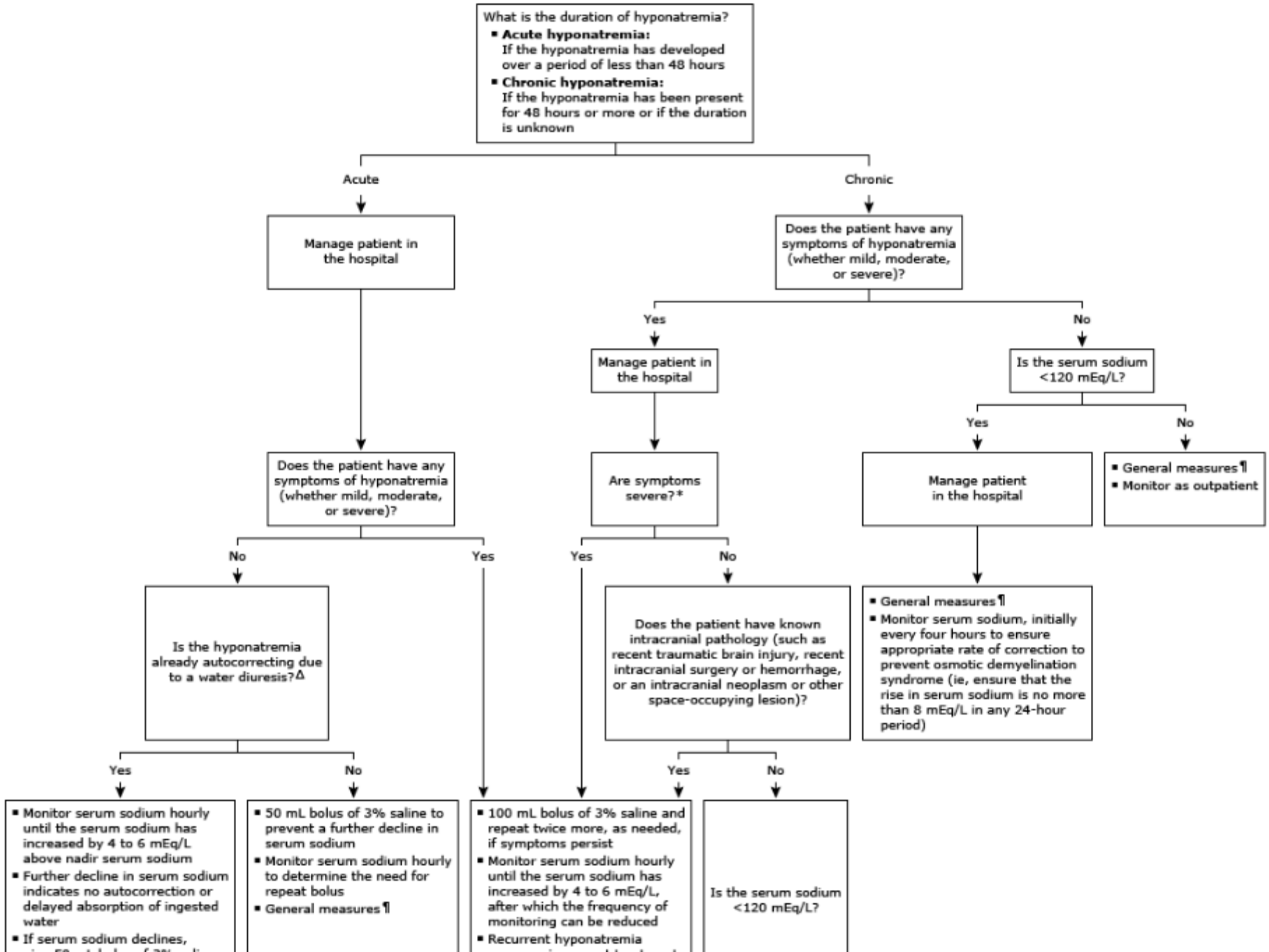
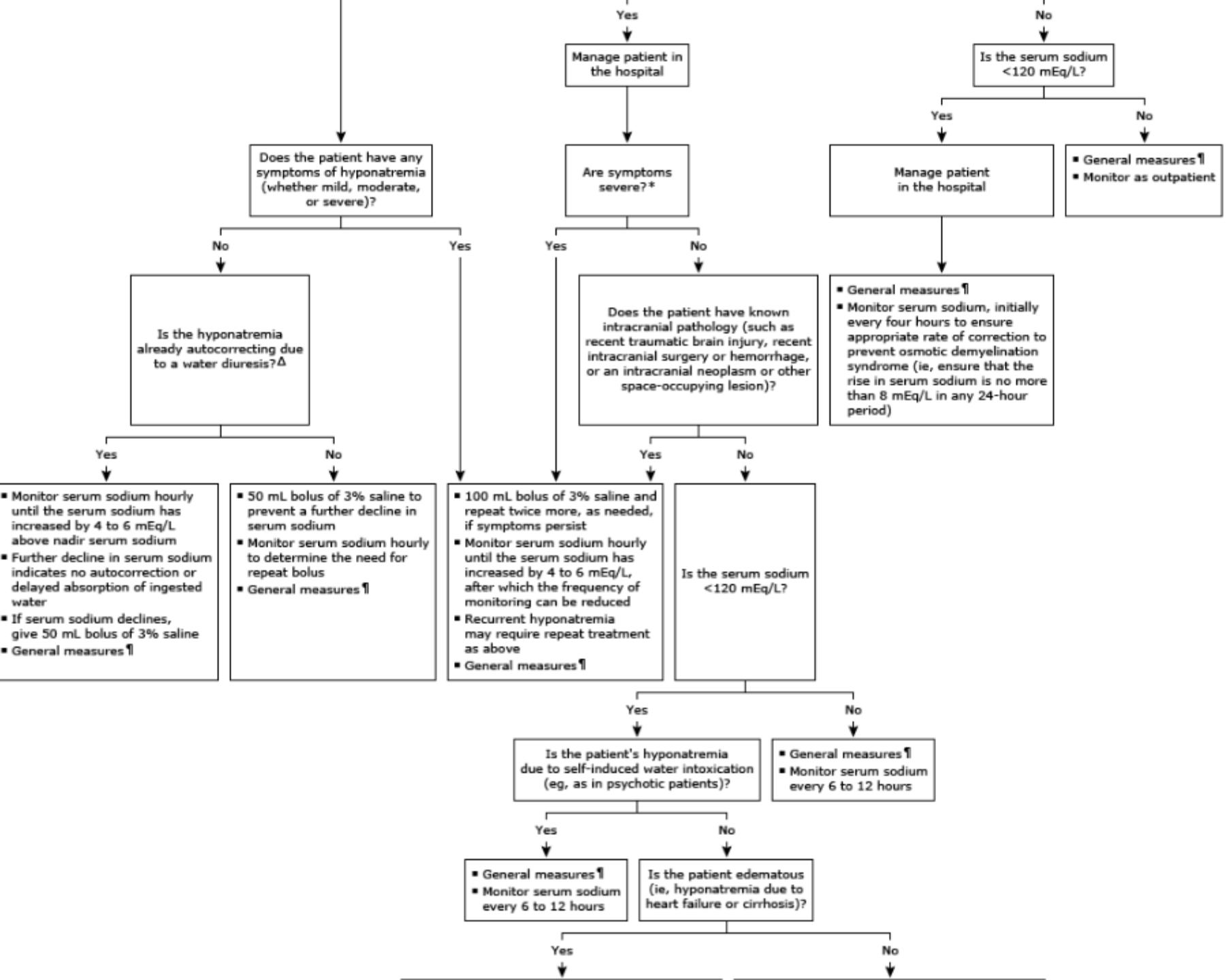
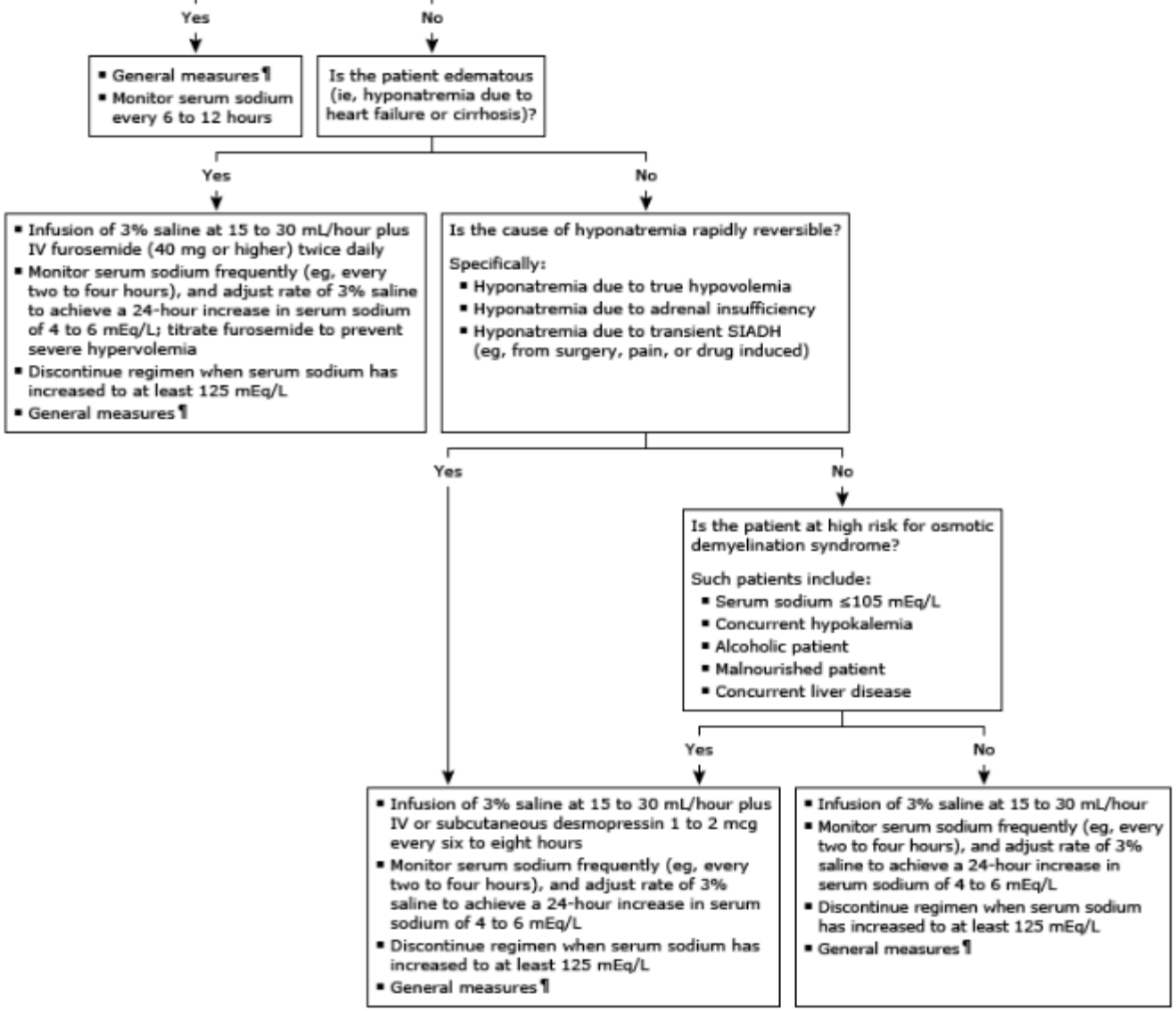
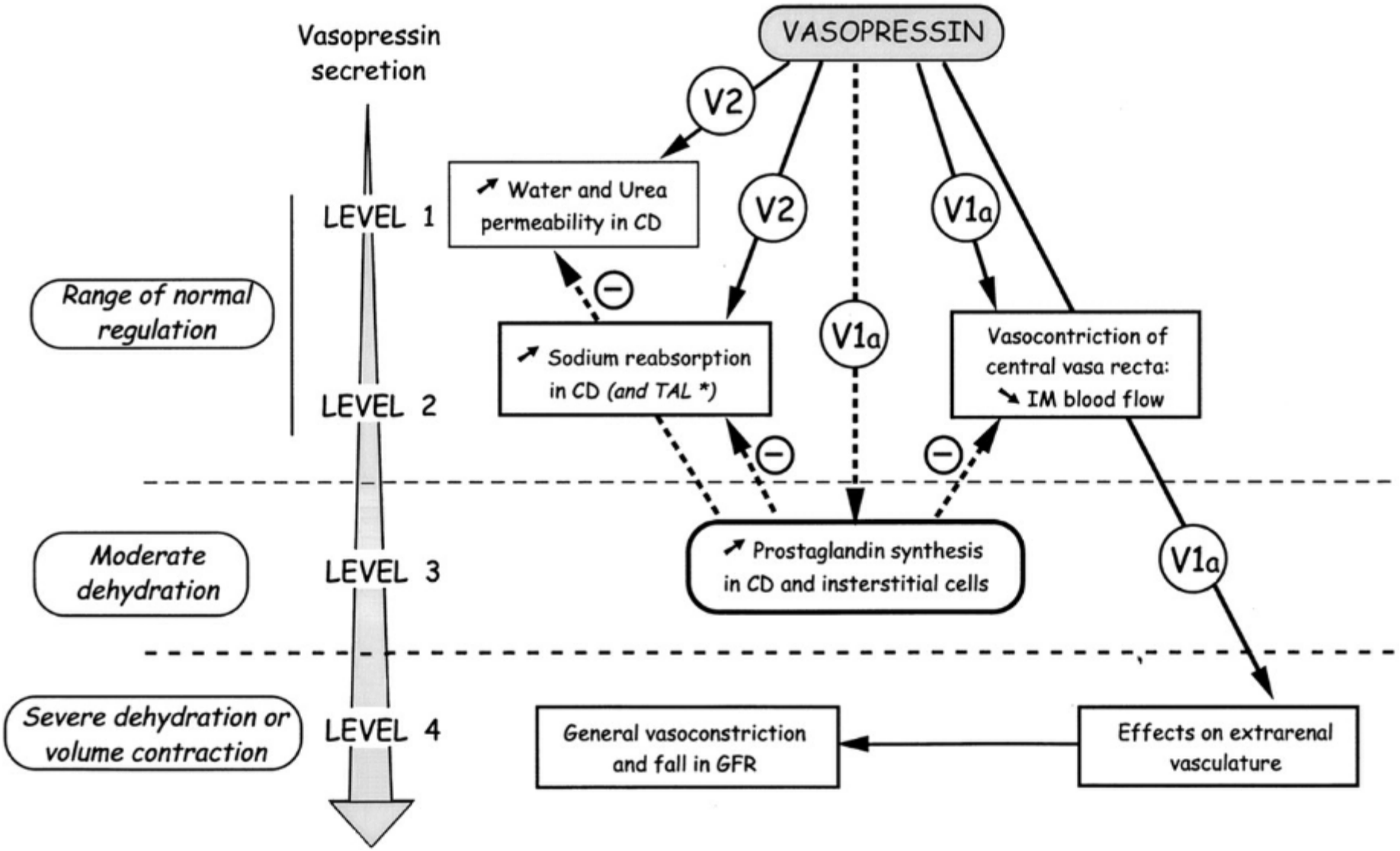

What is the duration of hyponatremia?
Acute hyponatremia: If the hyponatremia has developed over a period of less than 48 hours
Chronic hyponatremia: If the hyponatremia has been present for 48 hours or more or if the duration is unknown
Acute
Chronic
Manage patient in the hospital
Does the patient have any symptoms of hyponatremia (whether mild, moderate, or severe)?
Yes
No
Manage patient in the hospital
Is the serum sodium <120 mEq/L?
Yes
No
Manage patient in the hospital
General measures¶
Monitor as outpatient
Does the patient have any symptoms of hyponatremia (whether mild, moderate, or severe)?
Are symptoms severe?*
No
Yes
Yes
No
Is the hyponatremia already autocorrecting due to a water diuresis?Δ
Does the patient have known intracranial pathology (such as recent traumatic brain injury, recent intracranial surgery or hemorrhage, or an intracranial neoplasm or other space-occupying lesion)?
General measures¶
Monitor serum sodium, initially every four hours to ensure appropriate rate of correction to prevent osmotic demyelination syndrome (ie, ensure that the rise in serum sodium is no more than 8 mEq/L in any 24-hour period)
Yes
No
Yes
No
Monitor serum sodium hourly until the serum sodium has increased by 4 to 6 mEq/L above nadir serum sodium
Further decline in serum sodium indicates no autocorrection or delayed absorption of ingested water
50 mL bolus of 3% saline to prevent a further decline in serum sodium
Monitor serum sodium hourly to determine the need for repeat bolus
General measures¶
100 mL bolus of 3% saline and repeat twice more, as needed, if symptoms persist
Monitor serum sodium hourly until the serum sodium has increased by 4 to 6 mEq/L, after which the frequency of monitoring can be reduced
Recurrent hyponatremia
Is the serum sodium <120 mEq/L?


Yes
Manage patient in the hospital
Are symptoms severe?*
Yes
No
Does the patient have known intracranial pathology (such as recent traumatic brain injury, recent intracranial surgery or hemorrhage, or an intracranial neoplasm or other space-occupying lesion)?
Yes
No
100 mL bolus of 3% saline and repeat twice more, as needed, if symptoms persist
Monitor serum sodium hourly until the serum sodium has increased by 4 to 6 mEq/L, after which the frequency of monitoring can be reduced
Recurrent hyponatremia may require repeat treatment as above
General measures¶
Is the serum sodium <120 mEq/L?
Yes
No
Is the patient's hyponatremia due to self-induced water intoxication (eg, as in psychotic patients)?
Yes
No
General measures¶
Monitor serum sodium every 6 to 12 hours
Is the patient edematous (ie, hyponatremia due to heart failure or cirrhosis)?
Yes
No
No
Is the serum sodium <120 mEq/L?
Yes
No
Manage patient in the hospital
General measures¶
Monitor serum sodium, initially every four hours to ensure appropriate rate of correction to prevent osmotic demyelination syndrome (ie, ensure that the rise in serum sodium is no more than 8 mEq/L in any 24-hour period)
General measures¶
Monitor as outpatient
Does the patient have any symptoms of hyponatremia (whether mild, moderate, or severe)?
No
Yes
Is the hyponatremia already autocorrecting due to a water diuresis?Δ
Yes
No
Monitor serum sodium hourly until the serum sodium has increased by 4 to 6 mEq/L above nadir serum sodium
Further decline in serum sodium indicates no autocorrection or delayed absorption of ingested water
If serum sodium declines, give 50 mL bolus of 3% saline
General measures¶
50 mL bolus of 3% saline to prevent a further decline in serum sodium
Monitor serum sodium hourly to determine the need for repeat bolus
General measures¶

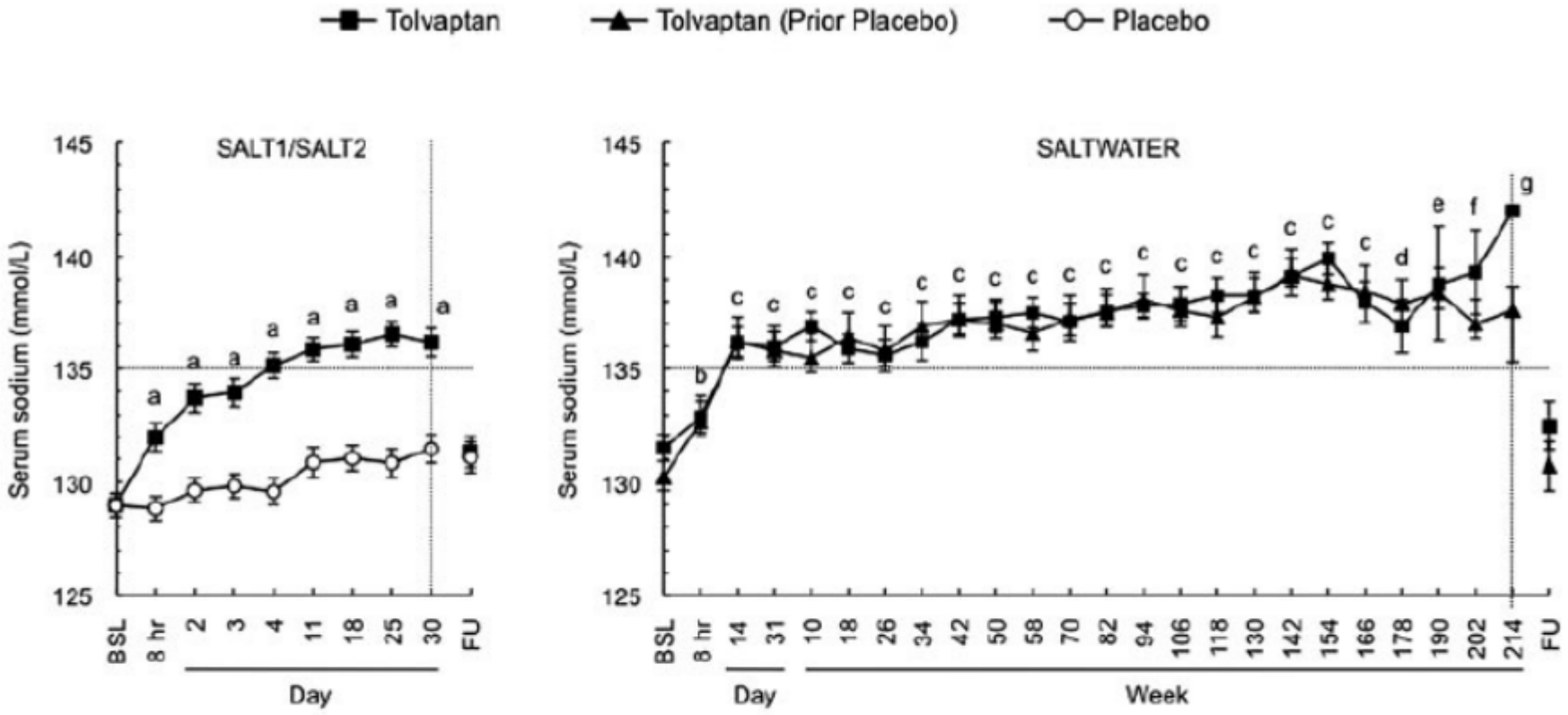

Yes
No
General measures¶
Monitor serum sodium every 6 to 12 hours
Is the patient edematous (ie, hyponatremia due to heart failure or cirrhosis)?
Yes
No
Infusion of 3% saline at 15 to 30 mL/hour plus IV furosemide (40 mg or higher) twice daily
Monitor serum sodium frequently (eg, every two to four hours), and adjust rate of 3% saline to achieve a 24-hour increase in serum sodium of 4 to 6 mEq/L; titrate furosemide to prevent severe hypervolemia
Discontinue regimen when serum sodium has increased to at least 125 mEq/L
General measures¶
Is the cause of hyponatremia rapidly reversible?
Specifically:
Hyponatremia due to true hypovolemia
Hyponatremia due to adrenal insufficiency
Hyponatremia due to transient SIADH (eg, from surgery, pain, or drug induced)
Yes
No
Is the patient at high risk for osmotic demyelination syndrome?
Such patients include:
Serum sodium ≤105 mEq/L
Concurrent hypokalemia
Alcoholic patient
Malnourished patient
Concurrent liver disease
Yes
No
Infusion of 3% saline at 15 to 30 mL/hour plus IV or subcutaneous desmopressin 1 to 2 mcg every six to eight hours
Monitor serum sodium frequently (eg, every two to four hours), and adjust rate of 3% saline to achieve a 24-hour increase in serum sodium of 4 to 6 mEq/L
Discontinue regimen when serum sodium has increased to at least 125 mEq/L
General measures¶
Infusion of 3% saline at 15 to 30 mL/hour
Monitor serum sodium frequently (eg, every two to four hours), and adjust rate of 3% saline to achieve a 24-hour increase in serum sodium of 4 to 6 mEq/L
Discontinue regimen when serum sodium has increased to at least 125 mEq/L
General measures¶


Vasopressin secretion
VASOPRESSIN
V2
V2
V1a
V1a
V1a
Water and Urea permeability in CD
Sodium reabsorption in CD (and TAL *)
Vasocontriction of central vasa recta: IM blood flow
Prostaglandin synthesis in CD and insterstitial cells
LEVEL 1
LEVEL 2
LEVEL 3
LEVEL 4
Range of normal regulation
Moderate dehydration
Severe dehydration or volume contraction
General vasoconstriction and fall in GFR
Effects on extrarenal vasculature

ADH antagonists

- Tolvaptan → V2 receptor
- Conivaptan → both the V2 and V1a receptors.

- Double-blind, placebo-controlled multicenter trials (SALT-1 and SALT-2) in 448 patients with hyponatremia (mean serum sodium 129 meq/L) caused by SIADH, heart failure, or cirrhosis
- Open-label extension (called SALTWATER),111 patients were treated with tolvaptan for a mean follow-up of 2 years. The mean serum sodium was maintained at more than 135 meq/L, compared with 131 meq/L at baseline. The responses were similar in SIADH and heart failure and were more modest in cirrhosis.



Tolvaptan n =	56	54	56	55	54	55	55	56	56	54
Placebo n =	55	54	55	55	53	55	55	52	54	52

Tolvaptan n =	56	52	55	53	46	45	44	44	40	39	38	30	28	27	27	23	23	20	17	14	9	6	3	2	33
Tolvaptan n = (Prior placebo)	55	47	52	51	48	43	46	41	40	40	37	27	28	26	23	20	19	18	17	13	8	8	5	5	35

- The US Food and Drug Administration (FDA) warns that tolvaptan should **not** be used in any patient for longer than 30 days and should **not** be given at all to patients with liver disease (including cirrhosis)

A case worthy of Dr House…

82 yr WF brought to ER by husband for altered mental status, worse over the past week.

ROS: poor PO intake, 20 pound weight loss x 2 months

PMH: CAD, PVD

PSH: L fem-pop bypass

SH: married, no etoh/RDA, 120 p.y. tobacco use, still smokes.

Meds on admission: none

Examination

- Vital signs: T 36.6 deg. cent, BP 105/54, HR 90, Wt. 50kg, Ht. 63 in.
- Gen: thin, poorly nourished
- HEENT: edentulous, no jvd
- Lungs: coarse bs bilaterally with decreased BS left base and mild exp wheeze.
- Cor: rrr, no mrg
- Abd: soft, nt nd
- Extr: no C/C/E.
- Neuro: non focal, a and o x 1 (person)

Labs

- Na 123, K 4.0, Cr 0.8, BUN 12, Uric Acid 4.
- Hgb 13, hct 38, wbc 7, plt 200
- Plasma osmolarity 250
- Urine osmolarity 616, urine sodium 40.

Tests

- CT head age appropriate cerebral atrophy
- CXR: possible left lower lobe mass
- Renal US: increased echogenicity bilateral, normal sized kidneys without hydronephrosis.

Sodium Deficit

- Na+ deficit = TBW X [Na+] deficit per liter.
- Na+ deficit = 0.5 X lean body weight (kg) X (135 -123).
  = (0.5 X 50 kg) X 12 = 300 mEq
- The patient is admitted to the hospital and started on 0.9% NS @ 150 cc/hr X total of 2 liters (Na = 154 meq/L, 2L = 308 meq/L) by the intern.
- chest ct is scheduled and a nephrology consultation is requested.

The intern checks the Na next morning.

- serum sodium is 118
- patient is lethargic and has increased confusion.

What happened?

- U Osm = 616 mosmol/liter
- 0.9% NS is 154 mEq/L of Na + Cl = 308 mosmol/kg.
- For each L of 0.9 NS administered, the NaCl (308 mOsm/L) is excreted @ 616 mosm/L ie in 500 cc of urine output (and 500 cc of free water is retained!)

Hindsight is 20/20

- s/p 2L 0.9NS → 1L free H2O has been retained.
- Initial TBW (0.5 X 50 kg) = 25L
- Initial TBNa = 123 meq/L x 25 L = 3075 meq.
- New TBW = 25 + 1L = 26L (TBNa unchanged)
- New serum Na = 3075 meq/26L = **118 meq/L!**

Given the worsening hyponatremia and increased symptoms, the resident decides to raise her serum Na to 125 meq/liter over the next 12 hours.

How do we accomplish that?

Goal Na = 125 meq/L (Current Na = 118 meq/L)

Na Deficit (7 meq/L x 26L) = 182 meq

Since 3% NaCl = 514 meq/L, volume needed = 182/514 = 354 mL

She was started on 354/12 = 30 cc/hr

Urine 616 mosmol/kg	NaCl, mosmol	Water, mL
Isotonic Saline		
In	**308**	**1000**
Out	**308**	**500**
Net	**0**	**500**
Hypertonic Saline		
In	**1026**	**1000**
Out	**1026**	**1660**
Net	**0**	**-660**
Hypertonic Saline + Loop diuretic (decrease U Osm to 300 mosm/L)		
In	**1026**	**1000**
Out	**1026**	**3420**
Net	**0**	**-2420**

Questions?

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