

FLUID AND ELECTROLYTE DISORDERS

Jennifer Bryan, MD, FRCP

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with slides From Dr. Carly Ng, MD, CCFP(EM) and Dr. Howard Ovens, MD, CCFP (EM)

Toronto Addis Ababa Academic Collaboration in Emergency Medicine

THIS SESSION WILL BE RECORDED

We are recording this zoom session so that it can be watched again at your convenience, and so that we can share it with your colleagues who were not able to join us today.

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We will share the presentation slides and other materials (journal articles, etc.) By email; you will have access to all materials regardless of whether the recording is shared.

PLEASE ALSO NOTE:

The information in this presentation and the video recording is up to date as of the date it was recorded (September 29, 2020)

It has not been updated to include any subsequent advances in practice, and the information presented in this video does not replace hospital, health centre, or governmental guidelines.

DISCLOSURE STATEMENT

I have not received any financial or in-kind support from any commercial organization and have no conflicts of interest to declare.

RESOURCES

- **Readings:** Tintinalli's 9th edition, ch. 17 (fluids and electrolytes)
- **Optional additional resource:** EM Cases episode 86
(<https://emergencymedicinecases.com/emergency-management-hyperkalemia/>)

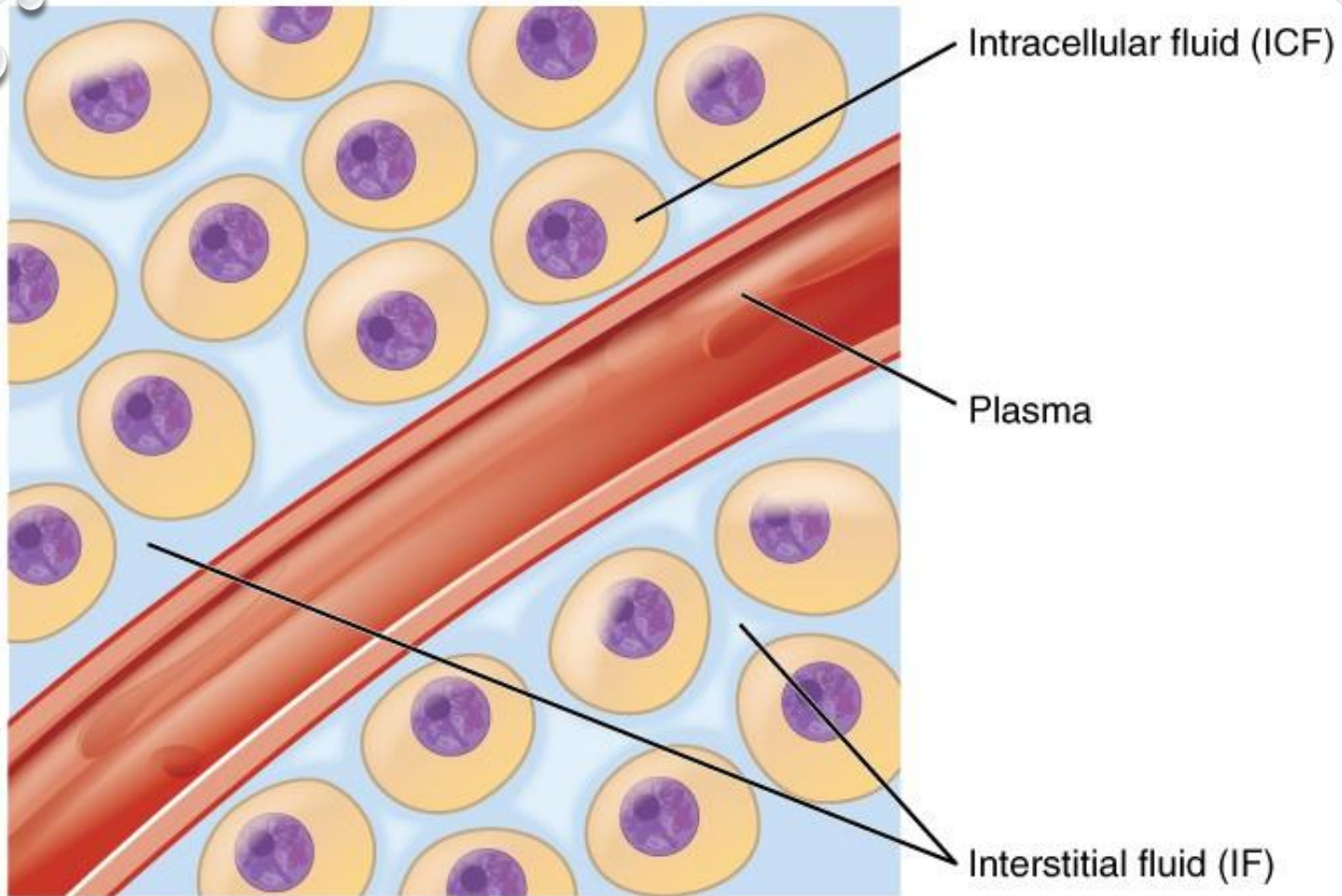
OBJECTIVES

- Review fluid replacement
- Case based approach to common electrolyte disorders in the ED
- Emphasis on practical applications
- ...this is not an internal medicine or nephro review

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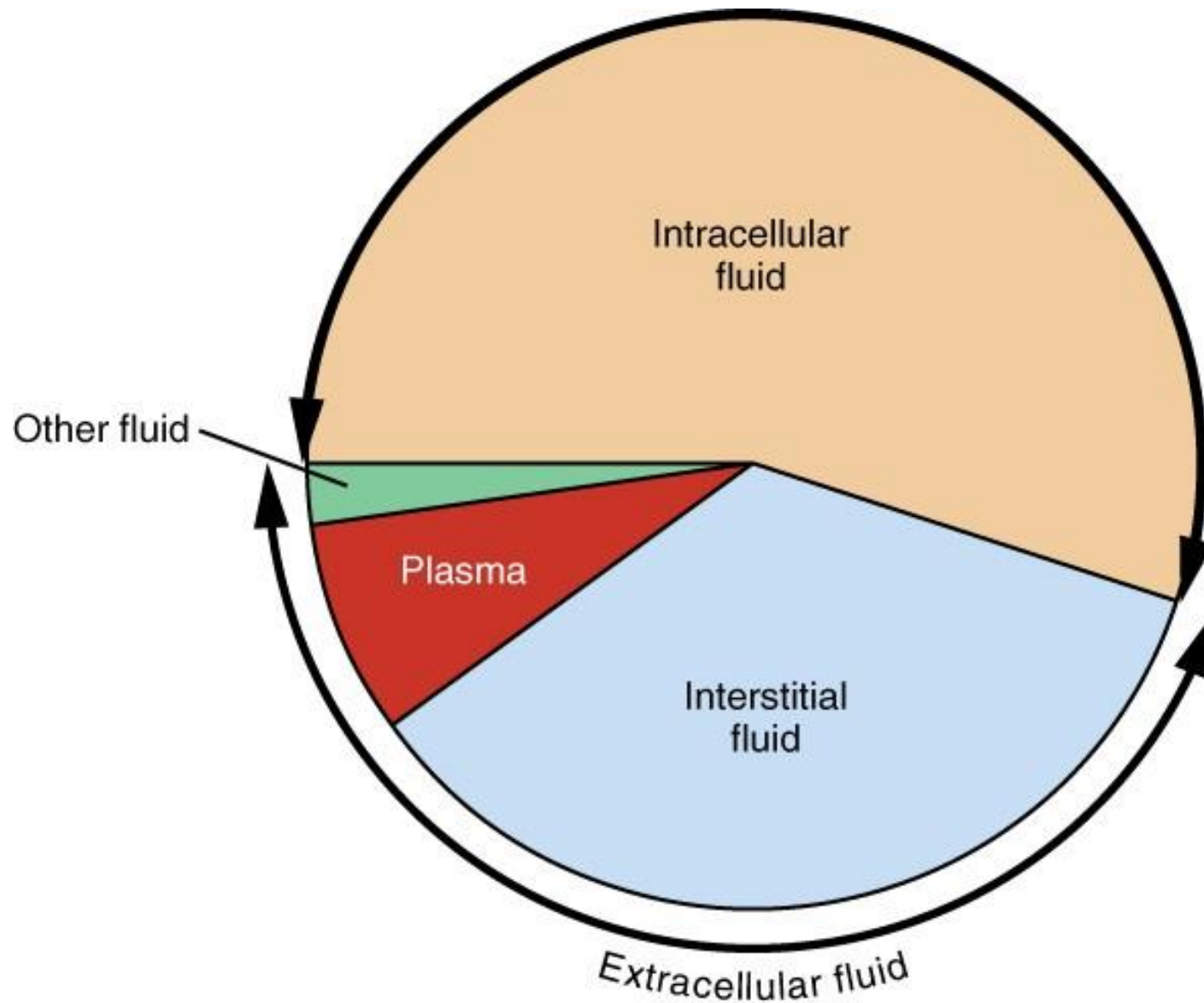
FLUID REPLACEMENT AND MAINTENANCE

FLUID COMPARTMENTS



- Extracellular
 - Intravascular (plasma)
 - Interstitial
- Intracellular

<https://courses.lumenlearning.com/suny-ap2/chapter/body-fluids-and-fluid-compartments-no-content/>



	Plasma	Interstitial	Intracellular	NS	RL
Sodium mEq/L	142	144	10	154	130
Potassium mEq/L	4	4.5	150	5	5

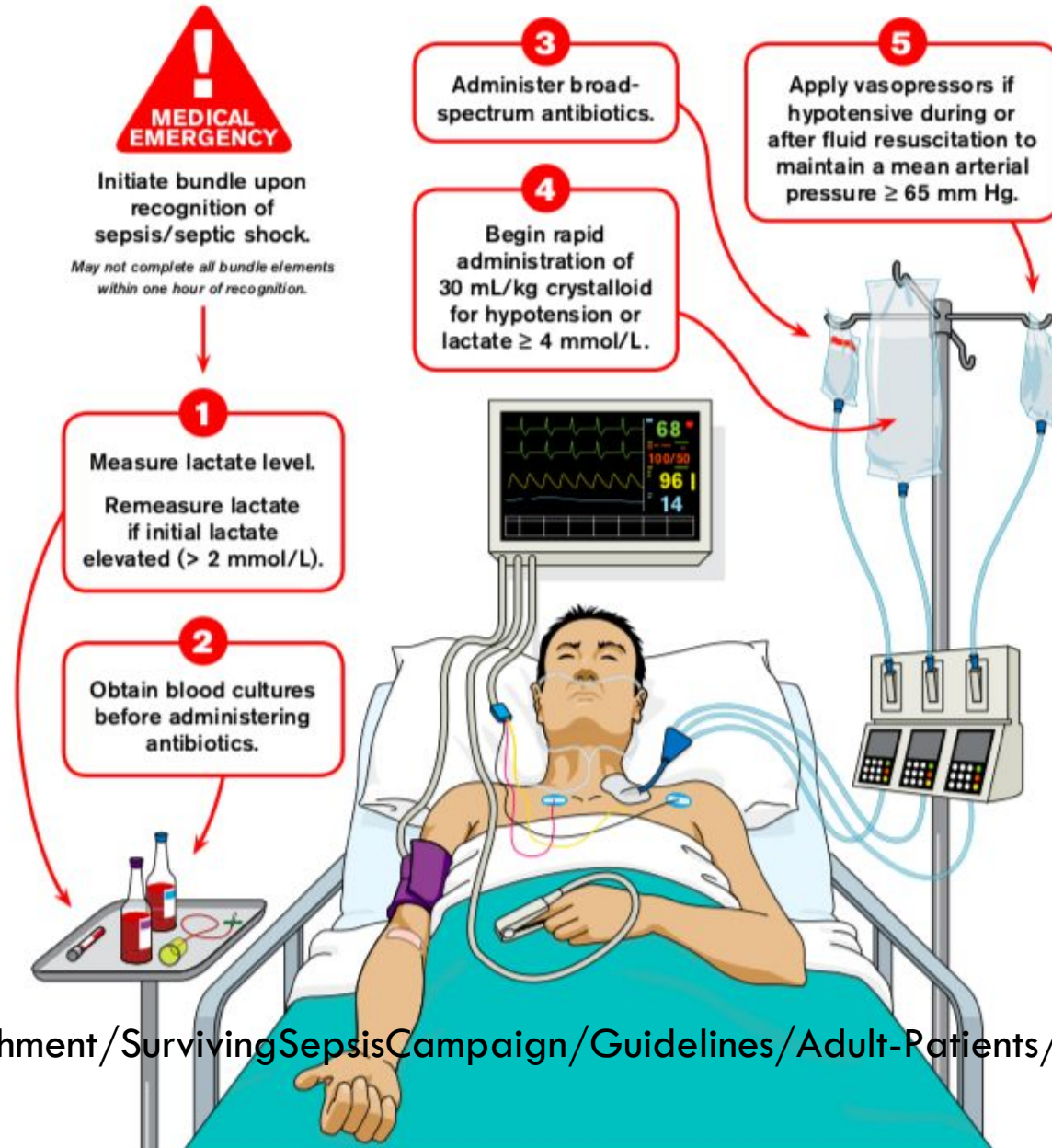
TONICITY

- Osmotic pressure gradient between two solutions across a semipermeable membrane

	Plasma	Interstitial	intracellular	NS	RL
Osmolality	285-295	285-295	285-295	286	254

Hour-1 Bundle

Initial Resuscitation for Sepsis and Septic Shock





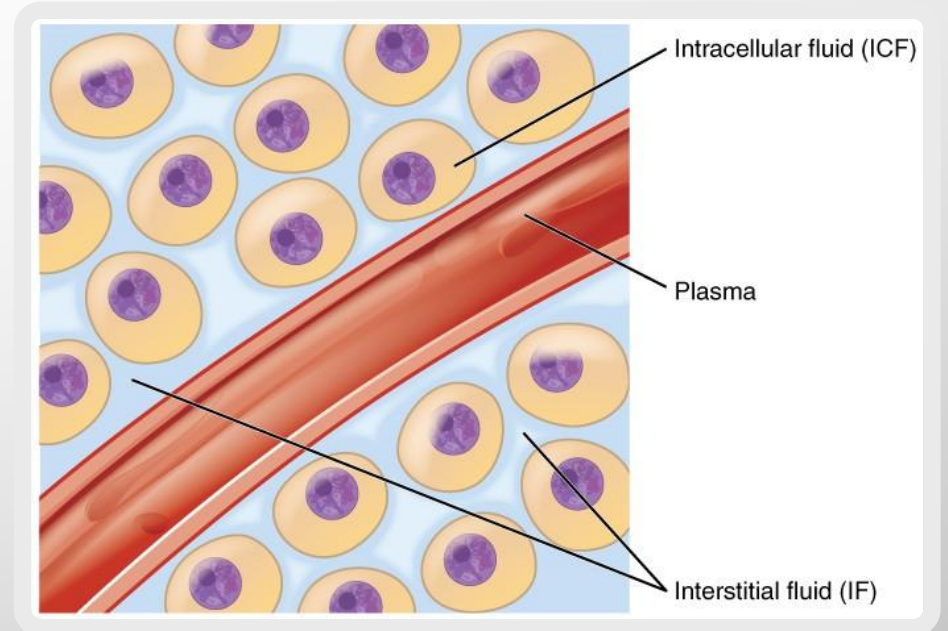
**WHY DO WE NEED VOLUME RESUSCITATION?
WHERE DOES THE FLUID GO?**

WHY DO WE NEED VOLUME RESUSCITATION? WHERE DOES THE FLUID GO?

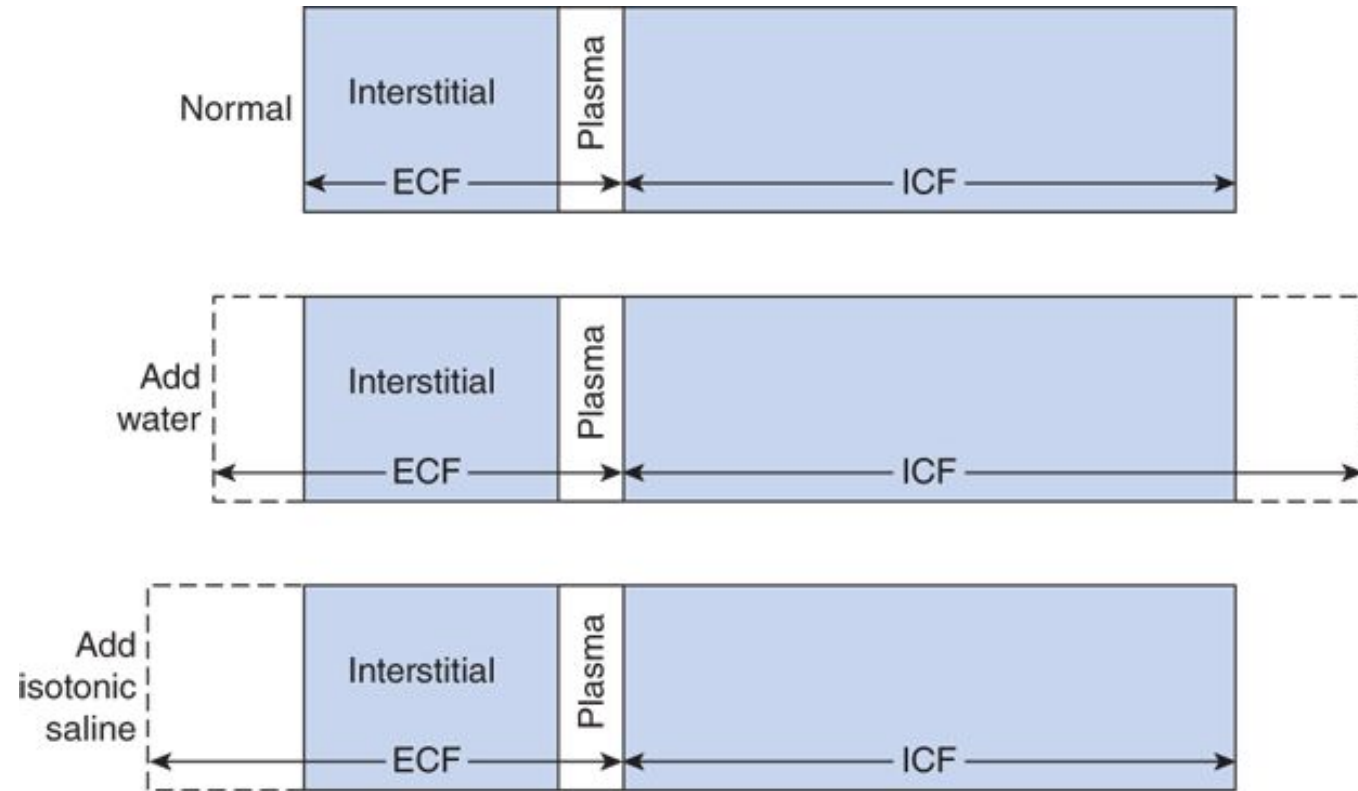
- Decreased intake
- Increased output (renal, GI)
- Hemorrhage
- Increased insensible losses (sweating, respiratory losses)
- Alteration in vascular resistance (“vascular leak”)

CHOOSING A RESUSCITATION FLUID

- Why don't we just use free water?



<https://courses.lumenlearning.com/suny-ap2/chapter/body-fluids-and-fluid-compartments-no-content/>



CRYSTALLOIDS vs COLLOIDS

Crystalloids

Cheap

Easy to use

Increased edema

Colloids

More expensive

Potentially more directed intravascular fluid

Risk of allergic rxn, clotting, AKI

Lewis, Sharon R., et al. "Colloids versus crystalloids for fluid resuscitation in critically ill people." *Cochrane Database of Systematic Reviews* 8 (2018).

<https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD000567.pub7/full>

CASE 1

- 65y M with urosepsis
- Appears dry – BP 100/60, HR 120, RR 16, T37, wt 70kg
- Confused
- How to calculate fluid replacement?



How dry is he?

- Estimate degree of dehydration
- Assess potential for fluid responsiveness
- Calculate free water deficit using serum sodium

Estimating degree of dehydration

- Mild (~ 5%)
 - Minimal loss of skin turgor, semidry mucous membranes, normal eye
- Moderate (~ 8-10%)
 - Moderate loss of skin turgor, dry mucous membranes, weak rapid pulses, enophthalmos
- Severe (> 10%)
 - Considerable loss of skin turgor, severe enophthalmos, tachycardia, extremely dry mucous membranes, weak/thready pulses, hypotension, altered level of consciousness

poor sensitivity and specificity

FLUID REPLACEMENT

- Correct volume to normalize hemodynamics with IV NS
- Fluid needs = Maintenance + Losses + Deficit
- Losses
 - Urine loss $\sim 1 \text{ cc/kg/hr} \sim 1500 \text{ cc/day}$
 - Insensible loss (lung, skin) $\sim 1000 \text{ cc/day} + 500 \text{ cc}$ for each degree of fever
 - Add emesis/NG or other losses

FLUID REPLACEMENT CONT'D

- Estimate deficit
 - TBW ~ 60% of weight, ~ 42 L for 70kg patient
 - E.g., 10% dehydrated = 4 litres
- In this case total fluid needs in 1st 24 hrs = 6.5L (4L + 1.5L urine + 1L insensible loss)
- After vitals normalized give half in 1st 8 hours, rest in 16 hours (avoid cerebral edema from rapid correction), titrate to urine output
 - E.g., 500 cc bolus, then 350 cc/hr X 8h (~3L), 200cc/hr X 16h (4L) = total 6.5L
- Replace K if needed

PRACTICALLY SPEAKING...

Start with a fluid bolus based on clinical presentation and physical exam (30cc/kg)

Check IVC for potential fluid responsiveness

Check biomarkers if available

Encourage po fluid intake

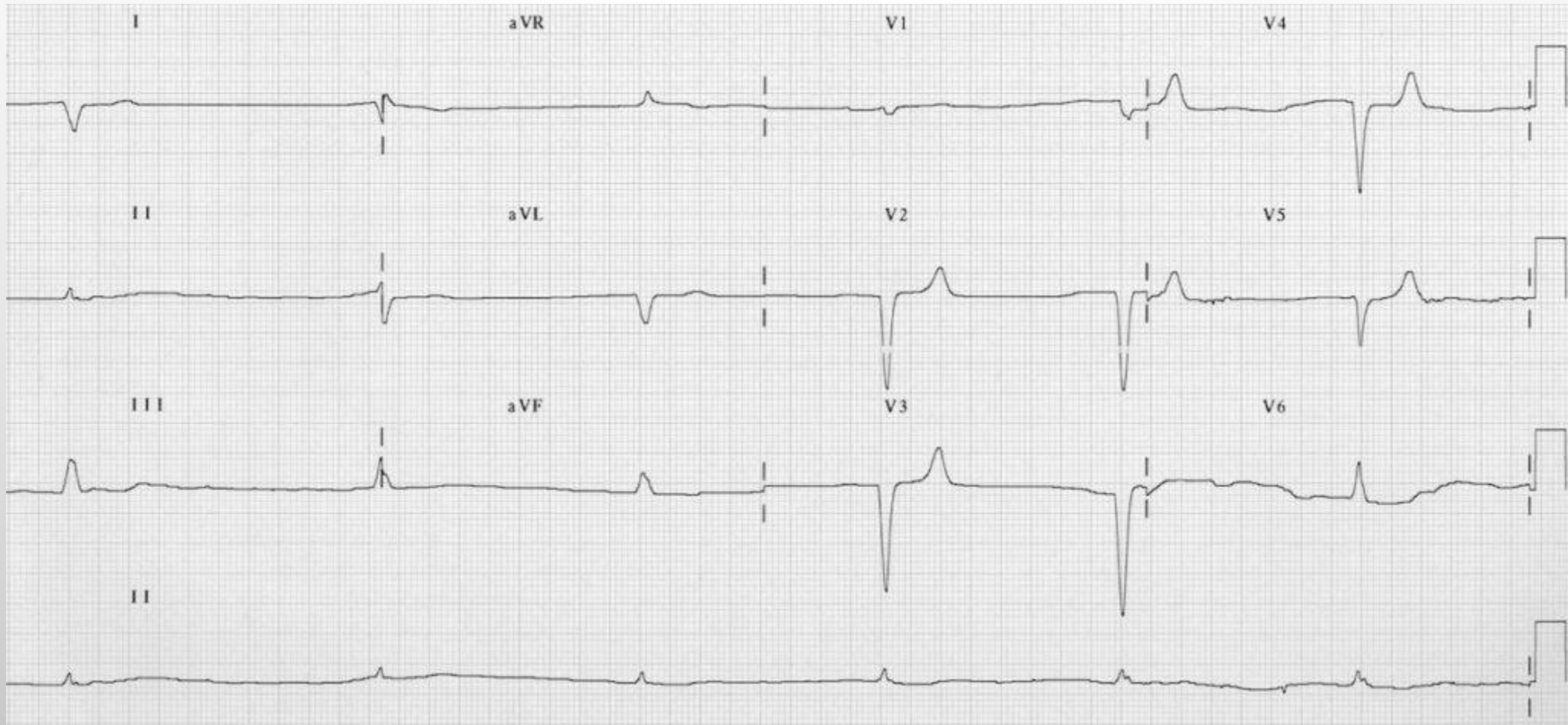
Track urine output

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ELECTROLYTE ABNORMALITIES

CASE 2

- 55y F cc: syncope
- MedHx: CRF due to hypertension, no cardiac hx
- Medications: enalapril, ibuprofen for knee pain
- HPI: Feeling increasingly fatigued, two episodes syncope without prodrome today
- Vitals: HR 40, BP 80/60, T 36.5, RR 18, random 116



<https://litfl.com/>

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WHAT'S CAUSING HER BRADYCARDIA?

POTASSIUM



<https://commons.wikimedia.org>

HYPERKALEMIA - CAUSES

- Most Common Causes
 - Pseudohyperkalemia – hemolysis from blood draw, lab error
 - **Renal failure**
 - Cell death - rhabdo, burns, crush, hemolysis
 - Drugs - spironolactone, ACEI, K supplements

Indication for dialysis of AKI patients, SPHMMC, Addis Ababa, May 2015

Dialysis indication ^a	Frequency (Percent)	
	Yes	No
Refractory Fluid Overload	135 (89.4 %)	16 (10.6 %)
Uremic signs and symptoms	93 (61.6 %)	58 (38.4 %)
Hyperkalemia	44 (29.1 %)	107 (70.9 %)
Metabolic Acidosis	14 (9.3 %)	137 (90.7 %)

Ibrahim, Ahmed, et al. "Clinical profile and outcome of patients with acute kidney injury requiring dialysis—an experience from a haemodialysis unit in a developing country." *BMC nephrology* 17.1 (2016): 1-5. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4957374/>

- A cross-sectional study was conducted from September to November 2017, on 163 CV patients attending the emergency department (ED) of TASH.
- 59 (36.2%) had serum Na⁺ imbalance and 37 (22.7%) had serum K⁺ imbalance
- Diribsa, Getahun Chala, Yekoye Abebe Kinfe, and Senbeta Guteta Abdissa. "Assessment of renal function and electrolyte balance in patients with cardiovascular disease at Tikur Anbessa Specialized Hospital, Addis Ababa, Ethiopia." *Asian Journal Of Research In Nephrology* (2019): 1-12. <https://www.journalajrn.com/index.php/AJRN/article/view/30092>

HYPERKALEMIA - SIGNS AND SYMPTOMS

- Common and life threatening
- Cardiac
 - K 6.5-7.5 – peaked T waves, short QT, prolonged PR
 - K 7.5-8.0 – widened QRS, flat P waves
 - K > 8.0 – V fib
- Neuromuscular – nonspecific
- GI – vomiting, abdo pain, diarrhea

5 STEPS TO TREAT HYPERKALEMIA

1. Stop exogenous potassium or potassium increasing medications
2. Stabilize the myocardium if there are ECG changes
3. Shift potassium into cells
4. Eliminate potassium from the body
5. Monitor

5 STEPS TO TREAT HYPERKALEMIA

1. Stop exogenous potassium or potassium increasing medications
2. Stabilize the myocardium if there are ECG changes
 - Calcium gluconate 1g of 10% over 2-3 min; can repeat in 5-15 minutes (lasts only about 20 minutes)
3. Shift potassium into cells
4. Eliminate potassium from the body
5. Monitor

5 STEPS TO TREAT HYPERKALEMIA

1. Stop exogenous potassium or potassium increasing medications
2. Stabilize the myocardium if there are ECG changes
3. Shift potassium into cells
 - Humulin R 10 units IV with 2 amp D50
 - Salbutamol 20mg neb or up to 12 puffs MDI (give after insulin/D50)
 - Sodium bicarb 1 amp IV over 15 minutes (in acidosis)
 - Consider re-shifting in 4 hours if indicated
4. Eliminate potassium from the body
5. Monitor

5 STEPS TO TREAT HYPERKALEMIA

1. Stop exogenous potassium or potassium increasing medications
2. Stabilize the myocardium if there are ECG changes
3. Shift potassium into cells
4. Eliminate potassium from the body
 - Urine
 - If hypervolemic, furosemide 40mg IV
 - If euvolemic, NS IV 500cc/h + furosemide 40mg IV
 - If hypovolemic, NS IV 500cc/h
 - Stool
 - Sodium polystyrene sulfonate (kayexalate) 30g in 30-60ml 70% sorbitol (falling out of favour)
 - Dialysis
5. Monitor

5 STEPS TO TREAT HYPERKALEMIA

1. Stop exogenous potassium or potassium increasing medications
2. Stabilize the myocardium if there are ECG changes
3. Shift potassium into cells
4. Eliminate potassium from the body
5. Monitor
 1. Random, ECG (or rhythm on monitor), repeat lytes, Cr

SODIUM POLYSTYRENE SULFONATE (KAYEXALATE)

Risk of intestinal necrosis:

“It would be wise to exhaust other alternatives for managing hyperkalemia before turning to these largely unproven and potentially harmful therapies.”

Sterns RH et al. Ion-Exchange Resins for the Treatment of Hyperkalemia: Are They Safe and Effective? J Am Soc Nephrol 2010; 21: 733-5. PMID: 20167700



<https://rebelem.com/kayexalate-useful-treatment-hyperkalemia-emergency-department/>

HYPERKALEMIA PEARLS

- Prevention!
 - Caution whenever prescribing a new K sparing drug or adding drugs to pt's on K sparing drugs
 - E.g., NSAID to someone on ACEI
- Get an ECG
- Ca first choice in life threatening emergencies
- Insulin/dextrose, beta agonist for shift

WHAT ABOUT THE OPPOSITE, HYPOKALEMIA?

- Causes?

WHAT ABOUT THE OPPOSITE, HYPOKALEMIA?

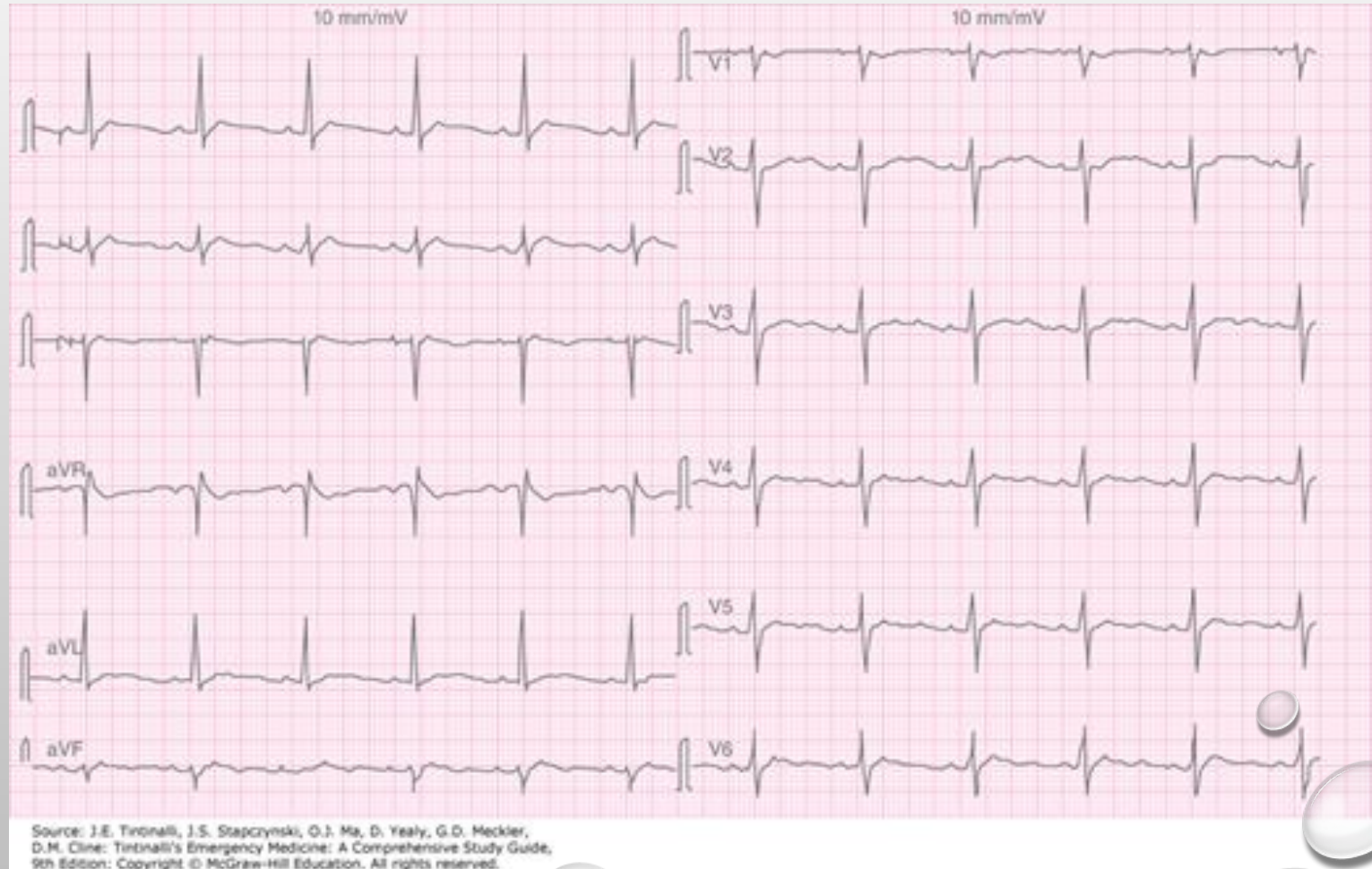
- Causes?
 - Reduced intake
 - Shift into cells (i.e., hyperK treatment)
 - Increased losses – loop diuretics , GI losses
 - Misc – Mg deficiency, licorice use

HYPOKALEMIA

- Symptoms (non-specific)
 - Neuromuscular – severe muscle weakness
 - GI – ileus
 - Cardiac – tachydysrhythmias

ECG FINDINGS OF HYPOKALEMIA

- Delayed repolarization
 - Prolonged QTc, flattened T waves, and the appearance of U waves
- Tachyarrhythmias (atrial fibrillation,³⁵ torsades de pointes, ventricular tachycardia, and ventricular fibrillation)



HYPOKALEMIA: TREATMENT

- **K 20mEq/dose = increases serum K by ~0.25mmol/L**
- Replace Mg if Mg low (Mg 0.5-1g/h)
- IV vs PO
 - Max infusion rate 20Eq/h
- If K 3.0-3.4: KCl 20-40mEq PO/day
- If K<3.0: KCl 40mEq PO q6-8h.
 - If IV infusion also running, KCl 20mEq PO q3h
- Diet: fruits, dried fruits, nuts, vegetables, and meat

CASE 3

- 22y M
- First time marathon runner
- Seizure near the end of the race, previously well
- GCS13 (E3, V4, M6), HR 60, BP 130/80, O₂100% on room air
- DDx?



SODIUM DISTURBANCES

EXERCISE-ASSOCIATED HYPONATREMIA

- As high as 23% in Ironman
- Overhydration with hypotonic fluids
- Increased antidiuretic hormone
- Possibly a role of sodium loss due to sweating



Bennett, brad L., Et al. "Wilderness medical society clinical practice guidelines for the management of exercise-associated hyponatremia: 2019 update." *Wilderness & environmental medicine* 31.1 (2020): 50-62.

https://www.Sciencedirect.Com/science/article/pii/S1080603219302066?Casa_token=erqmg_4m0rmaaaaa:knz4mfwvh6xeh1qpjedmczdrkbctzralu9zp-er_bcirecmntrtkwjx4mstumtfkefrbjcxm

HYPONATREMIA

- Most common electrolyte disorder – 20% of all admissions
- Independent predictor of mortality, $\text{Na} < 135$ at admission
- Nonspecific Symptoms:
 - Mild ($\text{Na} 130-135$): asymptomatic
 - Mod ($\text{Na} 120-129$): Nausea, weakness, headache, cramps, lethargy
 - Severe ($\text{Na} < 120$): seizures, ALOC, coma, respiratory arrest

HYPONATREMIA - CAUSES

- **Hypovolemic**

- Sweating
- Vomiting
- Diarrhea
- 3rd spacing
- Renal loss (diuretics, RTA, Addison's)

- **Euvolemic**

- SIADH
- Stress, pain
- Myxedema
- Polydipsia

- **Hypervolemic**

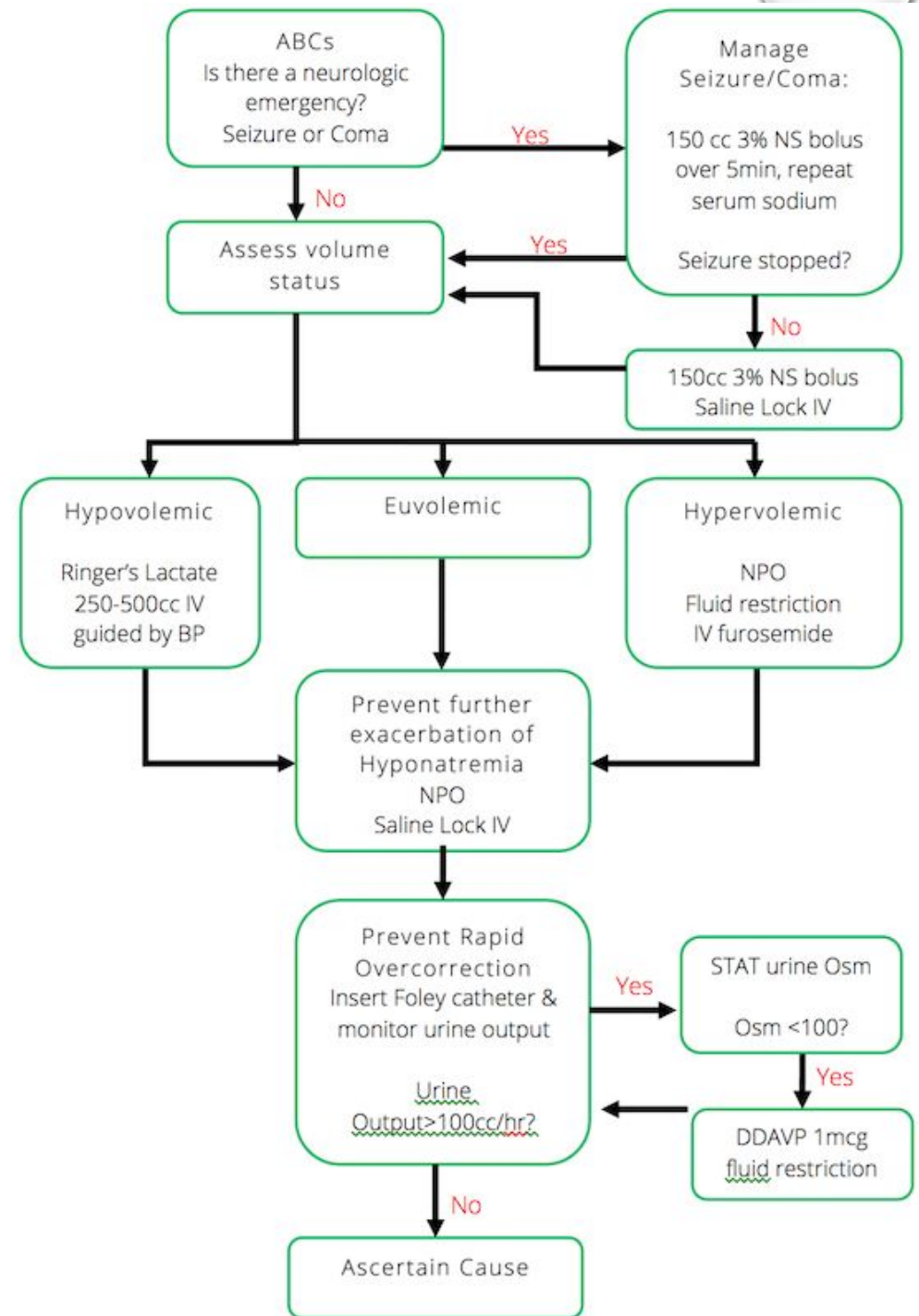
- CHF
- Cirrhosis
- Renal failure

- Common in hospitalized patients, e.g., 28.9% of admitted patients with heart failure in Jimma
- Ali, Khalid, Abdulhalik Workicho, and Esayas Kebede Gudina. "Hyponatremia in patients hospitalized with heart failure: a condition often overlooked in low-income settings." *International Journal Of General Medicine* 9 (2016): 267.

<https://www.Ncbi.Nlm.Nih.Gov/pmc/articles/PMC4977071/>

EMERGENCY APPROACH TO HYPONATREMIA

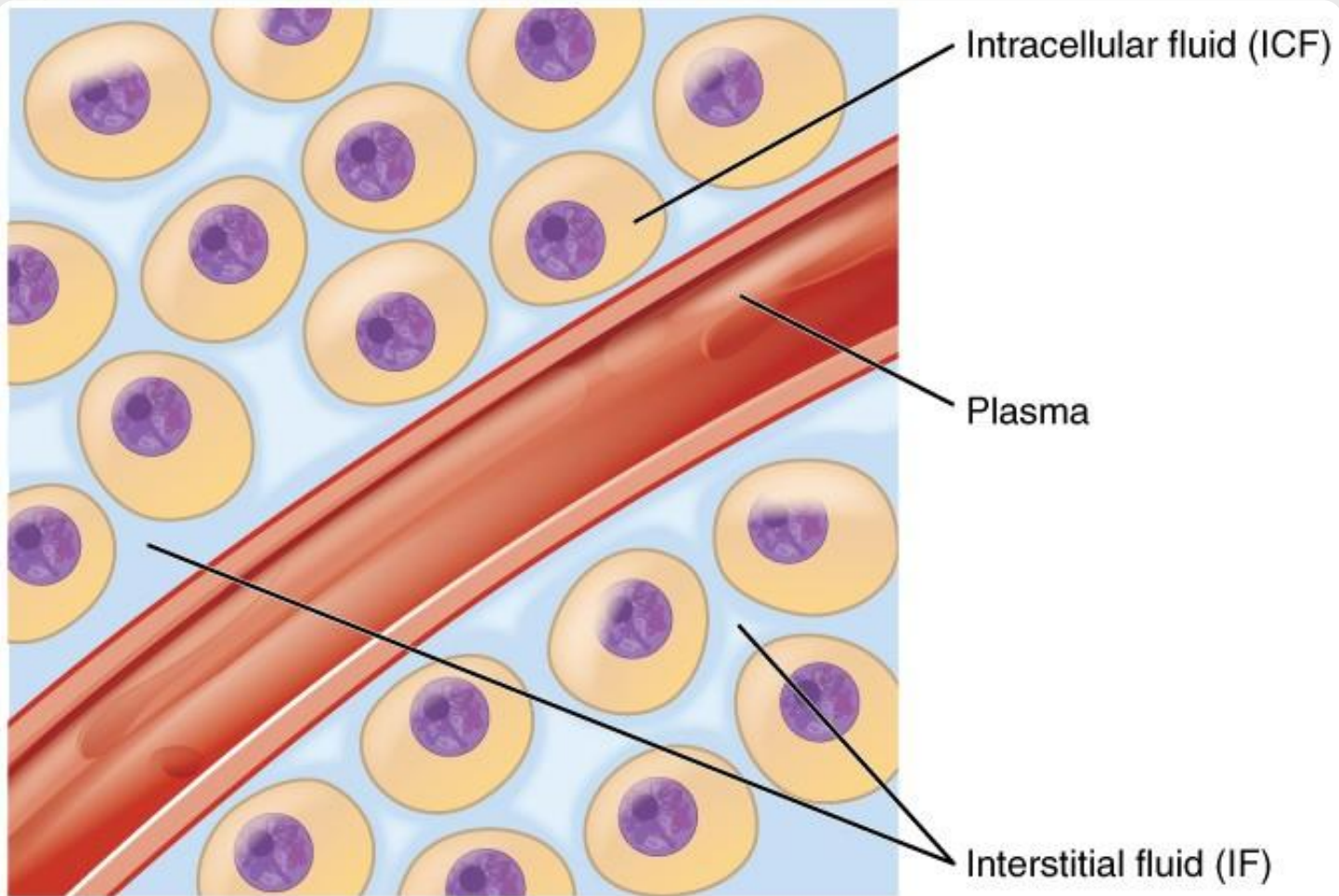
- FROM: EM CASES EPISODE 60: EMERGENCY MANAGEMENT OF HYPONATREMIA. AVAILABLE: [HTTPS://EMERGENCYMEDICINECASES.COM/EPISODE-60-EMERGENCY-MANAGEMENT-HYPONATREMIA/](https://EMERGENCYMEDICINECASES.COM/EPISODE-60-EMERGENCY-MANAGEMENT-HYPONATREMIA/)



HYPONATREMIA – NEURO EMERGENCY

- If coma/seizing/suspected herniation (from cerebral edema)
 - 3% hypertonic saline, 150cc over 3min, rpt x 1 in 30-60min prn
 - Then STOP all fluids
 - If no hypertonic saline – give 1 amp bicarb (Na!)
 - CT brain
 - Involved ICU

FLUID COMPARTMENTS



- Extracellular
 - Intravascular (plasma)
 - Interstitial
- Intracellular

<https://courses.lumenlearning.com/suny-ap2/chapter/body-fluids-and-fluid-compartments-no-content/>

HYPONATREMIA – FOCUS ON THE VOLUME

- Volume status: clinical (POCUS, edema)
 - Hypovolemic: restore the circulating volume, slowly
 - Hypervolemic: fluid restriction, diuretics
- Euvolemic: volume ok, so prevent worsening hypoNa

HYPONATREMIA – DON'T DO HARM

- Prevent overcorrection of Na
 - OSMOTIC DEMYELINATION SYNDROME
 - Dehydration of CNS cells and demyelination. Up to 7 days post rapid correction.
 - High risk: alcoholics, malnourished, liver failure, Addison's

HYPONATREMIA – RULE OF 6

- *“SIX IN SIX HOURS FOR SEVERE SYMPTOMS, THEN STOP. SIX A DAY MAKES SENSE FOR SAFETY.”*
- If neurologic emergency, correct Na max 6mmol in 6h
- All else: don't correct more than 6mmol/day

HOW MUCH SHOULD WE GIVE?

- Na^+ deficit = $0.6 \times \text{weight (kg)} \times (\text{change in Na}^+ \text{ desired})$
- 70kg patient, we want to increase by 6mmol in 6 hours
- $0.6 \times 70 \times 6 = 252$
- 3% saline has 513 mEq Na/L
- $(252/513) \times 1000 = 490\text{cc}$ over the first 6 hours

HYPONATREMIA - PEARLS

- Treat the neurologic emergency with 3% saline
- Defend the intravascular volume
 - Hypo? Give fluid
 - Hyper? Or Euvolemic? Fluid restrict
- Don't do harm
 - ODS
 - Monitor UO. If $UO > 100\text{cc/h}$ and $U_{\text{osm}} < 100$ – consider DDAVP
 - Rule of 6
- Find the cause
- $\text{Na} < 120$ requires inpatient management

HYPERNATREMIA

- Deficit in TBW (rarely increase in sodium)
- Usually would fix this ourselves by drinking water
 - Some underlying condition that: impairs the patient's sense of thirst, limits the availability of water, limits the kidney's ability to concentrate urine, or results in increased salt intake
 - Loss of free water in diarrhea
 - Acute onset can prompt intracranial hemorrhage
- Hypo/hyper/euvolemic
- Mortality 75% if Na greater than 150

HYPERNATREMIA

- Nonspecific symptoms
 - Irritability, lethargy, spasticity, coma, seizures
- High M&M in young and elderly – unable to respond to increased thirst
- Caused by
 - Decreased total body water
 - Decreased intake
 - Increased loss
 - V/D; sweating/fever; Diabetes Insipidus; meds (lithium, dilantin); burns
 - Inc'd Na (intake, endocrine)

HYPERNATREMIA: TREATMENT

- Correct volume deficit
 - $TBW \times (1 - \text{target Na} / \text{measured Na})$
- Max decrease = 10mmol/day

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CALCIUM AND MAGNESIUM

CALCIUM DISORDERS

- Hypocalcemia = $\text{Ca} < 1.5$
 - Paresthesias, cramps, weakness, confusion, seizures
 - ECG – long QT
 - Chvostek's sign = twitch over corner of mouth with tap at CN7 at zygoma
 - Trousseau's sign = carpal spasm when BP cuff inflated $> 3\text{min}$
 - Tx = Replace Ca (Po CaCarbonate, CaGluconate; IV Ca)
- Hypercalcemia = $\text{Ca} > 2.7$
 - Stones (renal), Bones (osteolysis), psychic Moans (lethargy, weakness, fatigue, confusion), Groans (abdo cramping)
 - ECG – ST dep, shortened QT, heart blocks
 - Tx = NS IV bolus, bisphosphonates, calcitonin

MAGNESIUM DISORDERS

- HypoMg Mg <0.7
 - Suspect clinically
 - Symptoms: CNS (confusion, vertigo, ataxia, weakness); cardiac (prolonged PR+QRS+QT)
 - Causes: Alcoholism, poor nutrition, cirrhosis, pancreatitis, GI, renal losses
 - Tx: replace Mg
 - PO if asymptomatic
 - IV MgSO₄ 2g over 1h if arrhythmias, seizures, DTs
- HyperMg Mg >1.1
 - Very rare! Usually in renal failure with inc'd intake (antacids, Li)
 - Nausea □ weakness □ DTRs disappear when Mg>4.0 □ resp depression Mg > 8 □ cardiac Mg >12
 - Tx: stop Mg, fluids, consider Calcium

HYPERVENTILATION INDUCED EFFECTIVE HYPOCALCEMIA

- Respiratory alkalosis shifts ionized Ca^{2+} to the protein-bound form, producing a transient effective hypocalcemia



<https://www.youtube.com/watch?v=6UsU6gzBONo>

KEY TIPS

- If hyperK, GET AN ECG
 - Ca
 - Shift with insulin and dextrose
- If hypoK
 - Correct with PO KCl
 - Check Mg
- If hypoNa
 - Volume assessment
 - Don't overcorrect!!
- If hyperNa
 - Fluids
- Check Mg and Ca

TIPS CONT'D

- Nonspecific signs and symptoms
- Always consider electrolyte disturbances in your DDx

KEY TOPIC REFERENCES

- Emergency Medicine Cases Episode 60: Emergency Management of Hyponatremia. Available from: <https://emergencymedicinescases.com/episode-60-emergency-management-hyponatremia/>
- Emergency Medicine Cases Episode 86: Emergency Management of Hyperkalemia. Available from: <https://emergencymedicinescases.com/emergency-management-hyperkalemia/>
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<https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD000567.pub7/full>

CASE EXAMPLE REFERENCES

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- Ibrahim, Ahmed, et al. "Clinical Profile And Outcome Of Patients With Acute Kidney Injury Requiring Dialysis—an Experience From A Haemodialysis Unit In A Developing Country." *BMC Nephrology* 17.1 (2016): 1-5.
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- Ali, Khalid, Abdulhalik Workicho, and Esayas Kebede Gudina. "Hyponatremia in patients hospitalized with heart failure: a condition often overlooked in low-income settings." *International Journal Of General Medicine* 9 (2016): 267.
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