Nephrology for the Hospitalist

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Section of Nephrology

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Disclosures

- None

Objective

- Management of Hyperkalemia
- Acute Kidney Injury Evaluation
- Approaching Acid Base Disturbances
- Indications for Dialysis
- Evaluation of Hyponatremia

- 71 year old female presents to the ER via EMS for altered mental status. The previous 3 days she has had nausea, vomiting, diarrhea.
- PMH: HTN, HLD, DM type 2
- Medications
  - ASA 81mg
  - Lisinopril 40mg
  - Metformin 1,000mg BID
  - Atorvastatin 80mg

Vitals:

Temp: 99.3 F

BP: 100/55

HR: 127

RR: 33

SpO_2: 98% on 2L NC

- Na 138 mmol/L
- K 8.7 mmol/L
- Cl 100 mmol/L
- CO_2 8 mmol/L
- AG 30 mmol/L
- BUN 65 mg/dL
- Cr 7.9 mg/dL (previously 1.2
- GFR 9 cc/min (previously GFR 68cc/min)
- Albumin 2.5g/dL

- VBG
  - pH 7.05
  - pCO_2 30 mmHg
  - Bicarbonate 7 mmol/L
  - Lactate 15 mmol/L

Hyperkalemia

- Ensure it is a true hyperkalemia
- Look for
  - Hemolysis
  - Leukocytosis (>100,000)
  - Thrombocytosis (1,000,000)[1]

EKG changes present in 65% of K >6.5[2]

THE RENAL ASSOCIATION
founded 1950

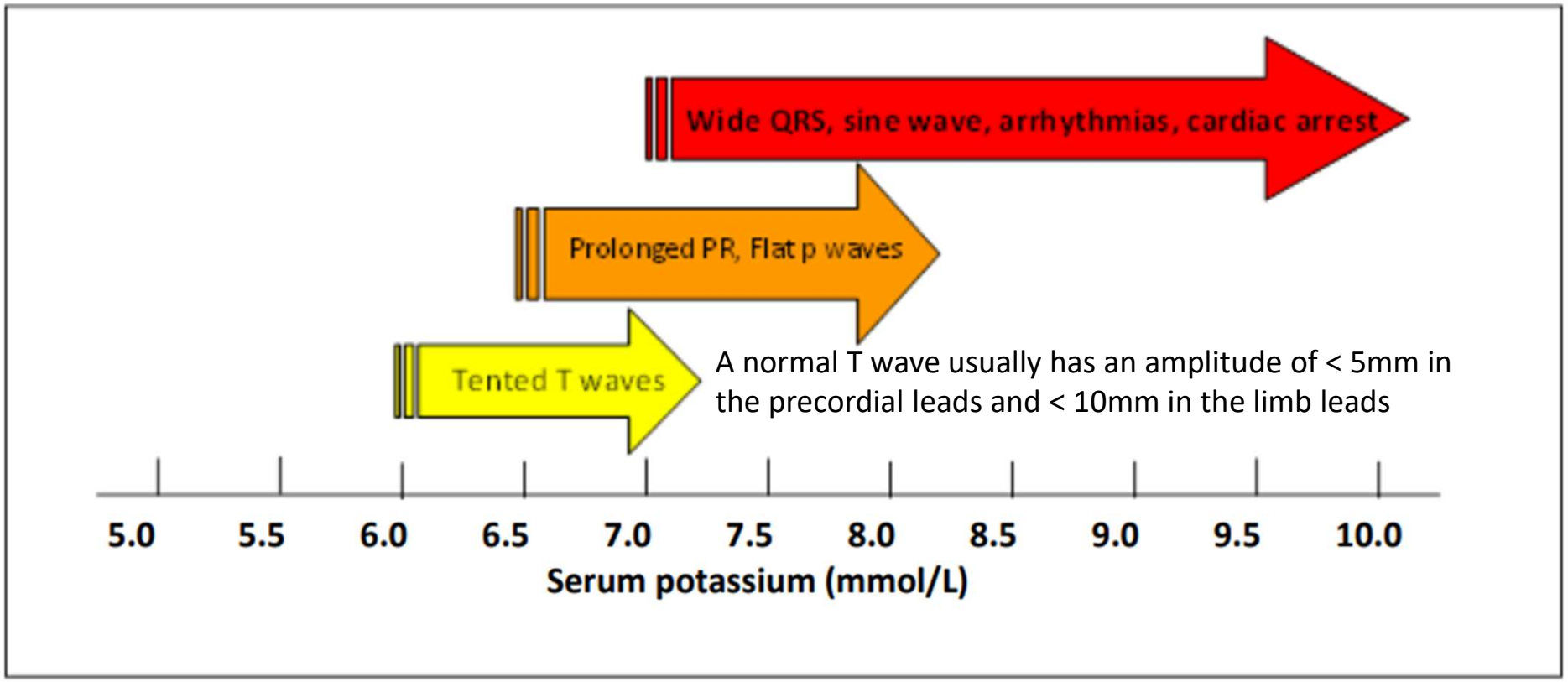


Figure 1: Progressive changes in ECG with increasing severity of hyperkalaemia.

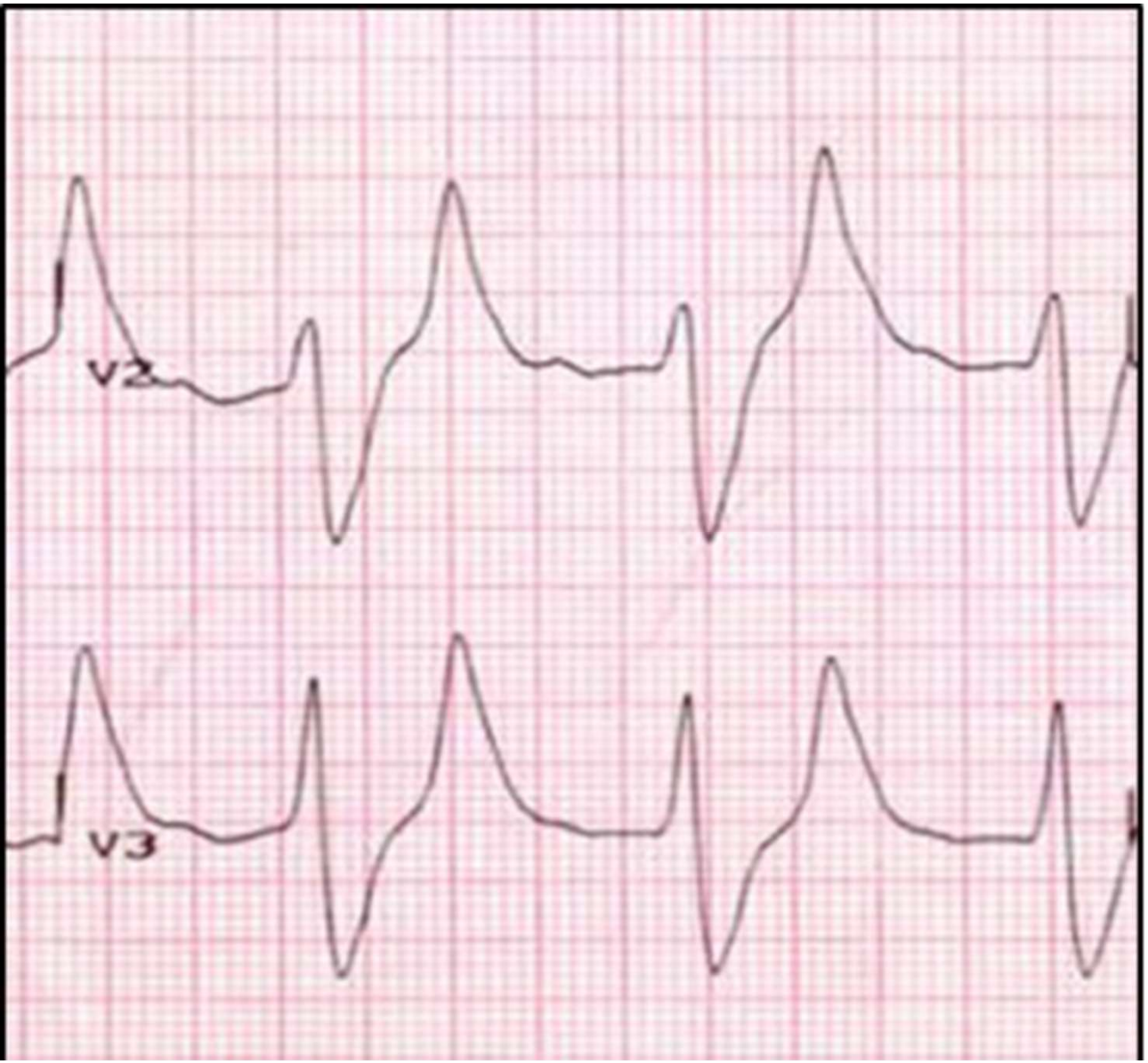
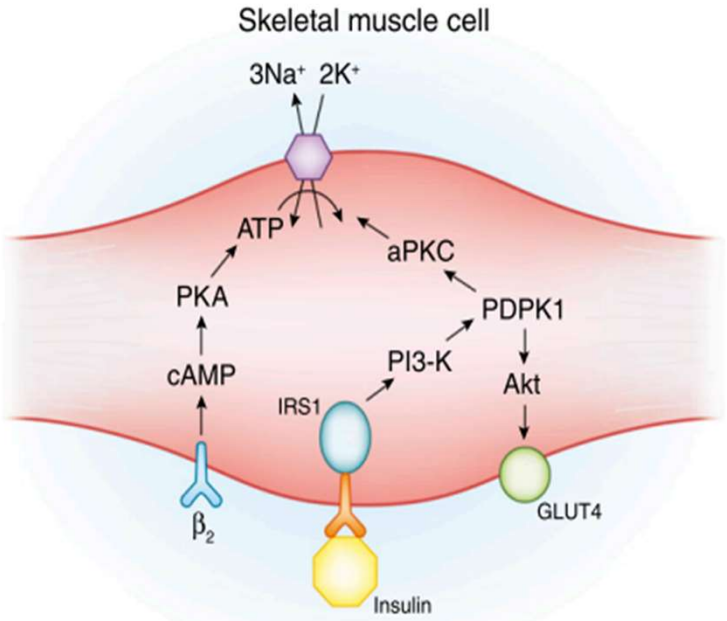


Figure 2: K 9.1 mmol/l

- Peaked T waves
- Diminished P waves
- Wide QRS complexes[1]

Hyperkalemia: Temporizing Measures

- **IV Calcium**
  - Gluconate or Chloride
  - Repeat every 30-60 minutes
  - Antagonizes the cardiac membrane excitability
- **Cell shifting**
  - Insulin/Dextrose
  - Albuterol
  - Bicarbonate[1,2]
- **These therapies buy time to remove potassium**



- **Clin J Am Soc Nephrol 10: 1050–1060, 2015**

Potassium Removal

- **Loop Diuretics**
- **Potassium Binders**
  - Patiromer (Veltassa)
  - Sodium Zirconium Cyclosilicate (Lokelma)
  - Sodium Polystyrene Sulfonate (Kayexalate)
- **Dialysis**[3]

Acute Kidney Injury

- KDIGO
  - ↑creatinine by 0.3 mg/dL within 48 hours **OR**
  - ↑creatinine to 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days **OR**
  - Urine volume <0.5 ml/kg/h for 6 hours.

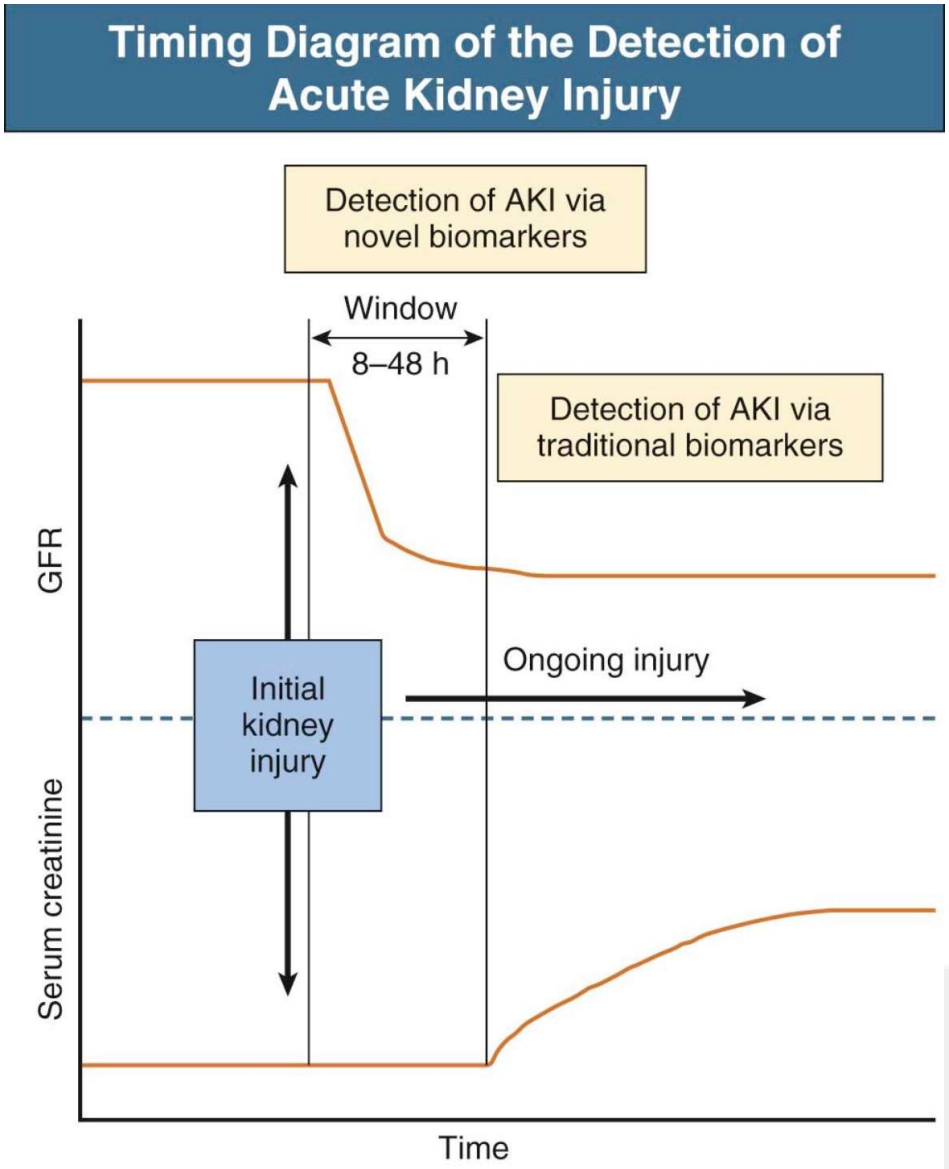
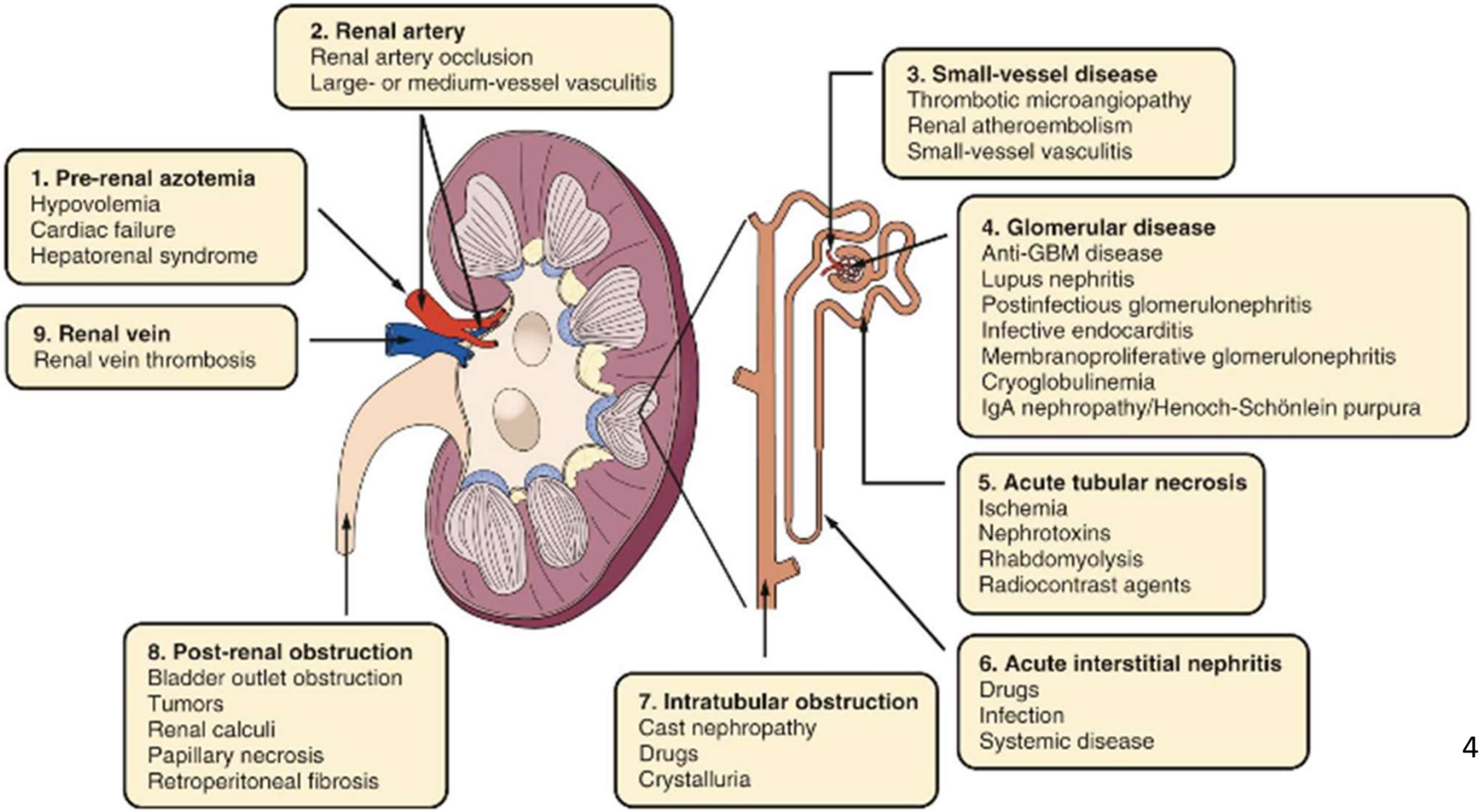


Figure 71-1: Timing diagram of the detection of acute kidney injury (AKI). *GFR,* Glomerular filtration rate.

(Adapted from reference 15.)

Causes of AKI



Nephrotic syndrome

- Proteinuria > 3.5g/day
- Hypoalbuminemia <3.5g/dL
- Edema
- Hypercholesterolemia
- Lipiduria

Nephritic syndrome

- Oliguria
- Hematuria
- Proteinuria (classically <3g/day)
- Edema
- Hypertension
- Abrupt onset

Rapidly progressive glomerulonephritis (RPGN)

- Renal failure over days to weeks
- Hematuria
- Proteinuria
- May have other systemic signs/symptom

Nephrotic

- Minimal change disease
- Focal segmental glomerulosclerosis
- Membranous nephropathy
- Amyloidosis/Myeloma
- Diabetic nephropathy
- Membranoproliferative GN

Nephritic

- ANCA vasculitis
- Anti-GBM
- Post infectious GN
- Lupus nephritis
- IgA nephropathy
- Membranoproliferative GN
- Thrombotic microangiopathy (TTP/HUS/ Compliment mediated)

Urinary Evaluation

- Sodium
- Urea
- Creatinine
- Osmolality
- Urinalysis
  - Macroscopic (dipstick)
  - Microscopic
- Protein to Creatinine ratio

- FeNa = $\frac{(U_{Na}/P_{Na})}{(U_{Cr}/P_{Cr})}$ x100
  - FeNa will be impacted by the use of diuretics or in CKD
  - <1% pre-renal
  - >2% intrinsic or ATN

- FeUrea= $\frac{(U_{Ur}/P_{Ur})}{(U_{Cr}/P_{Cr})}$ x 100
  - Is not impacted by diuretic use
  - <35% pre-renal
  - >50% intrinsic or ATN

How well does the fractional excretion of sodium (FENa) distinguish intrinsic from prerenal AKI?

CJASN
Clinical Journal of the American Society of Nephrology

Methods

Systematic review of studies utilizing FENa until December 31, 2021

Only studies of intrinsic vs prerenal AKI evaluated

Meta-analysis performed

Findings

		Pooled sensitivity FENa cutoff 1%	Pooled specificity FENa cutoff 1%
Overall	15 studies 872 patients	90% (95% CI: 81-95%)	82% (95% CI: 70-90%)
Subgroups	**Studies with CKD/diuretics** 6 studies 511 patients	83% (95% CI: 64-93%)	66% (95% CI: 51-78%)
Subgroups	**Diuretics** 5 studies 238 patients	80% (95% CI: 69-87%)	54% (95% CI: 31-75%)
Subgroups	**Oliguric w/o CKD/diuretics** 8 studies 264 patients	95% (95% CI: 82-99%)	91% (95% CI: 83-95%)

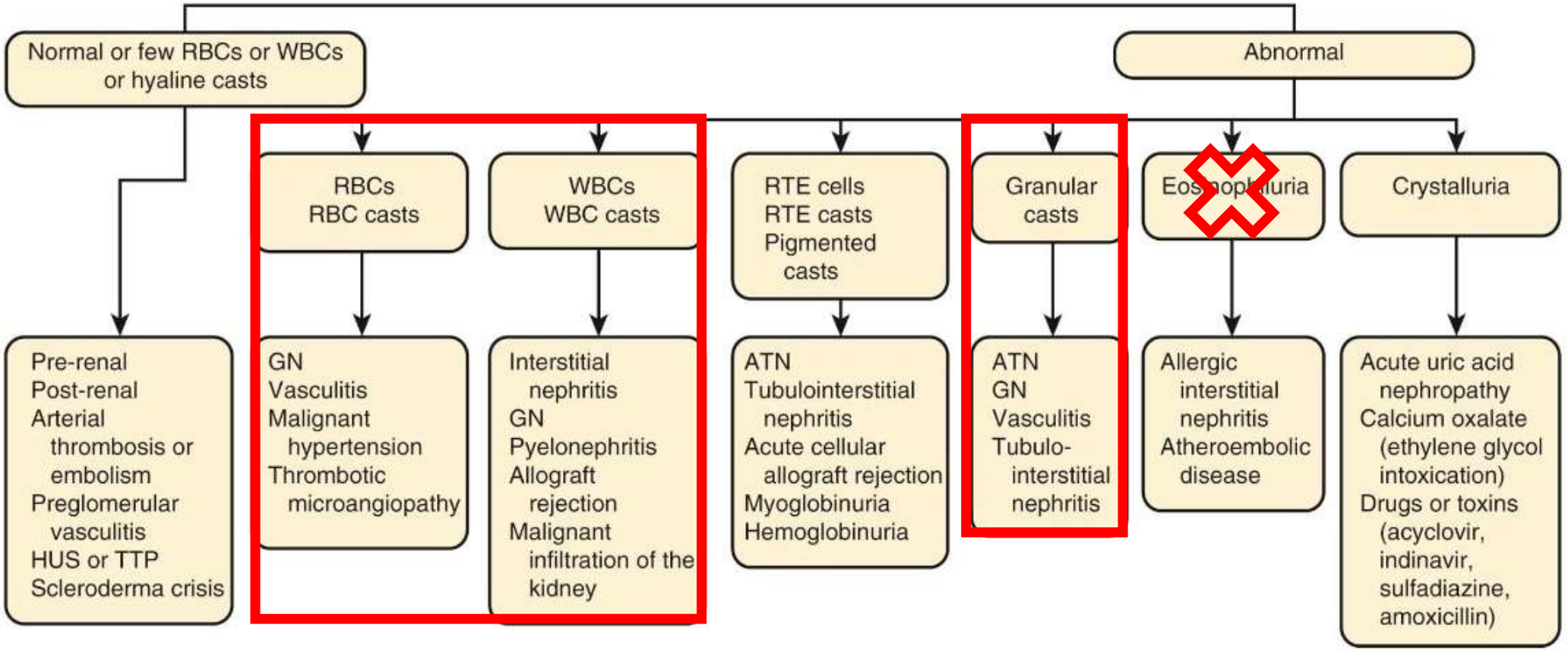
Conclusions: Fractional excretion of sodium (FENa) has a limited role for AKI differentiation in patients with a history of CKD or diuretic therapy. It is most valuable when oliguria is present.

Mohammad Abdelhafez, Tarek Nayfeh, Anwar Atieh, et al. ***Diagnostic Performance of Fractional Excretion of Sodium for the Differential Diagnosis of Acute Kidney Injury***. CJASN doi: 10.2215/CJN.14561121. **Visual Abstract by Gerren Hobby, MD**

Interpretation of Urine Studies in AKI

- Low urine sodium <20mmol/L
  - Decreased renal perfusion → Increased sodium reabsorption
    - Volume overload or depleted
- High urine sodium >20mmol/L
  - Diuretics
  - Acute Tubular Necrosis (ATN)
- Urine osmolality
  - Variable 50-1,200 mOsm/kg
  - Vasopressin activity → Increased water reabsorption → Higher urine osmolality

Urinary Sediment in AKI



URINALYSIS MACROSCOPIC WIT...	
APPEARANCE	Clear
COLOR	Normal (Yell...
SPECIFIC GRAVITY, URINE	>1.030 ▲
GLUCOSE	Negative
BILIRUBIN	Negative
KETONES	Negative
BLOOD	Negative
PH URINE	6.0
PROTEIN	30 !
UROBILINOGEN	Negative
NITRITE	Negative
LEUKOCYTES	Negative
URINALYSIS, MICROSCOPIC	
RBC'S	1.0
WBC'S	3.0
BACTERIA	Occasional o...

High specific gravity = concentrated urine = more vasopressin activity

- Urine Osmolality 552
- Urine Sodium <20
- FeNa = 0.2%
- FeUrea = 14%
- Protein/Creatinine ratio: 300mg/g
- Bland urine
- CT A/P without obstruction
- This patient is looking more Pre-Renal AKI

Acid-Base Disturbance

Acid-Base Disturbance

1. **pH- Normal 7.35-7.45**
   - **pH 7.0 is low so it is acidotic**

Acid-Base Disturbance

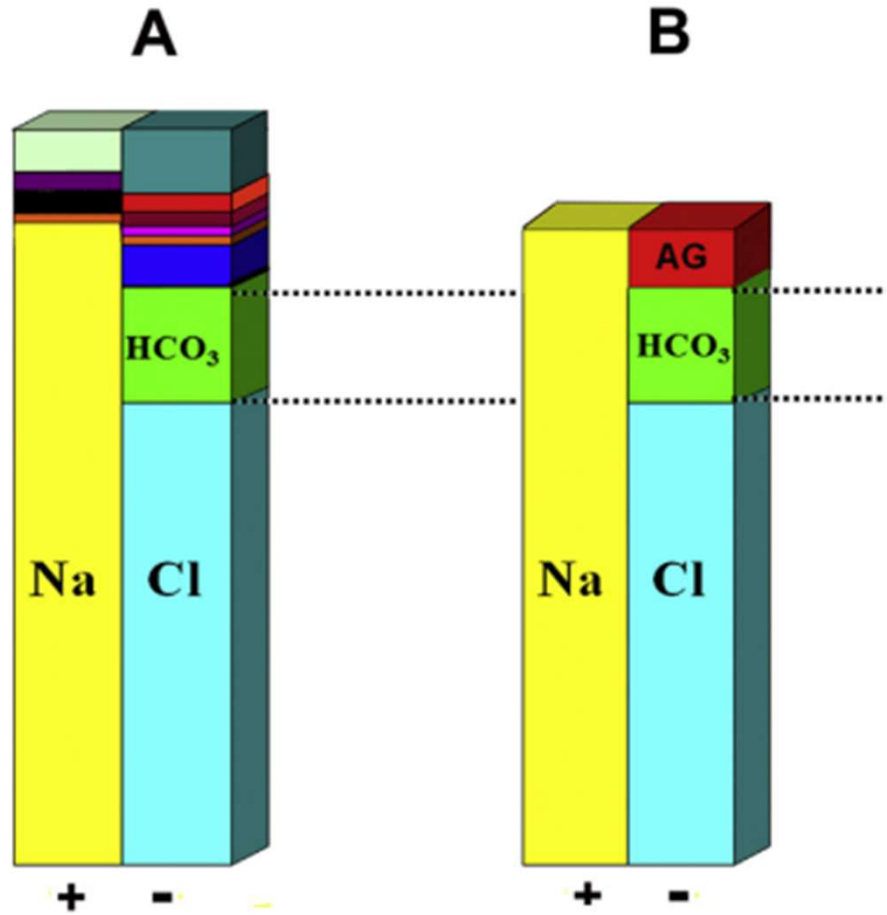
1. pH- Normal 7.35-7.45
   - pH 7.0 is low so it is acidotic
2. **What is the primary disturbance?**

Primary Disturbance

A/B Disturbance	pH 7.35-7.45 7.0	pCO_2 35-45mmHg 30	HCO_3^- 22-26mEq/L 8
Metabolic Acidosis	↓	↓	↓
Metabolic Alkalosis	↑	↑	↑
Respiratory Acidosis (HCO_3^- acute/chronic)	↓	↑	**Normal/↑**
Respiratory Alkalosis Always acute	↑	↓	**Normal**

Acid-Base Disturbance

1. pH- Normal 7.35-7.45
   - ➢pH 7.0 is low so it is acidotic
2. What is the primary disturbance?
   - ➢Metabolic Acidosis
3. **What is the AG? Na - (Cl+ Bicarb)**
   - ➢**High at 30**



Approach to Patients With High Anion Gap Metabolic Acidosis: Core Curriculum 2021 *AJKD* Vol 78 | Iss 4 | October 2021

Acid-Base Disturbance

1. pH- Normal 7.35-7.45
   - pH 7.0 is low so it is acidotic
2. What is the primary disturbance?
   - Metabolic Acidosis
3. **What is the AG? Na - (Cl+ Bicarbonate)**
   - **High at 30**
   - **What is the albumin? 2.5**
   - **Correcting for the albumin**
   - **AG Corrected = AG uncorrected + (2.5(4.5 – Albumin))**
   - **AG Corrected = 30 + (2.5(4.5-2.5)) = 35**

Acid-Base Disturbance

1. pH- Normal 7.35-7.45
   - pH 7.0 is low so it is acidotic
2. What is the primary disturbance?
   - Metabolic Acidosis
3. What is the AG? Na - (Cl+ Bicarbonate)
   - High at 35
4. **Is there respiratory compensation?**

Compensation

Acute Respiratory		
Acidosis	↑ 10 pCO_2	↑1 HCO_3^-
Alkalosis	↓ 10 pCO_2	↓2 HCO_3^-
Chronic Respiratory		
Acidosis	↑ 10 pCO_2	↑ 3 HCO_3^-
Alkalosis	↓ 10 pCO_2	↓ 4 HCO_3^-
Metabolic		
Acidosis	Winters formula: Expected $pCO_2 = 1.5(HCO_3^-) + 8$ (+/- 2)	

Expected $pCO_2 = 1.5(8) + 8 =$ **20 +/- 2**

Acid-Base Disturbance

1. pH- Normal 7.35-7.45
   - pH 7.0 is low so it is acidotic
2. What is the primary disturbance?
   - Metabolic Acidosis
3. What is the AG? Na - (Cl+ Bicarbonate)
   - High at 30
4. **Is there compensation?**
   - **Expected pCO_2 20 +/- 2**
   - **Actual pCO_2 30**
   - **No**

Acid-Base Disturbance

1. pH- Normal 7.35-7.45
   - pH 7.0 is low so it is acidotic
2. What is the primary disturbance?
   - Metabolic Acidosis
3. What is the AG? Na - (Cl+ Bicarbonate)
   - High at 30
4. Is there compensation?
   - Expected pCO_2 20 +/- 2
   - Actual pCO_2 30
   - No
5. **Primary HAGMA with Respiratory Acidosis**

Table 1. GOLDMARK Mnemonic for the High Anion Gap Metabolic Acidoses

Letter	Parameter	Potential causes
G	Glycols	Ingestion/infusion of ethylene, propylene, or diethylene glycol; metabolism generates glyoxylic, oxalic, D and L lactic acid.
O	5-Oxoproline	Chronic acetaminophen use can generate 5-oxoproline (a strong acid that is also called pyroglutamic acid).
L	L-Lactic acidosis	Multiple etiologies of types A and type B lactic acidosis.
D	D-Lactic acidosis	Carbohydrate loading in patients with short gut syndromes.
M	Methanol	Metabolism generates formic acid.
A	Aspirin	Toxic levels generate multiple organic acids including keto acids.
R	Renal failure	Accumulation of multiple inorganic and organic acids including sulfuric and phosphoric acid.
K	Ketoacidosis	B-OH butyric and acetoacetic acid.

Based on mnemonic proposed in Mehta et al, *Lancet*. 2008;372(9642):892.

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Our Differentials

- L-Lactate
- Renal failure
- Ketoacidosis

Table 3. Causes of lactic acidosis

Type A	Type B
Decreased oxygen delivery	Impaired oxygen utilization or defective lactate metabolism
Systemic hypoperfusion	Underlying disease
Shock	Liver failure
Sepsis	Kidney failure
	Malignancy
	Sepsis
	Pheochromocytoma
	Thiamine deficiency
	Diabetic ketoacidosis
	Alkalosis
Local hypoperfusion	Drugs/toxins
Thrombus/emboli	Acetaminophen
Volvulus/torsion	Salicylate
Compartment syndrome	Metformin
Sepsis	Catecholamines
	HARRT (first generation)
	Linezolid
	Propofol
	Cocaine
Profound hypoxia	Congenital metabolic defects
ARDS, COPD, asthma	
Carbon monoxide	
Methemoglobinemia	

HAART, highly active antiretroviral therapy; ARDS, acute respiratory distress syndrome; COPD, chronic obstructive pulmonary disease.

Acid-Base Disorders in the Critically Ill Patient

Anand Achanti and Harold M. Szerlip

CJASN 18: 102–112, January, 2023

Dialysis Indications

Acidosis

Electrolytes

- Hyperkalemia

Ingestions

- Methanol, Ethylene glycol, Metformin, ASA, Lithium

Oliguria

Uremia

- Constellation of symptoms caused by the build up of uremic toxins

Dialysis Indications for This Patient

- Hyperkalemia
- Oliguria
- Acidosis
  - Metformin toxicity
  - AKI
  - Ketosis from poor PO intake
  - Diarrhea
  - Elevated Lactate

88 year old male presents to the ER after a syncopal episode. He has felt dizzy and weak for the last 4 days prior to his syncope. He also complains of right hip pain. PMHx include smoking 1ppd for 60 years, COPD and HTN

- Medications:
  - HCTZ 25mg
  - Albuterol PRN
- Vitals:
  - Temp: 97.3 F
  - BP: 140/75
  - HR: 127
  - RR: 33
  - SpO_2: 98% on RA
- PE:
  - Euvolemic
  - R hip TTP/movement

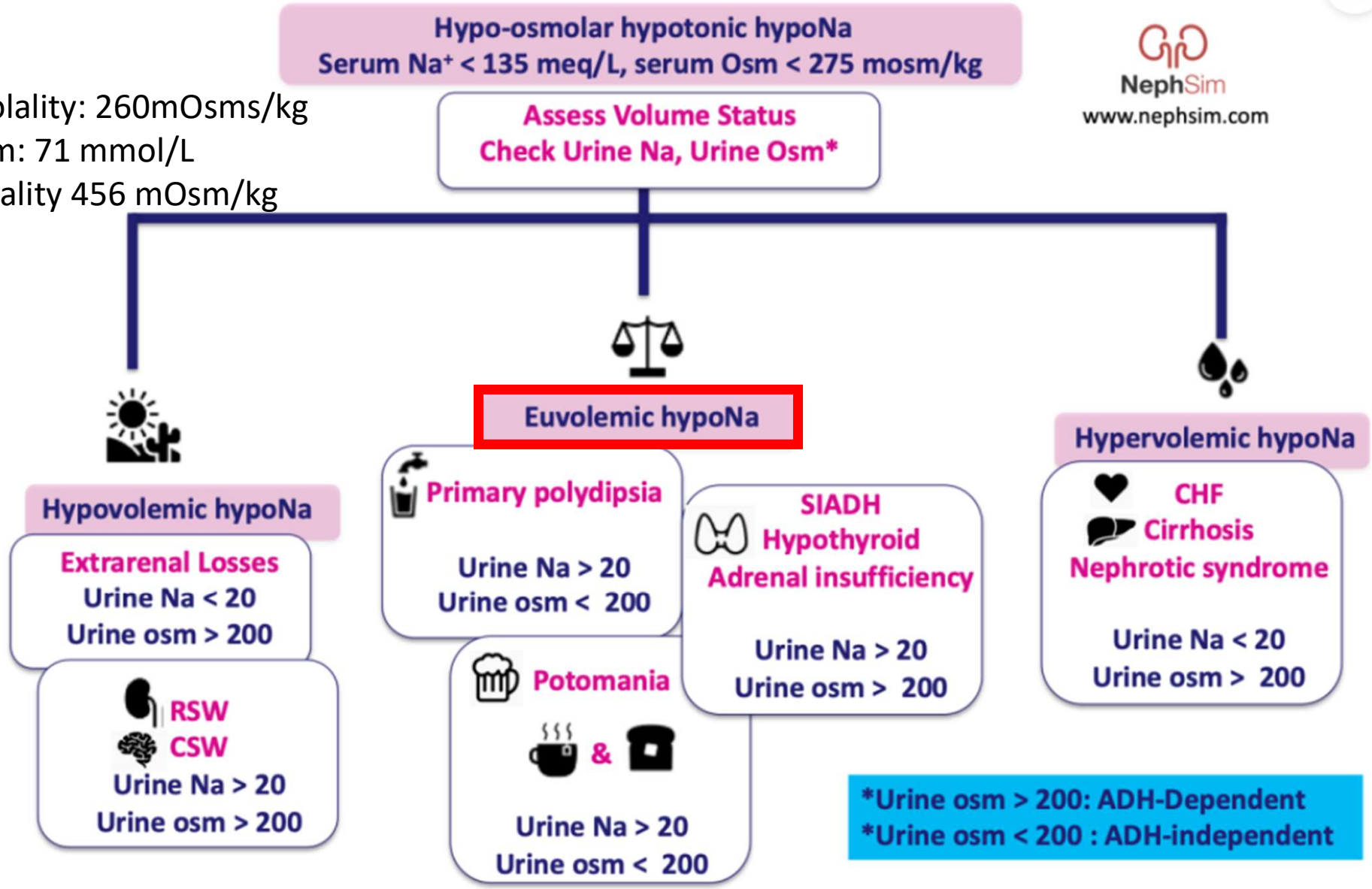
SODIUM	114 ⌄⌄
POTASSIUM	4.7
CHLORIDE	80 ⌄
CARBON DIOXIDE	22 ⌄
BUN	13
CREATININE	0.85
GLUCOSE	124
ANION GAP	12
BUN/CREAT RATIO	15
ESTIMATED GLOMERULAR FILTR...	69
CALCIUM	9.9

- Pelvic XR shows a right intertrochanteric fracture
- Orthopedics recommends ICU admission

Hyponatremia-Is it True?

- **True Hyponatremia has a serum osmolality <280mOsm**
- Normal serum osmolality ~280mOsm (Pseudohyponatremia)
  - Hypertriglyceridemia
  - Hyperglobulinemia
- Elevated serum osmolality >290mOsm
  - Hyperglycemia
  - Mannitol
  - IVIG
  - Contrast[4]
- This patient has none of these

Serum Osmolality: 260mOsms/kg
Urine Sodium: 71 mmol/L
Urine Osmolality 456 mOsm/kg



Interpretation of Urine Studies in Hyponatremia

- Low urine sodium <20mmol/L
  - Low solute intake
  - Decreased renal perfusion → Increased sodium reabsorption
    - Volume overload or depleted
- High urine sodium >20mmol/L
  - Diuretics
  - Normal renal perfusion
  - Acute Tubular Necrosis (ATN)
- Urine osmolality
  - Variable 50-1,200 mOsm/kg
  - Vasopressin activity → Increased water reabsorption → Higher urine osmolality

Triggers for ADH release

- Increased serum osmolality
- Non-osmotic stimuli for ADH release
  - Decreased effective circulating volume
  - Pain
  - Stress
  - Pulmonary disease
  - Cancer
  - Nausea
  - Medications (SSRI, Antipsychotics, etc.)
- Thiazide diuretics can cause hyponatremia as well

SIADH Treatment

- **Fluid restriction**
  - Decreased water intake → decreased water reabsorption
- **Tolvaptan**
  - Blocks the reabsorption of water
  - Must remove fluid restriction
- **3% saline**
- **Increased solute intake**
  - Salt tablets
  - Urea
  - High protein diet
- **Loop diuretics**
- This combination changes the concentration gradients between the interstitium and tubules → decreased water reabsorption

- **Goal correction for symptomatic hyponatremia is around 4mEq/L within a few hours**
- **Over a 24 hour period goal is no more 8mEq/L**
- This patients sodium begins to correct with a bolus of 100cc of 3% saline initially.
- He is transitioned to Furosemide 10mg BID, NaCl tabs 2g TID and fluid restriction of 1.2L
- His hospital course is complicated by aspiration pneumonia requiring intubation for 4 days.

Day 13 Labs:

- Na 140 mmol/L
- K 3.5 mmol/L
- Cl 100 mmol/L
- CO_2 10 mmol/L
- AG 30 mmol/L
- BUN 65 mg/dL
- Cr 0.65 mg/dL

- VBG
  - pH 7.22
  - pCO_2 23 mmHg
  - Bicarbonate 9 mmol/L
  - Lactate normal

Primary Disturbance

A/B Disturbance	pH 7.35-7.45 7.22	pCO_2 35-45mmHg 23	HCO_3^- 22-26mEq/L 10
Metabolic Acidosis	↓	↓	↓
Metabolic Alkalosis	↑	↑	↑
Respiratory Acidosis (HCO_3^- acute/chronic)	↓	↑	**Normal/↑**
Respiratory Alkalosis Always acute	↑	↓	**Normal**

Acid-Base Disturbance

1. pH- Normal 7.35-7.45
   - pH 7.22 is low so it is acidotic
2. What is the primary disturbance?
   - Metabolic Acidosis
3. What is the AG? Na - (Cl+ Bicarbonate)
   - High at 30, albumin normal at 4
4. Is there compensation?
   - Expected pCO_2 23 +/- 2
   - Actual pCO_2 23
   - Yes
5. Primary HAGMA--so why is this happening?

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- Lactate normal
- B-OH normal
- Unlikely ingestion

- MAR review: Acetaminophen 650mg 3-4 times a day for the last week.
- Glutathione and cysteine deficiency accelerates generation and accumulation of 5-Oxoproline (pyroglutamic acid)
- Occurs in malnourish, ill patients who have chronic Acetaminophen ingestion
- Treatment: Stop Acetaminophen

- 34 year old female comes to the ER with 40 pound weight gain, lower extremity edema and progressive dyspnea on exertion over the last 2 weeks. She has run out of all her medications
- PMH: Non-ischemic cardiomyopathy LVEF 15% from cocaine
- Medications:
  - ASA 81mg
  - Atorvastatin 40mg
  - Carvedilol 12.5mg BID
  - Valsartan 40mg
  - Spironolactone 12.5mg
  - Furosemide 40mg daily
  - Empagliflozin 25mg

- Vitals:
  - Temp 98.2
  - BP 147/90
  - HR 101
  - RR 30
  - O_2 92% 4L NC

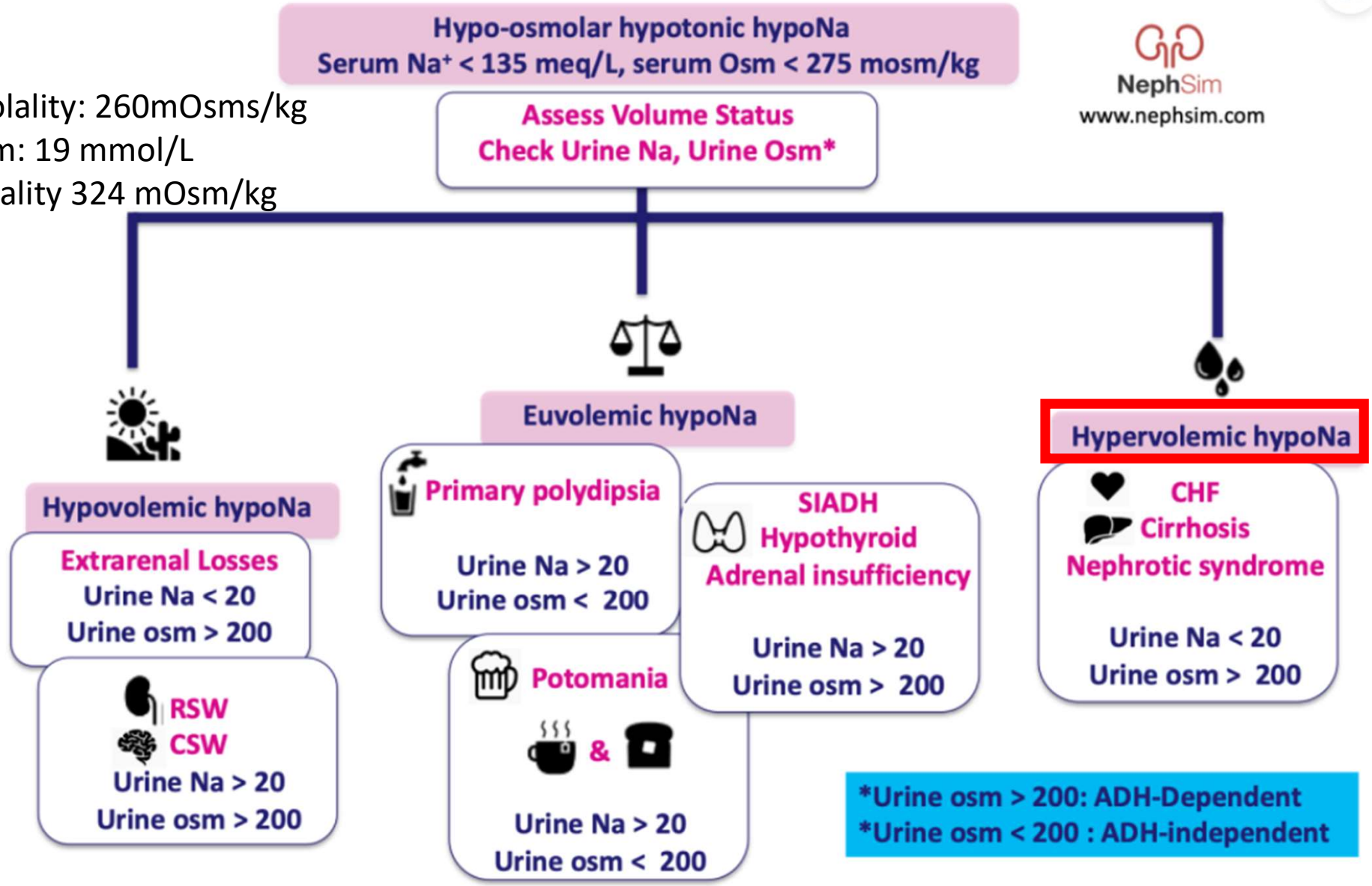
- PE:
  - General: Appears in distress
  - Cardiac: Tachycardic
  - Respiratory: Bibasilar crackles
  - Abdomen: Distended
  - Extremities: 3+ pitting edema

- Na 122 mmol/L
- K 5.3 mmol/L
- Cl 90 mmol/L
- CO_2 25 mmol/L
- AG 7 mmol/L
- BUN 37 mg/dL
- Cr 1.9 mg/dL (previously 0.8
- GFR 44 cc/min (previously GFR 75cc/min)

- VBG
  - pH 7.12
  - pCO_2 79 mmHg
  - Bicarbonate 25 mmol/L
  - Lactate 1 mmol/L

Hyponatremia

Serum Osmolality: 260mOsms/kg
Urine Sodium: 19 mmol/L
Urine Osmolality 324 mOsm/kg



Hypervolemic Hyponatremia Management:

DIURESIS

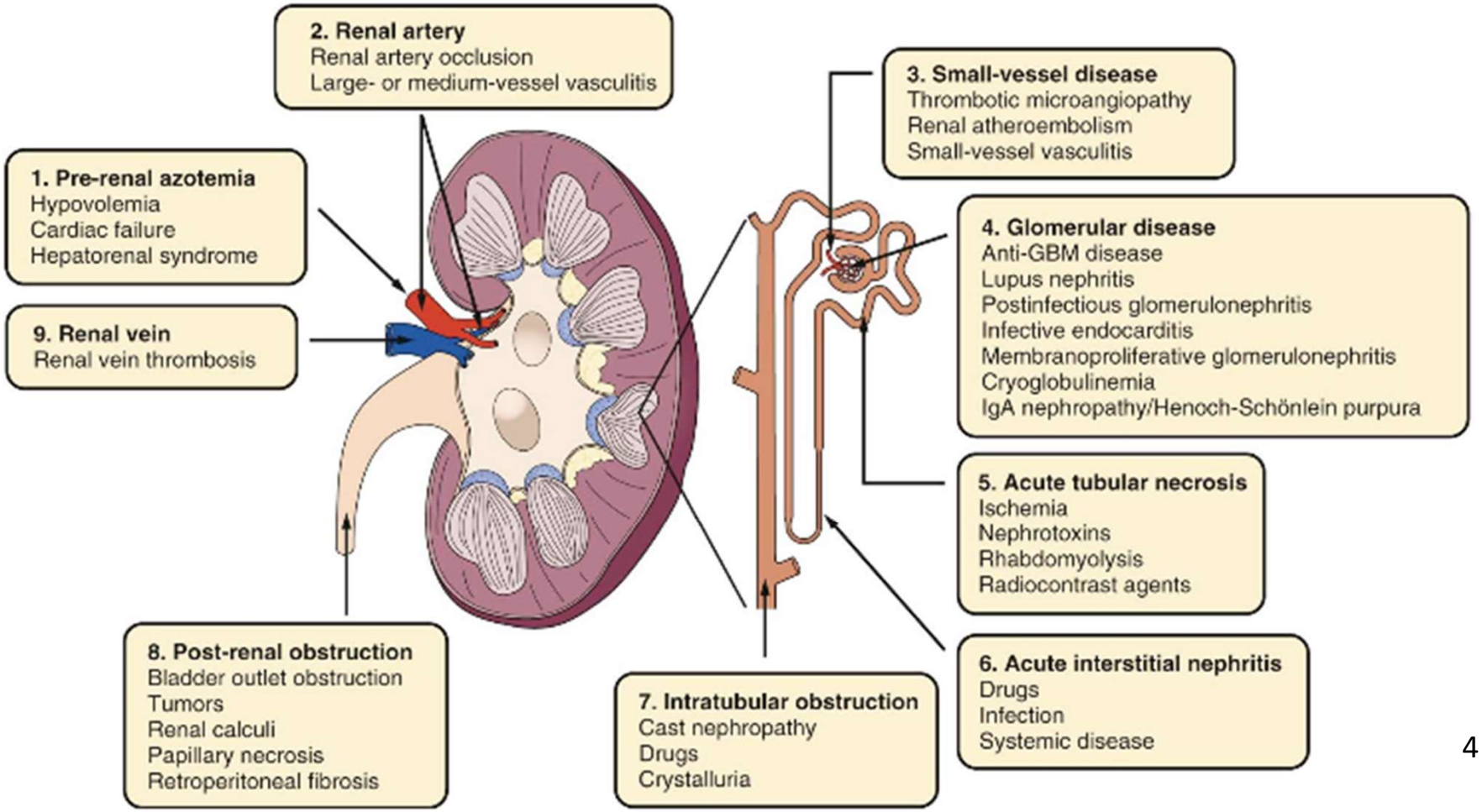
- IV Intermittent Bolus (BID-TID)

OR

- IV **Bolus** followed by Continuous Infusion

Acute Kidney Injury

Causes of AKI



URINALYSIS, MACROSCOPIC AN...	!
URINALYSIS, MACROSCOPIC	!
APPEARANCE	Cloudy !
COLOR	Normal (Yell...
SPECIFIC GRAVITY, URINE	1.014
GLUCOSE	Negative
BILIRUBIN	Negative
KETONES	Negative
BLOOD	Negative
PH URINE	7.0
PROTEIN	Negative
UROBILINOGEN	Negative
NITRITE	Negative
LEUKOCYTES	Negative
URINALYSIS, MICROSCOPIC	
RBC'S	2.0
WBC'S	1.0
BACTERIA	Occasional o...

- Urine Osmolality 324mOsm/kg
- Urine Sodium 19 mmol/L
- FeNa = 0.4%
- FeUrea = 23%
- Protein/Creatinine ratio: 200mg/g
- Bland urine
- Renal US: No obstruction
- Pre-renal: Volume overload

Acid-Base Disturbance

Primary Disturbance

A/B Disturbance	pH 7.35-7.45 7.12	pCO_2 35-45mmHg 79	HCO_3^- 22-26mEq/L 25
Metabolic Acidosis	↓	↓	↓
Metabolic Alkalosis	↑	↑	↑
Respiratory Acidosis (HCO_3^- acute/chronic)	↓	↑	**Normal/↑**
Respiratory Alkalosis Always acute	↑	↓	**Normal**

Rough Compensation Rules

Acute Respiratory		
Acidosis	↑ 10 pCO_2	↑1 HCO_3^-
Alkalosis	↓ 10 pCO_2	↓2 HCO_3^-
Chronic Respiratory		
Acidosis	↑ 10 pCO_2	↑ 3 HCO_3^-
Alkalosis	↓ 10 pCO_2	↓ 4 HCO_3^-
Metabolic		
Acidosis	Winters formula: Expected $pCO_2 = 1.5(HCO_3^-) + 8$ (+/- 2)	

Acid-Base Disturbance

1. pH- Normal 7.35-7.45
   - pH 7.1 is low so it is acidotic
2. What is the primary disturbance?
   - Respiratory Acidosis
3. Is there compensation?
   - Not yet, this can take some time
4. Primary Respiratory Acidosis
5. Treatment—Ventilation (BiPAP or Intubation) and Diuresis

55 year old male with a PMH of HLD on Atorvastatin presents to the ER. His friend states they were at a party last night and when he went to check on his friend and he found him with altered mental status.

Vitals:

Temp 99.1

SpO_2 88% 5L NC

RR 12

HR 115

BP: 105/73

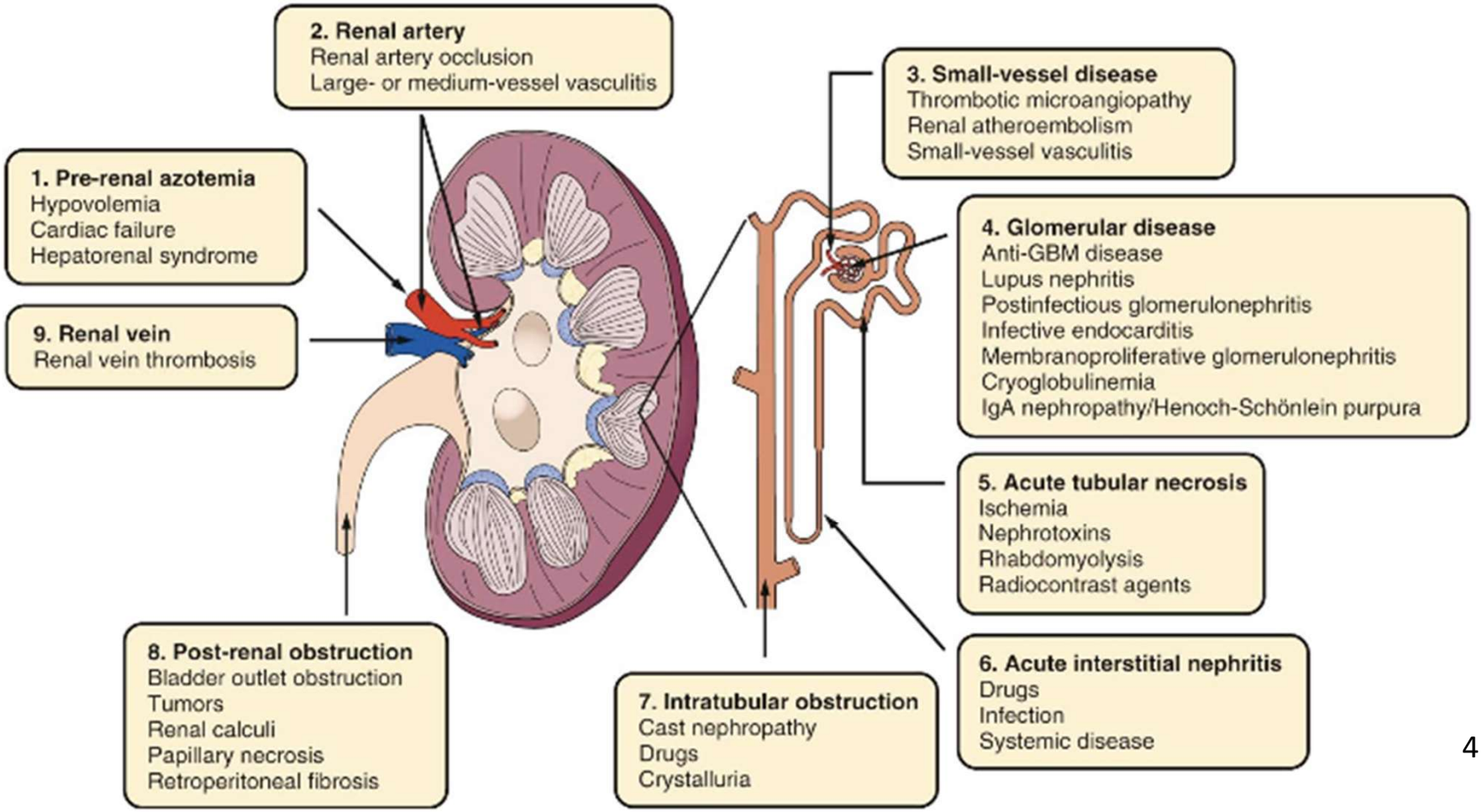
Arterial Blood Gas

- pH 6.8
- pCO_2 47
- Bicarbonate 7

CHEM 1	
SODIUM	132 ▼
POTASSIUM	5.1
CHLORIDE	98
CARBON DIOXIDE	7 ▼
BUN	51 ▲
CREATININE	10.12 ▲
GLUCOSE	81
ANION GAP	27 ▲
BUN/CREAT RATIO	19
ESTIMATED GLOMERULAR FILTR...	5 ▼
CALCIUM	8.6 ▼

Acute Kidney Injury

Causes of AKI



URINALYSIS, MACROSCOPIC AN...	!
URINALYSIS, MACROSCOPIC	!
APPEARANCE	Cloudy !
COLOR	Normal (Yell...
SPECIFIC GRAVITY, URINE	1.014
GLUCOSE	Negative
BILIRUBIN	Negative
KETONES	Negative
BLOOD	Negative
PH URINE	7.0
PROTEIN	Negative
UROBILINOGEN	Negative
NITRITE	Negative
LEUKOCYTES	Negative
URINALYSIS, MICROSCOPIC	
RBC'S	2.0
WBC'S	1.0
BACTERIA	Occasional o...

- Urine Osmolality 374mOsm/kg
- Urine Sodium 47 mmol/L
- FeNa = 2.7%
- FeUrea = 63%
- Protein/Creatinine ratio: 245mg/g
- Bland urine
- Oxalate crystals are noted
- Renal US: No obstruction
- Likely ATN/Oxalate deposition

Acid-Base Disturbance

1. pH- Normal 7.35-7.45
   - pH 6.8 is low so it is acidotic
2. What is the primary disturbance?
   - Metabolic Acidosis
3. What is the AG? Na - (Cl+ Bicarbonate)
   - High at 27, albumin is normal at 4
4. Is there compensation?
   - Expected pCO_2 18 +/- 2
   - Actual pCO_2 47
   - No
5. Primary HAGMA with Respiratory Acidosis

Table 1. GOLDMARK Mnemonic for the High Anion Gap Metabolic Acidoses

Letter	Parameter	Potential causes
G	Glycols	Ingestion/infusion of ethylene, propylene, or diethylene glycol; metabolism generates glyoxylic, oxalic, D and L lactic acid.
O	5-Oxoproline	Chronic acetaminophen use can generate 5-oxoproline (a strong acid that is also called pyroglutamic acid).
L	L-Lactic acidosis	Multiple etiologies of types A and type B lactic acidosis.
D	D-Lactic acidosis	Carbohydrate loading in patients with short gut syndromes.
M	Methanol	Metabolism generates formic acid.
A	Aspirin	Toxic levels generate multiple organic acids including keto acids.
R	Renal failure	Accumulation of multiple inorganic and organic acids including sulfuric and phosphoric acid.
K	Ketoacidosis	B-OH butyric and acetoacetic acid.

Based on mnemonic proposed in Mehta et al, *Lancet.* 2008;372(9642):892.

Approach to Patients With High Anion Gap Metabolic Acidosis: Core Curriculum 2021 *AJKD* Vol 78 | Iss 4 | October 2021

- Lactate: 2.7 mmol/L (normal <1.5)
- Serum Ethanol: 0
- Serum ASA/Acetaminophen: 0
- Beta-Hydroxybutyrate: 0.2mmol/L (normal <0.4)
- Serum Methanol: Pending
- Serum Glycol: Pending
- Serum Osmolality: 324 mOsm/kg
- Calculated Osmolality: 287 mOsm/kg
- Osmolar Gap: 37 mOsm/kg

Osmolar Gap

- Measured serum osmolality – calculated osmolality
  - >10 mosmol/kg is abnormal
- Calculated serum osmolality = 2(Na) + Glucose/18 + BUN/2.8
- When an osmolar gap is present that means there are osmoles detected by the lab that have not been identified yet
- More commonly seen in
  - Ethylene glycol ingestion
  - Methanol ingestion
  - Diethylene glycol
  - Propylene glycol infusion
  - IVIG
  - Mannitol

The Role of the Nephrologist in Management of Poisoning and Intoxication: Core Curriculum 2022

Michael E. Mullins and Jeffrey A. Kraut *AJKD* Vol 79 | Iss 6 | June 2022

B Time Course of Changes in the Osmolal and Anion Gaps

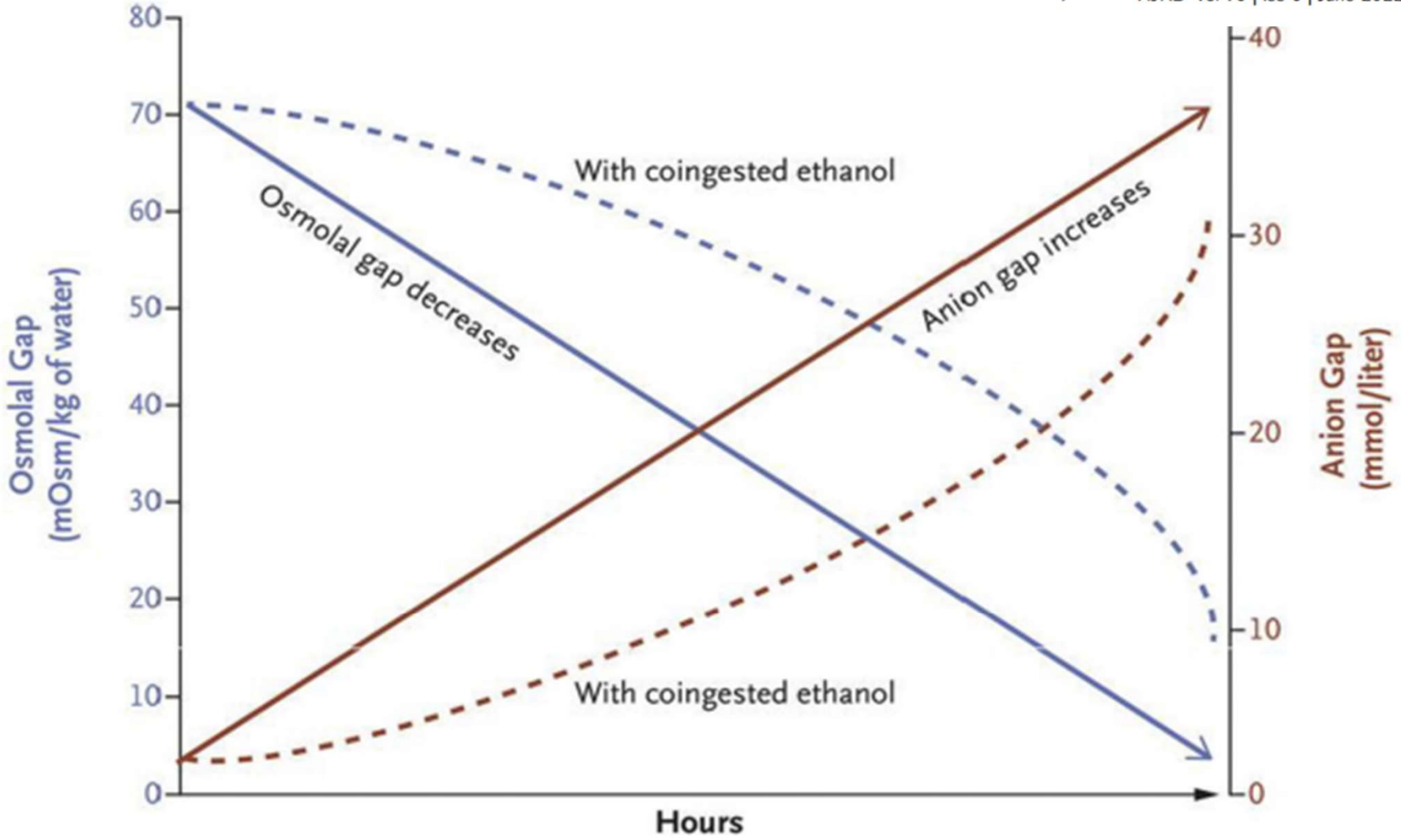


Figure 1

The Role of the Nephrologist in Management of Poisoning and Intoxication: Core Curriculum 2022

Michael E. Mullins and Jeffrey A. Kraut *AJKD* Vol 79 | Iss 6 | June 2022

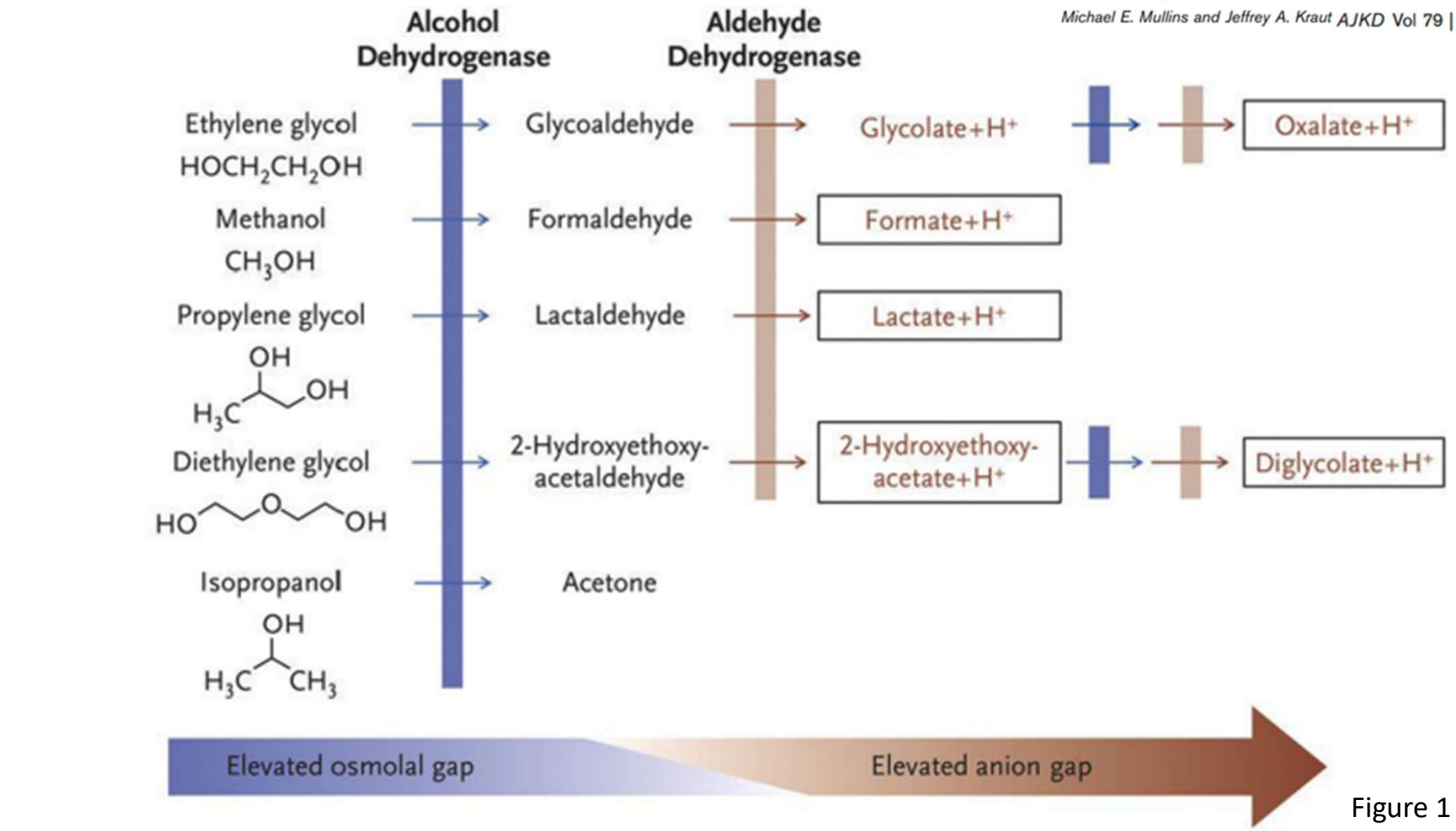


Figure 1

Toxic Alcohol Management

The Role of the Nephrologist in Management of Poisoning and Intoxication: Core Curriculum 2022
Michael E. Mullins and Jeffrey A. Kraut *AJKD* Vol 79 | Iss 6 | June 2022

Box 1. Indications for Hemodialysis

Toxic Alcohols

- Ethylene glycol or methanol concentration > 50 mg/dL without ADH inhibitor (fomepizole or ethanol)
- Ethylene glycol concentration > 200-300 mg/dL *with* ADH inhibitor *and* normal kidney function
- Methanol concentration > 70 mg/dL *with* ADH inhibitor *and* normal kidney function
- Isopropanol concentration > 400-500 mg/dL
- Any toxic alcohol: severe acidemia (pH < 7.2) *or* AKI

- **Dialysis removes methanol and ethylene glycol**

Dialysis Indications for This Patient

Acidosis

Electrolytes

- Hyperkalemia

Ingestions

- Methanol, Ethylene glycol, Metformin, ASA, Lithium

Oliguria

Uremia

- Constellation of symptoms caused by the build up of uremic toxins

30 year old female with PMHx of endometriosis is POD #6 from hysterectomy. She comes to the ER with worsening abdominal pain, nausea and vomiting.

- Vitals:
  - Afebrile
  - HR 113
  - BP 110/60
- Physical Exam:
  - Abdomen: distended, hypoactive bowel sounds, diffusely TTP
- CT A/P shows dilated small bowel loops with transition point in the distal small bowel with post operative changes in the pelvis.
- Surgery recommends NG tube is placement

Admission Labs

- Na 137 mmol/L
- K 4.1 mmol/L
- Cl 104 mmol/L
- CO_2 23 mmol/L
- BUN 37 mg/dL
- Cr 0.9 mg/dL

- VBG
  - pH 7.41
  - pCO_2 36 mmHg
  - Bicarbonate 22 mmol/L

Labs 2 days into Admission

- Na 133 mmol/L
- K 2.9 mmol/L
- Cl 80 mmol/L
- CO_2 37 mmol/L
- BUN 42 mg/dL
- Cr 1.1 mg/dL

- VBG
  - pH 7.5
  - pCO_2 47 mmHg
  - Bicarbonate 37 mmol/L

Acid-Base Disturbance

Primary Disturbance

A/B Disturbance	pH 7.35-7.45 7.5	pCO_2 35-45mmHg 47	HCO_3^- 22-26mEq/L 37
Metabolic Acidosis	↓	↓	↓
Metabolic Alkalosis	↑	↑	↑
Respiratory Acidosis (HCO_3^- acute/chronic)	↓	↑	**Normal/↑**
Respiratory Alkalosis Always acute	↑	↓	**Normal**

Metabolic Alkalosis Pathogenesis, Diagnosis, and Treatment: Core Curriculum 2022 *AJKD* Vol 80 | Iss 4 | October 2022

Catherine Do, Pamela C. Vasquez, and Manoocher Soleimani

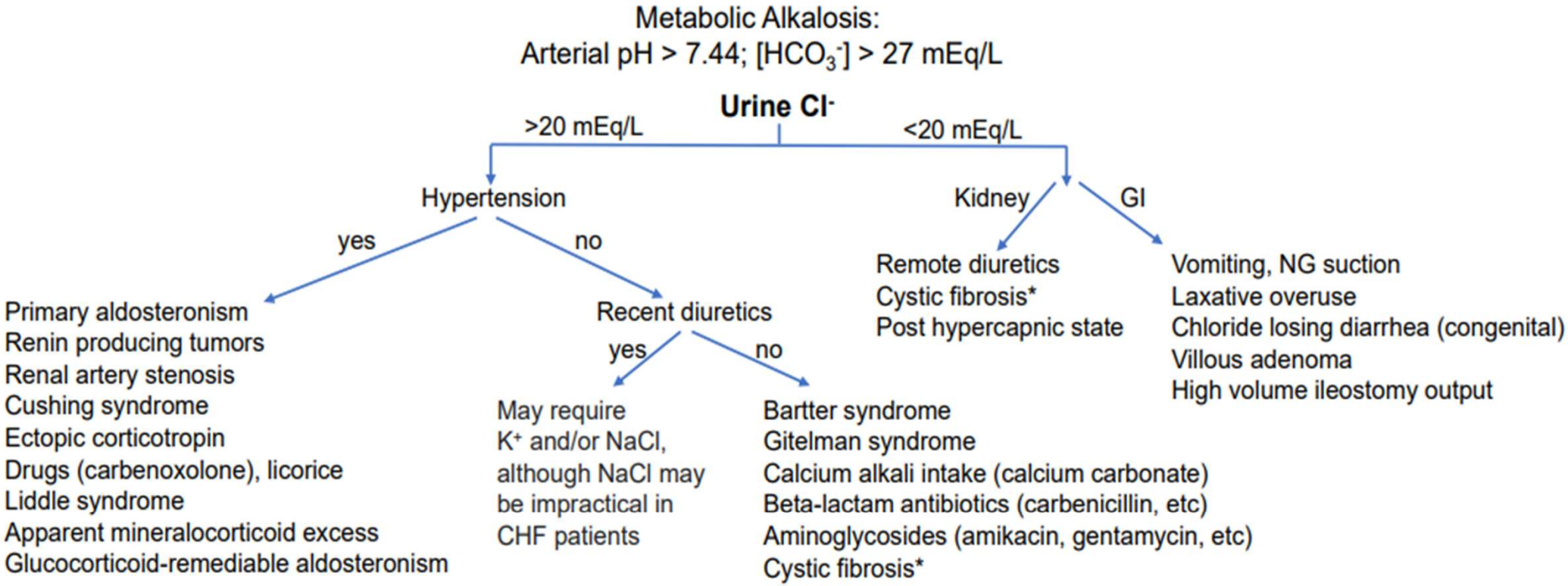


Figure 6. Algorithm for an approach to metabolic alkalosis based on urine chloride. Abbreviations: AME, apparent mineralocorticoid excess; CHF, congestive heart failure; GI, gastrointestinal; GRA, glucocorticoid-remediable aldosteronism; NG, nasogastric. *Cystic fibrosis may present with either low (<20 mmol/L) or high (>20 mmol/L) urine chloride.

Evaluation and Management

- Her urine Cl^- is <20mmol/L
- Management
  - Giving chloride rich IVF
    - **Normal Saline**
    - Lactated Ringers
    - Plasma-Lyte
  - Replacing potassium
  - Getting NG tube out when possible

27 year old male comes to the ER with bloody, perfuse watery diarrhea for 3 days. He is feeling fatigued and having cramping abdominal pain.

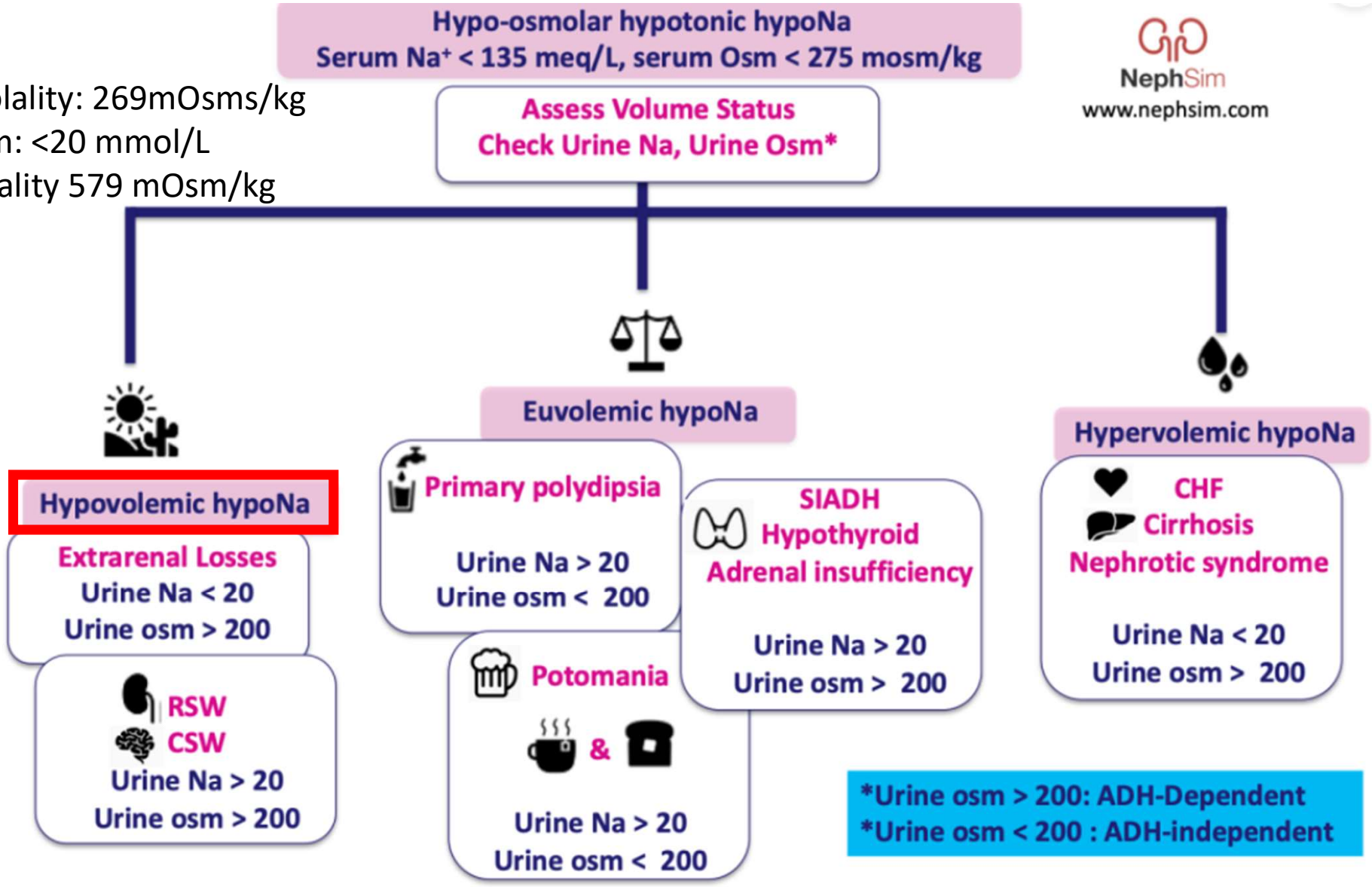
- Vitals:
  - Temp: 100 F
  - BP 80/50
  - HR 119
  - RR 24
  - Weight: 100kg
- Physical exam:
  - Sick appearing
  - Abd: soft, diffuse TTP, no rebound or guarding

- Labs:
  - Na 115
  - K 3.2
  - Cl 91
  - CO_2 15
  - AG 9
  - Creatinine 1.7

  - pH 7.35
  - pCO_2 26
  - Bicarbonate 15

Hyponatremia

Serum Osmolality: 269mOsms/kg
Urine Sodium: <20 mmol/L
Urine Osmolality 579 mOsm/kg



Acid-Base Disturbance

Primary Disturbance

A/B Disturbance	pH 7.35-7.45 7.35	pCO_2 35-45mmHg 28	HCO_3^- 22-26mEq/L 15
Metabolic Acidosis	↓	↓	↓
Metabolic Alkalosis	↑	↑	↑
Respiratory Acidosis (HCO_3^- acute/chronic)	↓	↑	**Normal/↑**
Respiratory Alkalosis Always acute	↑	↓	**Normal**

Acid-Base Disturbance

- Primary Metabolic Acidosis
- Normal Anion Gap: 9—normal albumin
- Respiratory Compensation
- Final diagnosis
  - Non-Anion Gap Metabolic Acidosis

Acid-Base Disorders in the Critically Ill Patient

Anand Achanti and Harold M. Szerlip

CJASN 18: 102–112, January, 2023

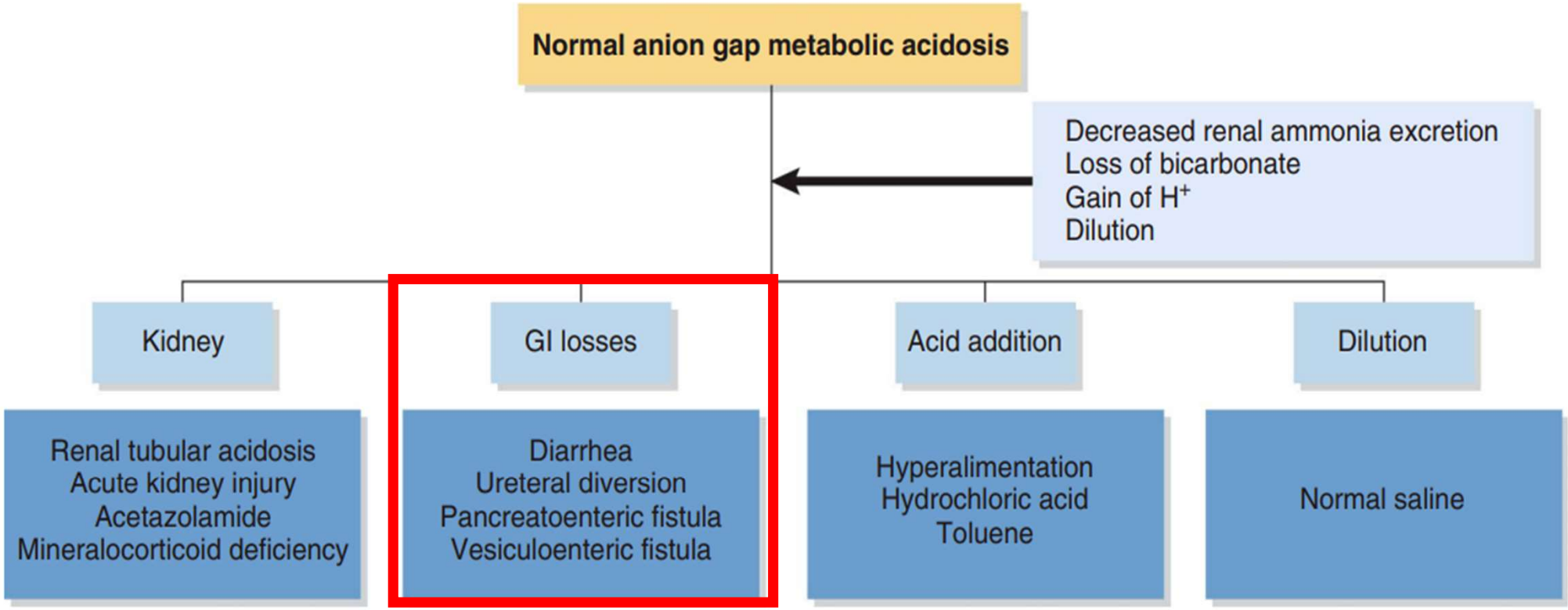


Figure 6. | Etiology of a non–gap metabolic acidosis.

Acute Kidney Injury

URINALYSIS, MACROSCOPIC AN...	!
URINALYSIS, MACROSCOPIC	!
APPEARANCE	Cloudy !
COLOR	Normal (Yell...
SPECIFIC GRAVITY, URINE	>1.030 ▲
GLUCOSE	Negative
BILIRUBIN	Negative
KETONES	Negative
BLOOD	Negative
PH URINE	7.0
PROTEIN	Negative
UROBILINOGEN	Negative
NITRITE	Negative
LEUKOCYTES	Negative
URINALYSIS, MICROSCOPIC	
RBC'S	2.0
WBC'S	1.0
BACTERIA	Occasional o...

- Urine Osmolality 465mOsm/kg
- Urine Sodium <20 mmol/L
- FeNa = 0.7%
- FeUrea = 13%
- Protein/Creatinine ratio: 256mg/g
- Renal US: No obstruction
- Pre-renal: Volume depletion

Management

Metabolic Acidosis

- Treat diarrhea
- Replace bicarbonate
  - LR, Plasma-Lyte

AKI

- Replace volume
  - NS, LR, Plasma-Lyte

Hyponatremia

- IVF
  - NS, LR, Plasma-Lyte
- **Overcorrection is a big concern**
- Replacing potassium will cause sodium to rise

Hyponatremia Overcorrection Management

- The patient receives 3L NS and repeat serum sodium 4 hours later is now 125.

1. Stop NS
2. Is this acute or chronic hyponatremia?
   - <48 hours acute
   - >48 hours chronic
   - If unknown treat as chronic
3. Give D5W bolus and IV D5W
4. Give DDAVP

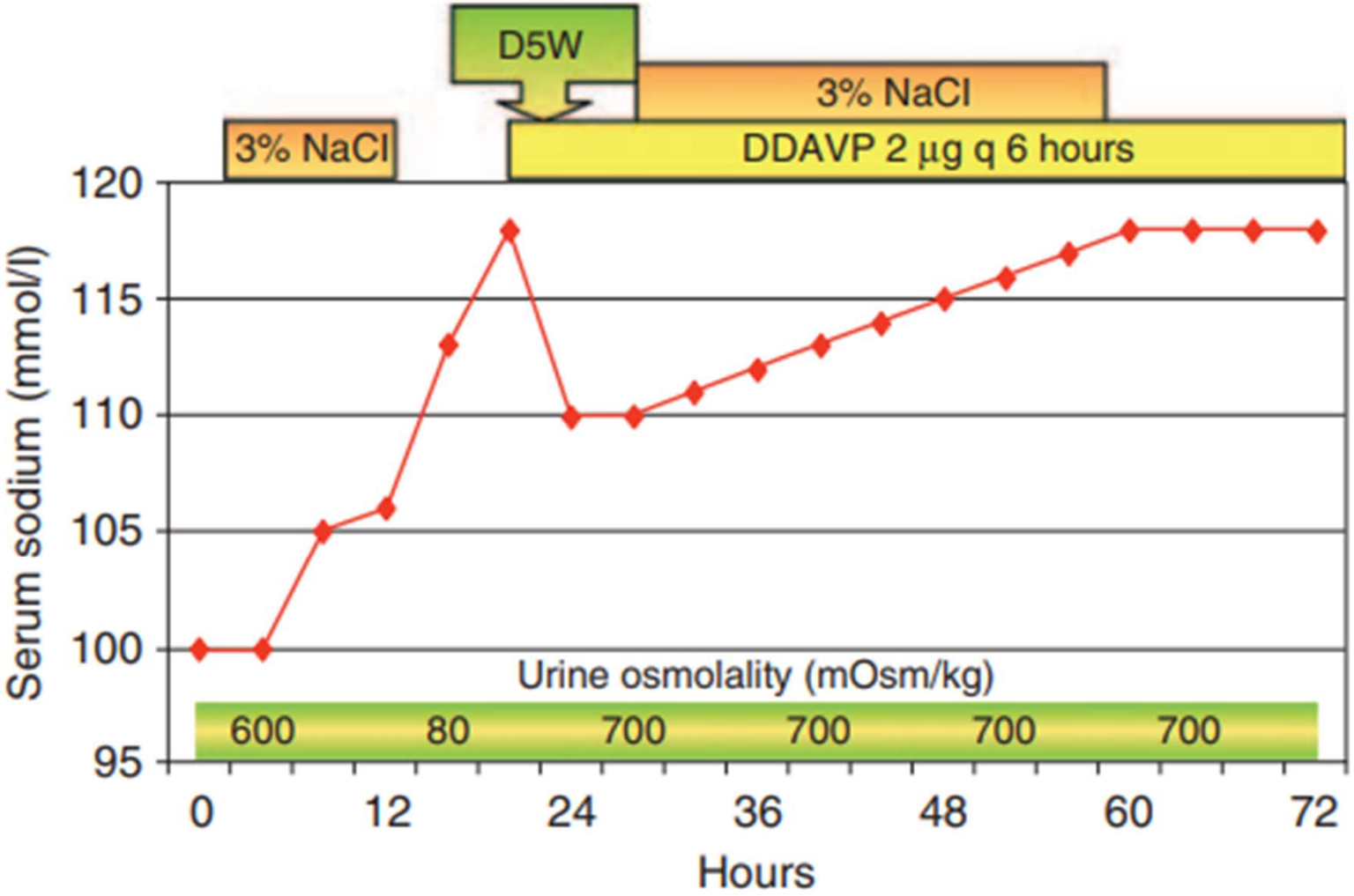
How much D5W to give?

- Change in serum Na^+ = (Infusate Na^+ - Serum Na^+) ÷ (TBW + 1)
- TBW = weight (kg) x correction factor
  - Water composition correction factor
    - 50% (young) and 45% (elderly) of lean weight (kg) in females
    - 60% (young) and 50% (elderly) of lean weight (kg) in young males[6]
- Change in serum Na^+ = (0- 125)÷ (50L + 1) =2.5mmol/L D5W given
- So they would need roughly 2L D5W
- Giving DDAVP along with the D5W helps prevent water diuresis

Overcorrection of hyponatremia is a medical emergency

Kidney International (2009) **76**

Richard H. Sterns[1,2] and John K. Hix[1,2]



Take Home Points

- Hyperkalemia
  - Temporizing measures vs potassium removal
- Acid-Base
  - Walking through in an orderly fashion
- Hyponatremia
  - Check serum/urine osmolality and urine sodium
  - Be careful of over correction in the hypovolemic hyponatremia
- Dialysis indications
  - A,E,I,O,U

References

1. Renal Association Clinical Practice Guidelines – Treatment of Acute Hyperkalemia in Adults. July 2020. 1-161
2. Montague, B. (2008). Retrospective Review of the Frequency of ECG Changes in Hyperkalemia. *CJASN*, *3*, 324–330.
3. Palmer, B. (2021). Clinical Management of Hyperkalemia. *Mayo Clinic Proceedings*, *96*(3), 744–762.
4. Johnson, R. (2015). *Comprehensive Clinical Nephrology*(5th ed.). Philadelphia, PA: Elsevier Saunders.
5. Rose, B. D., & Post, T. W. (2001). *Clinical physiology of acid-base and electrolyte disorders*. (5th ed.). New York: McGraw-Hill.
6. Reilly, R., Jr., & Perazella, M. (2014). *Nephrology in 30 Day*. McGraw-Hill. ISBN 978-0-07-178840-3