Nephrology for the Hospitalist

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Disclosures

- Non-Declaration Statement: I have no relevant relationships with ineligible companies to disclose within the past 24 months. (Note: Ineligible companies are defined as those whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients.)

Objectives

- Approaching the patient with an AKI
- Acid-Base Disturbances
- Management of Hyperkalemia
- Management of Hyponatremia
- Contrast exposure and minimizing AKI
- Diuresis in the fluid overloaded patient

- 82 year old female presents to the ER after she is found down by her daughter for an unknown time.
- PMH: HTN, HLD, DM, hx CVA, CAD s/p CABG
- Medications
  - ASA 81mg
  - Carvedilol 12.5mg BID
  - Lisinopril 40mg
  - Metformin 1,000mg BID
  - Atorvastatin 80mg

Vitals:

Temp: 97.3 F

BP: 90/45

HR: 107

RR: 16

SpO_2: 98% on 2L NC

- Na 133 mmol/L
- K 6.2 mmol/L
- Cl 97 mmol/L
- CO_2 18 mmol/L
- AG 18 mmol/L
- BUN 65 mg/dL
- Cr 1.9 mg/dL (previously 1.0)
- Creatinine kinase 64,876 mg/L
- VBG
  - pH 7.19
  - PCO_2 43 mmHg
  - Bicarbonate 17 mmol/L
  - Lactate 6.5 mmol/L

Hyperkalemia

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

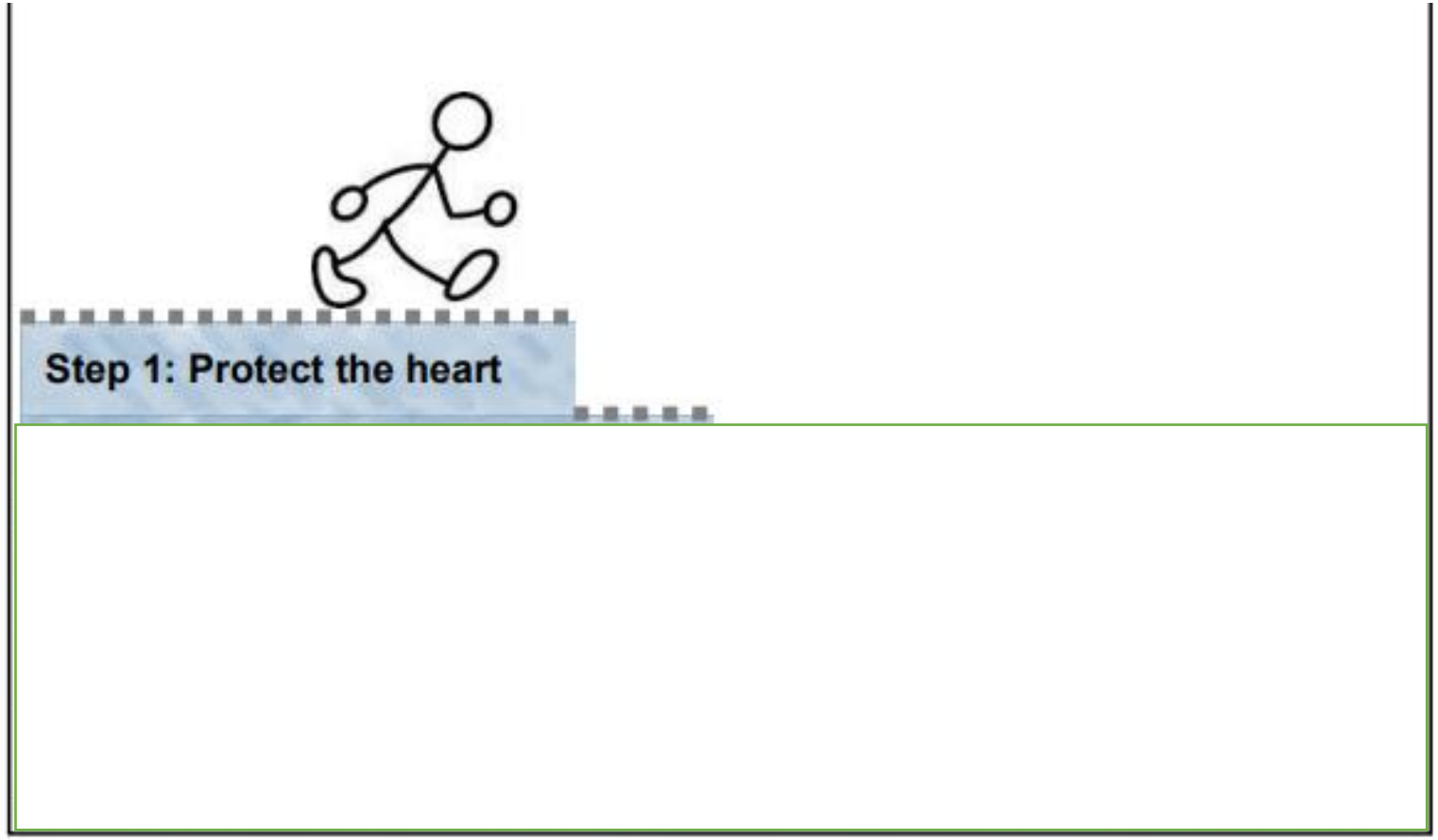


Figure 4: There are five key steps in the treatment of hyperkalaemia (*never walk away without completing all of these steps*).

EKG= poor sensitivity and specificity[2]

THE RENAL ASSOCIATION
founded 1950

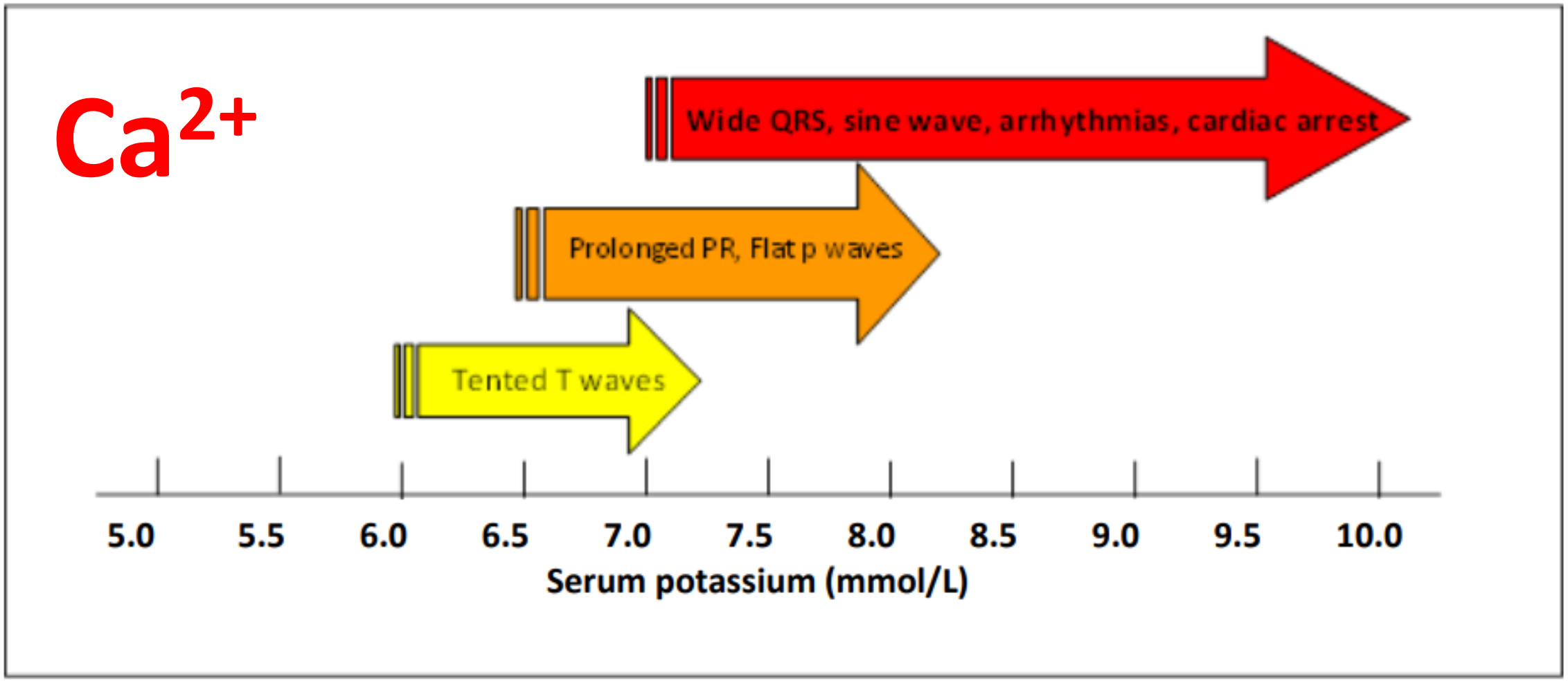


Figure 1: Progressive changes in ECG with increasing severity of hyperkalaemia.

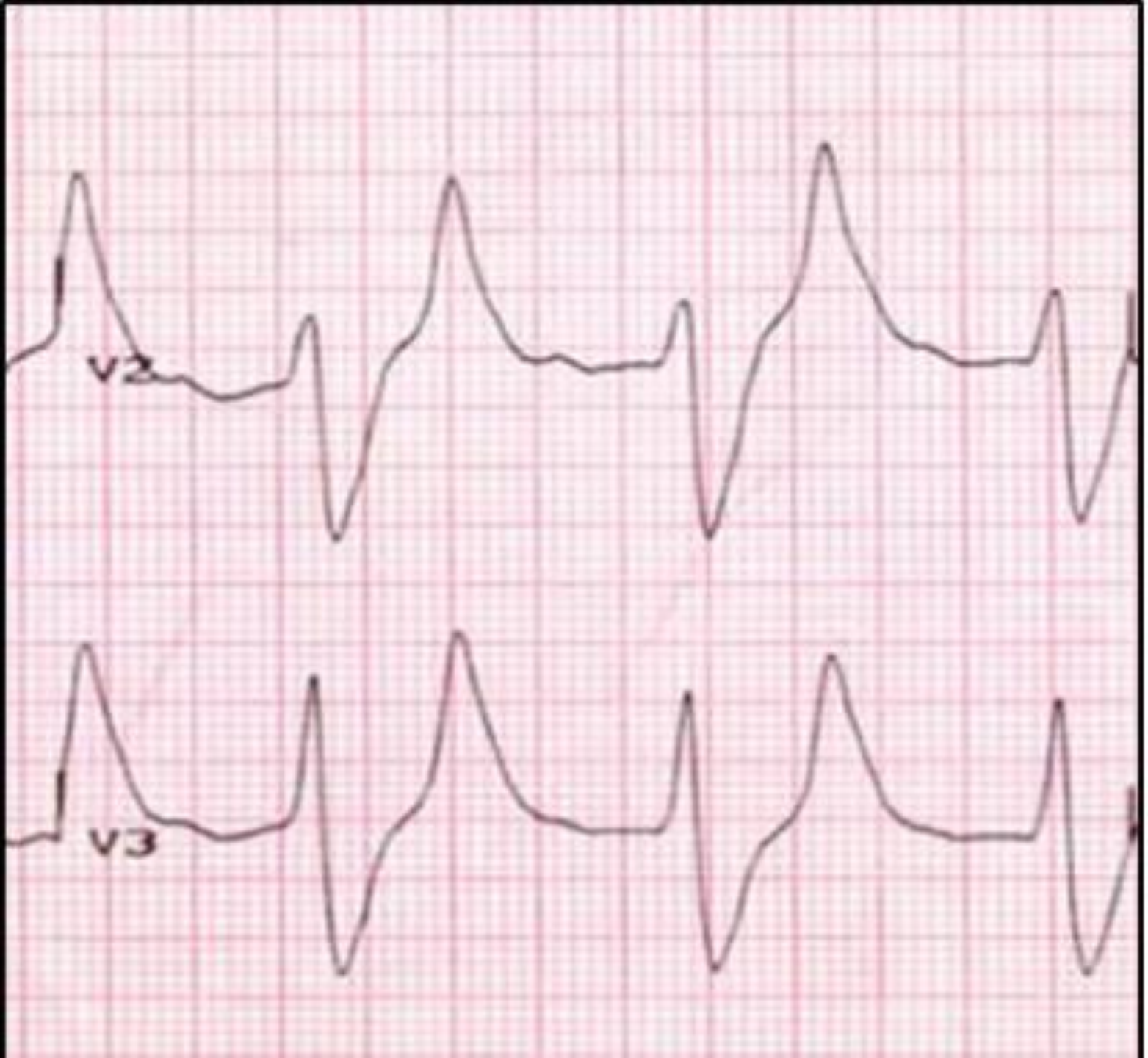
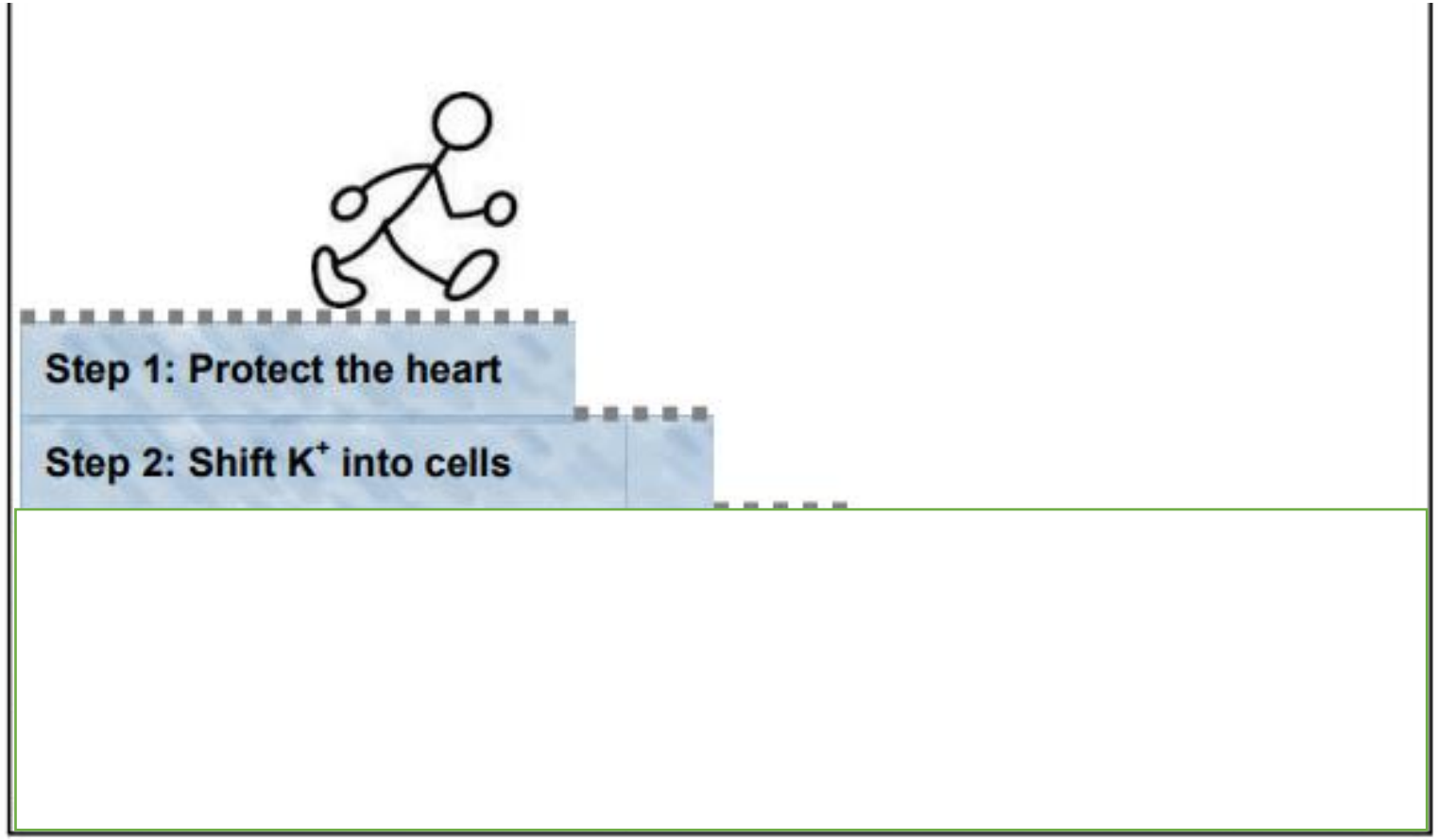


Figure 2: K 9.1 mmol/l

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



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Cell Shifting

Insulin/Dextrose and Albuterol

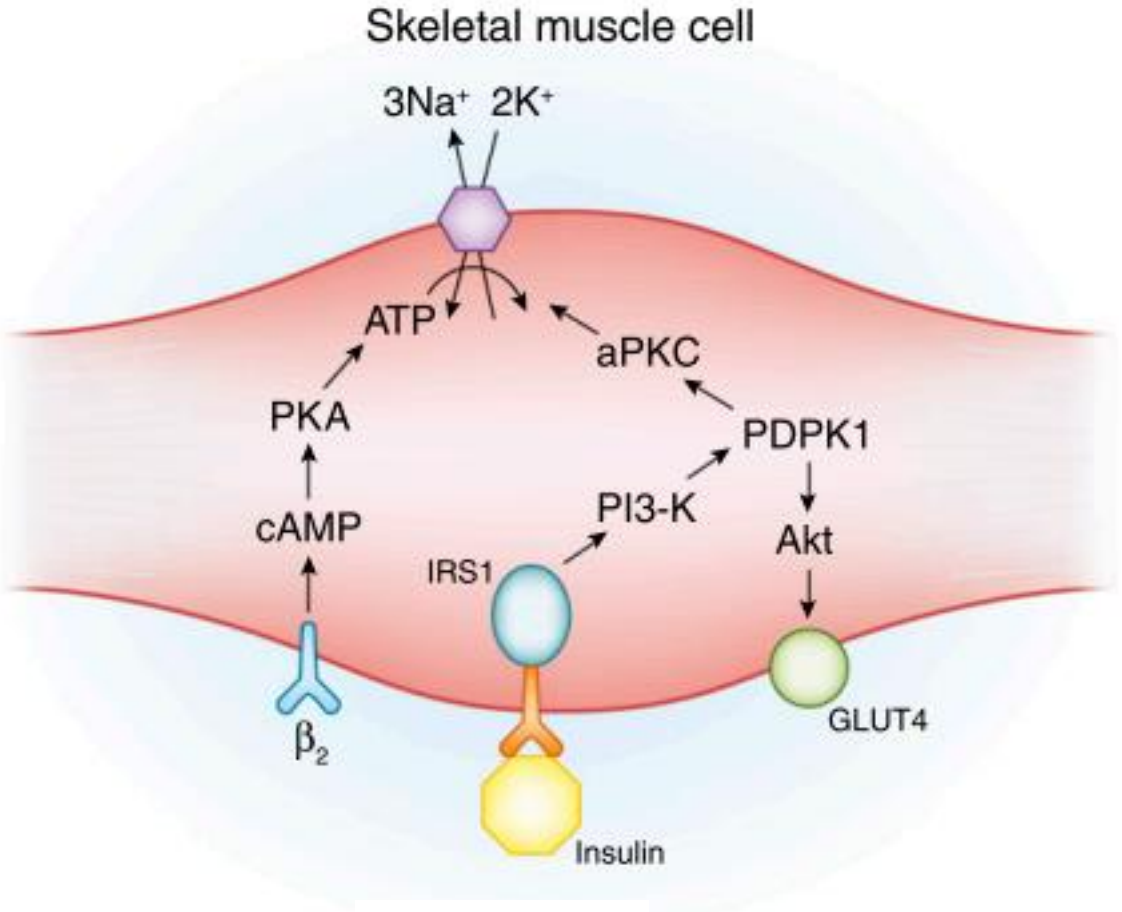


Figure 1.

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

Bicarbonate

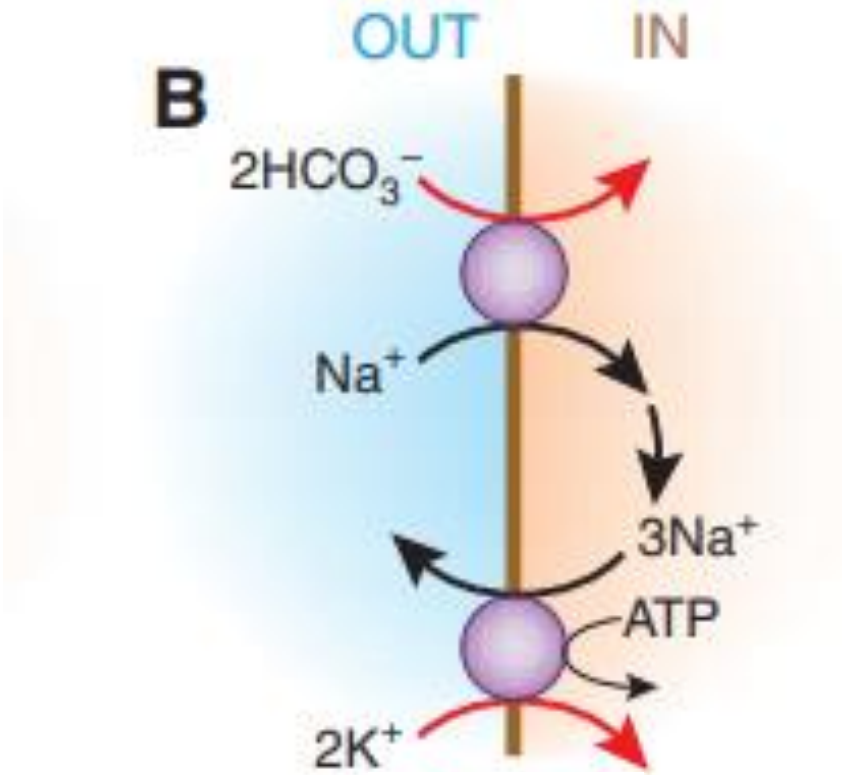


Figure 3.

J Am Soc Nephrol 22: 1981–1989, 2011

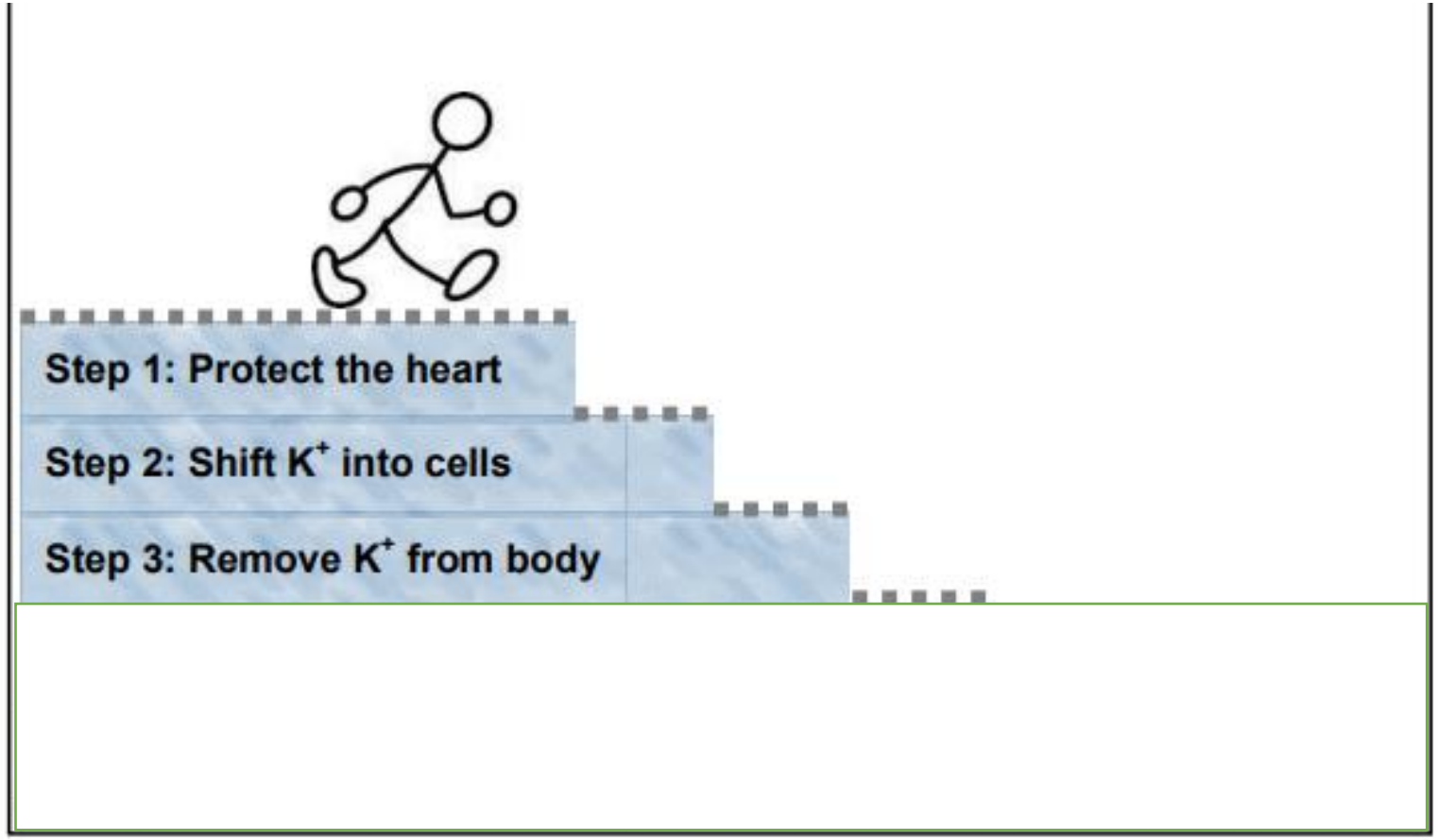
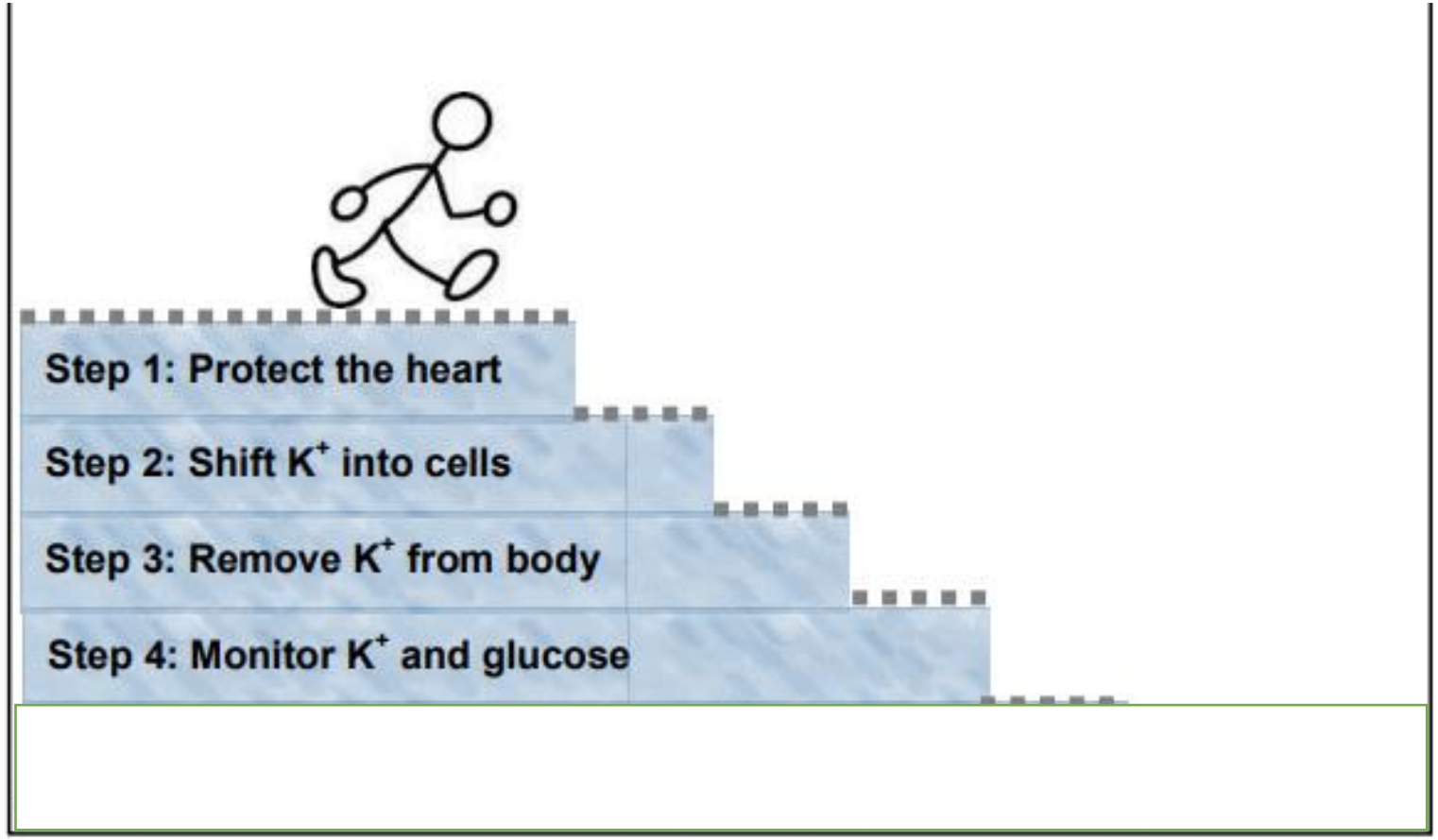


Figure 4: There are five key steps in the treatment of hyperkalaemia (*never walk away without completing all of these steps*).

Potassium Removal

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



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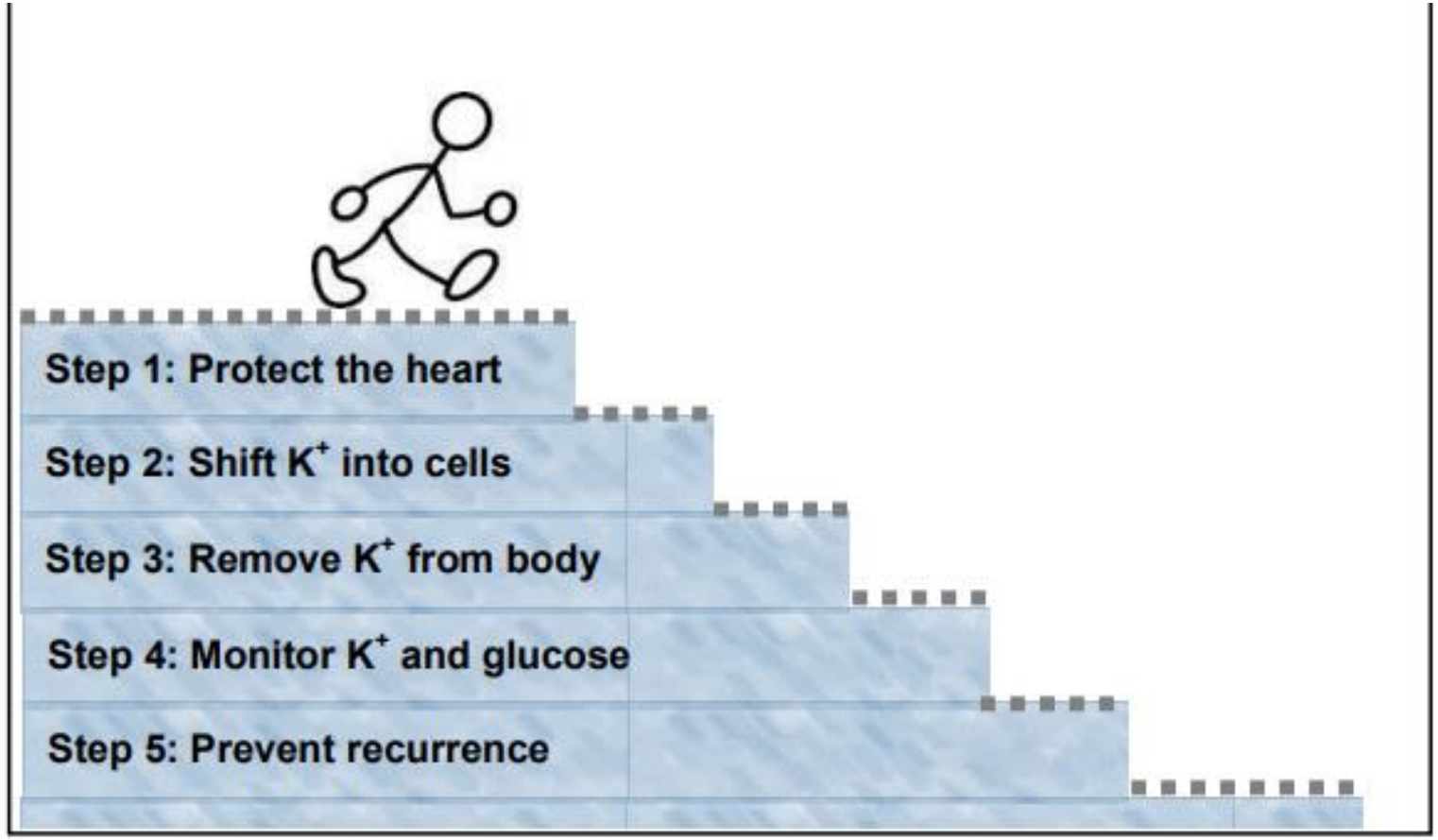


Figure 4: There are five key steps in the treatment of hyperkalaemia (*never walk away without completing all of these steps*).

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.

Heme-Pigment Nephropathy

- Renal injury due to
  - Volume depletion → ischemic injury
  - Tubular cast formation
  - Direct tubular toxicity of myoglobin
- IVF are imperative
  - Alkalinization of urine can decrease cast formation
  - No strong data in using one specific type of IVF[4]

Anion Gap Acidosis

- Anion Gap 18
- VBG
  - pH 7.19
  - PCO_2 43 mmHg
  - Bicarbonate 17 mmol/L
  - Lactate 6.5 mmol/L

Glycols

Oxoproline

L-Lactate

D-Lactate

Methanol

Aspirin

Renal failure

Ketoacidosis

- 55 year old male is admitted to your service for NSTEMI. The patient has and AKI (Cr. 2.1mg/dL, GFR 32cc/min). Cardiology is wanting to take the patient for left heart catheterization due to ongoing chest pain. What can you do to minimize the risk contrast induced nephropathy?

  A. Give him normal saline 100cc/hr
  B. Give him N-Acetylcysteine and Sodium Bicarbonate
  C. Recommend not doing the heart catheterization until AKI resolves
  D. A and B

- Contrast-associated acute kidney injury (CA-AKI):
  - AKI occurring within 48 hours after the administration of contrast, but not exclusively due to contrast (other factors at play-sepsis, nephrotoxic medications)
- Contrast-induced acute kidney injury (CI-AKI):
  - AKI linked to contrast administration

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 15, 2018 VOL. 378 NO. 7

Outcomes after Angiography with Sodium Bicarbonate and Acetylcysteine

S.D. Weisbord, M. Gallagher, H. Jneid, S. Garcia, A. Cass, S.-S. Thwin, T.A. Conner, G.M. Chertow, D.L. Bhatt, K. Shunk, C.R. Parikh, E.O. McFalls, M. Brophy, R. Ferguson, H. Wu, M. Androsenko, J. Myles, J. Kaufman, and P.M. Palevsky, for the PRESERVE Trial Group*

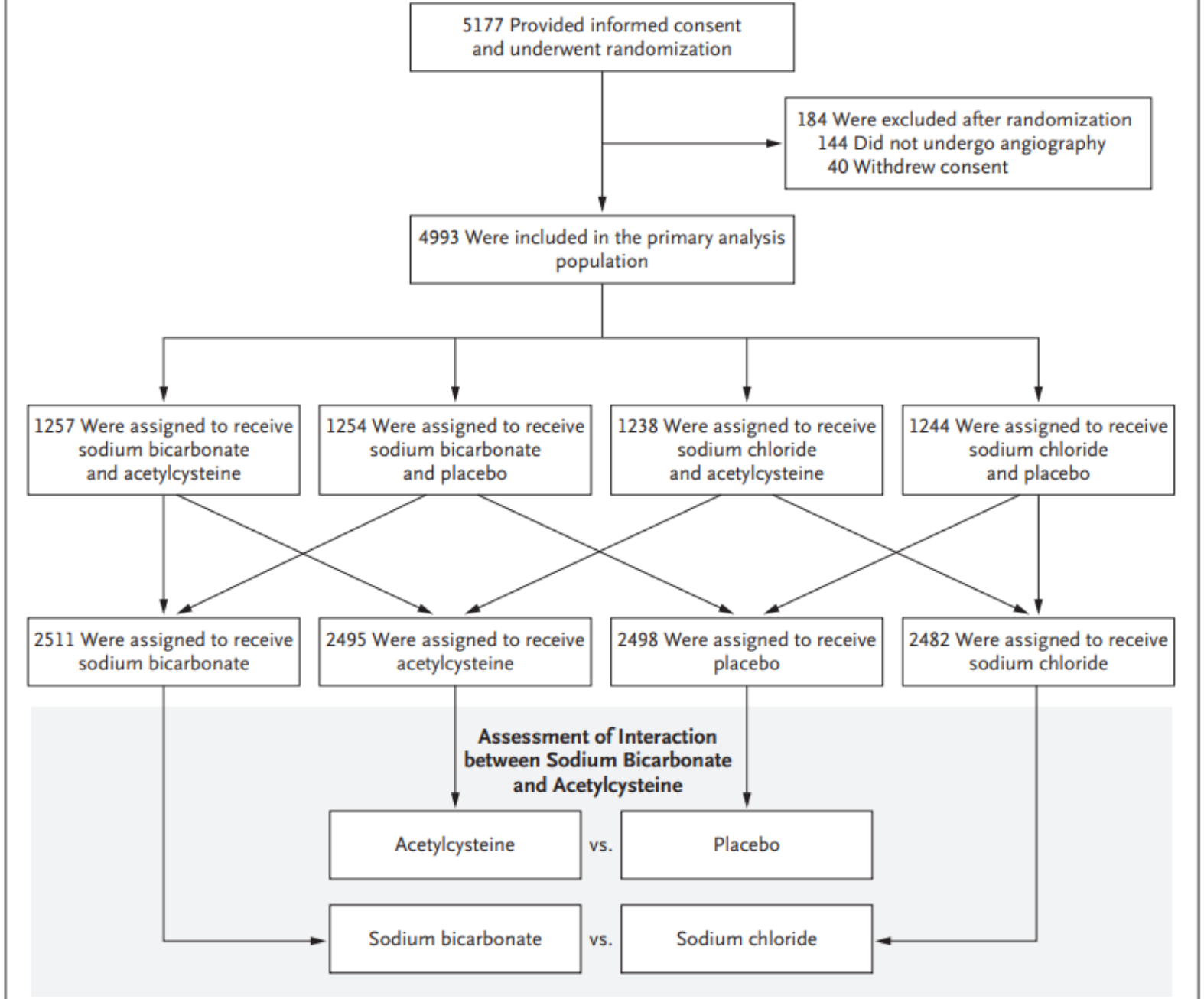
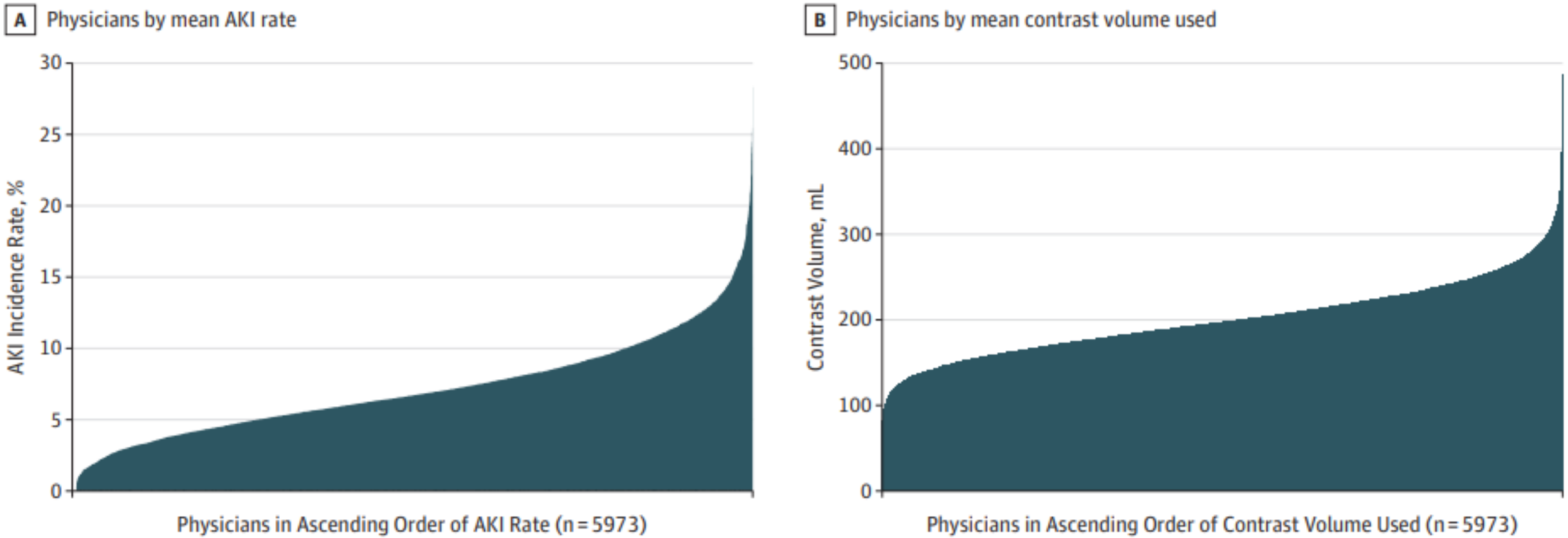


Figure 1. Enrollment and Randomization.

Association of Variation in Contrast Volume With Acute Kidney Injury in Patients Undergoing Percutaneous Coronary Intervention
JAMA Cardiology September 2017 Volume 2, Number 9

Figure 1. Variation in Acute Kidney Injury (AKI) Incidence Rate and Mean Contrast Volume per Percutaneous Coronary Intervention Among US Physicians



A, Physicians (on the x-axis) are listed in ascending order according to their AKI rate, lowest to highest. B, Physicians (on the x-axis) are listed in ascending order according to their mean contrast volume used, lowest to highest.

Which is more likely to result in AKI- intravenous vs intra-arterial contrast?

Kidney Injury after Intravenous versus Intra-arterial Contrast Agent in Patients Suspected of Having Coronary Artery Disease: A Randomized Trial

Radiology 2019; 292:664–672

Eva Schönenberger, MD • Peter Martus, PhD • Maria Bosserdt, MD • Elke Zimmermann, MD • Rudolf Tauber, MD • Michael Laule, MD • Marc Dewey, MD

Investigative Radiology • Volume 51, Number 12, December 2016

Acute Kidney Injury After Intravenous Versus Intra-Arterial Contrast Material Administration in a Paired Cohort

Jennifer S. McDonald, PhD, Caleb B. Leake, BS,† Robert J. McDonald, MD, PhD,* Rajiv Gulati, MD, PhD,‡ Richard W. Katzberg, MD,|| Eric E. Williamson, MD,* and David F. Kallmes, MD*§*

Use of Intravenous Iodinated Contrast Media in Patients with Kidney Disease: Consensus Statements from the American College of Radiology and the National Kidney Foundation

Radiology 2020; 294:660–668

- CI-AKI lower than previously thought
- IV fluids in:
  - AKI
  - eGFR less than 30 mL/min
- Holding potentially nephrotoxic medications if feasible for 24 to 48 hours before and after contrast exposure
- Holding ACE/ARB is controversial

Gadolinium

- Nephrogenic systemic fibrosis (NSF)
  - Fibrosing disorder of skin, connective tissue, and internal organs
  - Seen in CKD 4/ESRD
  - Delayed clearance allows Gadolinium to dissociate from the chelating agent and deposit in tissue
  - Can appear weeks to months after exposure[5,6]

Table 1. Current or previously approved gadolinium-based contrast agents and their manufacturer, chemical structure and ionicity, American College of Radiology classification, and Canadian Association of Radiologists risk assessment

Gadolinium Agent	Manufacturer	Chemical Structure	ACR Classification	CAR Risk Assessment
Gadodiamide	GE Healthcare	Linear nonionic	Group 1	High risk
Gadoversetamide	Guerbet	Linear nonionic	Group 1	High risk
Gadopentetate dimeglumine	Bayer AG	Linear ionic	Group 1	High risk
Gadobutrol	Bayer Healthcare/Bayer AG	Macrocyclic nonionic	Group 2	Low risk
Gadoteridol	Bracco Diagnostics	Macrocyclic nonionic	Group 2	Low risk
Gadoterate meglumine	Guerbet	Macrocyclic ionic	Group 2	Low risk
Gadobenate dimeglumine	Bracco Diagnostics	Linear ionic	Group 2	Medium risk
Gadoxetate disodium	Bayer Healthcare	Linear ionic	Group 3	Medium risk

ACR, American College of Radiology; CAR, Canadian Association of Radiologists.

5

If Gadolinium is needed in CKD stage 4/5 or ESRD try to avoid Group 1 or 3[5,6]

Contrast Take Home Points

- Iodinated contrast
  - Are there alternative studies?
  - IV less risk than direct arterial contrast
- Gadolinium
  - Ask which class is going to be given or alternative studies
  - Time HD to immediately after exposure for 3 days in a row
- Discuss with patient about possible risks vs benefits

- 67 year old female comes to the ER with 40 pound weight gain over the last 2 months, lower extremity edema and progressive dyspnea on exertion over the last 2 weeks.
- PMH: HTN, DM, HLD, CAD s/p PCI, CKD stage 3,Depression
- Medications:
  - ASA 81mg
  - Atorvastatin 40mg
  - Valsartan 80mg
  - Spironolactone 25mg
  - Furosemide 40mg daily
  - Insulin Glargine 25 units nightly
  - Sertraline 25mg

- Vitals:
  - Temp 98.2
  - BP 147/90
  - HR 101
  - RR 27
  - O_2 92% 4L NC

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

Labs 3 months ago

CHEM 1	
SODIUM	137 *
POTASSIUM	4.5 *
CHLORIDE	103 *
CARBON DIOXIDE	28 *
BUN	36 * ▲
CREATININE	1.11 * ▲
GLUCOSE	114 *
ANION GAP	6 *
BUN/CREAT RATIO	32 * ▲
ESTIMATED GLOMERUL...	48 * ▼
CALCIUM	9.5 *

Admission labs

CHEM 1	
SODIUM	128 ▼
POTASSIUM	5.0
CHLORIDE	97
CARBON DIOXIDE	22 ▼
BUN	33 ▲
CREATININE	1.56 ▲
GLUCOSE	166 ▲
ANION GAP	9
BUN/CREAT RATIO	21
ESTIMATED GLOMERUL...	23 * ▼
CALCIUM	10.0

Hyponatremia-Is it True?

- Normal serum osmolality ~280mOsm (Pseudohyponatremia)
  - Hypertriglyceridemia
  - Hyperglobulinemia
- Elevated serum osmolality >290mOsm
  - Hyperglycemia
  - Mannitol
  - IVIG
  - Contrast[7]

Serum Osmolality <280mOsm

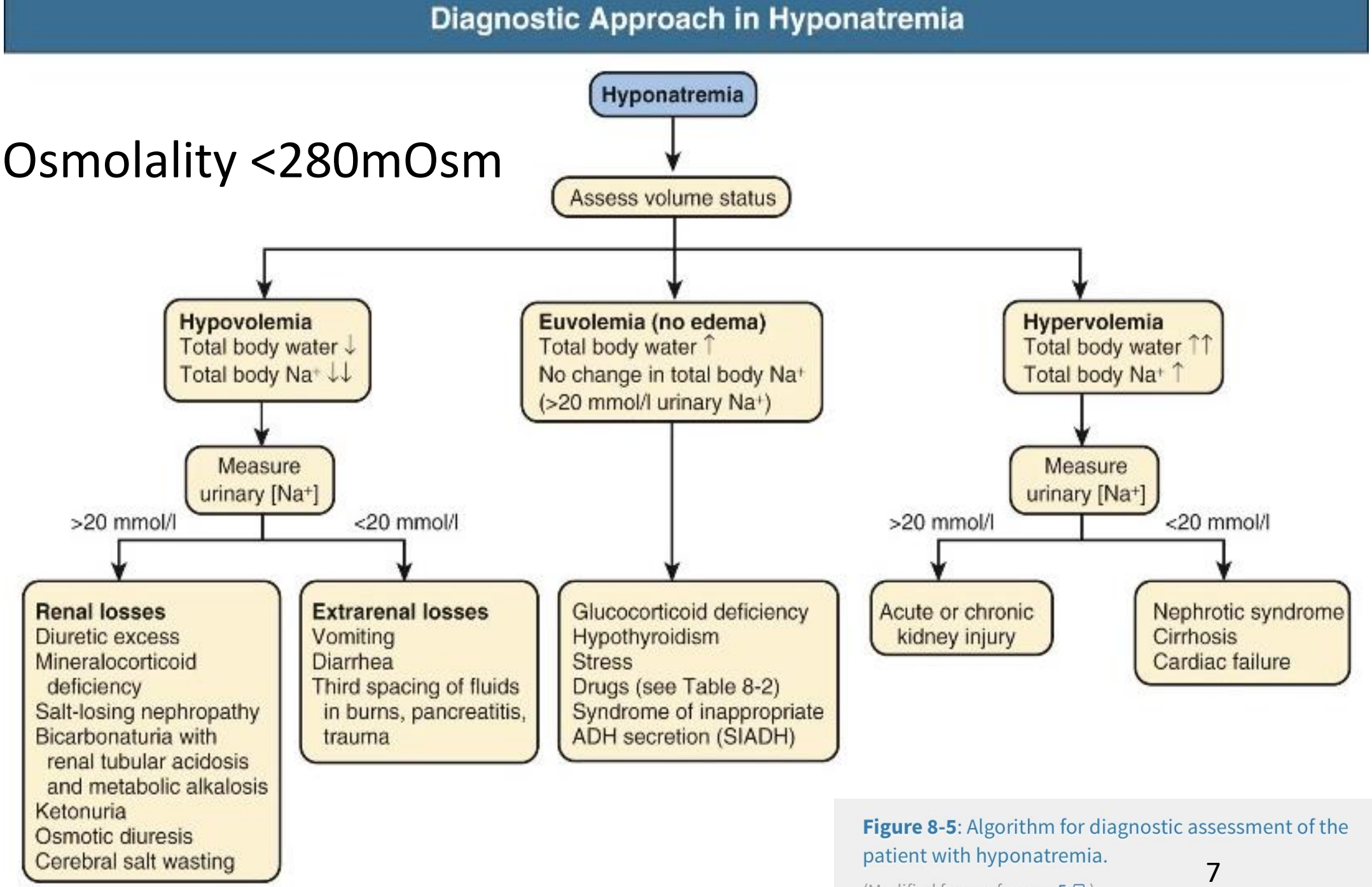


Figure 8-5: Algorithm for diagnostic assessment of the patient with hyponatremia.

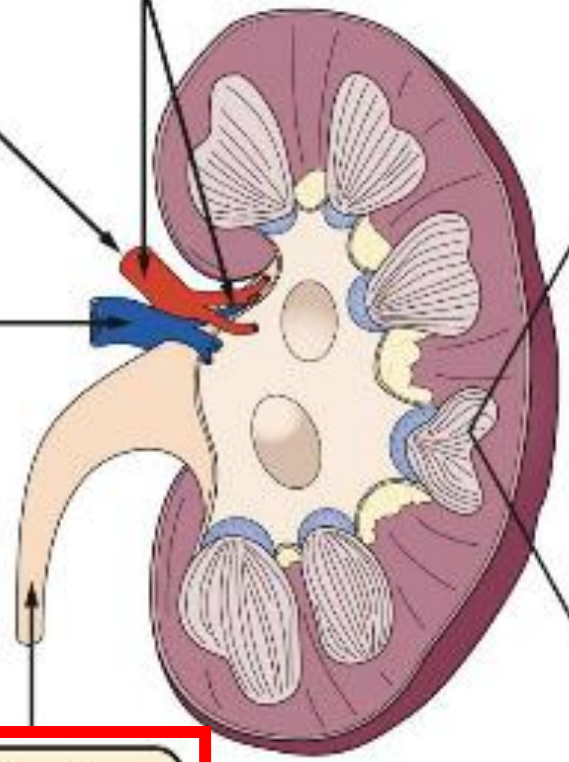
(Modified from reference 5.)

- Serum Osmolality: 270mOsms/kg
- Urine Sodium: 20 mmol/L
- Urine Osmolality 456 mOsm/kg

Interpretation of Urine Studies

- Low urine sodium <20mmol/L
  - Poor renal perfusion → increased sodium reabsorption
- High urine sodium >20mmol/L
  - Diuretics
  - Euvolemic
  - ATN
- Urine osmolality
  - Variable 50-1,200 mOsm/kg
  - Vasopressin activity → increased water reabsorption → higher urine osmolality

Causes of AKI



1. Pre-renal azotemia
Hypovolemia
Cardiac failure
Hepatorenal syndrome

2. Renal artery
Renal artery occlusion
Large- or medium-vessel vasculitis

3. Small-vessel disease
Thrombotic microangiopathy
Renal atheroembolism
Small-vessel vasculitis

4. Glomerular disease
Anti-GBM disease
Lupus nephritis
Postinfectious glomerulonephritis
Infective endocarditis
Membranoproliferative glomerulonephritis
Cryoglobulinemia
IgA nephropathy/Henoch-Schönlein purpura

5. Acute tubular necrosis
Ischemia
Nephrotoxins
Rhabdomyolysis
Radiocontrast agents

6. Acute interstitial nephritis
Drugs
Infection
Systemic disease

7. Intratubular obstruction
Cast nephropathy
Drugs
Crystalluria

8. Post-renal obstruction
Bladder outlet obstruction
Tumors
Renal calculi
Papillary necrosis
Retroperitoneal fibrosis

9. Renal vein
Renal vein thrombosis

Renal US shows no hydronephrosis

- Urine Sodium: 20 mmol/L
- Urine Osmolality 456 mOsm/kg
- Urine Creatinine: 65 mg/dL
- Urine Urea:44 mg/dL
- FeNa: 0.3%
- FeUrea: 3%
- Hyponatremia/AKI due to volume overload and vascular congestion

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**

DIURESIS

Anaesthesia 2018, 73, 238–247 doi:10.1111/anae.14038

Review Article

Continuous infusion vs. intermittent bolus injection of furosemide in acute decompensated heart failure: systematic review and meta-analysis of randomised controlled trials

K. T. Ng[1] and J. L. L. Yap[2]

Diuretics in States of Volume Overload:
Core Curriculum 2022

James E. Novak and David H. Ellison

AJKD Vol 80 | Iss 2 | August 2022

Table 3. Stepwise Diuretic Dosing Algorithm From CARRESS-HF

Level	Furosemide Dose		Suggested Metolazone Dose
	Current	Suggested (Bolus, Infusion)	
A	≤80 mg/d	40 mg, 5 mg/h	0
B	81-160 mg/d	80 mg, 10 mg/h	5 mg/d
C	161-240 mg/d	80 mg, 20 mg/h	5 mg 2×/d
D	≥240 mg/d	80 mg, 30 mg/h	5 mg 2×/d

Therapy was escalated to the next level if 3 L/d urine output was not achieved; other medical and technical interventions were also allowed. Based on information in Bart et al, 2012 (*J Card Fail*. https://doi.org/10.1016/j.cardfail.2011.12.009).
Abbreviation: CARRESS-HF, Cardiorenal Rescue Study in Acute Decompensated Heart Failure.

Diuretic Resistance

- Increased RAAS/Aldosterone
- Increased sympathetic tone
- High sodium diet
- Gut edema
- Long standing diuretic use
- Not a high enough dose

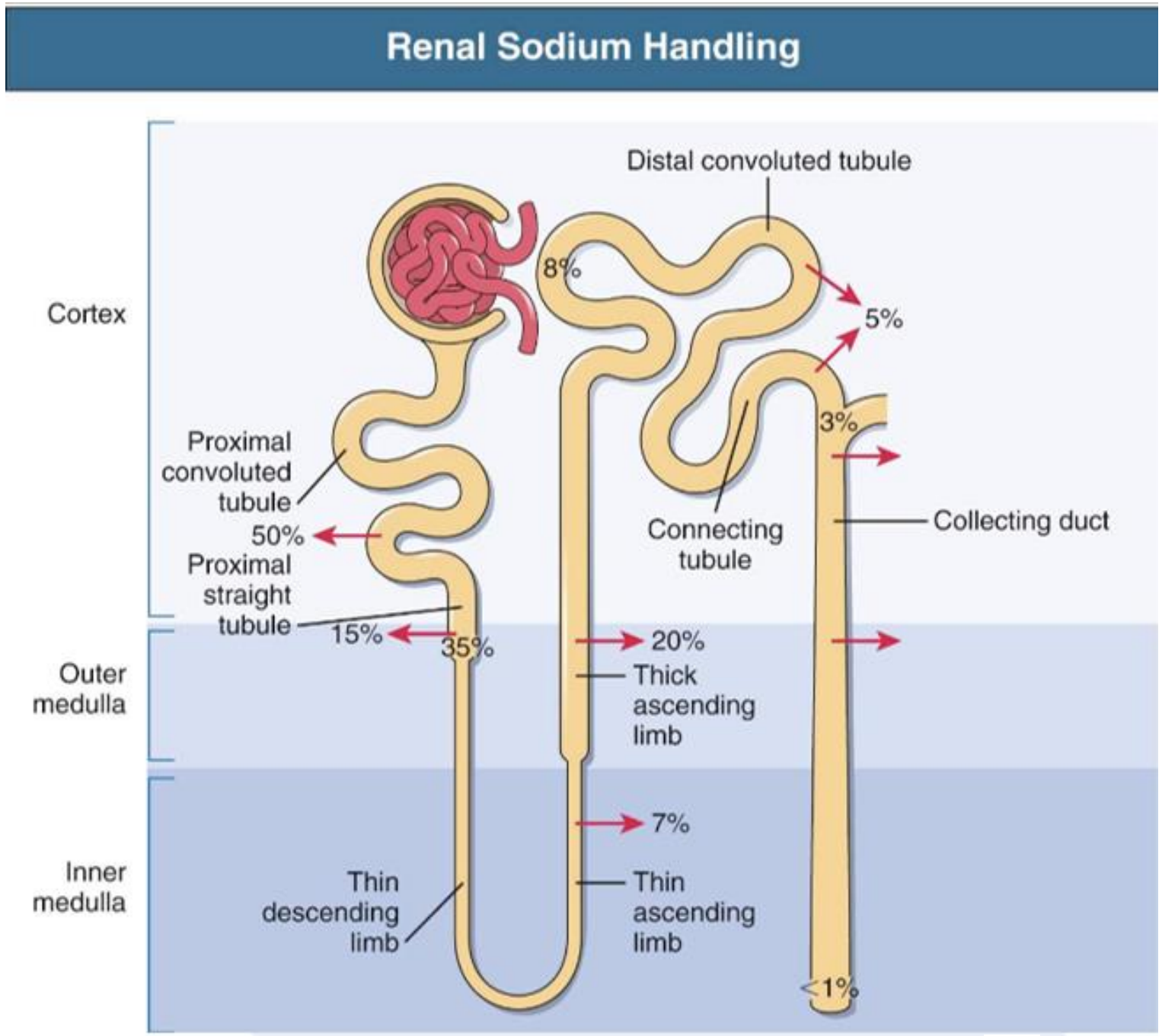


Figure 2-7: Renal sodium handling along the nephron.

Loop Diuretic Plus

- Thiazide diuretics
  - Hydrochlorothiazide
  - Chlorthalidone
  - Metolazone
  - Chlorothiazide

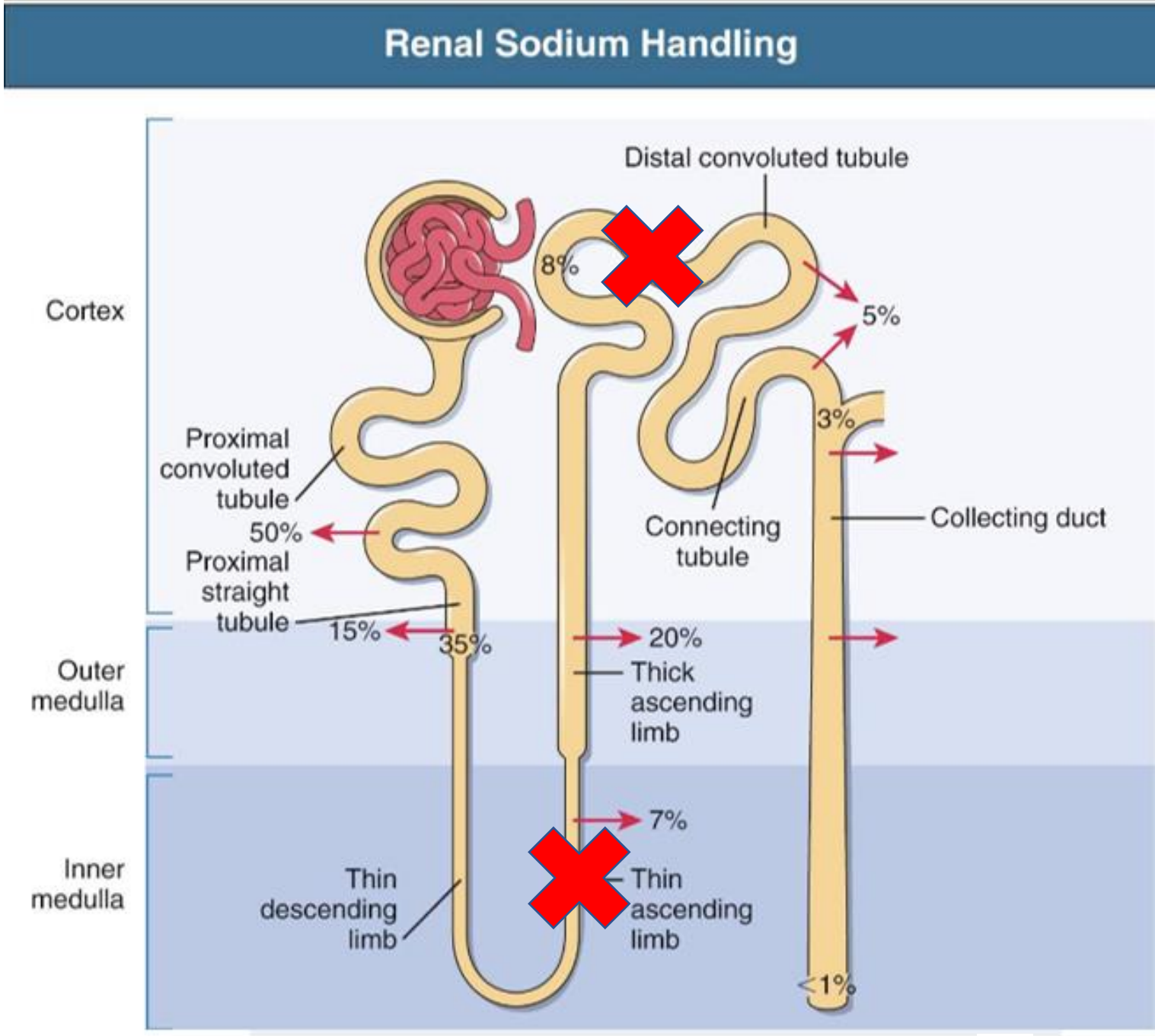


Figure 2-7: Renal sodium handling along the nephron.

Loop Diuretic Plus

- Thiazide diuretics
  - Hydrochlorothiazide
  - Chlorthalidone
  - Metolazone
  - Chlorothiazide
- Potassium sparing
  - Spironolactone
  - Eplerenone
  - Amiloride

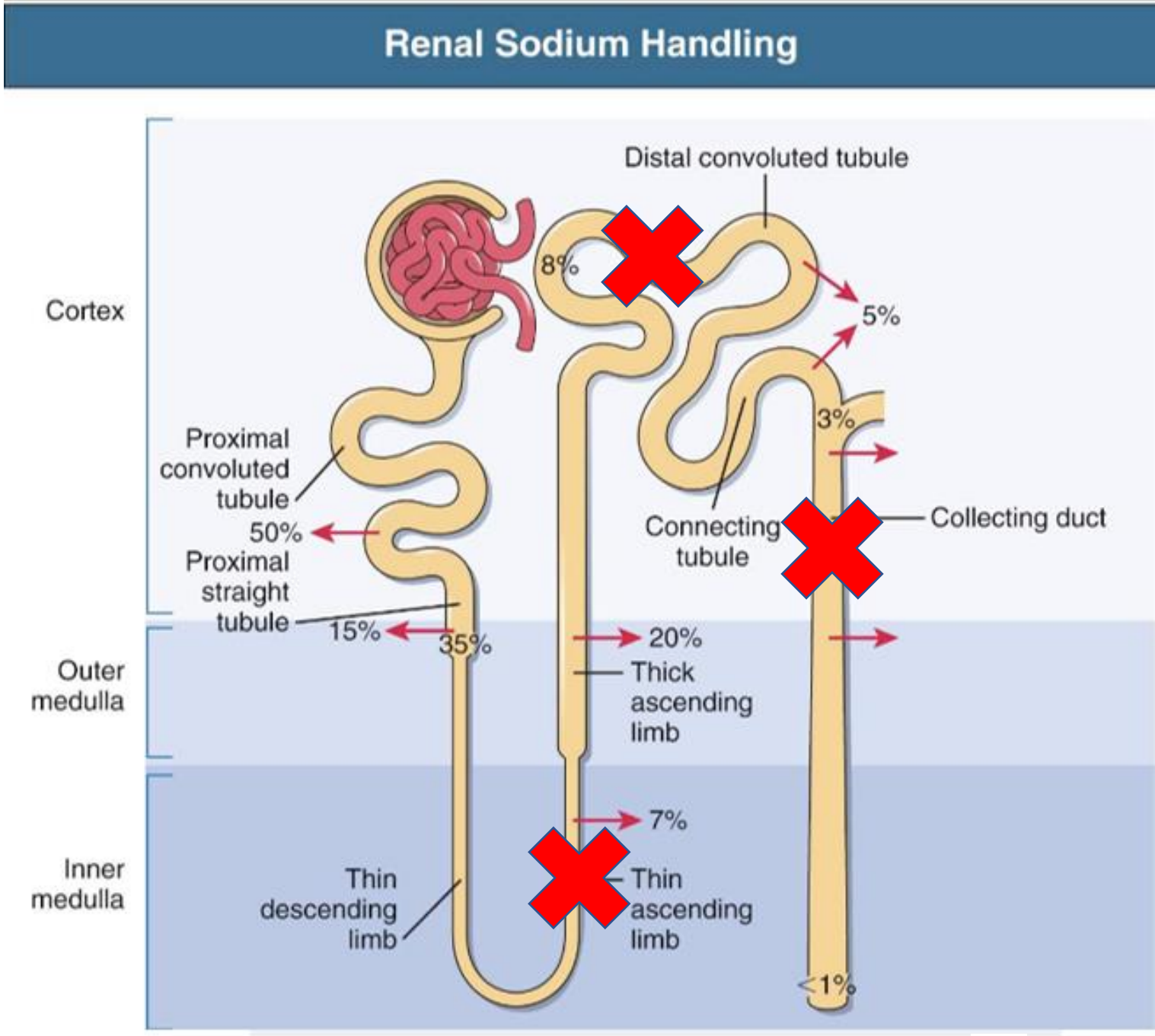


Figure 2-7: Renal sodium handling along the nephron.

Day 2

CHEM 1
SODIUM
POTASSIUM
CHLORIDE
CARBON DIOXIDE
BUN
CREATININE
GLUCOSE
ANION GAP
BUN/CREAT RATIO
ESTIMATED GLOMERUL...
CALCIUM

Day 4

CHEM 1		
SODIUM	138 *	
POTASSIUM	4.8 *	
CHLORIDE	104 *	
CARBON DIOXIDE	24 *	
BUN	31 *	▲
CREATININE	1.58 *	▲
GLUCOSE	94 *	
ANION GAP	10 *	
BUN/CREAT RATIO	20 *	
ESTIMATED GLOMERUL...	32 *	▼
CALCIUM	9.2 *	

What does a Rising Creatinine Mean?

- **Worsening Renal Function in Patients With Acute Heart Failure Undergoing Aggressive Diuresis Is Not Associated With Tubular Injury. Circulation. 2018 May 08; 137(19): 2016–2028**
  - Looked at ROSE-AHF trial
  - Found that rise in creatinine did not specifically correlate with tubular injury
- **Relevance of Changes in Serum Creatinine During a Heart Failure Trial of Decongestive Strategies: Insights From the DOSE Trial. J Card Fail. 2016 October ; 22(10): 753–760**
  - Look at DOSE trial data
  - Found that increase creatinine was associated with better outcomes

So what does it mean?

- **Hemoconcentration**
- **Hemodynamic changes**

Table 1. Pharmacokinetics of Diuretics

Diuretic	Bioavailability	Equivalent Dose, mg	Metabolism (Kidney/Liver)	Elimination $t_{1/2}$, h			
				Normal	CKD	CHF	ESLD
Loop							
Furosemide	50%-60% (10%-100%)[a]	40	100%/0%	1.5-2	2.6-2.8	2.7	2.5
Bumetanide	80%-100%	1	50%/50%	1	1.6	1.3	2.3
Torsemide	68%-100%	20	20%/80%	3-4	4-5	6	8
Thiazide							
HCTZ	65%-75%	25	100%/0%	6-15	↑	↔	↔
Chlorthalidone	60%-72%	12.5	100%/0%	40-60	↑	↔	↔
Metolazone	65%-90%	2.5	70%-95%/5%-30%	14-20	↑	↔	↔
Distal							
Amiloride	50%	10	50%/–[b]	6-26	100	?	↔
Triamterene	52%-80%	100	20%/80%	2-5	↑	?	–[c]
Spironolactone	>90%	25	0%/100%	>15[d]	↔	?	↔

Based on information in Hoorn and Ellison, 2017 (*Am J Kidney Dis.* https://doi.org/10.1053/j.ajkd.2016.08.027). Abbreviations: $t_{1/2}$, half-life; AKI, acute kidney injury; CHF, congestive heart failure; CKD, chronic kidney disease; EABV, effective arterial blood volume; ESLD, end-stage liver disease; FFA, free fatty acid; HCTZ, hydrochlorothiazide.

[a]The usual range is taken as 50%-60%, but some have reported a range as great as 10%-100%.

[b]Amiloride is 50% excreted in the stool.

[c]Triamterene requires hepatic activation and is considered inactive in ESLD.

[d]Spironolactone undergoes rapid hepatic breakdown to active metabolites; parent molecule elimination $t_{1/2}$ = 1.5 h.

24 year old female comes into the ER for nausea, vomiting and diarrhea for the last 3 days. She has been able to drink some water.

- Vitals:
  - Temp: 98.7
  - HR 102
  - BP 90/55
  - RR 22
- PE:
  - ENT: Dry mucus membranes
  - Cardiac: Tachycardic
  - Lungs: CTA
  - Abd: TTP in epigastric region
  - Skin: Tenting of the skin

- Na 122 mmol/L
- K 2.7 mmol/L
- Cl 87 mmol/L
- Bicarbonate 22 mmol/L
- BUN 33 mg/dL
- Cr 1.5 mg/dL

Serum Osmolality 265 mOsm/kg
Urine Sodium <20 mmol/L
Urine Osmolality 653 mOsm/kg

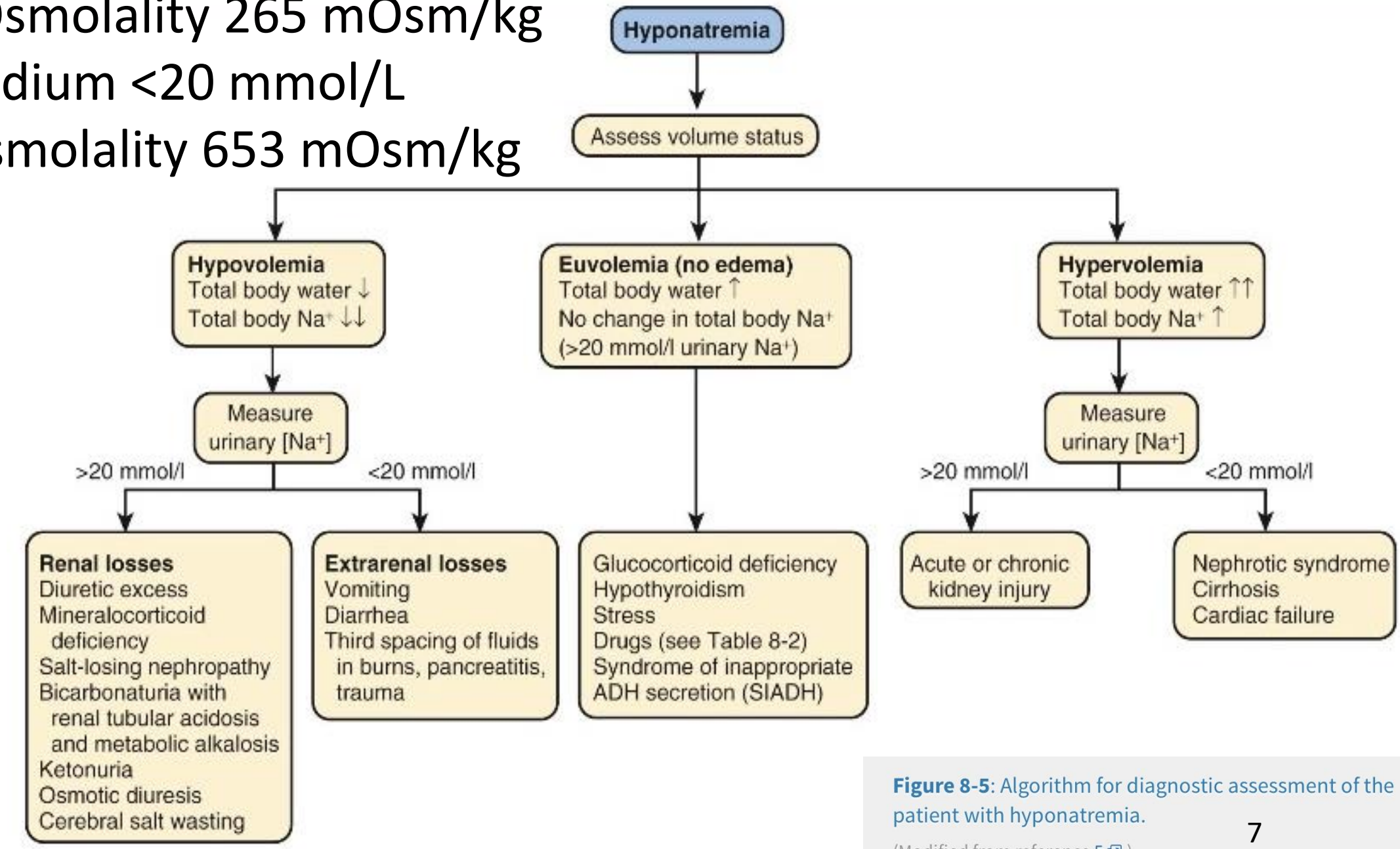


Figure 8-5: Algorithm for diagnostic assessment of the patient with hyponatremia.

(Modified from reference 5.)

- While waiting for labs:
  - Given 2L normal saline
- Labs return:
  - Given 20mEq KCL
- What is your initial concern?
- Overcorrection of hyponatremia

Hypovolemia

↓

Vasopressin + RAAS activation

↓

Leads to renal water and sodium retention

↓

IVF given—reaching euvolemia

↓

↓

Dilute urine output

↓

Rapid rise in serum Na

- You recheck her sodium after NS infusions and it is now 137
- Now what?
  - D5W bolus and continuous infusion
  - DDAVP to slow urine output
- What about replacing her potassium?
  - This will also cause the serum sodium to rise

Correction of Hyponatremia

- Acute <48 hours
- Chronic >48 hour
  - If unknown treat as chronic
  - Goal correction around 6-8mEq/24 hours
- Severe Symptomatic Patients
  - Seizure, AMS
  - ICU→3% Saline
  - Correct ~4mEq/L[7]

Osmotic Demyelination (Central Pontine Myelinolysis)

- Risk factors
  - Malnourished
  - Alcoholics
  - Hypokalemia
  - Liver disease
- Seen in 13-29% at autopsy
- Signs/Symptoms
  - Encephalopathy, behavioral problems, cranial nerve palsies, quadriplegia[7]

- 64-year-old Caucasian male with a PMH of GERD on Famotidine and adenocarcinoma of the colon treated with partial colectomy 20 years ago presents to clinic with unexplained weight loss of 20 pounds in the last month, fatigue, nausea and periodic joint pain in which he has been taking PRN Tylenol/Ibuprofen.
- His PCP draws labs.

CBC	
WBC	5.0
HGB	7.6
HCT	24.3
PLATELET COUNT (AUTO)	268
PLATELET COUNT	268
SEDIMENTATION RATE	
RBC	2.77
MCV	87.7
MCHC	31.3
MCH	27.4
RDW	
RDW-CV	14.3

CHEM 1	
SODIUM	135
POTASSIUM	4.5
CHLORIDE	102
CARBON DIOXIDE	24
BUN	39
CREATININE	2.61
GLUCOSE	142
GLUCOSE,NONFAST	
ANION GAP	9
BUN/CREAT RATIO	15
ESTIMATED GLOMERUL...	23 *
CALCIUM	9.5

Baseline labs 6 months ago: Hgb 12, Cr 0.7

- You are called by the PCP for a direct admission and accept.
- You interview the patient and they have not had any fevers, vomiting, diarrhea, constipation, abdominal pain, edema, dysuria or gross hematuria. He has only taken 4 doses of Ibuprofen in the last month.
- PE:
  - Vitals unremarkable.
  - ill appearing but otherwise unremarkable

Causes of AKI

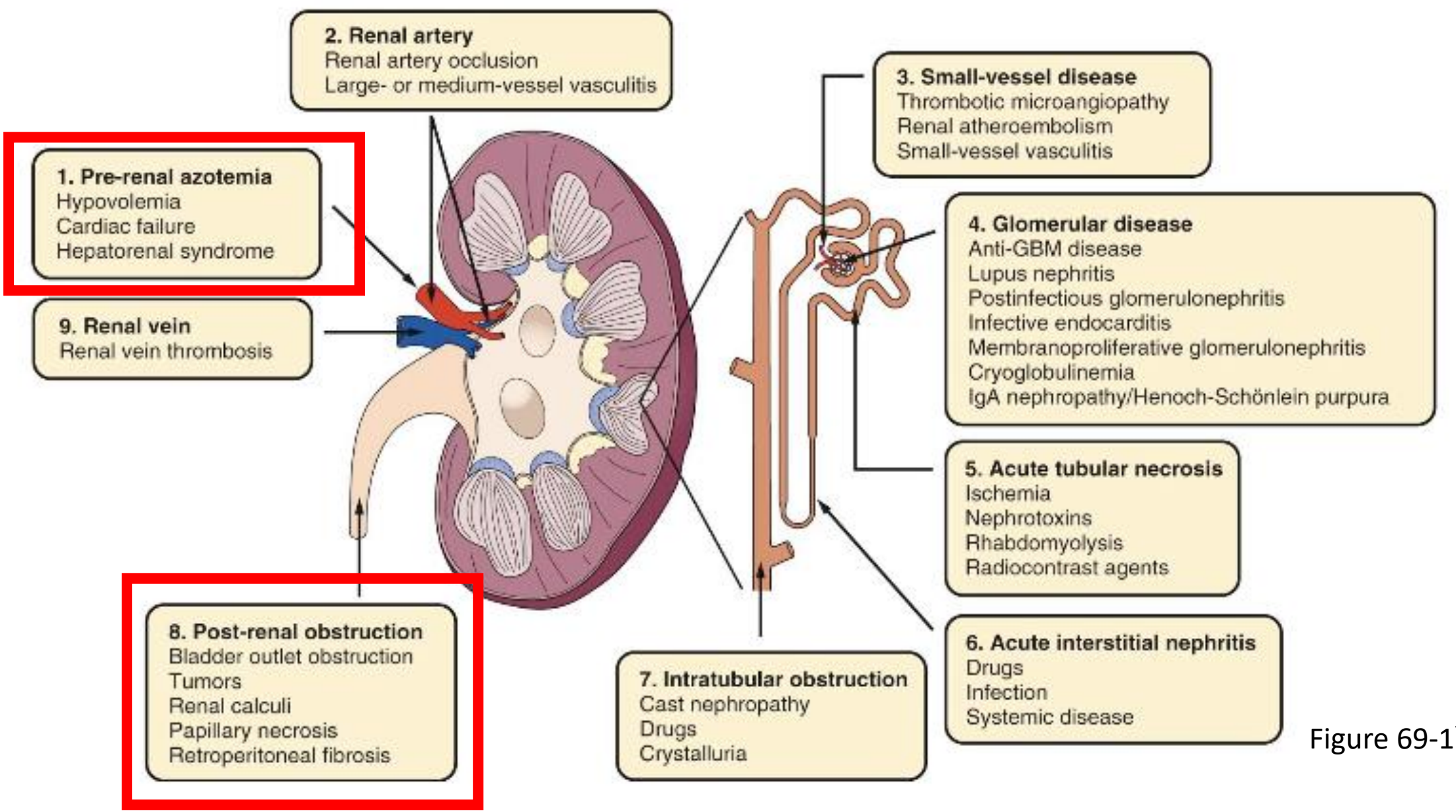


Figure 69-1[7]

APPEARANCE	Clear *	
COLOR	Normal (Yellow) *	
SPECIFIC GRAVITY, ...	1.010 *	
GLUCOSE	Negative *	
BILIRUBIN	Negative *	
KETONES	Negative *	
BLOOD	Moderate *	!
PH URINE	6.0 *	
PROTEIN	100 *	!
UROBILINOGEN	Negative *	
NITRITE	Negative *	
LEUKOCYTES	Negative *	
URINE,MICROSCOPIC		
RBC'S	26.0 *	▲
WBC'S	5.0 *	
BACTERIA	Occasional or ... *	

CT A/P unremarkable for malignancy or obstruction

- Urine Sodium 44
- FeNa = 3.1%
- FeUrea = 60%
- Urine protein/creatinine= 1.3g/g
- AKI with hematuria and/or proteinuria = glomerulonephritis

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

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  - Proteinuria > 3.5g/day
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  - Edema
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  - Lipiduria

- **Nephritic syndrome**
  - Oligiuria
  - Hematuria
  - Proteinuria (classically <3g/day)
  - Edema
  - Hypertension
  - Abrupt onset

Nephrotic syndrome

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

Nephritic syndrome

- Oligiuria
- 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

- Differentials for Nephritic syndrome
  - ANCA vasculitis
  - Anti-GBM
  - Post infectious GN
  - Lupus nephritis
  - IgA nephropathy
  - Membranoproliferative GN
  - Thrombotic microangiopathy (TTP/HUS and Compliment mediated HUS)
  - Cryoglobulinemic Vasculitis
- Renal biopsy is performed showing ANCA vasculitis

- 75-year-old quadriplegic in a nursing home presents with increased AMS and lethargy. Per nursing reports she has been having nausea and vomiting for the last 2 days.
- PMH of HTN on Lisinopril and stage 2 sacral wounds

- Vitals:
  - Temp 38.9˚C.
  - BP 90/40,
  - HR 99
  - Weight 80kg
- PE: Diaphoretic, Dry mucus membranes, abd TTP, BS hypoactive.
- Labs:
  - Na 165mmol/L, K 3.1mmol/L, Cl 88mmol/L, CO_2 32mmol/L, BUN 52 mg/dL, Cr 1.4 mg/dL
  - VBG pH 7.5, PCO2 44, Bicarb 30

Hypernatremia

- Free water loss
  - GI loss
  - Insensible loss
  - Diabetes insipidus
- Free water deficit
  - FWD= ($\frac{Plasma\ Na\ Concentration}{140}$ -1) x total body water[8]
- FWD = 7.1L

Insensible and Urinary Loss

- Skin-500cc
- Respiratory-400cc
- GI-200cc
- Urine free water clearance=urine volume x(1-$\frac{urine\ [Na]+urine[K]}{serum\ [Na]}$)[8]

Final FWD

- FWD =~7.1L
- Insensible loss = ~1.0L/day
- Urinary free water loss = ~600mL/day
- =8.7L

Fluid	mEq/L						g/L			
	Na	Cl	K	Ca	Mg	PO_4^{-3}	Dextrose	Buffers	pH	Osmolality
Plasma	140	103	4	5	2	3.5	-	HCO_3	7.4	290
NS	154	154	-	-	-	-	-	-	5.7	308
½ NS	77	77	-	-	-	-	-	-		154
LR	130	109	4	3	-	-	-	Lactate	6.4	273
D5W	-	-	-	-	-	-	50	-	7.4	
Plasma-Lyte	140	98	5	-	3	1		Acetate/ Gluconate	7.4	295

Is the rate of correction of hypernatremia associated with clinical outcomes?

CJASN
Clinical Journal of American Society of Nephrology

Methods and Cohort

MIMIC

Data from Medical Information Mart for Intensive Care-III (MIMIC-III)

Na >155 mmol/L

On admission
N = 122

Hospital-acquired
N = 327

Rapid correction (>0.5 mmol/L/hr)

VS

Slow correction (≤0.5 mmol/L/hr)

Findings

Rapid Correction		Slow Correction
30 day mortality 25%	NS P=0.80	30 day mortality 28%
30 day mortality 44%	NS P=0.50	30 day mortality 40%

0 cases of cerebral edema, seizures or alteration in consciousness attributable to rapid hypernatremia correction

Conclusions Rapid correction of hypernatremia was not associated with a higher risk for mortality, seizures, alteration of consciousness and/or cerebral edema in critically ill adults with either admission or hospital-acquired hypernatremia.

Kinsuk Chauhan, Pattharawin Pattharanitima, Niralee Patel, Aine Duffy, et al. ***Rate of Correction of Hypernatremia and Health Outcomes in Critically Ill Patients.*** CJASN doi: 10.2215/CJN.10640918. **Visual Abstract by Michelle Lim, MBChB**

Metabolic Alkalosis

- Labs:
  - Na 165mmol/L, K 3.1mmol/L, Cl 88mmol/L, CO_2 32mmol/L, BUN 52 mg/dL, Cr 1.4 mg/dL
  - VBG pH 7.5, PCO2 44, Bicarb 30
- Commonly due to GI loss or over diuresis
- If not
  - Look at BP
  - Urine Chloride
  - Renin/Aldosterone

Fluid	mEq/L						g/L			
	Na	Cl	K	Ca	Mg	PO_4^{-3}	Dextrose	Buffers	pH	Osmolality
Plasma	140	103	4	5	2	3.5	-	HCO_3	7.4	290
NS	154	154	-	-	-	-	-	-	5.7	308
½ NS	77	77	-	-	-	-	-	-		154
LR	130	109	4	3	-	-	-	Lactate	6.4	273
D5W	-	-	-	-	-	-	50	-	7.4	
Plasma-Lyte	140	98	5	-	3	1		Acetate/ Gluconate	7.4	295

Advanced CKD Medication Dosing

- AVOID in GFR <30cc/min
  - Baclofen
  - Morphine
  - Duloxetine
  - NSAIDs
  - Fenofibrate
  - Sucralfate
  - Metformin
  - Glyburide
- Careful use in CKD
  - Bactrim
  - Gabapentin
  - Codeine
  - NSAIDs

Dialysis Indications

Acidosis

Electrolyte abnormalities

Ingestion

Oliguria

Uremia

Take Home Points

- Hyperkalemia
  - Calcium is imperative
  - EKG not great
  - Monitoring potassium every 2-4 hours
- Work up hyponatremia
  - Serum osmolality, urine osmolality and urine sodium
  - Monitor for over correction
- AKI with hematuria/proteinuria = GN

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