

RENAL MANAGEMENT IN THE CARDIOTHORACIC/VASCULAR PATIENT

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OVERVIEW

- AKI in the CVICU/VTICU
- Electrolyte Disturbances
- Acid-Base Considerations
- Volume Assessment and Management
- Diuretic Regimens
- CRRT Indications and Prescriptions

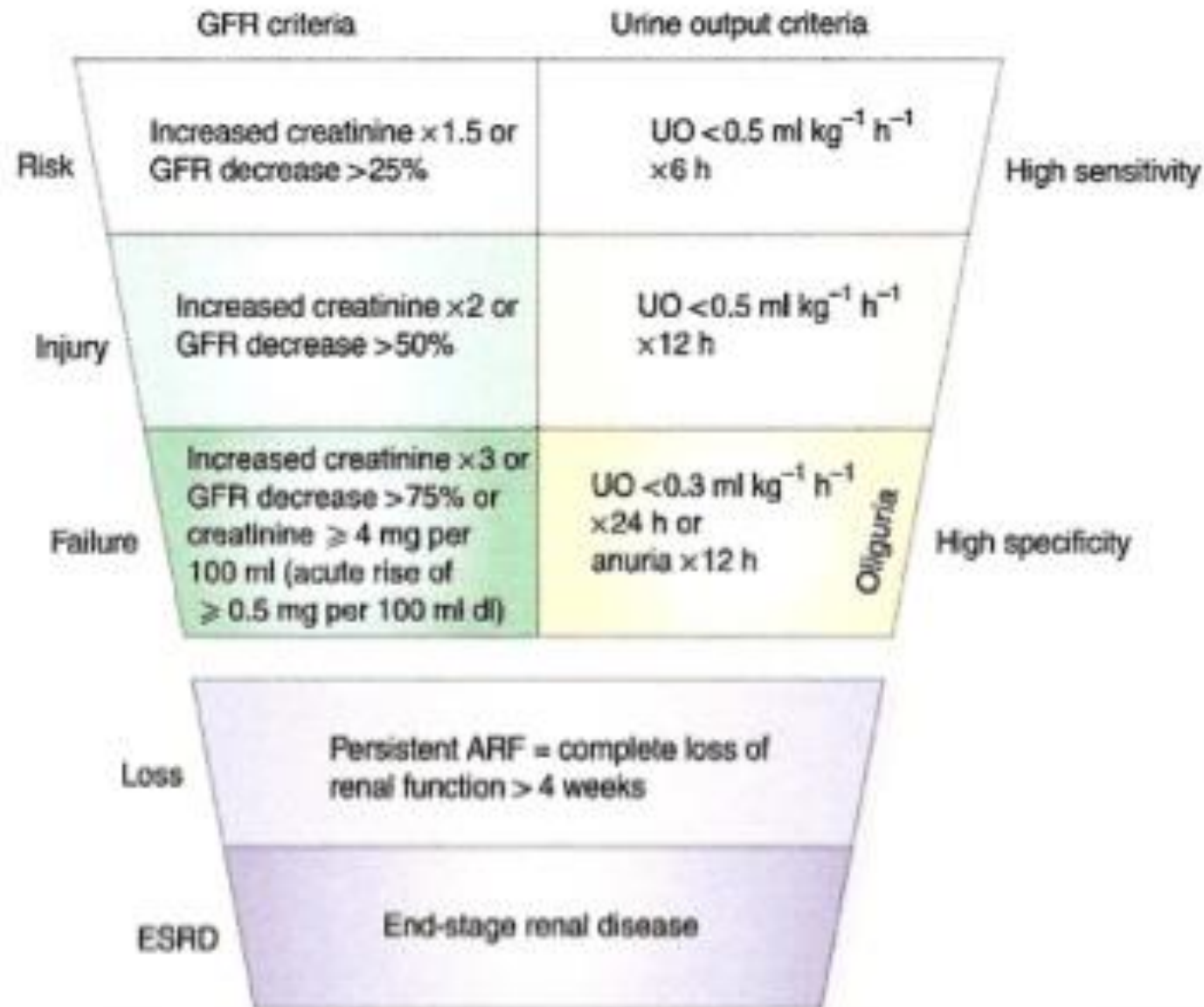
CLINICAL CASE

- 62 year-old male with PMH of HTN, DM, OSA and AS is admitted after a syncopal event. Patient undergoes echocardiography which reveals an AVA of 0.7. The patient undergoes AVR, and his intraoperative course was complicated by hypotension which requires vasoactive therapy. The following day, his serum creatinine increases from 1.0 (preop) to 1.5. His urine output has also decreased over the last 6 hours. What is the next step to confirm your diagnosis?

ACUTE KIDNEY INJURY

- RIFLE classification
 - Simple, readily available clinical tool to classify AKI in different populations
 - Good outcome predictor, with a progressive increase in mortality with worsening RIFLE class
 - Also suggests that even mild degrees of kidney dysfunction may have a negative impact on outcome

Classification of AKI according to RIFLE criteria

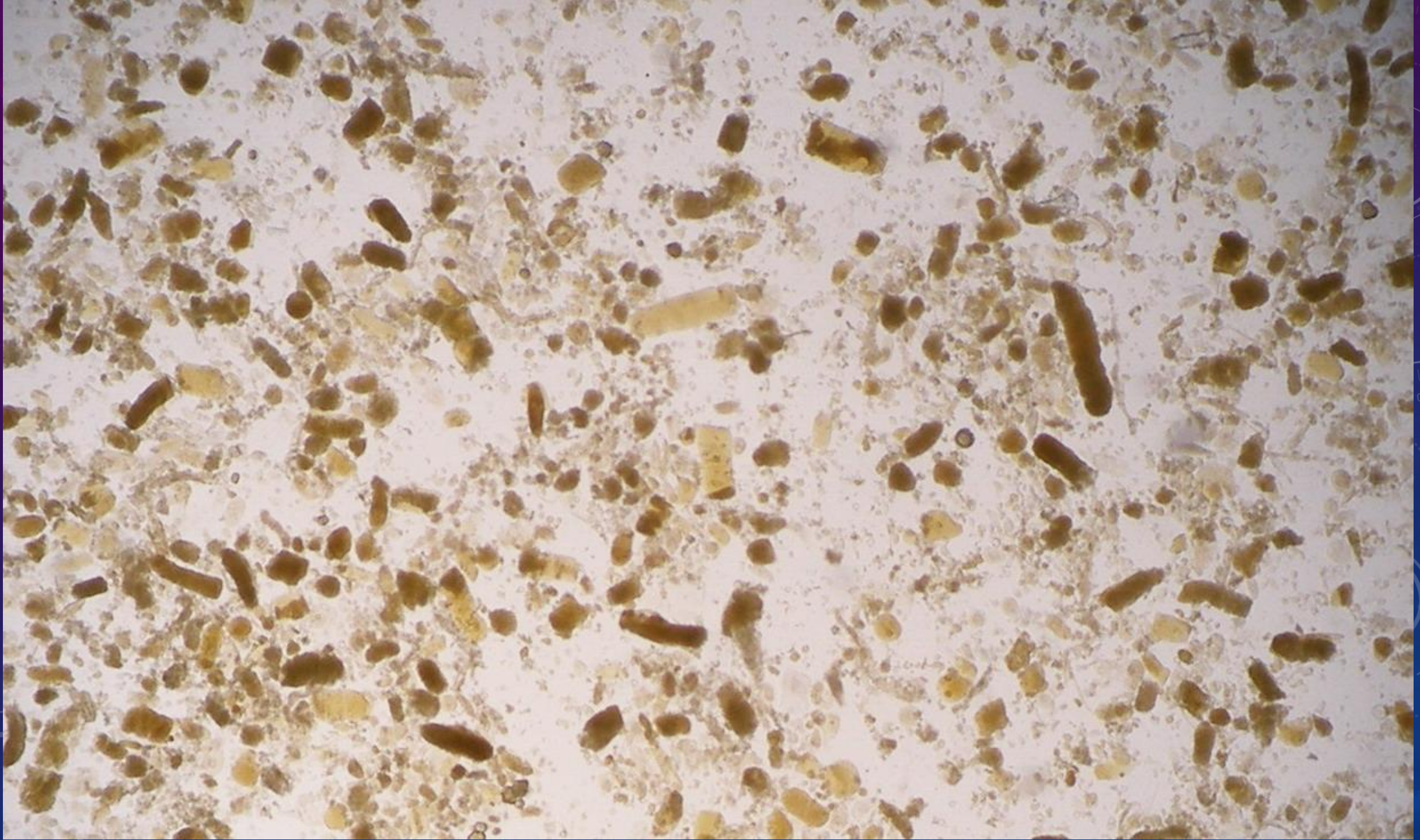


RIFLE defines three grades of severity of AKI on the basis of either:

- acute increase in serum creatinine
- decrease in GFR
- decreased urine output.

AKIN CRITERIA

Stage	Creatinine criteria	Urine output criteria
1	1.5 - 2 x baseline (or rise ≥ 26.4 $\mu\text{mol/L}$)	< 0.5 ml/kg/hour for > 6 hours
2	$>2 - 3$ x baseline	< 0.5 ml/kg/hour for > 12 hours
3	> 3 x baseline (or > 354 $\mu\text{mol/L}$ with acute rise ≥ 44 $\mu\text{mol/L}$)	< 0.3 ml/kg/hour for 24 hours or anuria for 12 hours



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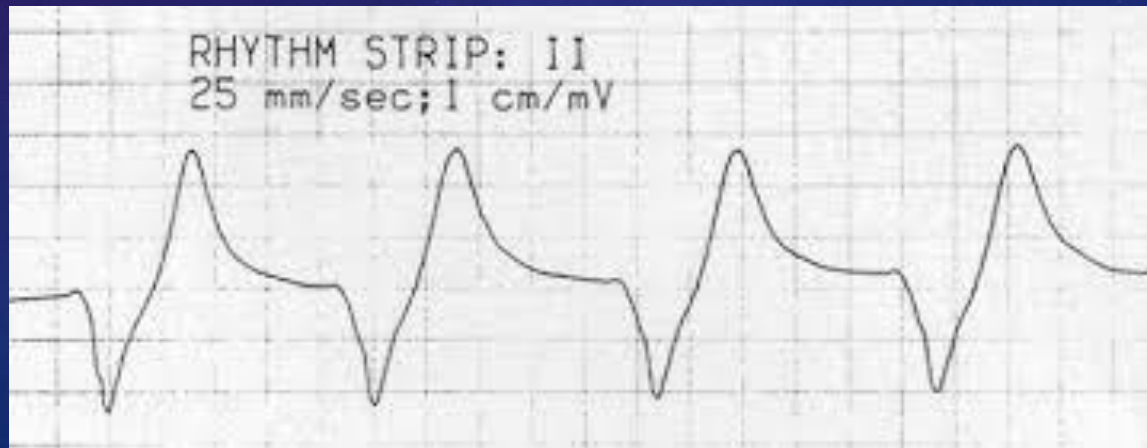
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- A. IVF challenge
- B. Diuretic trial
- C. Urine microscopy
- D. Renal US
- E. Echocardiogram

AKI MANAGEMENT

- Treat underlying disorder
- Avoid nephrotoxic insults
- Review dosing of renally cleared drugs
- Optimize hemodynamics (both MAP and CO)
- Monitor and treat volume overload or depletion, optimize electrolyte and acid-base status
- Assess for RRT indications: AEIOU
- No renal benefit to dopamine or mannitol

CLINICAL CASE

- 71 year-old female with PMH of CKD, HTN, DM and CAD presents to the ER with complaints of palpitations and chest pain. She admits to not adhering to a prescribed renal diet. Labs done in the ER are notable for a potassium of 8.4, bicarbonate of 18 and creatinine of 5.8. Rhythm strip is shown below. What is the next best step in management?



ELECTROLYTE DISTURBANCES

- Hyperkalemia

- Causes: transcellular shifts, decreased GFR, hypoaldosteronism, medications
- Symptoms: weakness, paresthesias, palpitations, myocardial irritability

- Hypokalemia

- Causes: transcellular shifts, GI or renal losses
- Symptoms: nausea, vomiting, weakness, muscle cramps

ELECTROLYTE DISTURBANCES

- Hypercalcemia
 - Symptoms: neuropsych disturbances, GI abnormalities, NDI, nephrolithiasis, AKI, CV disease, muscle weakness, bone pain
- Hypocalcemia
 - Symptoms: tetany, seizures, CV issues, papilledema, psych manifestations

ELECTROLYTE DISTURBANCES

- Hypophosphatemia

- Causes: increased insulin secretion, acute resp alkalosis, hungry bone, inadequate intake, medications, chronic diarrhea, vit D deficiency, HPTH, Fanconi, PD/CRRT, thiazide diuretics
- Symptoms: metabolic encephalopathy, paresthesias, seizures, coma, decreased myocardial contractility, diaphragmatic paralysis, proximal myopathy, dysphagia, ileus, rhabdomyolysis, hemolysis, heme dysfunction

ELECTROLYTE DISTURBANCES

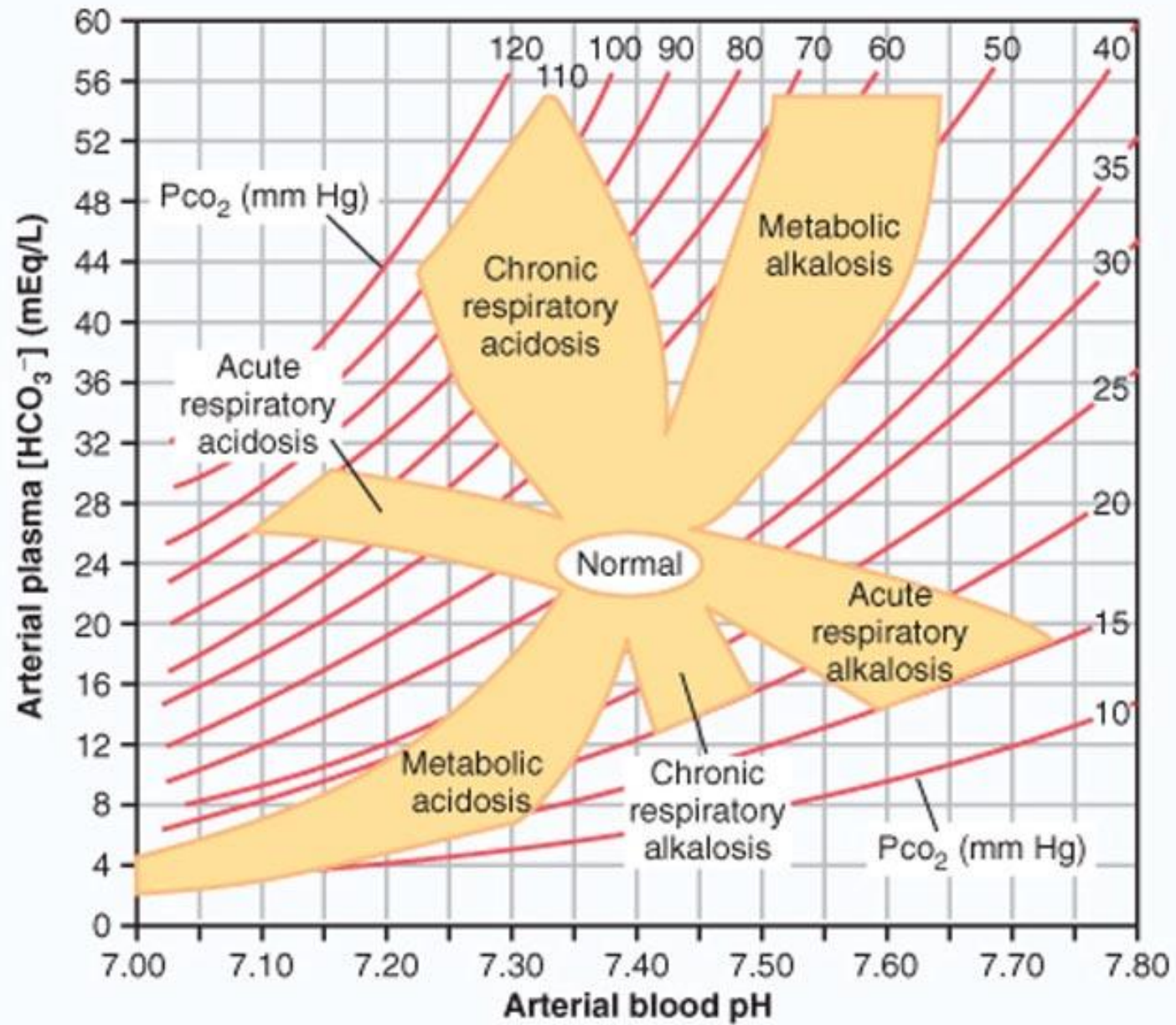
- Hypomagnesemia
 - Causes: PPIs, EtOH, DM, CNI, hypercalcemia, familial renal wasting syndromes, volume expansion, diuretics, aminoglycosides, amphotericin B, cisplatin, digoxin
 - Symptoms: NM excitability (similar to hypocalcemia), weakness, CV manifestations, hypokalemia

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- A. Ca gluconate administration
- B. Insulin/D50
- C. 1 ampule of sodium bicarbonate
- D. Albuterol NEB
- E. Hemodialysis

CLINICAL CASE

- 52 year-old male with PMH of HTN, CKD and CAD presents to the ER after having a VT arrest in the field. He is successfully resuscitated after 5 minutes of ACLS protocol, current intubated and sedated, on two vasoactive agents. Labs are notable for Na 130, K 5.1, Cl 100, HCO₃ 14, BUN 48, Cr 2.5, Glc 122. ABG is as follows: 7.22/38/13/100 on 60%FiO₂. What test would you order next to determine the etiology of this patient's acid-base disturbance?



ACID-BASE CONSIDERATIONS

- Metabolic Acidosis

- DDx: Lactic acidosis, ketoacidosis (starvation, DM, EtOH), ingestions, diarrhea, RTA, diuretics (CA inhibitor), ureteral diversion, CKD
- Rx: HCO_3^- (but only when indicated eg. $\text{pH} < 7.1-7.2$), fix underlying etio, RRT

- Metabolic Alkalosis

- DDx: Vomiting/NGT suction, diarrhea, mineralocorticoid excess, diuretics (loop or thiazide), posthypercapnea, hypercalcemia, severe hypokalemia, alkali therapy, contraction
- Rx: fix underlying etiology, rarely need to correct with acetazolamide

ACID-BASE CONSIDERATIONS

- Acute vs. Chronic
- Respiratory Acidosis
 - DDx: CNS depression, NM disorders, airway abnormalities, lung parenchyma abnormalities
 - Rx: treat underlying etiology
- Respiratory Alkalosis
 - DDx: hypoxia → hyperventilation, primary hyperventilation
 - Rx: oxygenate, fix underlying etiology

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- A. Methanol level
- B. Procalcitonin level
- C. Urinalysis
- D. Lactate
- E. BNP

CLINICAL CASE

- 80 year-old male with PMH of CHF (EF 15-20%), DM, CAD, HTN is admitted to the CVICU for cardiogenic shock following CABG surgery complicated by intraop cardiac arrest. Labs are relatively unremarkable, with the exception of a BNP of 74,000. PE shows 1-2+ edema, JVP is difficult to assess. Latest CVP is 16. Urine output has been 100-150cc/hr postop thus far on a lasix gtt at 10ml/hr. What is the next best step in management?

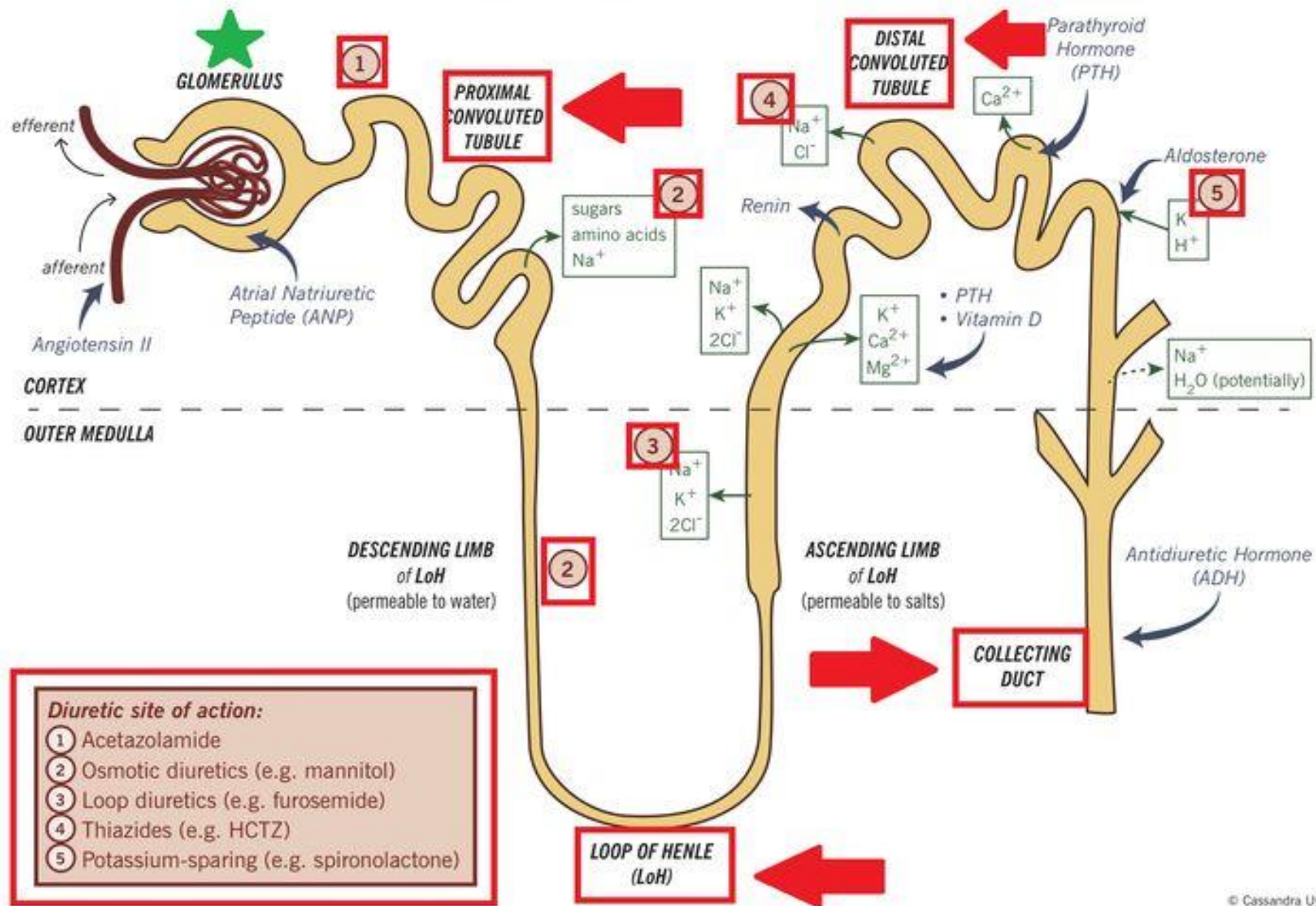
VOLUME ASSESSMENT

- Physical Exam
- CXR
- CVP
- PAC/SGC
- TTE
- SVV
- IVC diameter

VOLUME MANAGEMENT

- “To be (wet) or not to be (dry), that is the question”
- Surgical perspective
 - Offload an overworked, congested heart as quickly as possible
- Nephrologic perspective
 - Avoid rapid fluid shifts with consequent hypotension and/or intravascular volume depletion and subsequent AKI
- Use your data and clinical experience to arrive at a mutual endpoint (nephro, CCM and surgical teams)

Hormones Acting on the Nephron / Diuretics and Their Site of Action



INTERMITTENT VS. CONTINUOUS DIURETIC THERAPY

- In the setting of ADHF, continuous diuretic therapy resulted in
 - Greater net diuresis
 - Greater reductions in BNP from admission to discharge
 - Worsened renal filtration function
 - Use of additional treatment
 - Higher rates of rehospitalization or death at six months

AQUAPHERESIS

- As compared to maximal diuretic regimens
 - Higher rates of AKI
 - No significant differences in weight loss, mortality or rate of hospitalization at 60 days
 - Higher rates of AEs

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- A. Stop lasix gtt and change to alternative loop diuretic
- B. Increase lasix gtt to 20ml/hr and add metolazone 5mg PO daily
- C. Initiate aquapheresis therapy
- D. Start CRRT

CLINICAL CASE

- 42 year-old male with no PMH has a cardiac arrest while jogging, he is resuscitated after approximately 10 minutes and is taken straight to the cath lab upon arrival to the hospital. LHC shows no CAD and IABP is placed. Patient decompensates further and is on maximal vasoactive therapy and ventilator support, with inability to oxygenate adequately. The patient subsequently is started on ECMO therapy. His latest labs are notable for Na 130, K 5.6, HCO₃ 16, BUN 60, Cr 2.5 (on admission Cr was 1.3 approximately 24 hours ago). The patient has been anuric for the past 12 hours. What is the next best step for management of this patient's AKI?

CRRT

- Is an extracorporeal blood purification therapy intended to substitute for impaired renal function over an extended period of time and applied for or aimed at being applied for 24 hours a day.
- Indicated in any patient who meets criteria for hemodialysis therapy but cannot tolerate intermittent hemodialysis due to hemodynamic instability

GOALS OF CRRT

- Mimic the functions and physiology of the native organ
- Qualitative/quantitative blood purification
- Restoration/maintenance of homeostasis
- Avoid complications and achieve clinical tolerance
- Provide conditions favoring recovery of renal function

CRRT INDICATIONS

- Fluid overload
- AKI (either de novo or superimposed on progressive CKD)
- Life-threatening electrolyte/acid-base disturbances
 - Metabolic acidosis
 - Hyperkalemia
 - Hyper/hypocalcemia

PRINCIPLES OF CRRT

- Vascular access
- Semi-permeable membrane
- Transport mechanism
- Dialysate and replacement fluid

CRRT MODALITIES

- Slow Continuous Ultrafiltration (SCUF)
 - Ultrafiltration
- Continuous Veno-Venous Hemofiltration (CVVH)
 - Convection
- Continuous Veno-Venous Hemodialysis (CVVHD)
 - Diffusion
- Continuous Veno-Venous Hemodiafiltration (CVVHDF)
 - Diffusion and Convection

CRRT COMPLICATIONS

- Bleeding
- Hypothermia
- Vascular access issues
- Fluid volume deficit (intravascular volume depletion)
- Hypotension
- Inadequate electrolyte or bicarbonate replacement during CRRT
- Blood loss (clotting of filter or hemorrhage due to over-anticoagulation)
- Air embolism
- Arrhythmias or circulatory shock
- Hemolysis

CRRT PRESCRIPTIONS

- Modality
- Anticoagulation
- Blood Flow Rate
- Dose and Effluent Flow Rate
- Ultrafiltration Rate
- Solutions (Na/K/HCO₃/Ca/Glc)



CRRT AND ECMO

- Initiation of CRRT in ECMO patients is simply an indicator of severity of illness and fatality
- Mortality is higher in these patients as compared those receiving ECMO alone
- The combination of ECMO and CRRT in a variety of methods appears to be a safe and effective technique that improves fluid balance and electrolyte disturbances
- In the absence of primary renal disease, chronic renal failure did not occur after concurrent use of CRRT with ECMO

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- A. Administer lasix 80mg IV
- B. Give albumin 25g
- C. Repeat labs in 12 hours
- D. Initiate Dialysis
- E. Initiate CRRT

BOTTOM LINE

- AKI in the CT/VTICU is not rare
- Diagnose, identify underlying etiology and treat accordingly
- Pay close attention to electrolyte and acid-base status
- Volume assessment highly variable and management is extremely difficult
- CRRT may be necessary, but in general is overutilized in CT/VTICUs
- Use a multidisciplinary approach with nephrology, critical care and surgery involved

ANY QUESTIONS?

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