Acute Kidney Injury: Diagnosis and Management

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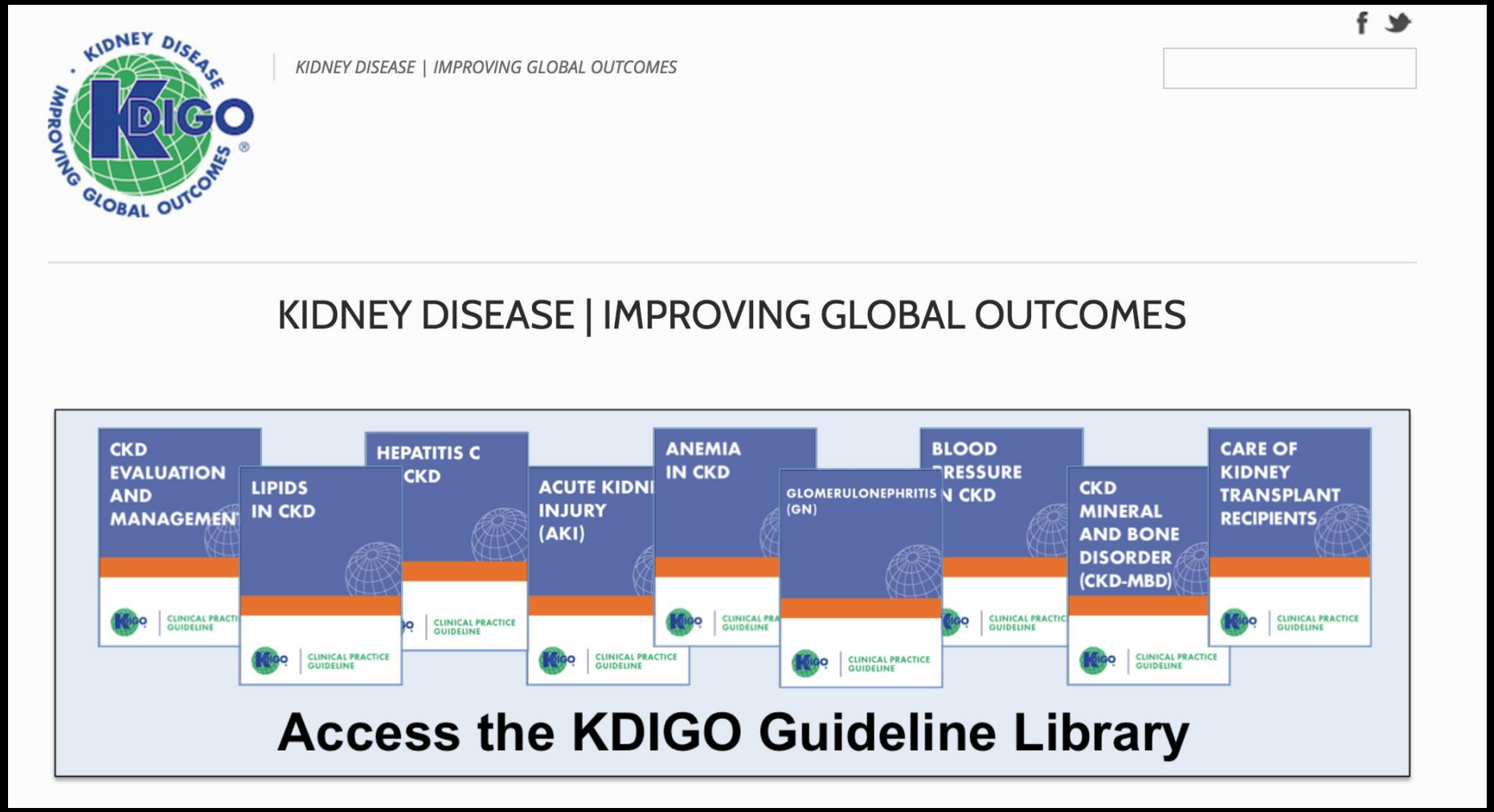
Disclosures

- Conflict of Interest: None

Objectives

- Explore KDIGO Guidelines as they relate to Acute Kidney Injury (AKI)
- Identify pharmacologic causes of AKI and medications used to treat the condition.
- Apply the KDGIO Guidelines for acute kidney injury to critical ill patients across multiple patient populations

What is KDIGO?



What is KDIGO?

- http://kdigo.org/home/
- Established in 2003 as an independently incorporated non-profit foundation governed by an international Executive Committee.
- KDIGO was managed by the National Kidney Foundation, a U.S. foundation experience in developing and implementing guidelines.
- Mission Statement: Improving the care and outcomes of kidney disease patients worldwide through the development and implementation of global clinical practice guidelines.

Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney inter., Suppl. 2012; 2: 1–138.

Purpose of Practice Guideline for AKI

- AKI is common. (1% - 25% of ICU patients)
- AKI imposes a heavy burden of illness (mortality rates from 15–60%)
- The cost per person of managing AKI is high.
- AKI is amenable to early detection and potential prevention.
- There is considerable variability in practice to prevent, diagnose, treat, and achieve outcomes of AKI.
- Clinical practice guidelines in the field have the potential to reduce variations, improve outcomes, and reduce costs.

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KDIGO Definition

- An abrupt decrease in kidney function that includes, but is not limited to, ARF.
- Broad clinical syndrome encompassing various etiologies, including specific kidney diseases (e.g., acute interstitial nephritis, acute glomerular and vasculitic renal diseases); non-specific conditions (e.g, ischemia, toxic injury); as well as extrarenal pathology (e.g., prerenal azotemia, and acute postrenal obstructive nephropathy)

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RIFLE vs AKIN

	Stage	Creatinine Criteria	Urine Output Criteria
RIFLE		GFR decrease ≥25% or sCr increase by 1.5x GFR decrease ≥50% or sCr increase by 2x GFR decrease ≥75% or sCr increase by 3x or >4 mg/dL Persistent failure >4 weeks Persistent failure >3 months	<0.5 mL/kg/h for 8 h <0.5 mL/kg/h for 16 h <0.3 mL/kg/h for 24 h (anuria 12 h)
AKIN	1 2 3	Increase >0.3 mg/dL or to 150%–200% baseline Increase to 200%–300% baseline Increase to >300% baseline or >4 mg/dL with an acute increase of 0.5 mg/dL	<0.5 mL/kg/h for 6 h <0.5 mL/kg/h for 12 h <0.3 mL/kg/h 24 h (anuria 12 h)
KDIGO	1 2 3	Increase >1.5–1.9x baseline (or >0.3 mg/dL increase) Increase >2–2.9x baseline >3x baseline Initiation of CRRT Decrease in eGFR to <35 mL/min/1.73 m^2	<0.5 mL/kg/h for 6–12 h <0.5 mL/kg/h for >12 h <0.3 mL/kg/h >24 h (anuria 12 h)

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Causes of AKI

- **Exposures:**
- Sepsis
- Critical Illness
- Circulatory Shock
- Burns
- Trauma
- Cardiac Surgery (bypass)
- Major non-cardiac Surgery
- Nephrotoxic Drugs
- Radiocontrast Agents
- Poisonous Plants and animals

- **Susceptibilities:**
- Dehydration
- Advanced age
- Female gender
- Black Race
- CKD
- Chronic disease (lung, heart, liver)
- Diabetes
- Cancer
- Anemia

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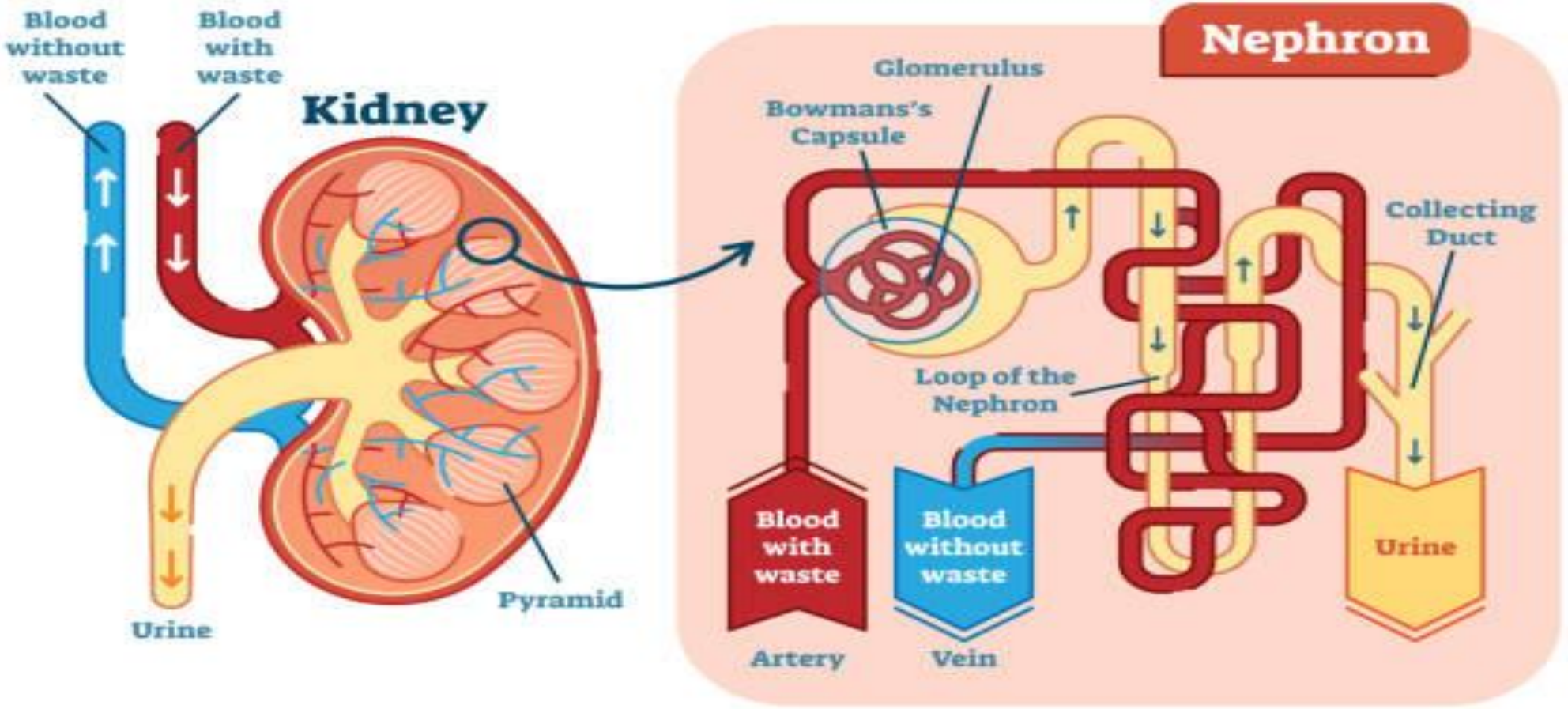
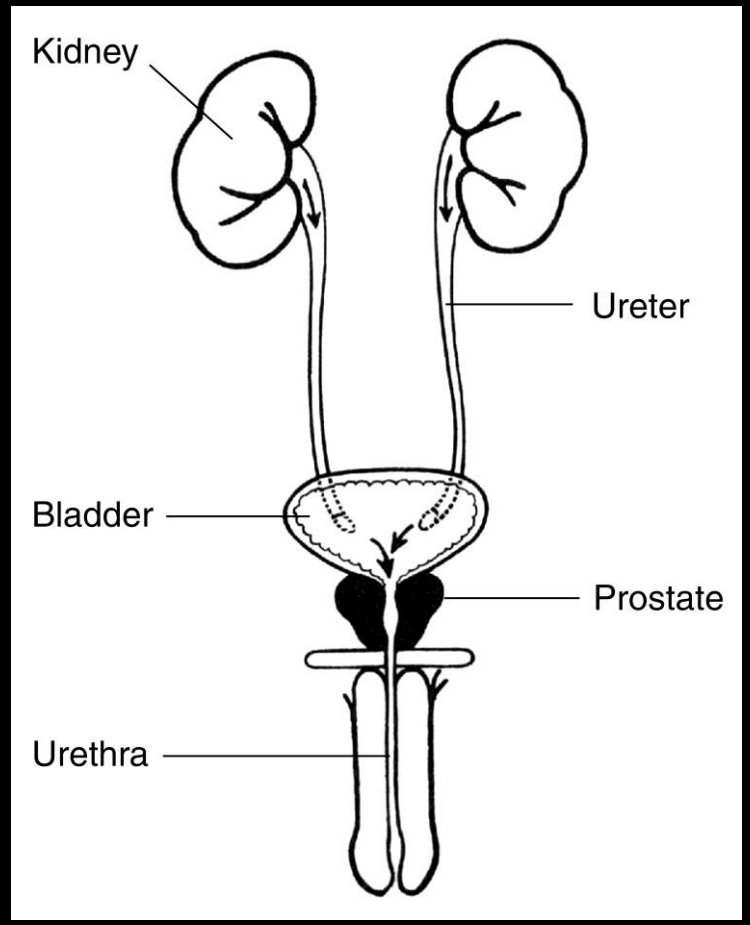
Nephron Anatomy



Causes of Acute Kidney Injury



PreRenal

- Acute drop in blood pressure and/or interruption in blood flow to the kidney from injury or illness

IntraRenal

- Direct damage to the kidney by inflammation, toxins, drugs, infection or reduced blood supply

PostRenal

- Obstruction of urine flow due to prostate, kidney stones, tumor, or injury

Diagnosis of AKI

RIFLE vs AKIN

	Stage	Creatinine Criteria	Urine Output Criteria
RIFLE		GFR decrease ≥25% or sCr increase by 1.5x GFR decrease ≥50% or sCr increase by 2x GFR decrease ≥75% or sCr increase by 3x or >4 mg/dL Persistent failure >4 weeks Persistent failure >3 months	<0.5 mL/kg/h for 8 h <0.5 mL/kg/h for 16 h <0.3 mL/kg/h for 24 h (anuria 12 h)
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Diagnosis: Case A

- Baseline: 1.0
- Day 1: 1.3
- Day 2: 1.5
- Day 3: 2.0
- Day 7: 1.0

- Criterion 1: 50% Baseline: Yes

- Criterion 2: Greater than or equal to 0.3 rise in less than or equal to 48 hours: Yes

- Stage: 2 (More than 2x baseline)

Diagnosis: Case B

- Baseline: 0.4
- Day 1: 0.5
- Day 2: 0.6
- Day 3: 0.7
- Day 7: 0.4

- Criterion 1: 50% Baseline: Yes

- Criterion 2: Greater than or equal to 0.3 rise in less than or equal to 48 hours: No

- Stage: 1

Diagnosis: Case C

- Baseline: 1.0
- Day 1: 1.1
- Day 2: 1.2
- Day 3: 1.4
- Day 7: 1.0

- Criterion 1: 50% Baseline: No

- Criterion 2: Greater than or equal to 0.3 rise in less than or equal to 48 hours: YES

- Stage: 1

Diagnosis: Case D

- Baseline: Unknown
- Day 1: 4.1
- Day 2: 2.6
- Day 3: 2.2
- Day 7: 1.0

- Criterion 1: 50% Baseline: ?/Yes

- Criterion 2: Greater than or equal to 0.3 rise in less than or equal to 48 hours: No

- Stage: 3 (More than 3x baseline or Creat >4mg/dL

Diagnosis Based on Urine Output

- Clinical Judgement
- UOP Range: 0.3 mL/hour/kg-0.5mL/kg/h

Urine Output Criteria

< 0.5 ml/kg/hr x 6-12 hours
<0.5 ml/kg/hr for >12 hours
<0.3 ml/kg/hr for >24 hours, OR Anuria > 12 hours

Diagnosis

Cause of AKI	Recommended Diagnostic Test
Decreased kidney perfusion	I/Os, volume evaluation, urinary diagnostics: urine sodium, urine lytes, urine osmol
Acute glomerulonephritis, vasculitis, TMA	Assess Urine for sediment, serological testing and hematological testing
Urinary Tract Obstruction	Renal Ultrasound

Diagnosis Summary

- The cause of AKI should be determined whenever possible.
- Stratify patients for risk of AKI according to their susceptibilities and exposures. Reduce exposures when possible
- Evaluate those at risk for AKI with measurements of SCr and urine output. Individualize the frequency
- Evaluate patients 3 months after AKI for resolution, new onset, or worsening of pre-existing CKD.

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Management of AKI

Supportive Management

- Target plasma glucose 110–149 mg/dl. Use insulin as necessary.
- Avoid restriction of protein intake
- Provide enteral nutrition
- Prevention
  - Off pump CABG vs On pump CABG

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Management of AKI

- Use isotonic crystalloids rather than colloids (albumin or starches) as initial management for expansion of intravascular volume in patients at risk for AKI or with AKI.
- Use protocol-based management of hemodynamic and oxygenation parameters to prevent development or worsening of AKI in high-risk patients.
  - perioperative setting
  - septic shock
- Use vasopressors in conjunction with fluids in patients with shock.

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Variations in Colloids and Crystalloids Formulations

Solution	pH	Na+	Cl-	K+	Ca++	Lactate	Glucose	Osmolality		Other
0.9% normal saline	5.0	154	154	0	0	0	0	308		0
Hartmann/CSL	5-7	131	112	5	2	28	0	255		0
Plasma lyte	7.4	140	98	5	0	0	0	294	27mmol Acetate	23mmol Gluconate
5% dextrose in water (D5W)		4.0	0	0	0	0	0	50 g/L	252	0
.45% normal saline with dextrose (D51/2 NS)	4.5	77	77	0	0	0	50 g/L	406	0	
Albumin (4%)	6.7-7.3	140	128	0	0	0	0	260	40 g/L albumin	
Albumin (20%)	6.4-7.3	48-100	130-160	0	0	0	0	130	200 g/L albumin	
Hetastarch 6%	5.5	154	154	0	0	0	0	310	60 g/L starch	
Pentastarch 10%	5.0	154	154	0	0	0	0	326	100 g/L starch	
Dextran-40 (10% solution)	3.5-7.0	154	154	0	0	0	0	311	100 g/L dextran	
Dextran-70 (6% solution)	3.0-7.0	154	154	0	0	0	0	310	60 g/L dextran	
Haemaccel 3.5%	7.4	145	145	5	6.25	0	0	293	35 g/L gelatin	
Gelofusine	7.4	154	125	0	0	0	0	308	40 g/L gelatin	

SMART-MED/SMART-SURG (NEJM 2018)

- Normal saline has been associated with hyperchloremic metabolic acidosis and renal injury
- Small difference in rates of major adverse kidney events, which occurred in 15.4% patients of the normal saline group and 14.3% in the balanced crystalloids group (P=0.04)


The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

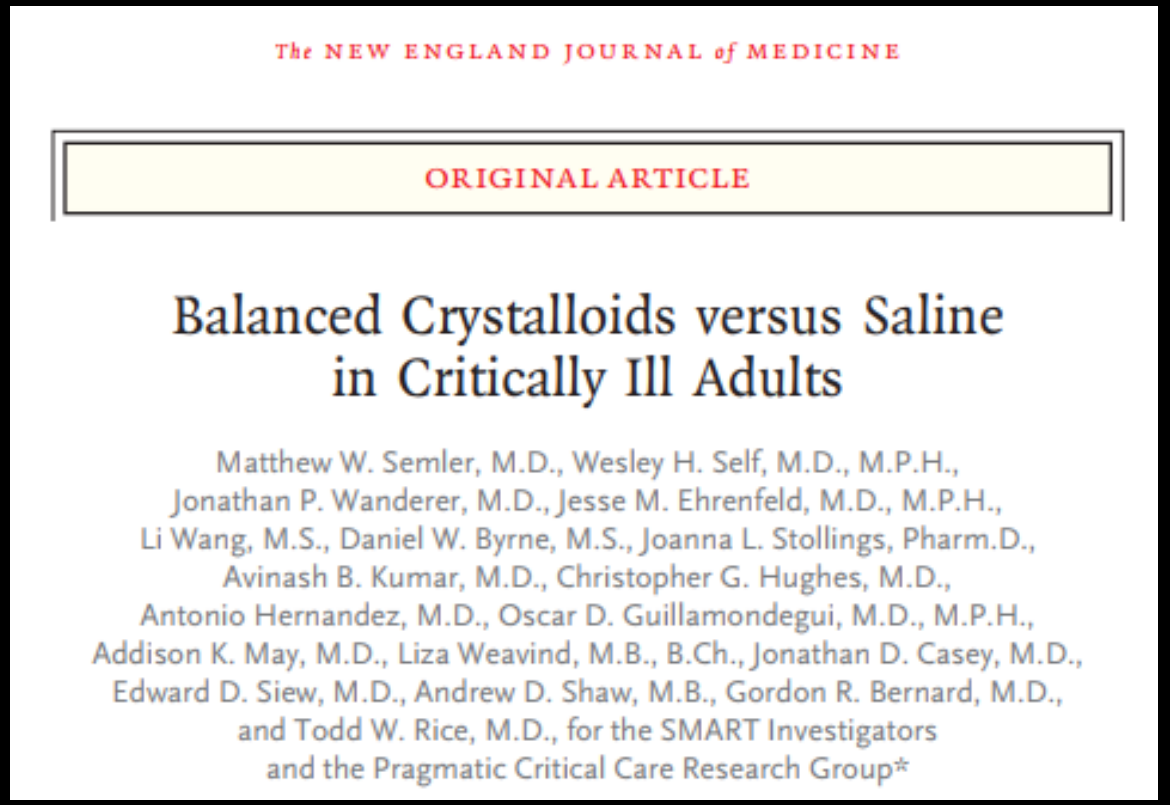
Balanced Crystalloids versus Saline in Critically Ill Adults

Matthew W. Semler, M.D., Wesley H. Self, M.D., M.P.H., Jonathan P. Wanderer, M.D., Jesse M. Ehrenfeld, M.D., M.P.H., Li Wang, M.S., Daniel W. Byrne, M.S., Joanna L. Stollings, Pharm.D., Avinash B. Kumar, M.D., Christopher G. Hughes, M.D., Antonio Hernandez, M.D., Oscar D. Guillamondegui, M.D., M.P.H., Addison K. May, M.D., Liza Weavind, M.B., B.Ch., Jonathan D. Casey, M.D., Edward D. Siew, M.D., Andrew D. Shaw, M.B., Gordon R. Bernard, M.D., and Todd W. Rice, M.D., for the SMART Investigators and the Pragmatic Critical Care Research Group*

Semler MW, et al. "Balanced Crystalloids versus Saline in Critically Ill Adults". The New England Journal of Medicine. 2018. 378(9):829-839.

SMART-MED/SMART-SURG (NEJM 2018)

- Benefit of balanced crystalloids were primarily seen in medical ICU patients, those with sepsis, those without traumatic brain injury, and those with prior renal-replacement therapy
- Incidence of new CRRT was 2.5% in balanced group and 2.9% in saline group (p=0.08)
- No statistically or clinically meaningful differences in secondary outcomes which included in-hospital death; ICU-, ventilator-, or vasopressor-free days



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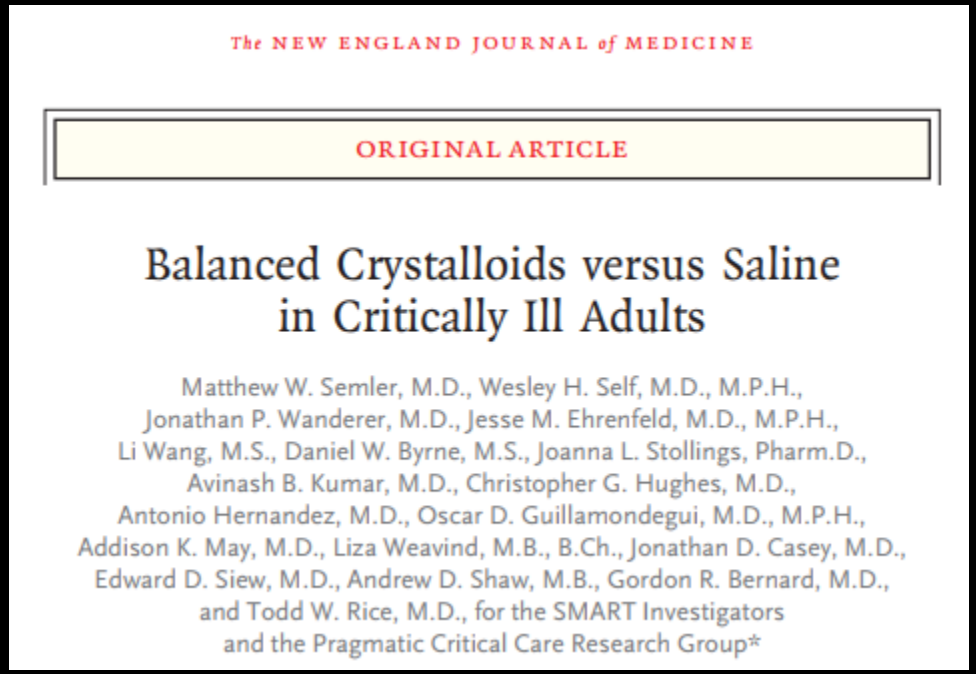
SMART-MED/SMART-SURG (NEJM 2018)

- Among medical and surgical ICU patients, balanced crystalloids such as LR or Plasma-Lyte reduce the rate of death, need for renal-replacement therapy, or persistent renal dysfunction, when compared to normal saline.
- Clinically meaningful?



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Albumin vs Starch Solutions

- Saline vs. Albumin Fluid Evaluation (SAFE) study
  - Albumin is safe, but no more effective than saline
  - No difference in renal outcomes
- A systematic review of RCTs on the use of starch solutions found an almost two-fold increased risk of AKI compared with crystalloids
- Meta-analysis Critical Care (2010)
  - Albumin decreased the odds of AKI by 76%
  - Starch solutions increased those odds by 92%
  - Mortality rates increased with use starch solutions
  - Albumin displays renoprotection whereas starch solutions display nephro-toxicity.

Finfer S, Bellomo R, Boyce N, et al. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. N Engl J Med 2004; 350: 2247–2256; Perel P, Roberts I, Pearson M. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev 2007; 4: CD000567. Wiedermann CJ, Dunzendorfer S, Gaioni LU, et al. Hyperoncotic colloids
and acute kidney injury: a meta-analysis of randomized trials. Crit Care
2010; 14: R191.

Diuretics in AKI

- Diuretics are frequently used in patients at risk of AKI
  - Fluid overload
- Approximately 59–70% of patients with AKI were given diuretics at the time of nephrology consultation or before the start of RRT
- Providers often prescribe diuretics to convert oliguric to nonoliguric AKI

- Do not use diuretics to prevent AKI.
- Do not use diuretics to treat AKI, except in the management of volume overload.—heart failure

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Mehta RL, Pascual MT, Soroko S, et al. Diuretics, mortality, and nonrecovery of renal function in acute renal failure. JAMA 2002; 288: 2547–2553.;

Diuretics in AKI

- Decrease oxygen consumption in the loop of Henle by inhibiting sodium transport
  - Decreasing ischemic injury.
- Inhibit sodium transport which reduces renal tubular oxygen consumption
  - Decreases ischemic injury
- Wash out necrotic debris blocking tubules
- Inhibit certain prostaglandins which reduces renovascular resistance and increases renal blood flow.

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Diuretics in AKI

- Furosemide has no significant effect on in-hospital mortality,
- Furosemide has no significant effect on risk for requiring RRT

- ”The House of God”

Vasodilators in AKI

- Do not use low-dose dopamine to prevent or treat AKI.
  - Does not reduce the chances of needing Renal replacement in therapy
  - Dopamine does increase UOP. But has no overall effect.
  - Does not have an impact on mortality

- Do not use fenoldopam to prevent or treat AKI.

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Antimicrobial Selection

- Do not use aminoglycosides for the treatment of infections unless no suitable, less nephrotoxic, therapeutic alternatives are available.
- Administer aminoglycosides as a single dose daily rather than multiple-dose daily treatment regimens.
- Monitor aminoglycoside drug levels when treatment with multiple daily dosing is used for more than 24 hours.
- Monitoring aminoglycoside drug levels when treatment with single-daily dosing is used for more than 48 hours.
- Use topical or local applications of aminoglycosides rather than IV, when feasible and suitable.
- Use lipid formulations of amphotericin B rather than conventional formulations of amphotericin B.
- Use azole antifungal agents rather than amphotericin B

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Aminoglycosides

- Gram-negative bacterial pathogens and Gram- positive bacterial pathogens.
- The nephrotoxicity of aminoglycosides has been very well studied
- Ex: Amikacin, Gentamycin, Tobramycin, Neomycin

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Aminoglycosides

- Nephrotoxicity is related to uptake of aminoglycosides through megalin receptor which is expressed on epithelial cells along the proximal convoluted tubule
- Aminoglycosides are concentrated in the proximal convoluted tubules
- Can cause direct glomerular injury and tubular impairment
- Single-dose daily regimens vs. multiple-daily dosing strategies have been evaluated and even meta-analyses indicate that once-daily dosing strategies generally tend to result in less AKI when compared to multiple-dose dosing strategies

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Aminoglycosides

- Aminoglycoside levels with single-daily dosing strategies is not standardized and remains somewhat controversial.
- Some providers do not measure therapeutic drug levels at all in patients receiving this dosing strategy.
- Others recommend at least a single peak measurement to ensure that the blood levels are adequate to cover the MIC of the infecting organism.
- Generally measure at 12, 18, or 24 hour intervals after the aminoglycoside dose
- Random level is also acceptable

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Amphotericin B

- Nephrotoxicity is related to multiple mechanisms, including ischemic injury and direct tubular and glomerular-cell membrane toxicity.
- Causes vasoconstriction of the afferent renal arteriole along with a systemic inflammatory response that may reduce renal blood flow.
- Directly inserts into human cellular membranes, where it disrupts membrane permeability and physiology
- Causes a loss of renal tubular concentrating ability, renal tubular acidosis, increasing urinary losses of potassium and magnesium, and decreased glomerular function, resulting in azotemia and decreased synthesis of erythropoietin.
- Can be accompanied by concomitant administration of other potentially nephrotoxic agents such as aminoglycosides, chemotherapeutic agents, and other potentially nephrotoxic agents (vancomycin)
- Lipid formulations of amphotericin are less nephrotoxic but require different dosing strategies (three- to five-fold higher doses than other formulations of amphotericin B)

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Contrast Induced AKI (CI-AKI)

- Assess the risk for CI-AKI and screen for pre-existing impairment of kidney function in all patients who will receive IV contrast
  - Remember to evaluate for ALL possible causes of AKI.
- Consider alternative imaging methods in patients at increased risk for CI-AKI.
- Use the lowest possible dose of IV contrast Administer isotonic sodium chloride or sodium bicarbonate solutions in patients at increased risk for CI-AKI.
  - Oral fluids are not sufficient
- Administer oral N-acetylcysteine (NAC) in addition to intravenous isotonic crystalloids, in patients at increased risk of CI-AKI.
- DO NOT provide intermittent hemodialysis for contrast-media removal in patients at increased risk for CI-AKI.

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Contrast Questionnaire

Do you have or have you been treated for the following:

- ☐ Kidney disease
- ☐ Kidney surgery
- ☐ Diabetes
- ☐ Kidney transplant or Single Kidney
- ☐ Cancer (type): _____
- ☐ Asthma – Meds _____
- ☐ Asthma attack in the last 3 months
- ☐ Change in asthma meds past 2 weeks
- ☐ Allergy to Iodine or contrast material

List all Allergies: _____

Please list all current medications: _____

_____

Yes	No	
☐	☐	Have you had a radiologic study / x-ray relating to this study? When / where: _____
☐	☐	Have you ever had an injection of IV contrast? If yes, any reaction to injection? _____
☐	☐	Have you ever had any major surgery? What / when: _____
		_____
☐	☐	Are you currently taking any medication containing Metformin?
☐	☐	Do you have or have you been treated for the following: Liver Dysfunction, history of alcohol abuse, Cardiac failure, Myocardial disease, Peripheral Muscle Ischemia or Peripheral Vascular disease (PVD)?
☐	☐	Are you currently breast feeding?
☐	☐	Is there any possibility that you are pregnant?
☐	☐	Are you or have you ever been a smoker? If you have quit, when? _____
☐	☐	Do you have a cardiac pacemaker or defibrillator?

AKI Risk Scoring

Risk Factors	Score
Hypotension	5
IABP	5
CHF	5
Age >75 y	4
Anemia	3
Diabetes	3
Contrast media volume	1 for each 100mL
eGFR <20 mL/min/1.73 m^2	6
eGFR 20–40 mL/min/1.73 m^2	4
eGFR 40–60 mL/min/1.73 m^2	2

Class of Risk	Risk Score
Low	≤5
Medium	6 to 10
High	11 to 16
Very high	≥16

Sodium Bicarbonate vs. Normal Saline

- No clear evidence for the optimal rate and duration of fluid infusion in CI-AKI prevention
- Most studies suggest that the fluids should be started at least 1h before and continued for 3–6 hours after contrast media administration.
- Mechanism by which sodium bicarbonate, beyond its volume-expanding effects, might further reduce CI-AKI remains poorly defined
  - Decrease generation of free radicals
  - Lowers intratubular viscosity caused by the contrast medium, compared to isotonic saline, because it causes less tubular sodium reabsorption than saline.

Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO Clinical Practice Guideline for Acute Kidney Injury. Kidney inter., Suppl. 2012; 2: 1–138.

Sodium Bicarbonate vs. Normal Saline

- In a major comprehensive meta-analysis, which included data from all available published and unpublished studies involving 3563 patients total, no clear evidence of overall benefit associated with the use of sodium bicarbonate to prevent CIN

- Smaller studies showed superiority for bicarbonate. However, there was not an overall benefit. Given the risk of adding this medication and the potential for error, either medication.

N-acetylcysteine

- Use oral n-acetylcysteine with isotonic crystalloids for those patients at increased risk of CI-AKI
- No FDA label is available for NAC as a preventive drug of CI-AKI.
- Dose: N-acetylcysteine 600-1200mg PO every 12 hours. 2 doses pre-contrast; 2 doses post contrast.
- Mechanism: (1) Minimizes vasoconstriction (2) reduces oxidative stress (antioxidant)

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N-acetylcysteine vs Others

- NAC with IV sodium bicarbonate reduced CI-AKI by 35% compared to other combinations
- However, the combination of NAC plus sodium bicarbonate did not significantly reduce renal failure requiring dialysis.
- CONCLUSION: combination prophylaxis with NAC and sodium bicarbonate substantially reduced the occurrence of CI-AKI overall, but not dialysis-dependent renal failure.

Contrast Induced AKI (CI-AKI)

- Those who develop CI-AKI have a greater risk for death or prolonged hospitalization, as well as for other adverse outcomes, including early or late cardiovascular events and increased mortality (1)
- When patients with CI-AKI require dialysis, the mortality is higher compared to those not requiring dialysis. (2)
- By 2 years, the mortality rate in patients who required dialysis was 81.2%.(3)
- Data on the association between risk of ESRD and CI-AKI are scarce. Only one study reported the incidence of new CKD Stage 4–5 (eGFRo30 ml/min) following percutaneous coronary interventions and found that this occurred in 0.3% of patients (4)

(1) McCullough PA. Radiocontrast-induced acute kidney injury. NephronPhysiol 2008; 109: pp 61–72.; (2) Weisbord SD, Chen H, Stone RA, et al. Associations of increases in serum creatinine with mortality and length of hospital stay after coronary angiography. J Am Soc Nephrol 2006; 17: 2871–2877. (3) McCullough PA, Wolyn R, Rocher LL, et al. Acute renal failure after coronary intervention: incidence, risk factors, and relationship to mortality. Am J Med 1997; 103: 368–375.(4) Vuurmans T, Byrne J, Fretz E, et al. Chronic kidney injury in patients after cardiac catheterization or percutaneous coronary intervention: a comparison of radial and femoral approaches (from the British Columbia Cardiac and Renal Registries). Heart 2010; 96: 1538–1542.

Renal Replacement Therapy (RRT)

- Do not use prophylactic intermittent hemodialysis (IHD) or hemofiltration (HF) for contrast-media removal in patients at increased risk for CI-AKI.
- Initiate RRT emergently when life-threatening changes in fluid, electrolyte, and acid-base balance exist.
- Trend lab tests rather than examining single BUN and creatinine thresholds when making the decision to start RRT.
- Discontinue RRT when it is no longer required
  - Kidney function has recovered
  - Because RRT is no longer consistent with the goals of care
- Do NOT use diuretics to enhance kidney function recovery, or to reduce the duration or frequency of RRT
- Use continuous and intermittent RRT as complementary therapies in AKI patients
- Use CRRT, rather than standard intermittent RRT, for hemodynamically unstable patients
  - acute brain injury or increased intracranial pressure or brain edema

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Indications for RRT

Acid-base problems

Electrolyte problems

Intoxications

Overload, fluid

Uremic symptoms

RRT Modality	Advantages	Disadvantages
Peritoneal Dialysis	• Minimal equipment needs • Minimal training needs • Feasible in small infants • Minimum hemodynamic compromise • No systemic anticoagulation • More physiological due to its continuous nature • No need for vascular access	• Slow and less efficient solute removal • Unreliable and variable ultrafiltration • Respiratory compromise – abdominal compartment syndrome • Peritonitis • Catheter obstruction, leakage
Hemodialysis	• Maximum solute clearance • Readily available • Short treatment time • Accurate ultrafiltrate	• Hemodynamic instability • Rapid fluid and solute compartment shifts • Difficult to use in small infants • Complex equipment • Requires vascular access • Heparin anticoagulation
CRRT	• Continuous control of fluid status • Accurate ultrafiltrate • Well tolerated by hemodynamically unstable patients • Excellent solute clearance • More physiological • Able to pump in huge amounts of fluids in oligo-anuric patients • Smaller circuit volumes	• Systemic anticoagulation (though regional anticoagulation may be used) • Frequent filter clogging • Hypotension in small infants • Cost • Requires expertise • Vascular access necessary

AKI, AKD, CKD

- What is acute?
  - A disease process that results in a change in SCr over many weeks is not AKI
- AKI is defined in terms of a process that results in a 50% increase in SCr within 1 week or a 0.3 mg/dl increase within 48 hours
  - When does the 1-week or 48-hour time periods occur?
    - —Hospital admission? ICU admission?
- The timeframe for AKI is somewhat arbitrary.

- Consider specialty consultation with nephrology
- Trend out for up to 3 months

Stage Based Management of DKA

High Risk:

- Discontinue nephrotoxic Agents
- Ensure volume status and Perfusion Pressure
- Consider functional hemodynamic monitoring
- Monitor serum creatinine and urine output
- Avoid hyperglycemia
- Consider alternatives to Contrast procedures

AKI Stage 1:

- Non-Invasive diagnostic workup
- Consider invasive diagnostic workup

AKI Stage 2:

- Check for changes in drug dosing
- Consider RRT
- Consider ICU admission

AKI Stage 3:

- Avoid subclavian catheters if possible

Future Direction

- Early detection with the use of biomarkers
- Prognosis where a biomarker is used to predict risk for AKI or risk for progression of AKI.
- Prognosis where a biomarker is used to predict recovery after AKI vs. death or need for long-term RRT.
- The influence of urinary output criteria on AKI staging.
- eGFR criteria on AKI staging

Case Study 1

- 34yo Male hospitalized with N/V/D abdominal pain X3 days
- PMHx: ETOH Abuse
- Na: 152, K: 2.8, Cl: 124, Bicarbonate: 14, Creat: 3.7, Glucose: 756
- UA: hyaline casts, glucosaria, ketonuria.
- BP: 80/40, HR 114, RR: 28
- CT Abdomen in ER reveals enteritis

- Diagnostics? Diagnosis?
- Causes?
- Management?
- Prevention?

Case Study 2

- 48 yo female admitted for respiratory failure.
- PMHx: bone marrow transplant Feb 2016 for leukemia
- Admitted with BUN: 20 Creat: 0.7
- Chest xray: small Left pleural effusion and right middle lobe infiltrates
- Antibiotics: piperacillin/tazobactam, gentamicin, vancomycin, acyclovir
- 3 days later Fio2 requirements double.
- CT Chest reveals Left Pulmonary embolus; Creat increases 1.2
- 5 days later Creat: 5.0

- Diagnostics? Diagnosis?
- Causes?
- Management?
- Prevention?
- Prognosis?

Case Study 3

- 56 yo cachectic male admitted with productive cough fever 3 weeks
- Bilateral UPPER lobe infiltrates
- PMHx: None. (+) smoker
- Cultures: (+) mycobacterium tuberculosis
- Creatinine: 1.0
- Started on: Clarithromycin, Rifampin, and ethambutol.
- 2 weeks later: Creatinine increases 1.0 to 1.5. Increases 0.2 daily.

- Diagnosis?
- Causes?
- Management?
- Prevention?

Case Study 4

- 45yo female admitted for acute respiratory distress.
- 2 nieces with chicken pox and no other sick contacts.
- Creatinine: 0.5
- Xray: bilateral diffuse infilltrates
- Dx: Varicella pneumonia. Tx: Acyclovir IV
- Day 4: BUN increases from 22 to 50; Creat: 1.0 to 1.6
- UA: (+) Crystals

- Diagnosis?
- Causes?
- Management?
- Prevention?

Questions?

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