



ACUTE KIDNEY INJURY

R. Cameron Herman – April 12 / 2019



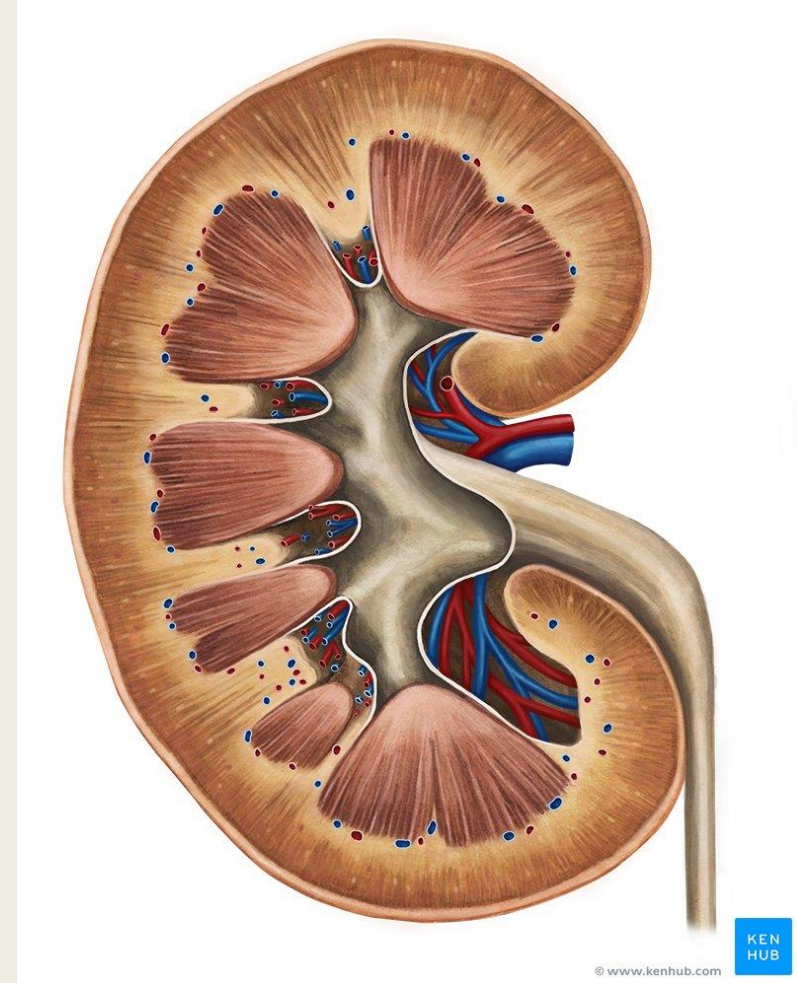
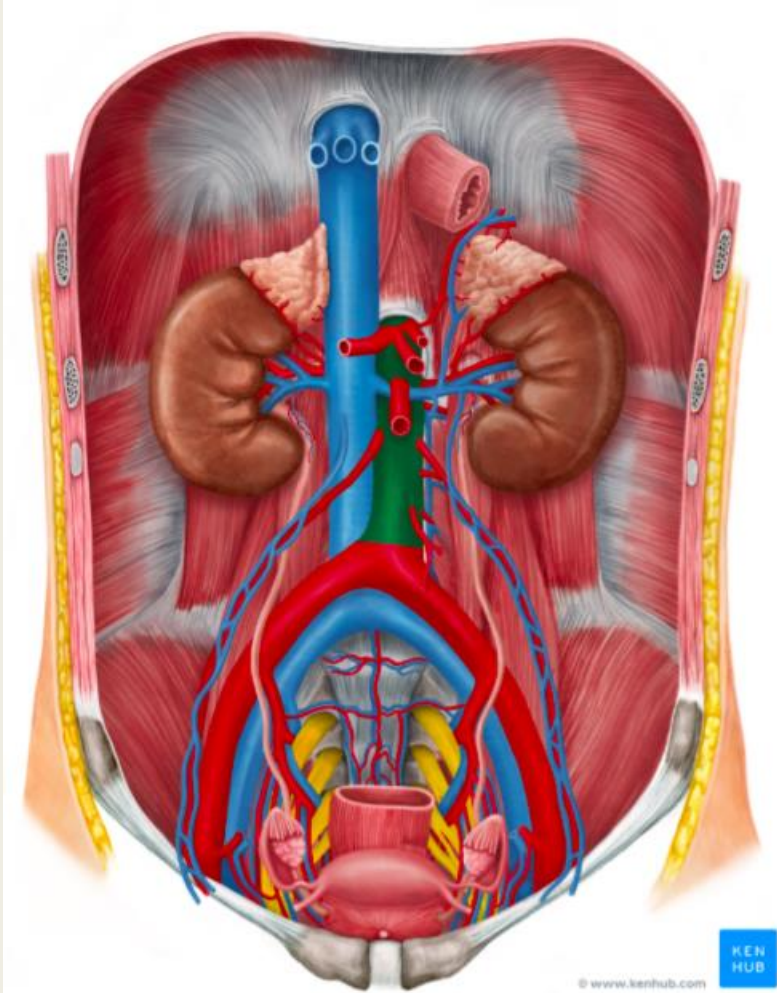
Disclosures / Conflicts of interest

- None

Objectives

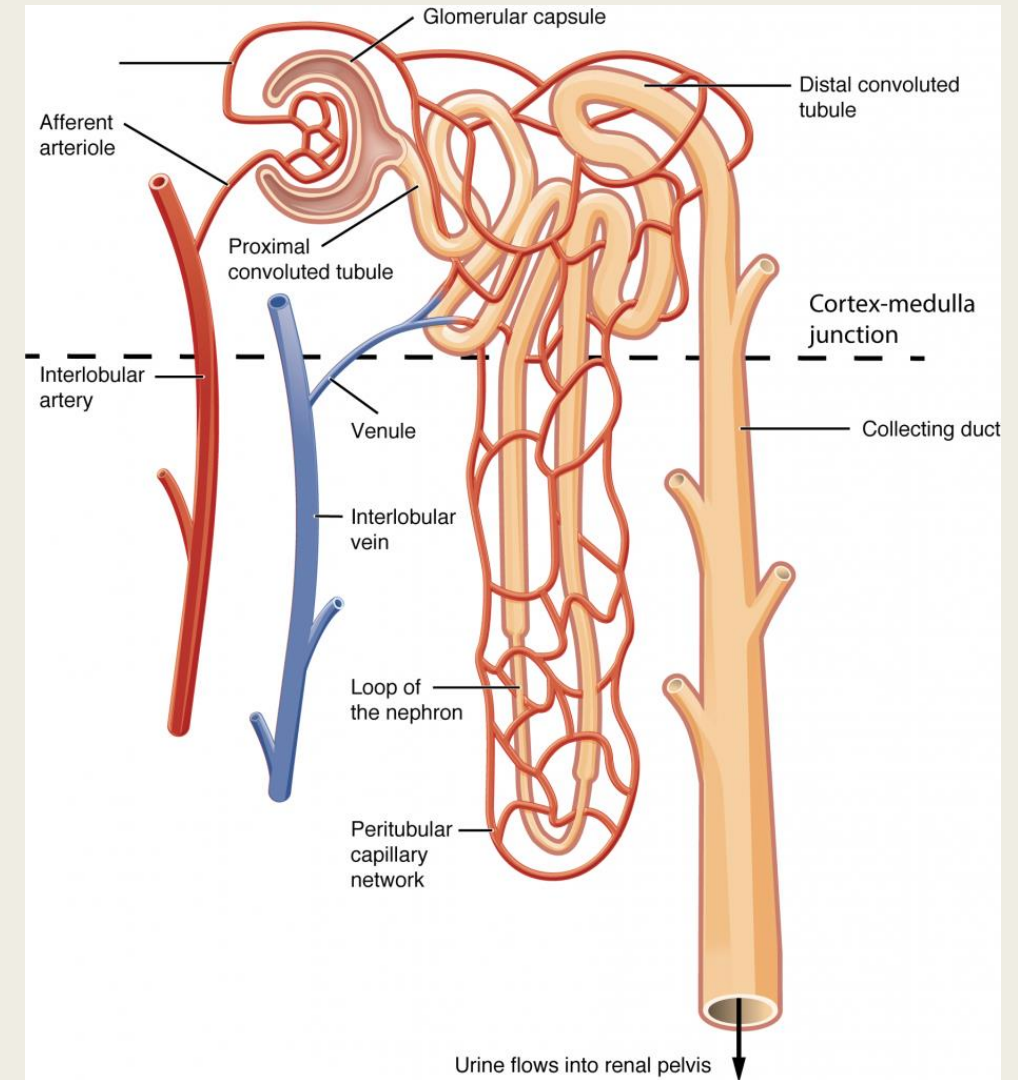
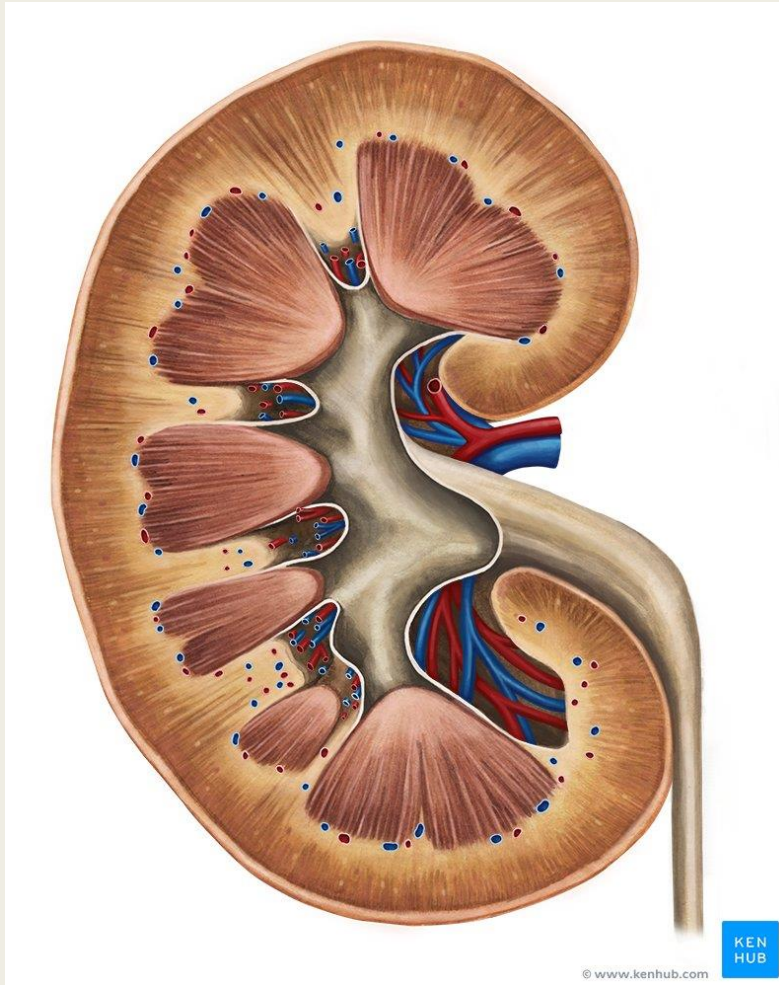
- Understand basic kidney functions and measurement of kidney function
- Understand definitions such as Acute Kidney Injury (AKI), Chronic Kidney Disease (CKD), and Acute on Chronic Kidney Injury (AoCKI)
- Recognize patients and clinical scenarios where AKI may occur
- Have a basic understanding of how physicians approach acute kidney injury re: etiology and management
 - *Clinical Exam*
 - *Urinalysis*
 - *Blood work*
 - *Imaging*
- Understand complications and management of complications from AKI
- Role of nursing in detection of AKI – “the front line”

Kidney anatomy



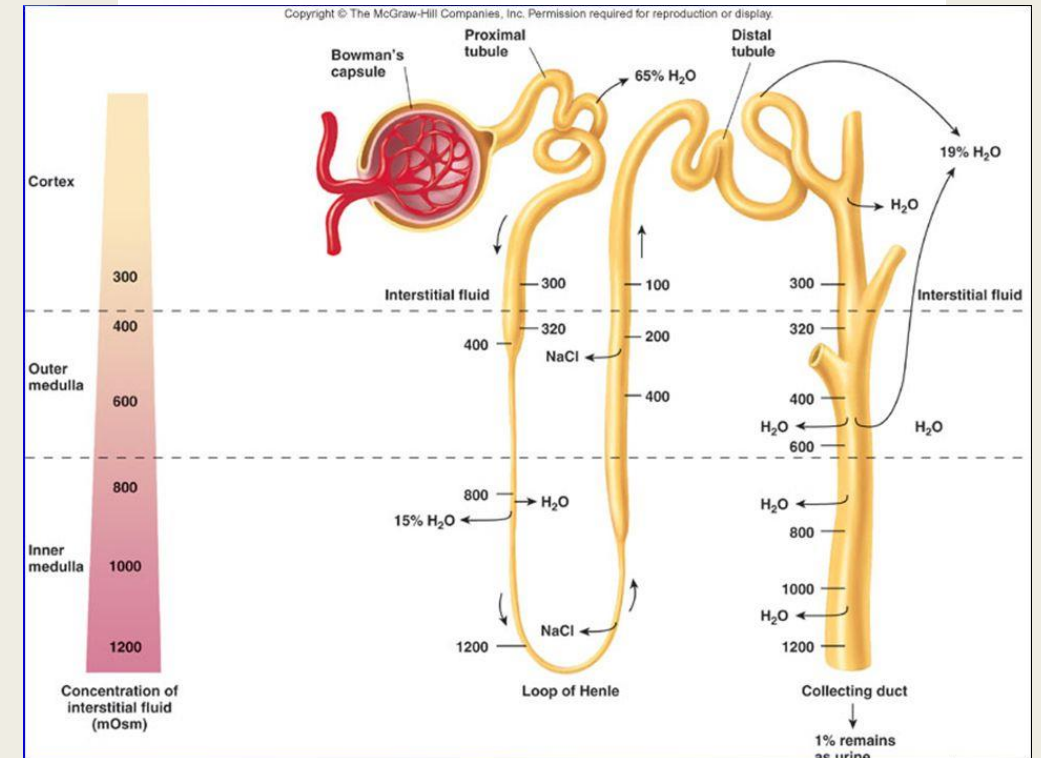
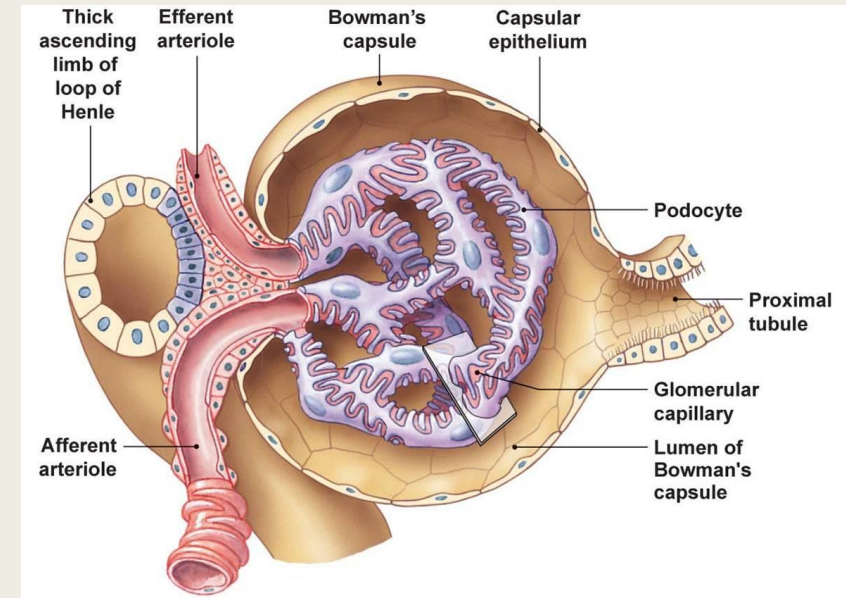
(Images from KENHUB anatomy, 20

Kidney anatomy



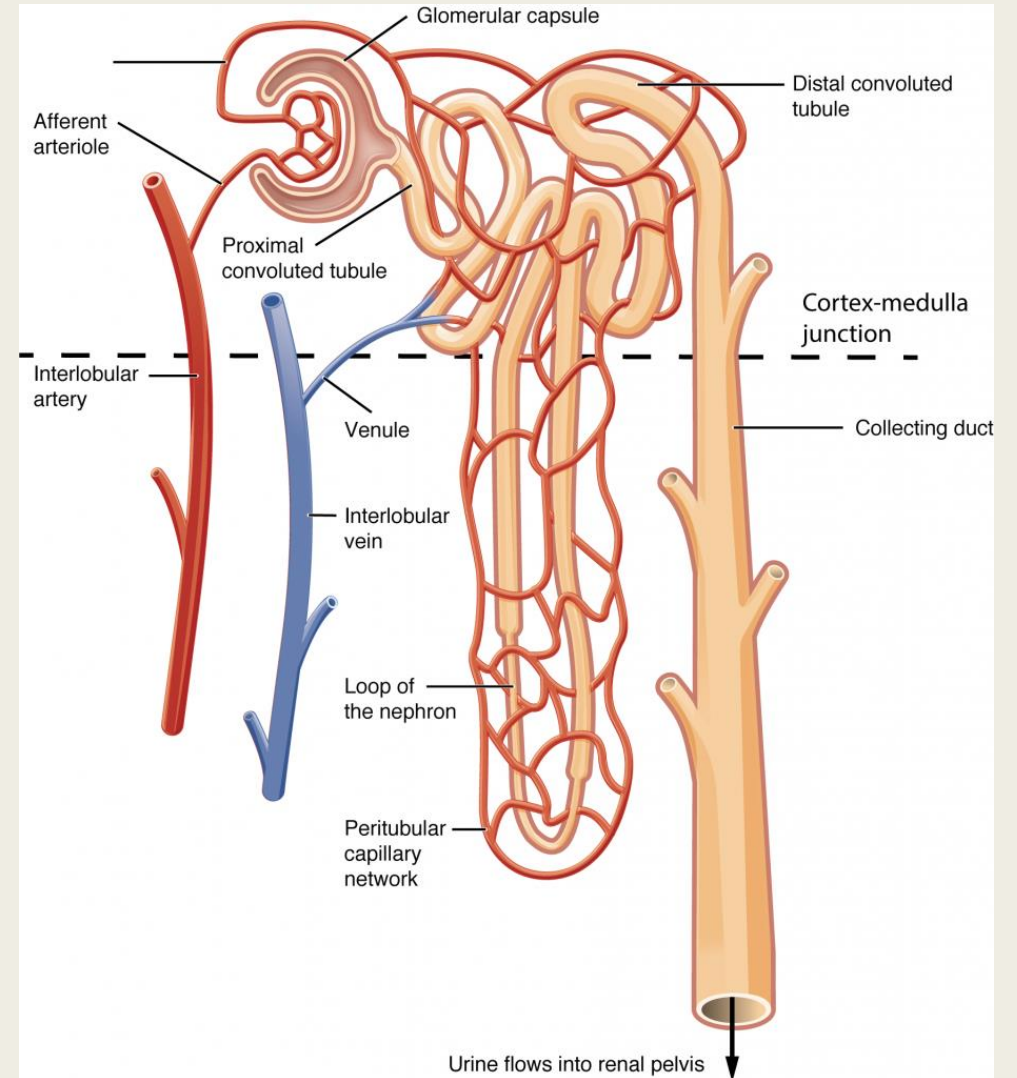
The nephron

- Composed of:
 - *Glomerulus (i.e., the filter)*
 - *blood enters and then forms a filtrate*
 - *Tubules – Filtrate from the glomerulus enters the tubules and then altered before becoming urine*
 - Reabsorb glucose, sodium
 - Secrete potassium



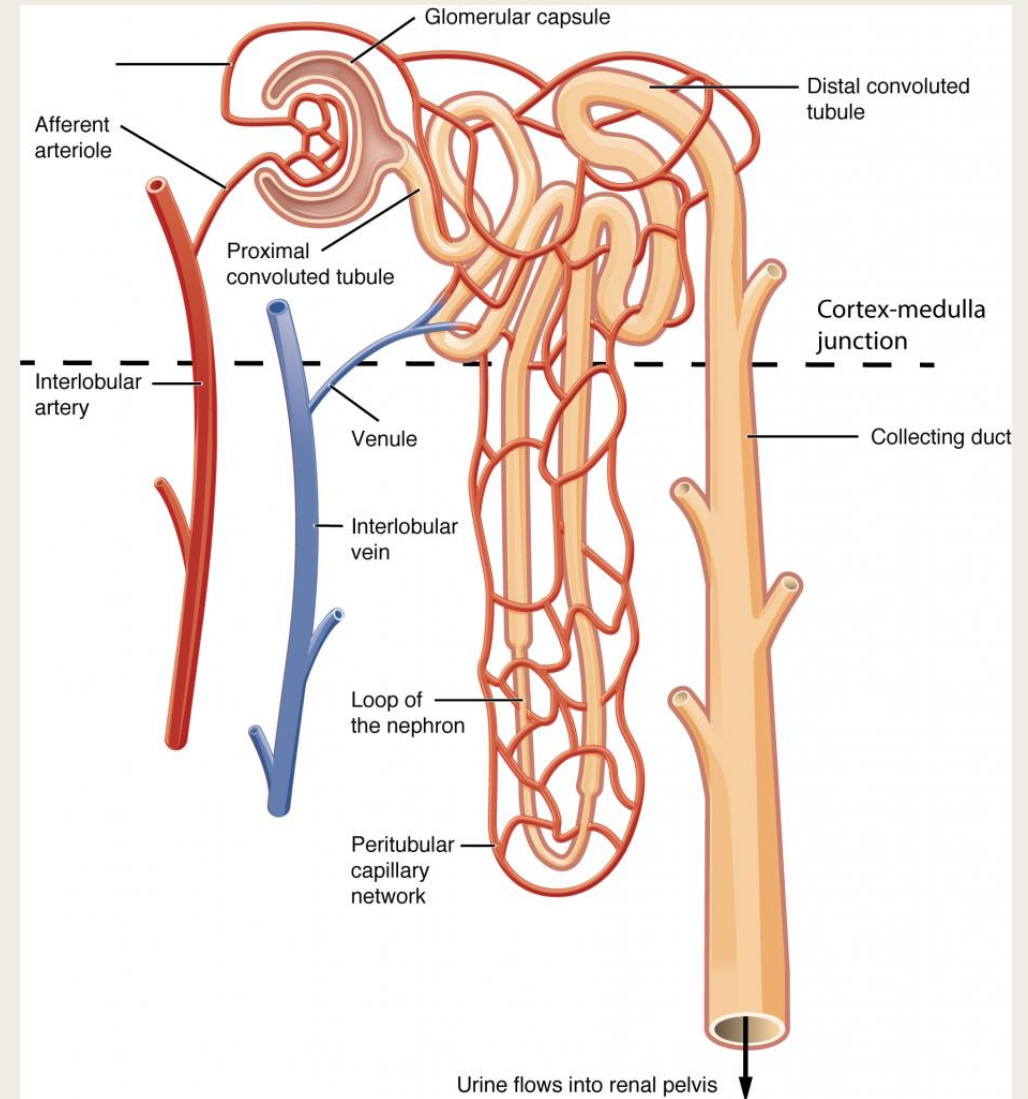
Kidney functions

- Maintaining the composition of the extracellular fluid
 - Maintenance of sodium, potassium, and water balance
 - Maintenance of normal pH with bicarbonate buffering
 - Removal of metabolic wastes
- Hormone functions
 - Converts 25-OH Vit D to active form
 - Erythropoietin and RBC production



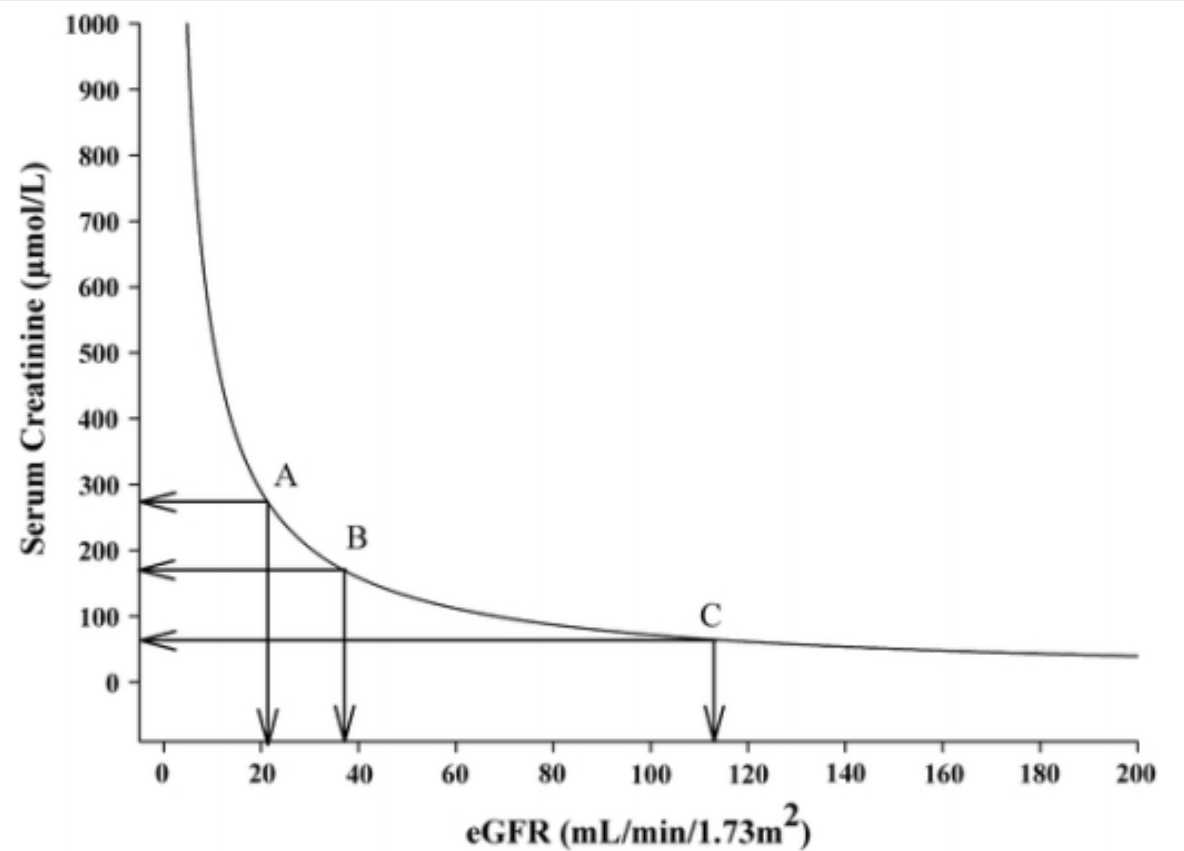
How do we measure kidney function?

- Use a blood test – **serum creatinine (Cr)** – to estimate glomerular filtration rate (**eGFR**) (i.e., how much the kidney is filtering)
 - How much the kidney is filtering (eGFR) is not the same as how much urine is being produced – urine is the final product as the filtrate is handled by the tubule
 - Kidney may, or may not, reabsorb water to make concentrated or dilute urine



Relationship between Cr and eGFR

- Consider an anephric (without kidney) individual...
 - GFR is zero as they do not have any kidneys
 - Cr will increase every day depending on how much is produced by the patient (no excretion, only production)
 - Formulas will give an eGFR that is inaccurate as actual GFR is zero



Urinalysis

- Essential in assessing kidney function
 - *Should be the first test ordered prior to more specialized tests*
- Kidney takes blood, makes a filtrate that is then modified as it travels the length of the nephron, before ultimately becoming urine
 - Red blood cells and protein **should not** appear in the urine
 - Presence may indicate damage to the filtering unit of the kidney

Macroscopic

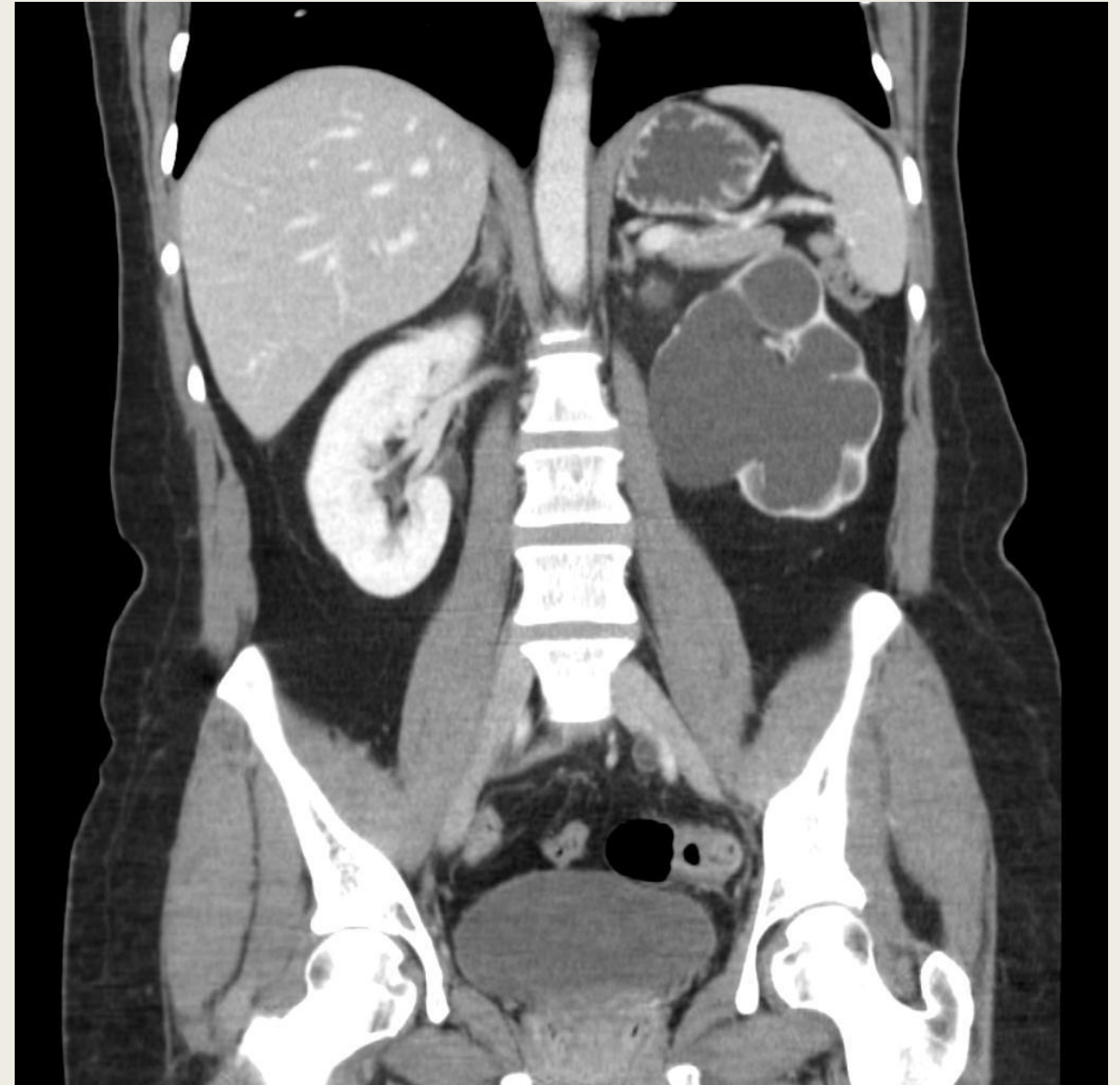
- Colour
- Turbidity
- **Specific Gravity** – *how concentrated is the urine?*
- **Protein**
- **Hemoglobin** – *≠ RBC*
- Ketones
- Leukocytes
- Nitrites

Microscopic

- **RBC**
- WBC
- Epithelial Cells
- Bacteria

Imaging

- Ultrasound or CT scan
 - Structural assessment
 - Number of kidneys
 - Stones
 - Evidence of chronic damage
 - **Is there an impediment to the drainage of urine?**



Practically, monitor kidney function with Creatinine and urine output

- In healthy person with normally functioning kidneys and absence of increased Cr release, Cr and eGFR will be stable
- 'Normal' Cr depends on the person
 - *Smaller people have lower Cr*
 - *Larger people have higher Cr*
- Most important questions are:
 - *1) is the baseline Cr normal or abnormal?*
 - *2) is the Cr changing?*
- Urine output is dynamic and changes to clinical scenario:
 - *When dehydrated – body wants to hold on to Na^+ and water*
 - *When high Na^+ and water intake, body will excrete same*
- As a general rule, low urine output concerning for worsening renal function and progressive inability to manage volume status and potassium
 - *$< 30 \text{ ml/hr}$ or $< 120 \text{ ml/4hrs}$*

Acute Kidney Injury (AKI)

- An acute impairment in kidney function, manifested by an increase in Cr, and/or a decrease in urine output
- Occurs over a period of days – weeks
- **Often thought of as being reversible, esp. when compared with chronic kidney disease**
- How much does the Cr have to increase to be significant?
 - *Increase of $\sim 30 \mu\text{mol/L}$, or $1.5\times$ baseline*
 - 40→70 vs. 200→230

Chronic Kidney Disease (CKD)

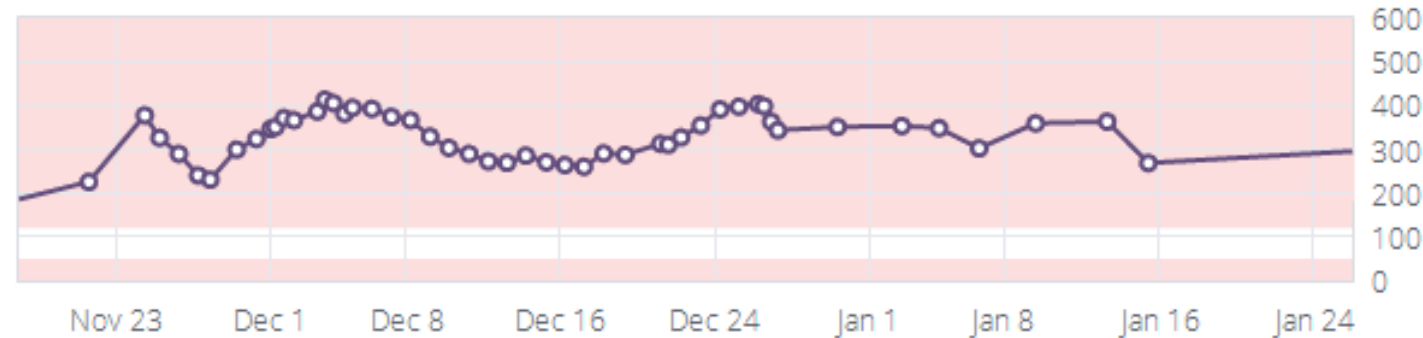
- Chronic (> 3 months) impairment in kidney function
- Staged on degree of impairment using: 1) eGFR, and 2) degree of albuminuria (i.e., the amount of protein in the urine)
- Generally start to see abnormalities in function as $\text{eGFR} \leq 30 \text{ ml/min}$

Prognosis of CKD by GFR and Albuminuria Categories: KDIGO 2012				Persistent albuminuria categories Description and range		
				A1	A2	A3
				Normal to mildly increased	Moderately increased	Severely increased
				<30 mg/g <3 mg/mmol	30-300 mg/g 3-30 mg/mmol	>300 mg/g >30 mg/mmol
GFR categories (ml/min/ 1.73 m ²) Description and range	G1	Normal or high	≥90			
	G2	Mildly decreased	60-89			
	G3a	Mildly to moderately decreased	45-59			
	G3b	Moderately to severely decreased	30-44			
	G4	Severely decreased	15-29			
	G5	Kidney failure	<15			

Acute on Chronic Kidney Injury (AoCKI)

- An acute deterioration in renal function in an individual with preexisting / baseline kidney dysfunction
- “Mr. Jones has longstanding diabetes, hypertension, and CKD with a baseline Cr of 175, now comes to hospital with 1 week of nausea, vomiting, and diarrhea with a Cr of 380”
- Kidney function may, or may not, return to prior baseline chronic impairment after reversal of acute insult

Creatinine (umol/L) Showing from 18-Nov-2018 to 25-Jan-2019



Where does AKI / AoCKI happen?

- Commonly occurs in the context of acute or chronic illness
- Estimated that ~10 - 20% of hospitalized patients will sustain AKI³
- If AKI is severe enough to require renal replacement therapy (i.e., dialysis), mortality approaches 50%
- Risk Factors for AKI:¹
 - *Older age*
 - *Volume depletion*
 - *CKD*
 - *DM*
 - *Other chronic disease (cardiac, respiratory, liver)*
 - *Shock*
 - *Sepsis*
 - *Trauma / burns*
 - *Major surgery (esp. cardiac)*
 - *Iodine based contrast*

**Bottom line: AKI happens in nearly every patient population –
You will regularly care for patients with AKI**

AKI has significant impacts

Health Care Costs Associated with Acute Kidney Injury

Methods



Alberta

Nov, 2002 to April, 2009
239,906 hospitalized adults



baseline Cr determined
prior to admission



AKI:
AKIN-determined
by peak Cr



Recovery:
determined by
Cr after D/C

Results

		N	hospital mortality	one year mortality	length of stay	hospital cost
	No AKI	206,650 (86%)	3%	12%	8.9 days	\$9,444 CAD
↕	Stage 1	25,495 (11%)	12%	25%	11.4 days	\$12,356 CAD
	Stage 2	4,598 (2%)	27%	41%	12.8 days	\$14,370 CAD
↕	Stage 3	2,493 (1%)	34%	47%	13.7 days	\$14,822 CAD
	Stage 3 + Dialysis	670 (0.3%)	50%	56%	16.5 days	\$24,260 CAD

Conclusion Severity of AKI, need for dialysis, and lack of kidney recovery are associated with significant health care costs in hospitalized patients and persist a year following admission.

How do physicians approach AKI?

- 1) Determine etiology: 3 possibilities of injury
 - *Pre-renal – problem with renal perfusion*
 - *Renal – problem with kidney*
 - *Post-renal – problem with urine drainage*
- 2) Therapy to preserve / restore kidney function
 - *Stop nephrotoxic medications*
 - *Dose adjust medications for renal function*
- 3) Manage complications
 - *Volume overload / edema / pulmonary edema*
 - *Hyperkalemia*
 - *Metabolic acidosis*

Pre-renal AKI

- Impairment in kidney function due to altered perfusion (blood flow) – not an intrinsic problem with kidney
- Low flow states
 - *Hypovolemia / hemorrhage*
 - *Decompensated heart failure*
 - *Liver failure*
- Medications that impact blood flow
 - *ACEi / ARBs*
 - *NSAIDs*
 - *IV contrast*

- *Clinical*
 - ***Low BP***
 - ***Volume contracted / expanded****
- *Blood work*
 - *Elevated Cr*
 - *Abnormalities as per underlying etiology (i.e., sepsis, bleed, etc.)*
- *Urinalysis*
 - *Kidney not damaged – no RBC or protein unless present before*
- *Imaging*

Treatment: Correct abnormal physiology (i.e., treat sepsis, heart failure, hypovolemia)

The most common cause of in-hospital AKI is pre-renal

- History will focus on:
 - *Any episodes of hypotension?*
 - *Any history of fluid losses (nausea, vomiting, diarrhea)?*
 - *Any low flow states (decompensated heart failure, cirrhosis, large volume ascites with paracentesis)?*
 - *Medication history / review*
- Sepsis has been proposed to cause AKI through several factors⁵
 - *Altered hemodynamics*
 - *Inflammatory mediators / cytokines*
 - *Cellular dysfunction & damage*

Post-renal AKI

- **Impairment in kidney function due to impediment of urine drainage**
- Internal obstruction of kidney / ureter / bladder
 - *Bilateral kidney stones*
 - *Atonic bladder*
 - *Prostate*
- External compression of ureter / bladder
 - *Abdominal malignancy*
- *Clinical*
 - *Older male*
 - *Post-op without Foley*
- *Blood work*
 - *Elevated Cr*
- *Urinalysis*
 - *Unremarkable (aka. Bland)*
- *Imaging*
 - *Hydronephrosis*
 - *Dilated ureter / collecting system*

Treatment: Relieve the obstruction, or make an alternative passage of urine (i.e., Foley, ureteric stent, nephrostomy).

Renal AKI

- Renal impairment from a collection of diseases that directly damage the components of the kidney (filter, tubules, blood vessels, etc.)
- Often involve internist and/or nephrologist in diagnosis and management
- "...**Lupus, ANCA vasculitis**, TTP, HUS, anti-GBM, membranoproliferative glomerulonephritis..."

- *Clinical*
 - *Depends on underlying disease*
- *Blood work*
 - *Elevated Cr*
 - *Abnormal serologies depending on cause*
- *Urinalysis*
 - ***Kidney damaged – Protein, RBCs / RBC casts***
- *Imaging*
 - *No specific abnormalities*

Treatment: Depends on etiology. Can include immunosuppression, plasmapheresis, blood pressure lowering, or supportive care

Important medications to stop / adjust in AKI

■ STOP

- *NSAIDS*
- *ACEi / ARBs*
- *Diuretics (Lasix, hydrochlorothiazide) unless renal dysfunction due to volume overload / CHF*

■ ADJUST

- *Antibiotics*
- *Antihypertensives*
- *Oral hypoglycemics*
- *Analgesia*
 - Prefer hydromorphone due to less accumulation of metabolites
- *Anticoagulation (heparins, DOACs)*

**When in doubt, look up whether appropriate given renal t
ask pharmacist / question prescriber whether safe**

Complications of AKI

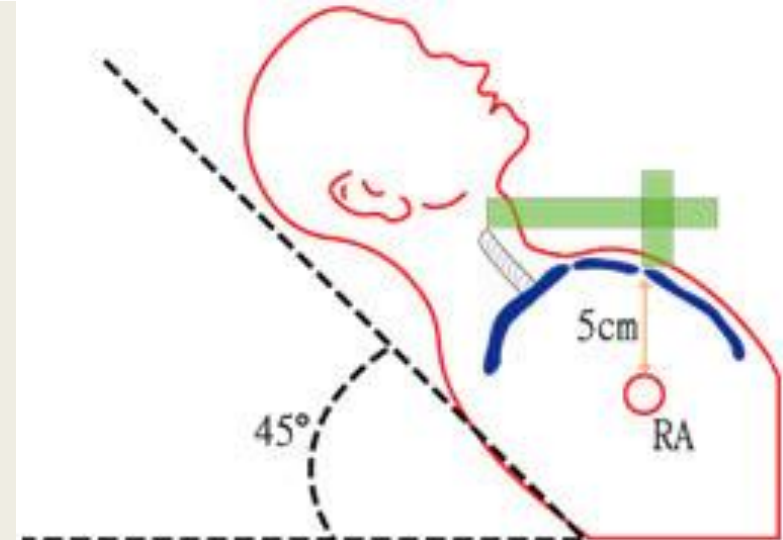
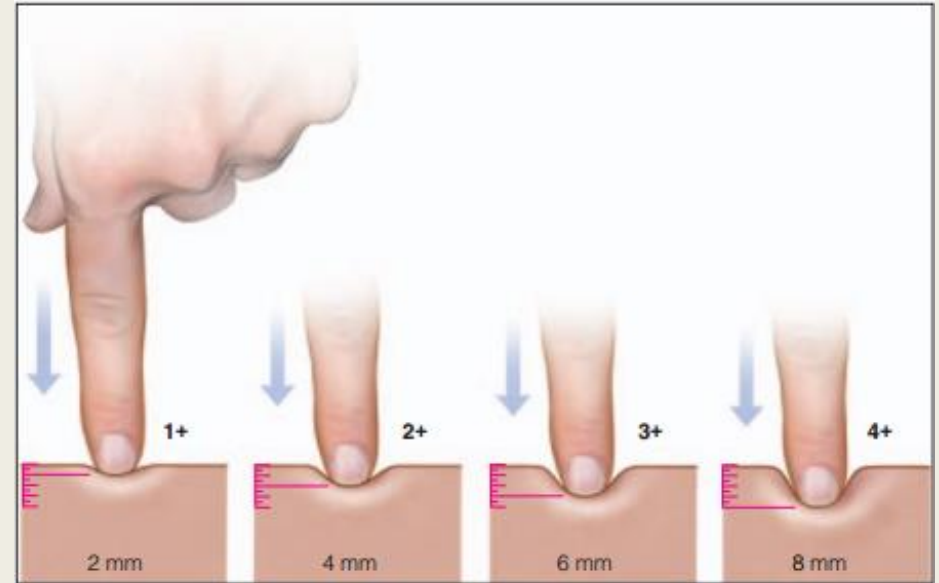
- **As eGFR / kidney function worsens, patients become:**
- Unable to manage sodium and water balance
 - *Edema, pulmonary edema*
- Unable to excrete potassium – leads to hyperkalemia
 - *Risk of cardiac arrhythmias*
- Unable to buffer acidosis generated from cellular functions
 - *Metabolic acidosis and cellular dysfunction*
- Unable to remove nitrogenous wastes

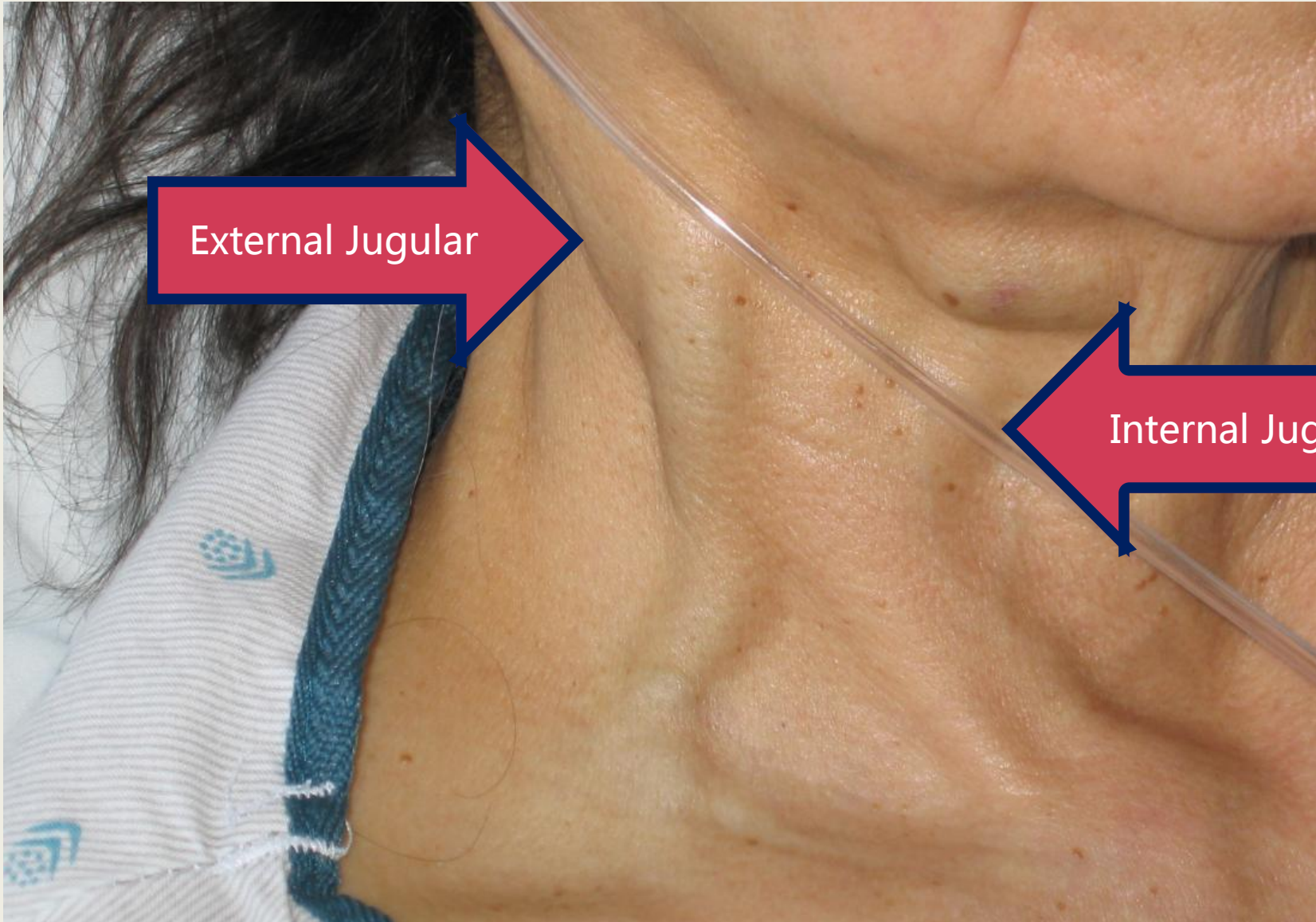
Treatment of AKI with complications:

Volume overload

■ Presents as:

- *Lower extremity or sacral pitting edema*
 - **How high does it go?** i.e., Ankles, shins, knees, thighs, etc.
- *Jugular venous distension*
 - Surrogate marker for right atrial venous pressure
 - **NB: elevated in other conditions** (tricuspid regurgitation, right heart failure, pulmonary hypertension, etc.)





(Image from Google Images, 20

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 - **NB: elevated in other conditions (tricuspid regurgitation, right heart failure, pulmonary hypertension, etc.)**

- Treat with:

- *Diuretics*
- *Na⁺ restriction*

Treatment of AKI with complications:

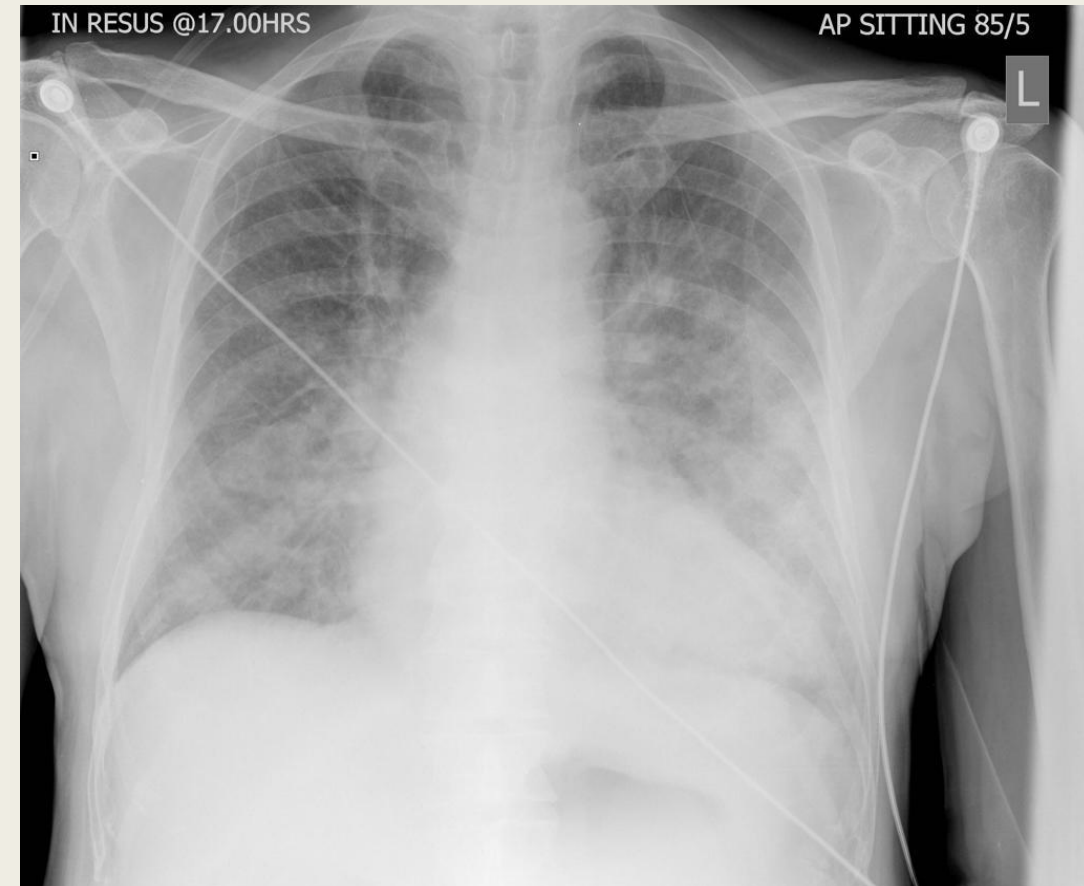
Pulmonary edema

■ Presents as:

- *Hypoxia / new O_2 requirement*
- *Shortness of breath / dyspnea*
- *Orthopnea – worsening SOB when supine*
- *Chest X-ray with bilateral infiltrates*

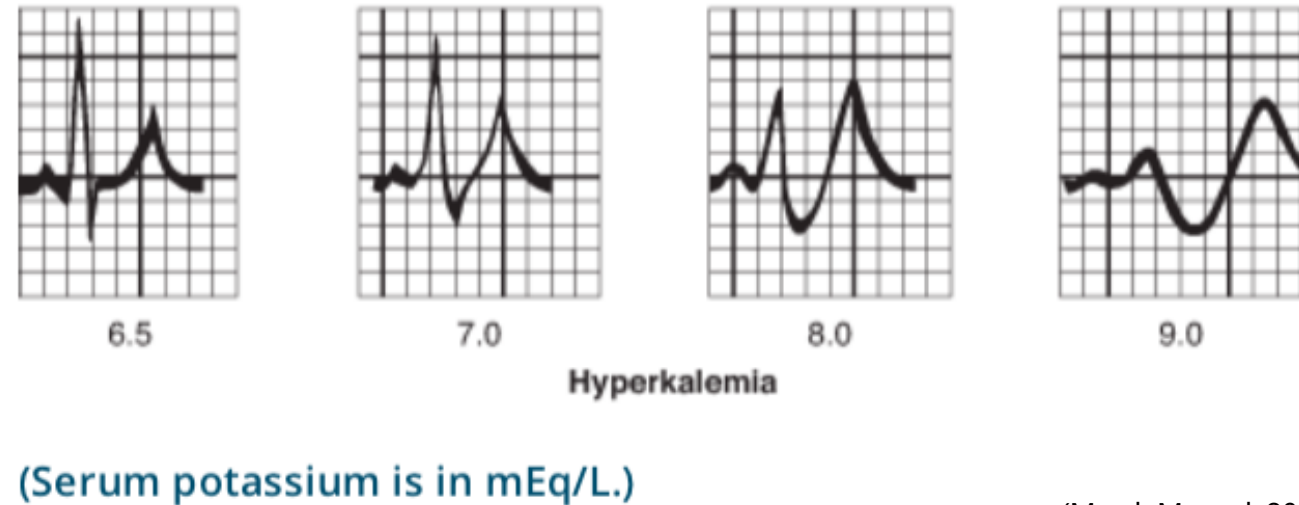
■ Treat volume pulmonary edema with:

- *Supplemental O_2*
- *Diuretics (esp. IV Lasix)*
- *BiPAP*



Treatment of AKI with complications: Hyperkalemia

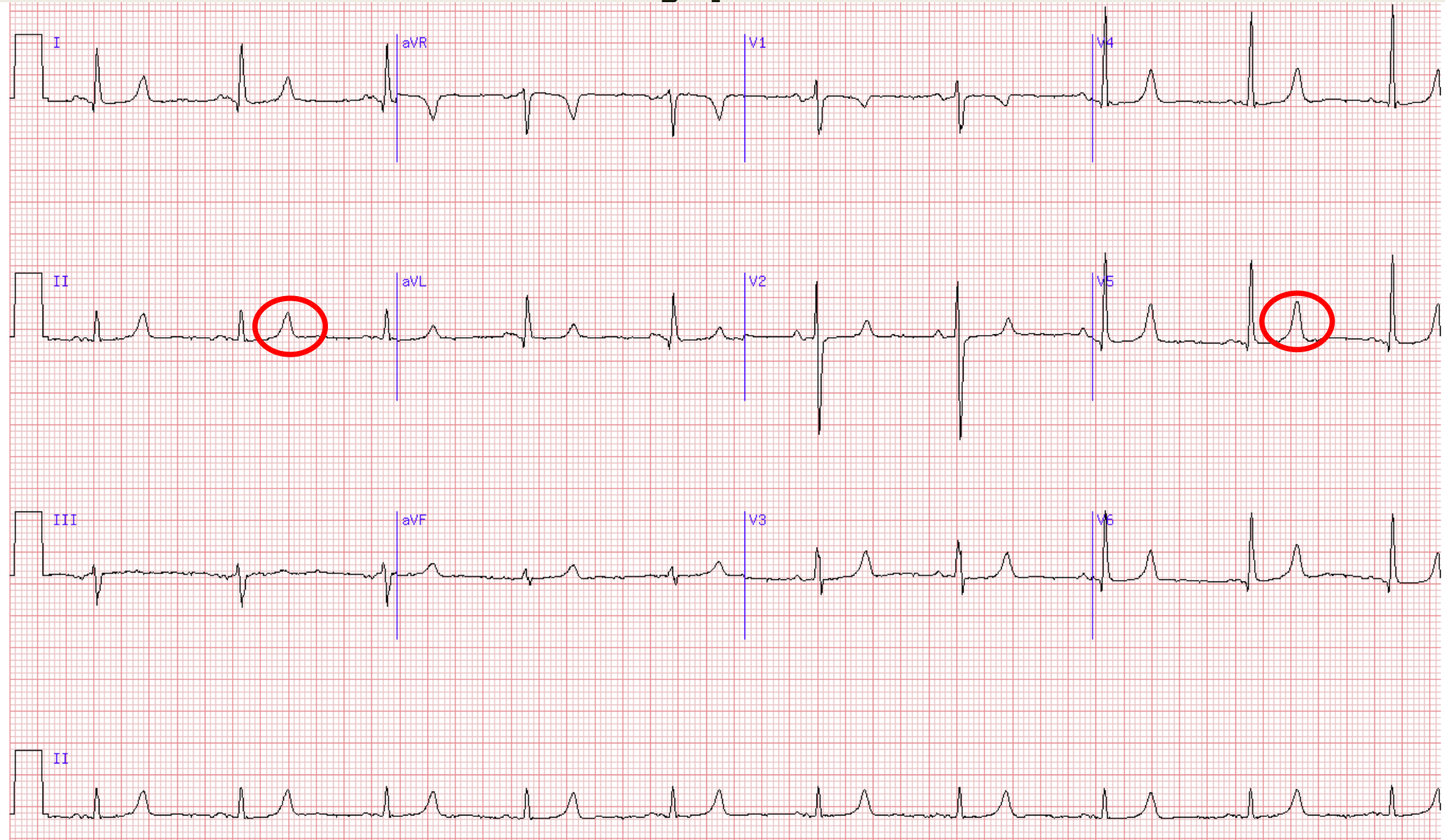
- Presents as:
 - *Spectrum of abnormalities based on 1) severity of elevation, and 2) rate of change*
 - *Asymptomatic – Peaked T waves, P wave flattening – QRS widening – ventricular tachycardia or fibrillation*
- Treat with:
 - *Acutely – stabilize myocardium with Ca^{2+} and shift potassium (insulin w/ glucose, bicarbonate)*
 - *Definitively – increase excretion of potassium through kidney or gut*



(Merck Manual, 2019)

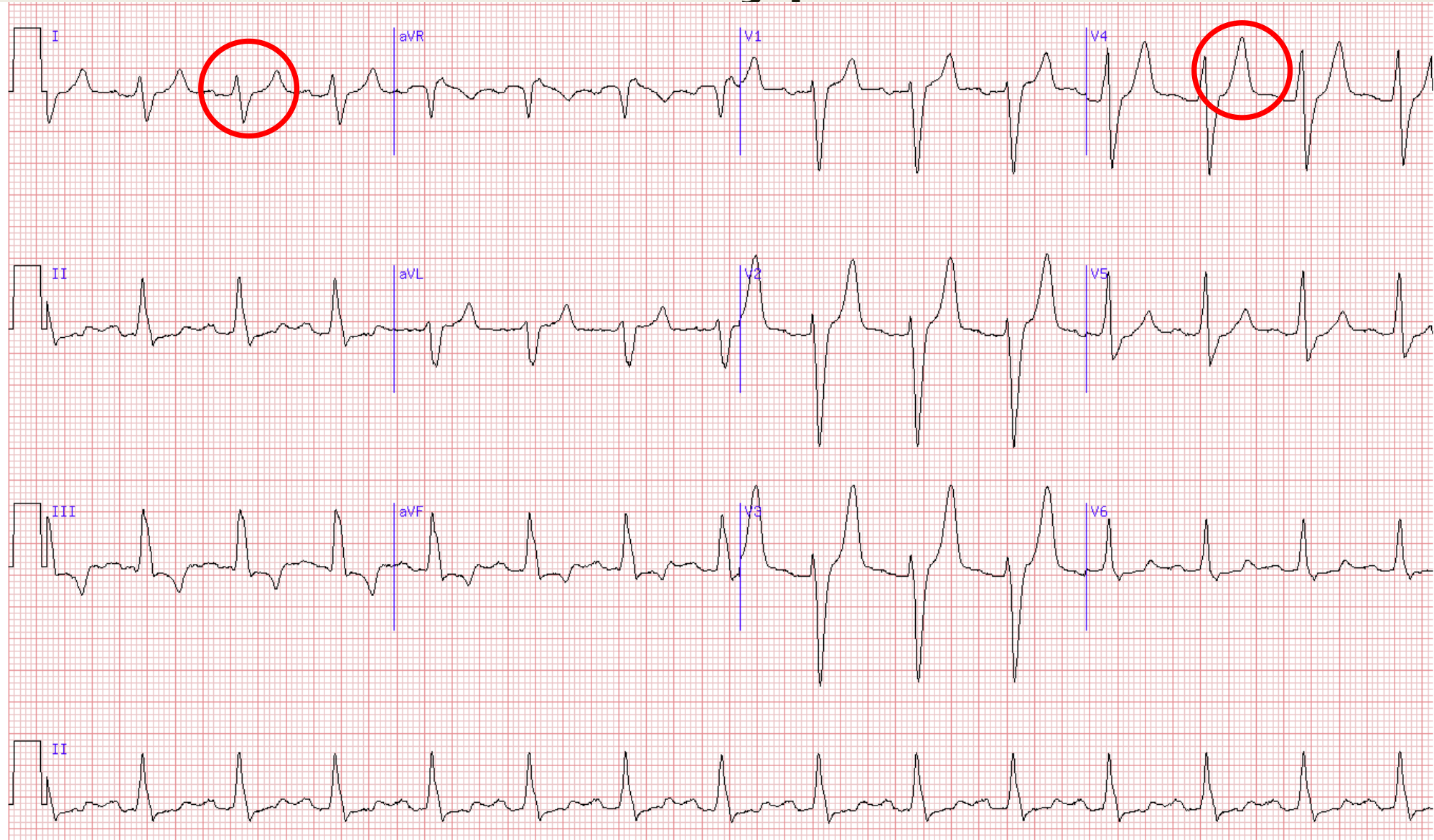
ECG with mild hyperkalemia

6



ECG with severe hyperkalemia

6



Treatment of AKI with complications:

Metabolic acidosis

- Presents as:

- *When severe, will cause cellular dysfunction – commonly manifest as hemodynamic instability, altered LOC*

- Treat with:

- *Improve renal function to excrete H^+ and generate bicarbonate*
- *Can use IV bicarbonate when severe*

When treatment fails and complications cannot be managed:

Dialysis

- Acute dialysis with hemodialysis
- Machine takes blood from patient, removes fluid, toxins, and electrolytes, then returns blood
- Typical session is 4 hrs, 3x / week
- Requires involvement of a nephrologist
- **Either supportive therapy until recovery of kidney**

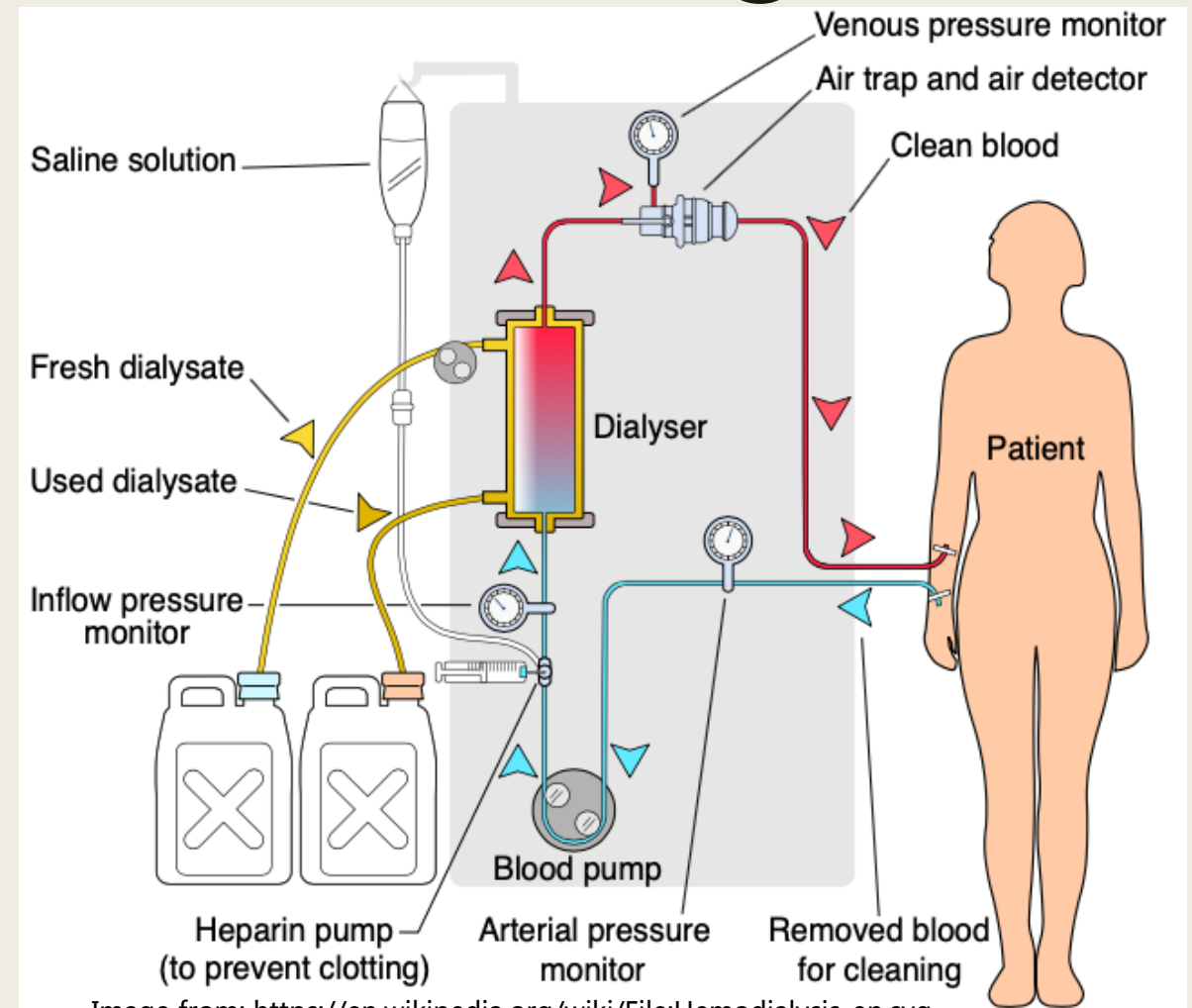


Image from: <https://en.wikipedia.org/wiki/File:Hemodialysis-en.svg>

Nursing and AKI

- In hospital, nurses are in a unique position to recognize and respond to AKI
 - *Decreased urine output often first noticed by nursing staff*
- Low urine output can then be interpreted in context of patient presentation, baseline kidney function, and clinical course
- **Early recognition is key**
 - *If offending pathophysiology, medications, etc., is recognized early, often can be reversed without significant short or long-term consequence*
- **Respond**
 - *Appropriate treatment of underlying cause and continued monitoring*
- **Refer**
 - *Communication with the other members of the team*

Nursing and AKI

- With change to Connect Care, expect automated flags that will alert users when a patient meets criteria for AKI/AoCKI
- Improve early recognition and early implementation of therapy to restore kidney function

The Problem:

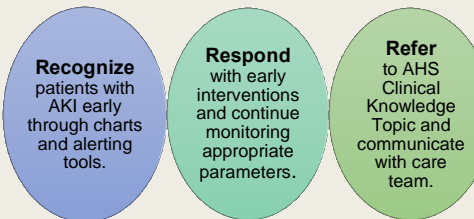
Acute kidney injury (AKI) is a common complication in hospital, as provincial data shows:

- Incidence of 1 in 10 hospitalized patients
- Associated with long-term risks of kidney failure requiring dialysis and mortality
- Increases length of hospital stay and costs of care

AKI is often reversible with early recognition and management.

What is SUPPORT AKI?

- A clinical decision support initiative
- Processes will be implemented to:



Who is the target population?

- Patients 18 years or older, not receiving dialysis
- Onset of acute kidney injury in hospital

What are the SUPPORT AKI resources?



Computerized and non-electronic tools will alert clinical staff when patients develop AKI on targeted surgery and medical units.



Clinical decision support resources are available to manage AKI using fluid therapies and in making appropriate medication prescribing decisions.



Additional guidance on monitoring, testing, and specialist consultation for AKI is available in the Alberta Health Services Clinical Knowledge topic on AKI, available at:

<http://insite.albertahealthservices.ca/assets/clin/et-klink-ckv-acute-kidney-injury-adult-inpatient.pdf>

What are goals of the initiative?

Trigger Early Processes of Care for AKI:

- Prompt goal directed fluid therapies
- Guide medication management

Reduce Adverse Outcomes:

- Reduce progression of AKI and downstream health consequences
- Reduce length of hospital admissions and costs



Study inquiries or feedback about the tools and processes?

Dr. Matthew James, Clinical Knowledge Topic Lead/Principal Investigator mjames@ucalgary.ca
Dr. Neesh Pannu, Principal Investigator npannu@ualberta.ca
Meha Bhatt, Project Coordinator (Calgary) meha.bhatt@ucalgary.ca
Nasreen Ahmed, Project Coordinator (Edmonton) nasreen2@ualberta.ca
Nancy Ruholl, Project Coordinator (Edmonton) nruholl@ualberta.ca

Questions?

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