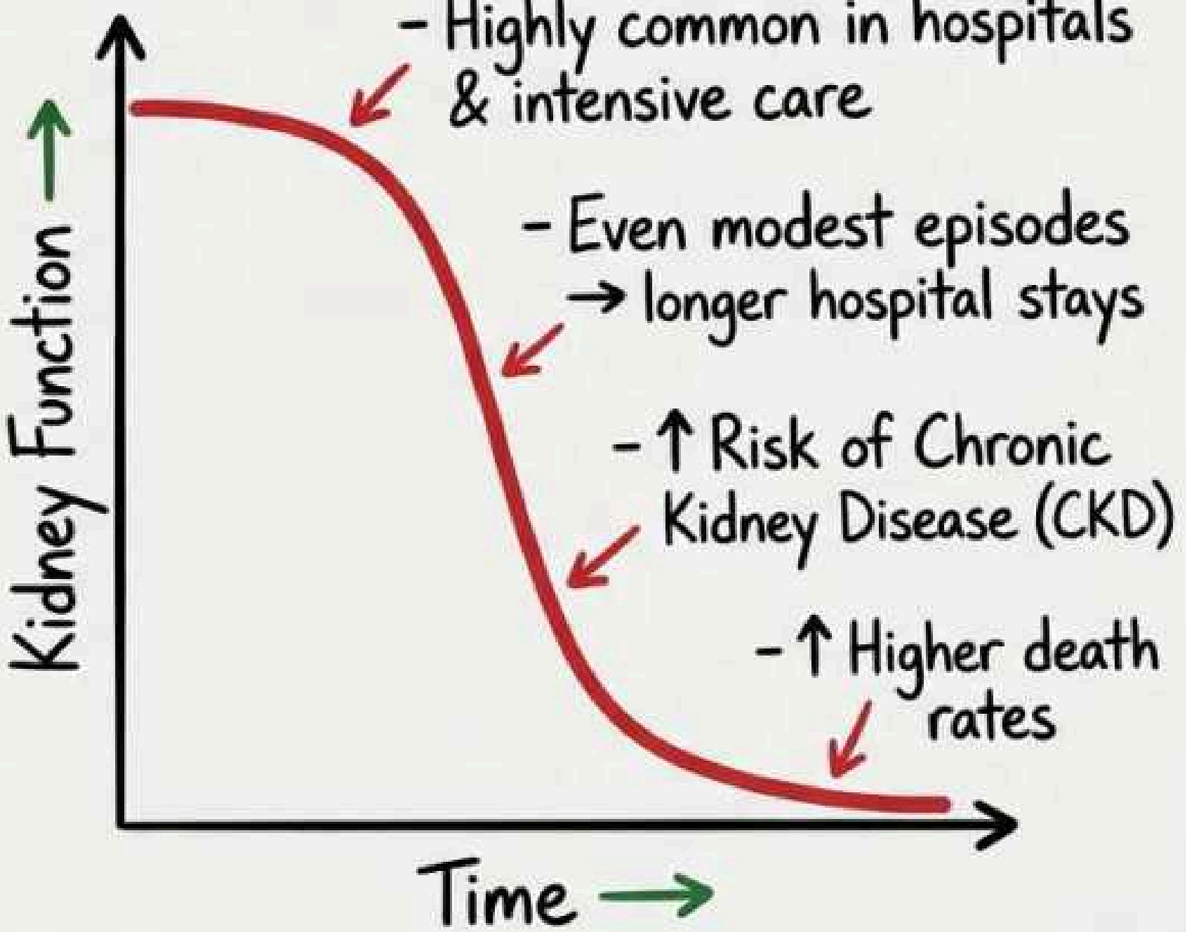
[¶ 4.1]

Defining Acute Kidney Injury

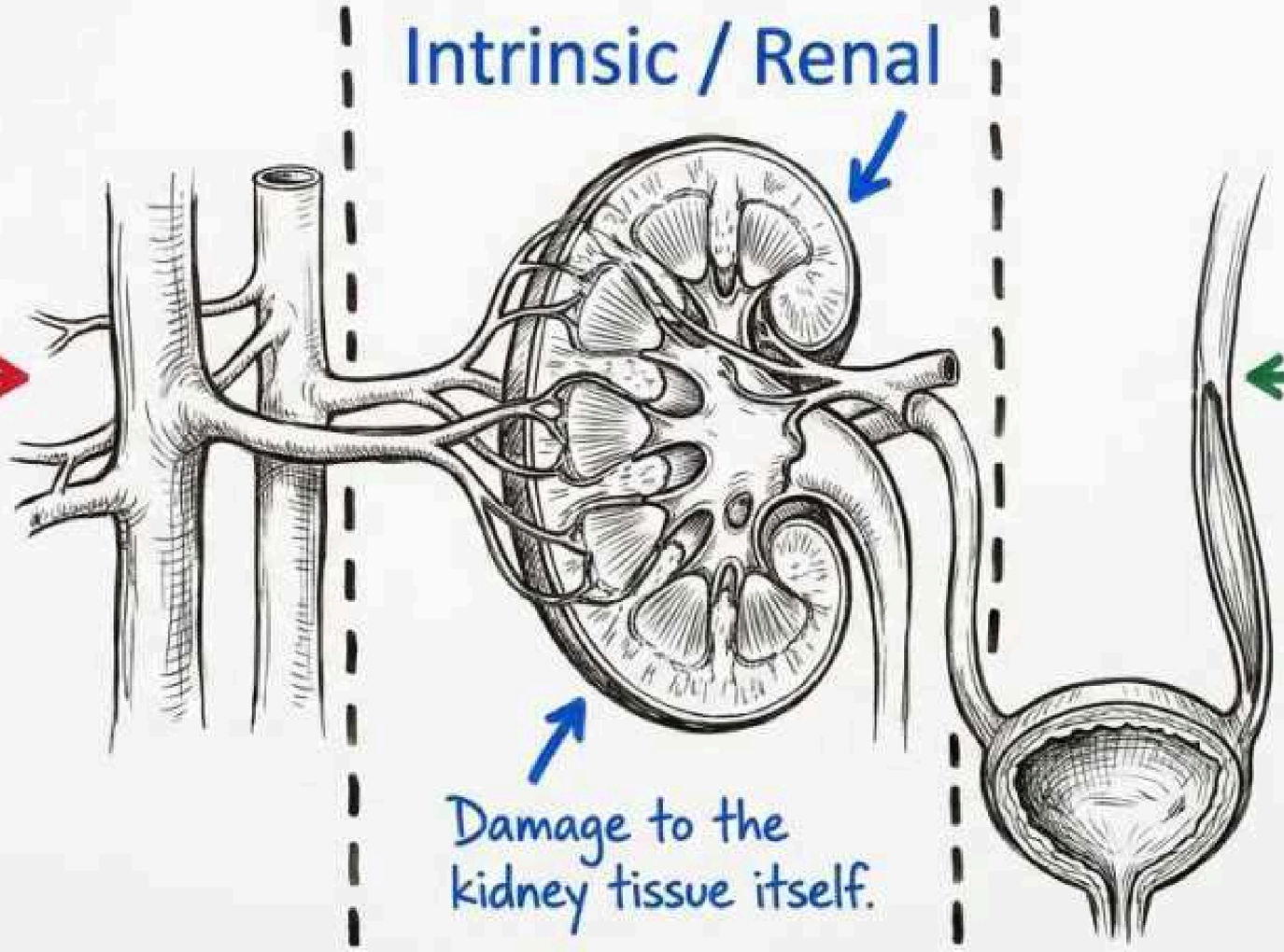
$$\text{AKI} = \frac{\left(\uparrow \text{Blood Creatinine} \;\text{OR}\; \downarrow \text{Urine Output}\right)}{\text{Over } \Delta \text{ Time (Hours} \rightarrow \text{Days)}}$$



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The Tripartite Anatomical Framework

[¶ 4.2]



This simple spatial division guides the first steps of all clinical assessment.

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Pre-Renal Causes: Perfusion and Pressure

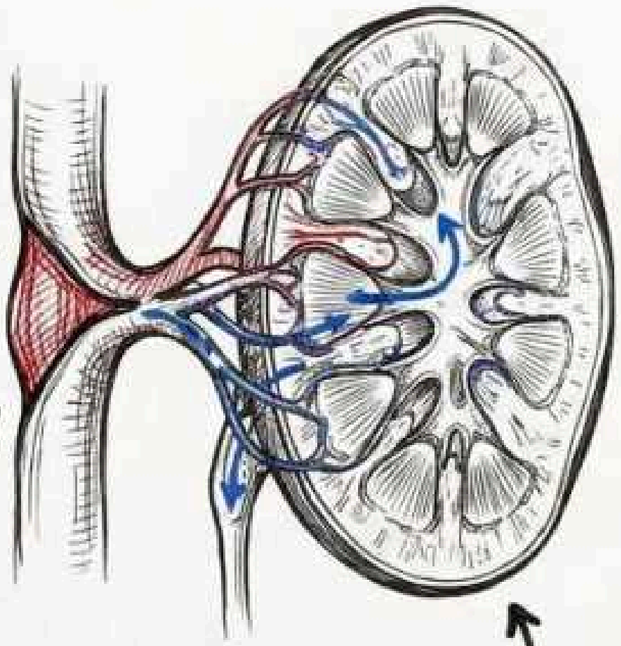
[¶ 4.3]

Dehydration

Bleeding

Heart Failure

Severe Infection with Low BP



Kidneys initially healthy; function recovers quickly if flow restored.

Medication Trap

[Vulnerable Patient] + [NSAID Painkillers OR RAS-Blockers] → Impaired Filtration Protection



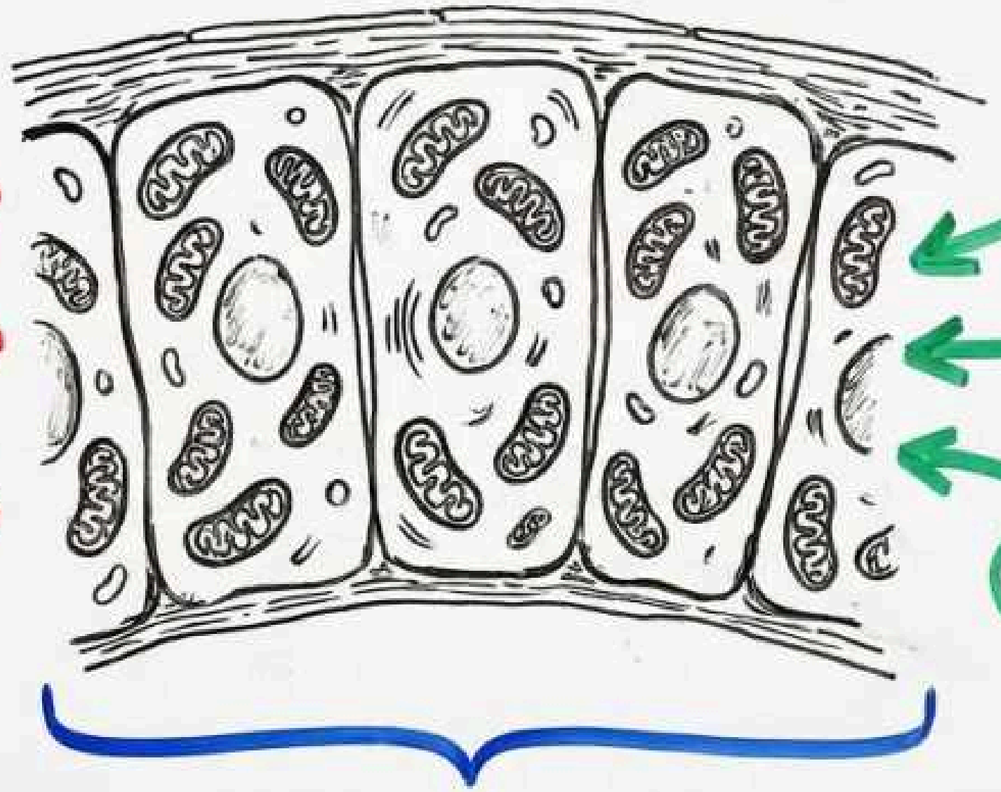
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Intrinsic Pathology:
Acute Tubular Injury (ATI)

[¶ 4.4]

Hypoxia

Prolonged Low Blood Pressure.



Toxins

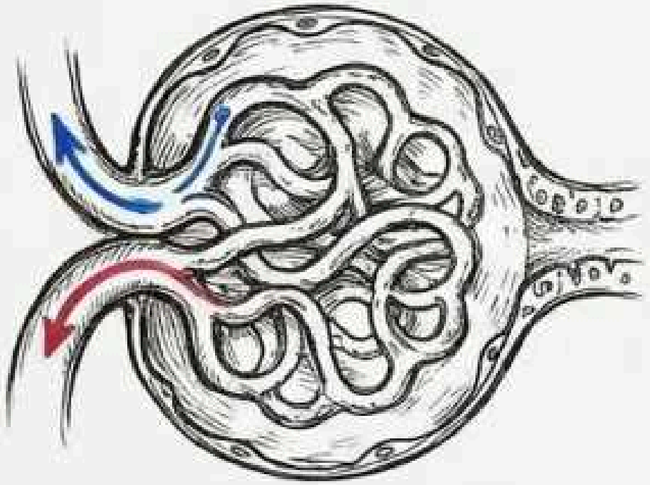
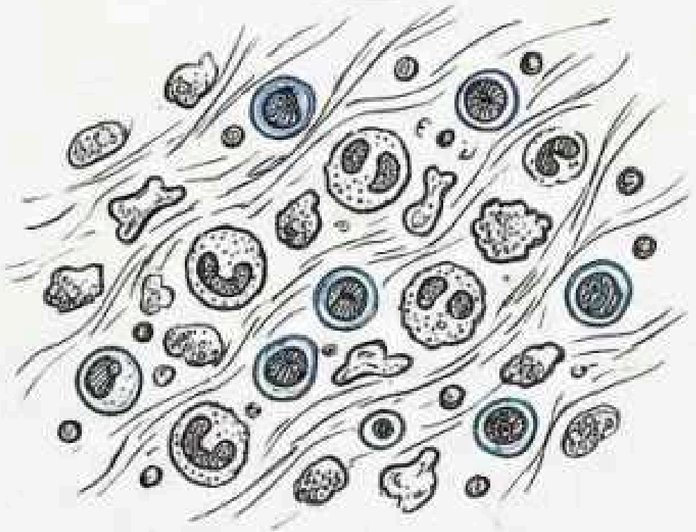
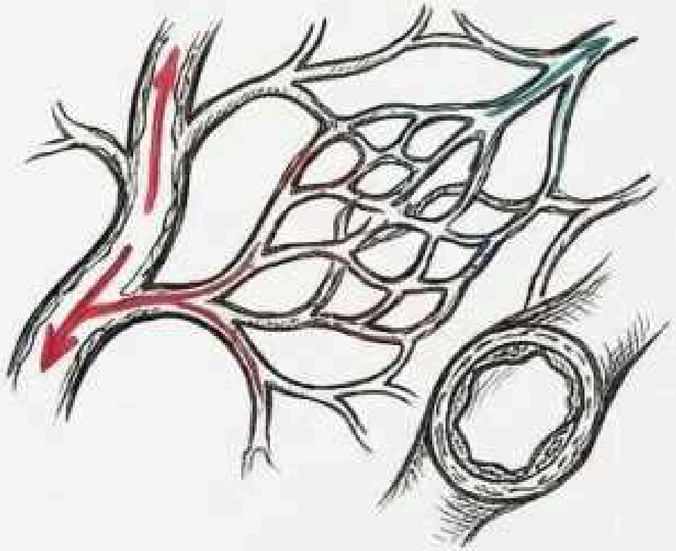
- Severe infection
- Specific Antibiotics & Chemotherapy drugs
- Contrast dye (used in scans)
- Muscle breakdown (from crush injuries)

Highly active cells
= Great deal of O_2 needed
= Highly Vulnerable.

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Intrinsic Pathology: Beyond the Tubules

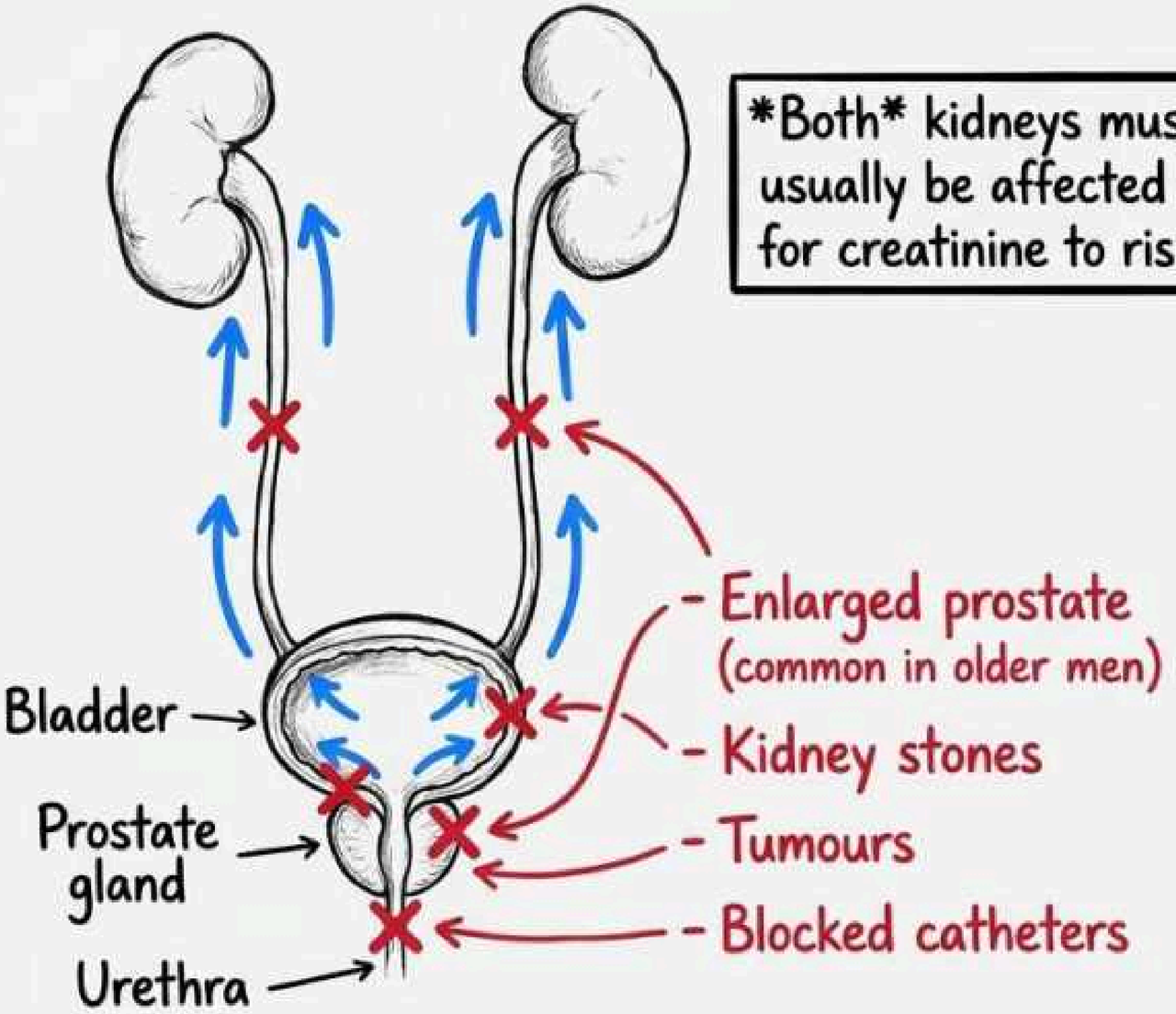
[¶ 4.5]

Glomeruli	Interstitium	Small Blood Vessels
		
Inflammation of the glomeruli. Can cause rapid loss of function.	Acute Interstitial Nephritis. Inflammatory reaction in kidney tissue, often an allergic reaction to a medicine.	Diseases of the small blood vessels injuring internal kidney structures.

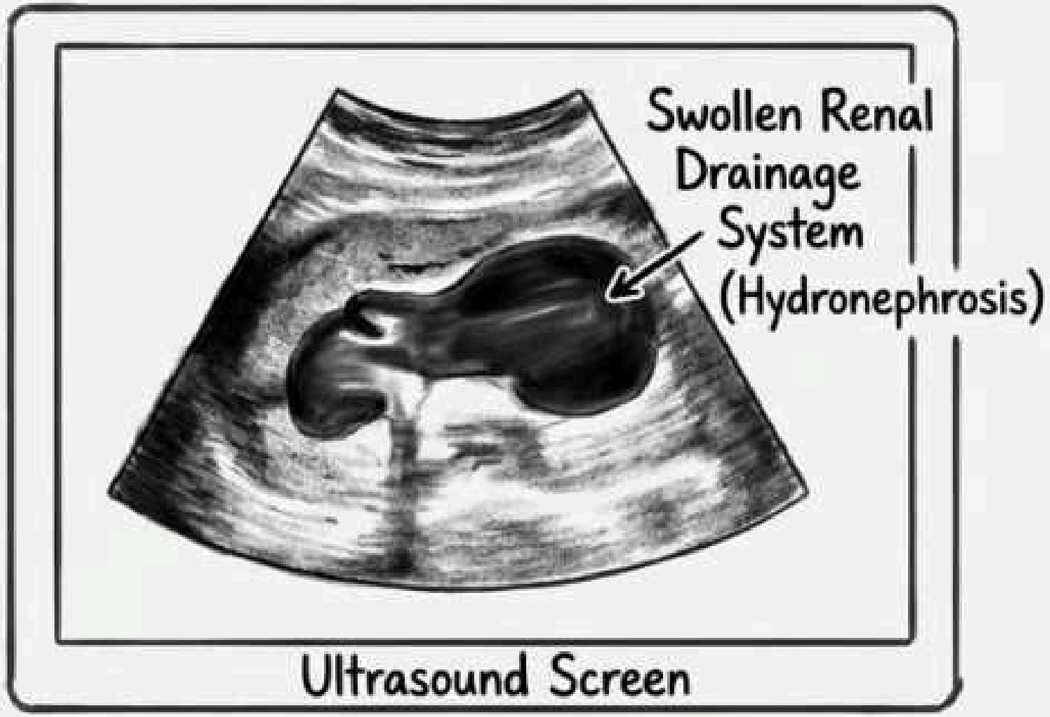
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Post-Renal Causes: The Obstructed Tract

[¶ 4.6]



Both kidneys must usually be affected for creatinine to rise.



→ Check for distended bladder

→ Order ultrasound for swelling

→ Relieve blockage to restore function.

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Clinical Assessment and Diagnosis

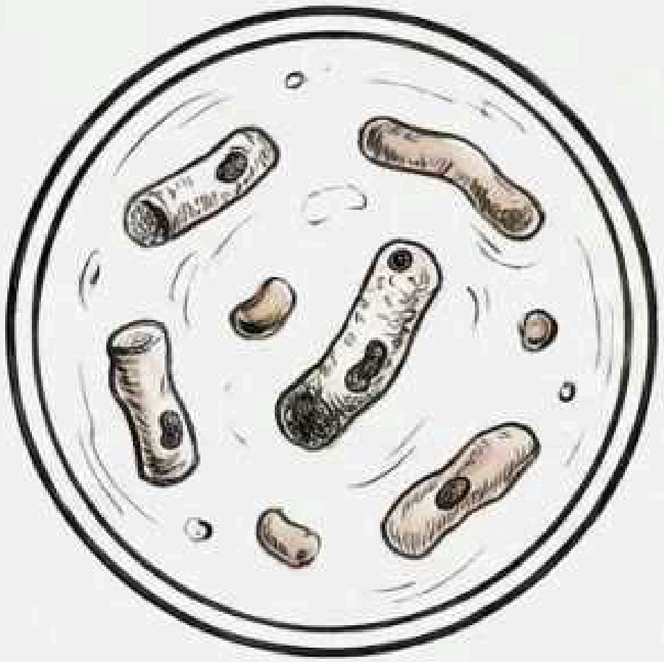
[¶ 4.7]



1. Patient Story & Medicines → 2. Examination (Focus on Fluid Status) →

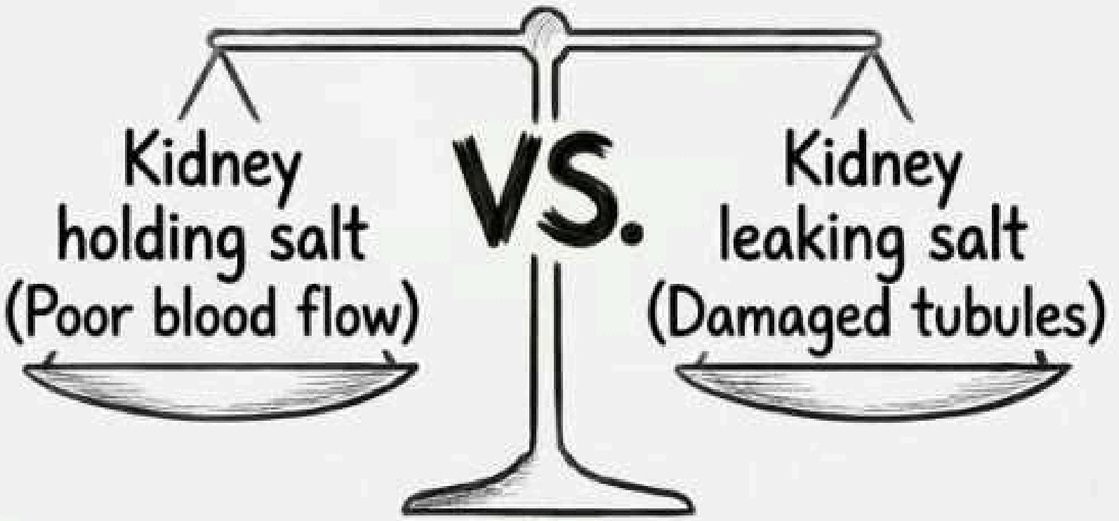


3. Blood & Urine Testing



Cells and casts point directly to tubular injury or glomerular inflammation.

Urine Salt Ratio helps distinguish:



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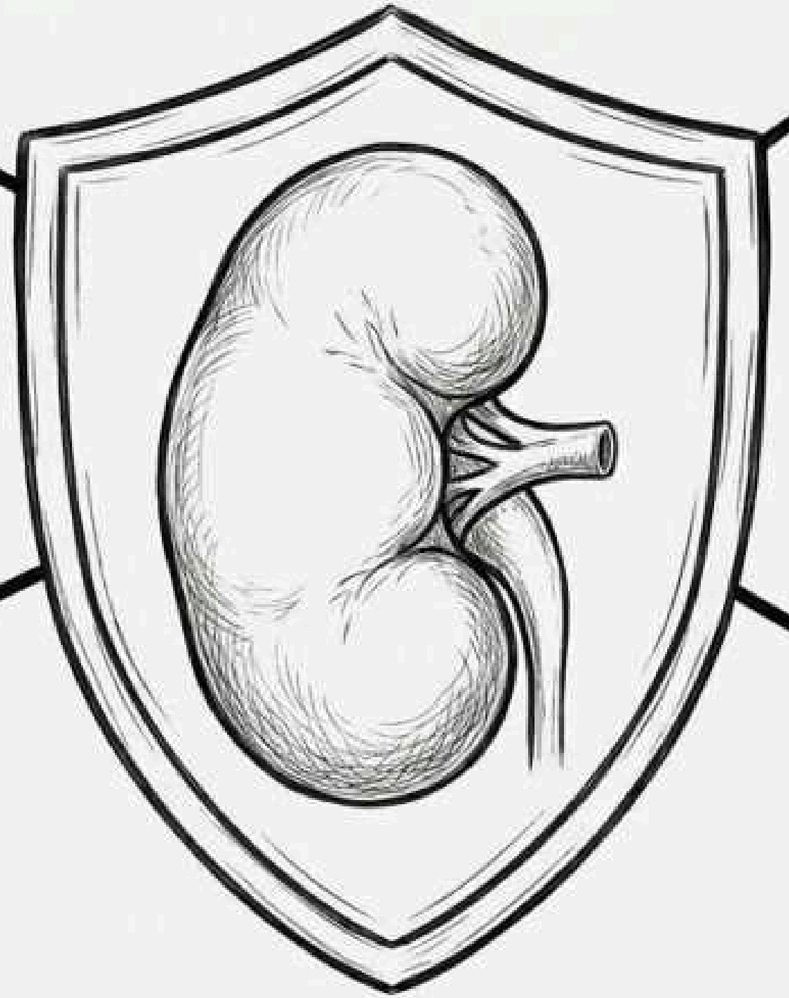
The Proactive Prevention Shield

[¶ 4.8]

Prevention matters greatly: There is NO drug that repairs an injured kidney.

1. Hydration
Keeping patients well-hydrated.

2. Toxin Management
Avoiding or adjusting kidney-harming medicines in at-risk patients.



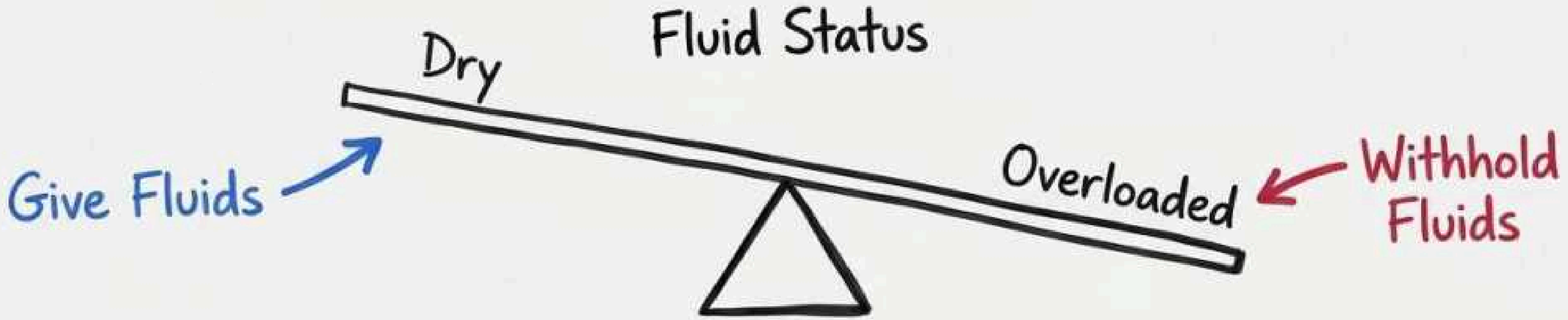
3. Vigilance
Watching kidney function closely in patients receiving risky drugs.

4. Rapid Response
Treating infection and low blood pressure promptly.

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Supportive Treatment and Clinical Balance

[¶ 4.9]



Rx

- Stop harmful drugs
- Adjust doses for reduced kidney function.

- High Potassium
- Severe Acidosis
- Fluid Overload

Require immediate treatment (as described in earlier lectures).

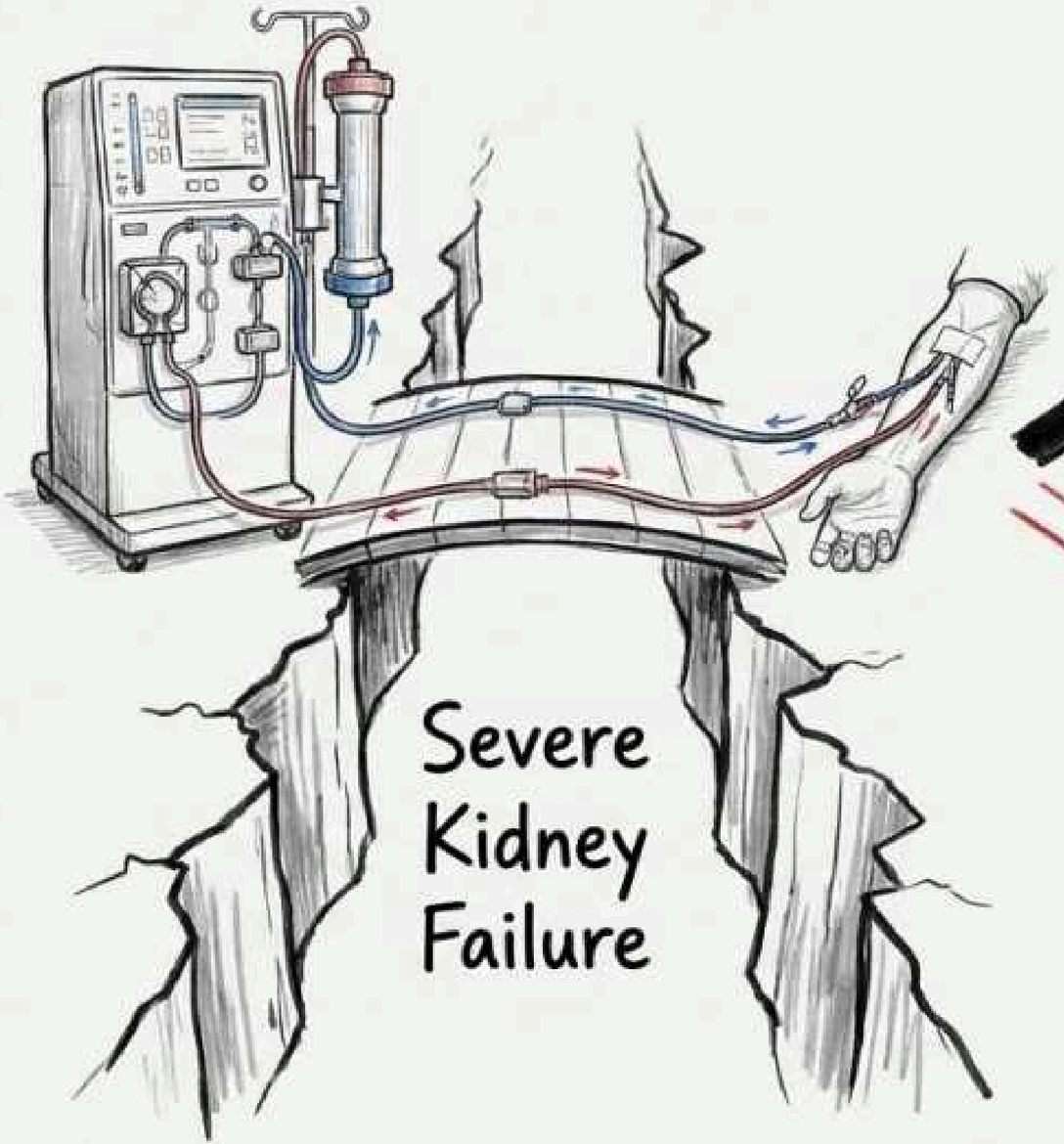
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Dialysis: Bridging Severe Failure

[¶ 4.10]

Indications Matrix (When to Initiate):

1. Dangerous potassium levels
2. Acidosis that cannot be controlled
3. Fluid overload in the lungs
4. Severe build-up of waste



Temporary bridge: Many recover enough function to stop dialysis.

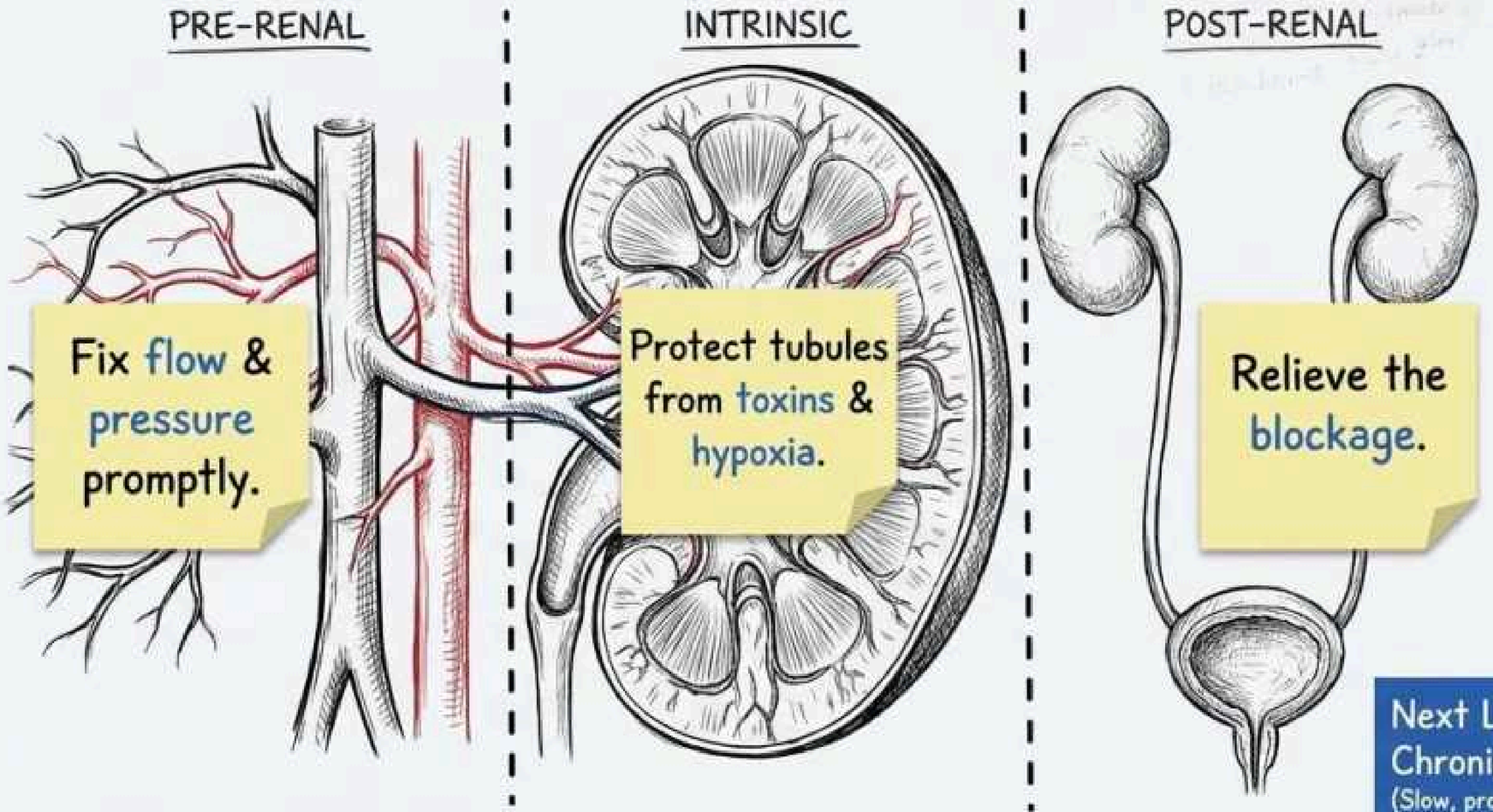
Some patients are left with lasting damage.

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Summary Synthesis: The AKI Landscape

[¶ 4.11]

AKI is a sudden loss of function. It is often preventable, and treatment relies on supporting the patient while the kidney recovers.



Next Lecture:
Chronic Kidney Disease
(Slow, progressive loss of function)

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