PEDIATRIC NEPHROLOGY SOCIETY OF THE PHILIPPINES

2024 TEACHING COURSE

BEDSIDE KIDNEY PHYSIOLOGY

CASE 1

Case 1

- A 9-month-old male presented with a 4-day history of non-productive cough, nasal congestion, and high-grade fever. Over the past day, his symptoms progressed to include fast breathing and poor oral intake.
- The patient was noted to be in cardiorespiratory distress on physical examination. His vital signs revealed tachycardia (HR 130 bpm), tachypnea (RR 58 cpm), and mild hypoxia with an oxygen saturation of 93% on room air. He was febrile at 39°C, with a blood pressure of 80/50 mm Hg, which is normal for his age.

Case 1

- On chest examination, there were subcostal and intercostal retractions indicative of increased work of breathing, and auscultation revealed bilateral crackles without wheezing.
- The heart examination showed a tachycardic but regular rhythm with no murmurs. His oral mucosa was dry, suggesting mild dehydration, but capillary refill time (CRT) was less than 2 seconds, and extremities were warm, indicating adequate perfusion.
- The abdominal exam was unremarkable, and pulses were full and equal in all extremities.

Case 1:

- Upon admission, he was immediately hooked to oxygen support at 10 lpm via face mask.
- D5IMB was started at FM + 20% and medications were started (Ampicillin at 100mkday Q6, Paracetamol at 10mkdose Q4 PRN for fever). He was transferred to the ward.
- An electrolyte panel, urinalysis, and chest x-ray were requested 4 hours after which revealed the following result.

Lab Results

	Reference values	Patient Result
Sodium (mmol/L)	**137-145**	126
Potassium (mmol/L)	**3.5-5.1**	3.5
Chloride (mmol/L)	**98-107**	90
BUN (mmol/L)	**2.5-6.1**	8.5
Creatinine (µmol/L)	**27-71**	33
Albumin (g/L)	**35-50**	40
Calcium (mmol/L)	**2.10-2.55**	2.1

Urinalysis

Dark yellow, slightly turbid, bilirubin negative, urobilinogen normal, ketone +, glucose normal, albumin negative, blood negative, PH 6.5, nitrite negative, leucocyte negative, specific gravity 1.018, RBC 0-1/HPF, WBC 2-4/HPF, EC 0, Bacteria 20, Mucus Thread 10

Chest X-ray

Hazy opacities on both middle lung fields. The heart is not enlarged. Aorta and pulmonary vascularity are within normal limits.

How would you approach the hyponatremia?

How would you approach the hyponatremia?

1. Assess the CNS status if warrants immediate management.

2. If normal, assess volume if euvolemic. If with severe neurologic manifestations, administration of hypertonic saline is warranted to increase sodium to safer levels

3. If euvolemic, check USG if concentrated or UNa is elevated. If NOT euvolemic, check FeNa and determine probable etiology

4. If concentrated, likely due to SIADH triggered by pneumonia. Non-osmotic ADH secretion leads to water retention and dilutional hyponatremia.

Points to Ponder

A urine electrolytes panel and repeat BUN and serum creatinine is requested showing the following:

- Urine Sodium ([Na]): 90 mmol/L (20-40)
- Urine Potassium ([K]): 20 mmol/L (25-125)
- Urine Urea: 5 mmol/L (20-40)
- Urine Creatinine: 15 mmol/L (9-16)
- Plasma Urea: 2.5 mmol/L (2.5-7)
- Plasma Creatinine: 40 umol/L (44-106)

Why is the urine sodium elevated in SIADH?

Why is the urine sodium elevated in SIADH?

- In SIADH, urine sodium concentration is typically **elevated** → This is a key diagnostic feature and reflects the body's inability to suppress renal sodium excretion despite hyponatremia.
- **Excess ADH** leads to water retention, which dilutes serum sodium (hyponatremia) but does not directly increase sodium reabsorption.
- The retained water expands extracellular volume, triggering **natriuresis** (increased sodium excretion) through suppression of the renin-angiotensin-aldosterone system (RAAS) and increased secretion of natriuretic peptides.
- As a result, the urine sodium remains high despite a low serum sodium level.
- *This finding helps differentiate SIADH from other causes of hyponatremia, such as hypovolemia, where urine sodium is usually low (<20 mmol/L).*

How else will you use the additional laboratory data?

- Check for urine osmolarity (indirectly):
  - Urine Osmolarity=2×([Na]+[K])+[Glucose]+[Urea]
  - Urine Osmolarity=2×(90+20)+0+5=2×110+5=225 mOsm/kg
- Compute for FE_{urea}:

$$\text{FE urea} = \frac{\text{Urine Urea} \times \text{Plasma Creatinine}}{\text{Plasma Urea} \times \text{Urine Creatinine}} \times 100$$

$$\text{FE urea} = \frac{5\,\text{mmol/L} \times 0.04\,\text{mmol/L}}{2.5\,\text{mmol/L} \times 15\,\text{mmol/L}} \times 100$$

$$\text{FE urea} = \frac{0.2}{37.5} \times 100 = 0.53\%$$

How would you interpret the results?

- **Urine osmolality >100 mOsm/kg**: Indicates inappropriately concentrated urine despite hyponatremia, consistent with SIADH.

- In SIADH, FE_{Urea} is typically >55%, reflecting the kidney's inability to conserve urea due to euvolemia and the suppression of renin-angiotensin-aldosterone activity. ADH increases water reabsorption but not urea reabsorption, leading to concentrated urine with urea retention.

- In contrast, in hypovolemia (a common differential diagnosis), the **FE_{Urea} is <35%** because the kidneys conserve urea as part of the volume conservation process.

What important aspect in the history should be elicited if this is due to SIADH?

What important aspect in the history should be elicited if this is due to SIADH?

- Identify triggers of SIADH release (i.e. stress, pulmonary and CNS disorders). In this case, patient has pneumonia
  - Risk factors or conditions that will have non-osmotic release for ADH
- Elicit large volume of hypotonic fluid intake. In this case, D5IMB plus 20% led to hyponatremia.
- In addition, humidified oxygen decreases insensible water loss so any high intake will worsen the hyponatremia.
- Remember: High levels of ADH alone do not cause hyponatremia. It should be paired with high intake of hypotonic fluids.

How would you manage the hyponatremia?

How would you manage the hyponatremia?

- Decrease hypotonic water intake.
- If you want to give an IVF, look for a fluid which has a higher osmolarity that the urine.
- In this case, this will be able to draw out excess water, increasing sodium levels.
- The mainstay of treatment in SIADH is fluid restriction.

Using 0.9% NaCl in SIADH

- Given:
  - ↑↑↑ ADH because of pneumonia
  - Euvolemic
  - Serum sodium 126 mmol/L
  - USG 1.018 ≈ 600 mOsm/kg (i.e., multiply last 2 digits x 36)

What do you think will happen if you infuse 0.9%NaCl at maintenance + deficit rates?

Using 0.9% NaCl in SIADH

- Osmolarity of 0.9% NaCl is 300 mOsm/L.
- With ADH, the kidneys (i.e. ultrafiltrate) will convert the 0.9% NaCl to match the current urine osmolarity of 600 mOsm/L.
- This is where mathematics will come in.

$$\frac{300}{x} = \frac{600}{1}$$

- Therefore, *x* will be 0.5 L or 500 ml.
- So, if you infuse 1 L of 0.9% NaCl, 500 ml of water will be retained just to produce a urine with 600 mOsm/L.
- The retained 500 ml of water will further lower the serum sodium.
- In order to correct the hyponatremia in SIADH, infuse a fluid which has an osmolarity greater than the urine osmolarity.

Using 3% NaCl in SIADH

- Osmolarity of 3% NaCl is 1052 mOsm/L.

$$\frac{1052}{x} = \frac{600}{1}$$

- Therefore, *x* will be 1.75 L or 1,750 ml.
- So, if you infuse 1 L of 3% NaCl, 750 ml of water will be removed to produce a urine with 600 mOsm/L.
- The 750 ml of water will now increase the serum sodium.

Using 0.9% NaCl in SIADH

- Given:
  - ↑↑↑ ADH because of pneumonia
  - Euvolemic
  - Serum sodium 126 mmol/L
  - USG 1.018 ≈ 600 mOsm/kg (i.e., multiply last 2 digits x 36)

What do you think will happen if you infuse 0.9%NaCl at maintenance + deficit rates?

Further lowers the serum sodium, worsening the hyponatremia.

Preventing Hyponatremia

- Given:
  - ↑↑↑ ADH because of pneumonia
  - Euvolemic

What will be your initial fluid of choice to prevent hyponatremia in this case?

Start with 0.9%NaCl BUT at limited rates of 70-80% maintenance.

What is the key physiological concept underlying hyponatremia?

Hyponatremia is NOT sodium ion loss BUT it is water retention.

Clinical Pearls:
Key Kidney Physiology Takeaways

1. **Hyponatremia = Water Problem**
   – Focus on fluid balance, not just sodium levels.
2. **In SIADH, prioritize fluid restriction**!
   - If IV fluids are necessary, ensure they are more concentrated than the urine osmolality
3. **Concentrated Urine + Hyponatremia = ADH Disorder**
   – Consider ADH-related causes when you see this pattern.

CASE 2

Case 2

An 8-year-old girl presents to a primary care clinic with complaints of dark-colored urine and mild abdominal discomfort. Her BP is elevated at 120/90 mmHg.

Her mother notes a recent fever that resolved, and the girl mentions feeling tired and having a "puffy" face in the morning.

Case 2

The clinician initially suspects a UTI based on the dark urine and mild abdominal symptoms and prescribes sulfamethoxazole-trimethoprim and advices increase fluid intake while waiting for the urinalysis result.

The urinalysis reveals moderate proteinuria (2+), mild pyuria (10-15 WBCs/hpf), and hematuria (15-20 RBCs/hpf).

Do you agree that the patient has UTI?

No, although there is pyuria but there are other signs and symptoms not related to UTI. UTI can not lead to edema and hypertension

What is the first thing to ask whenever you get a urinalysis result?

Always ask how it is collected especially check the USG.

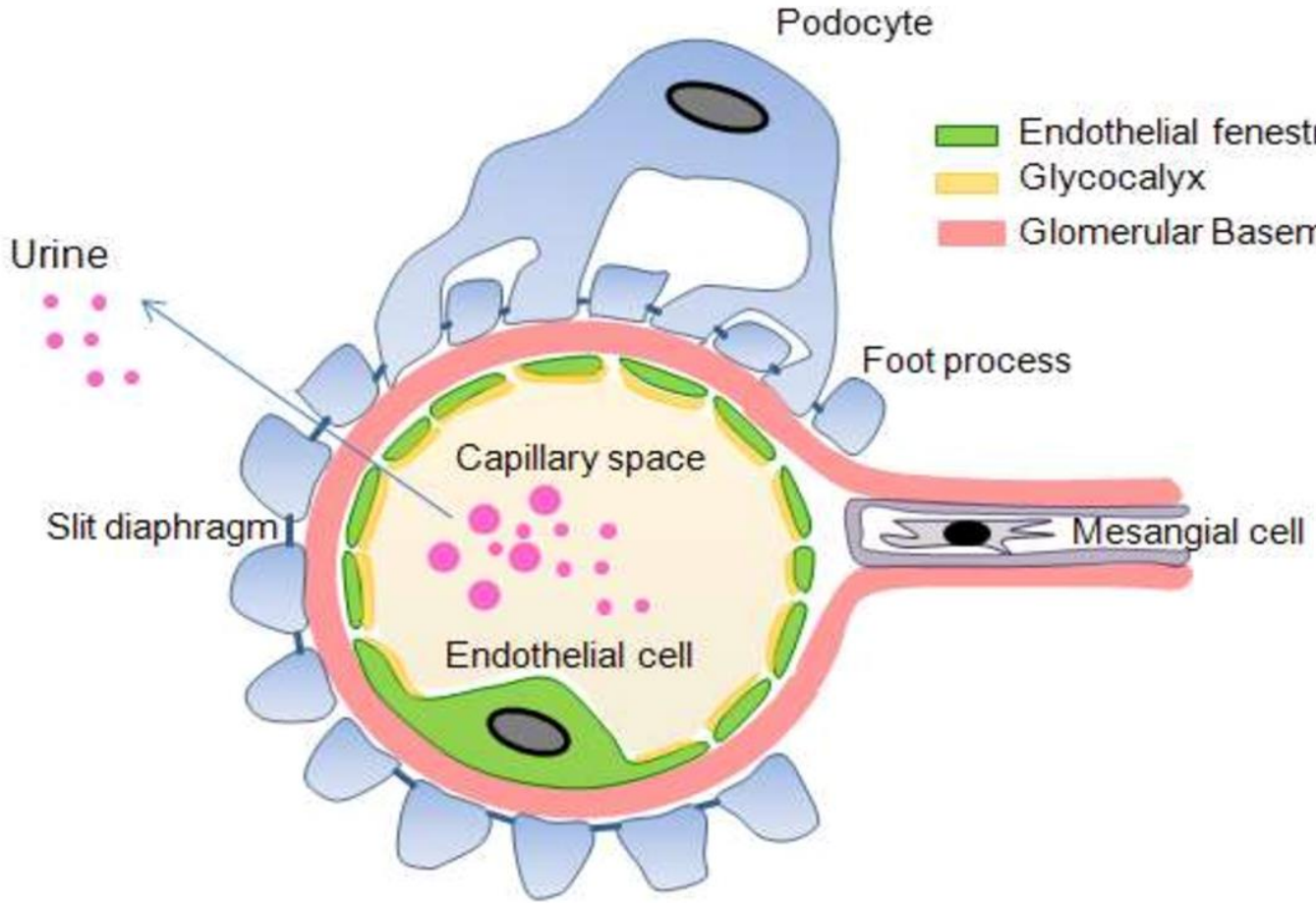
Do you agree that the patient has UTI?

Not all pyuria is UTI.

What does the presence of both proteinuria and hematuria mean?

If there is both proteinuria and hematuria, most of the time it is GN

- The filtration barrier which is composed of the epithelial, GBM, and endothelial layers, is affected.
- If the epithelial cell layer is affected, proteinuria is seen as in NS.
- If the GBM is affected, hematuria is seen as in PSGN.
- So, if both layers are affected to varying degrees, suspect GN.



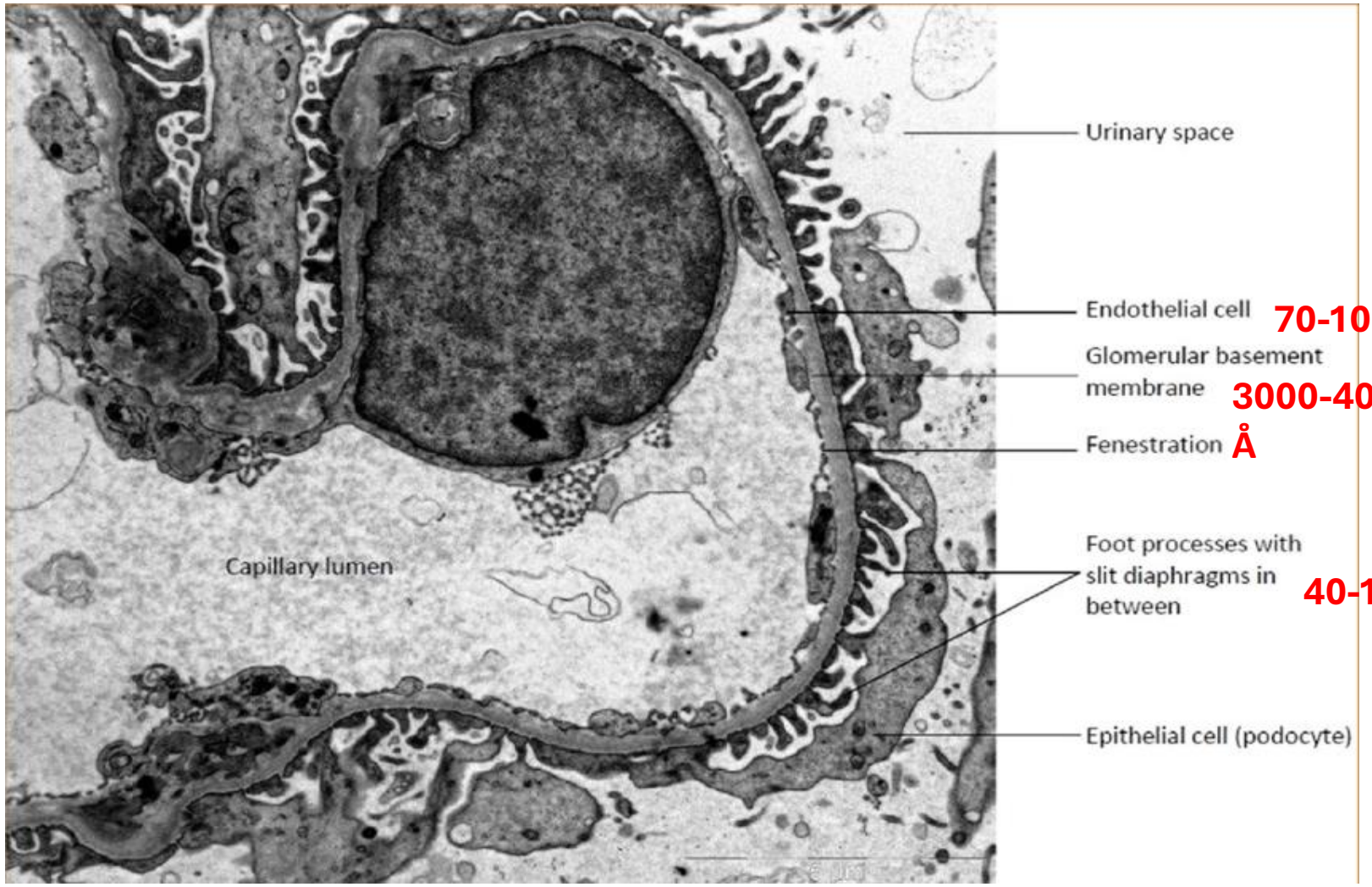
(SIZE BARRIER)

(CHARGE BARRIER)

If the **epithelial cell layer** is affected, **proteinuria** is seen as in Nephrotic Syndrome

If the **GBM is affected**, **hematuria** is seen as in **Post Strep Glomerulonephritis**

So, if both layers are affected to varying degrees, suspect GN.



The glomerular filtration barrier has size-selective properties and differentially handles molecules of varying size.

Small molecules up to the size of inulin, for example, which is at 10 to 14Angstroms filter freely, whereas large molecules such as plasma proteins are held back.

Case 2

Despite completing the prescribed antibiotics, the child's symptoms persist. The swelling worsens, and the frothy urine continues.

On follow-up, a repeat urinalysis reveals a USG of 1.005, negative for protein, RBC's and WBC's. The child is then referred to a pediatric nephrologist for further evaluation.

Case 2

Having inconsistent urinalysis findings, the nephrologist repeats the test. It now shows USG of 1.020, showing persistent 3+ proteinuria, 10-12 RBCs/HPF and sterile pyuria (10-15 WBCs per high power field).

What is the first thing you should check whenever you get a urinalysis result?

Check the urine specific gravity (USG).

If the urine is hypotonic, cell lysis can occur, leading to false-negative results, including on the protein dipstick test. In this case, excessive water intake diluted the urine, making the urinalysis appear deceptively normal.

How would you analyze the urinalysis results of this patient?

The dilute urine creates a false negative proteinuria and hematuria. Glucosuria with normal blood glucose means proximal tubule injury.

How would you approach the "puffy" face of the child? *Increase hydrostatic pressure.*

Remember the forces that govern filtration.

Anything that increases capillary filtration will lead to edema.

Either increase in hydrostatic pressure (P_{gc}) or decrease in oncotic pressure (π_{gc}).

$$GFR = Kf\,[(P_{gc} + \pi_{bs}) - (P_{bs} + \pi_{gc})]$$

GFR Formula

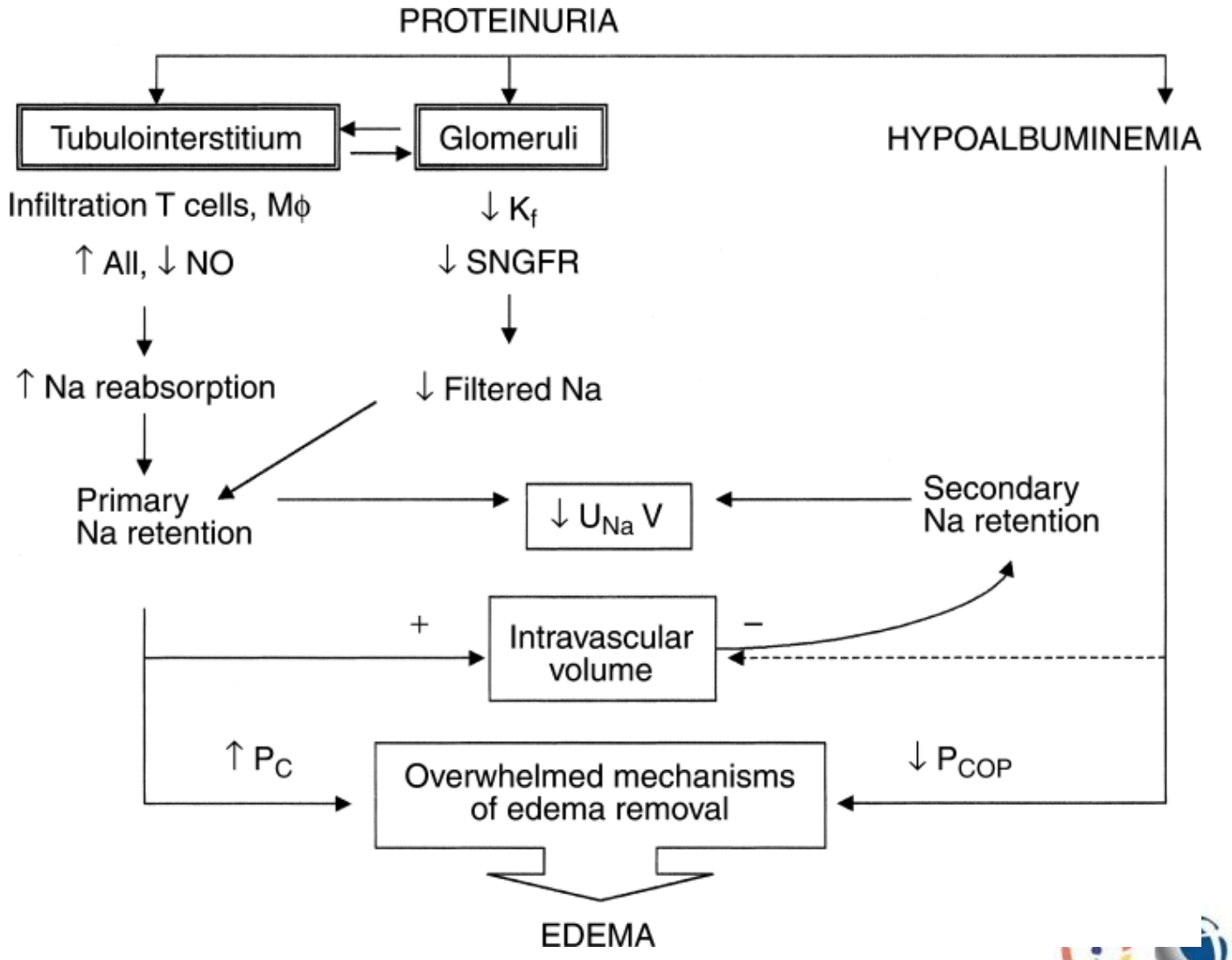
$$GFR = Kf\left[(P_{gc} + \pi_{bs}) - (P_{bs} + \pi_{gc})\right]$$

Favor

Oppose

- Where
  - Kf = glomerular filtration coefficient (product of glomerular surface times hydraulic conductivity)
  - P_{gc} = glomerular hydrostatic pressure
  - P_{bs} = hydrostatic pressure in Bowman's Space
  - Π_{gc} = glomerular capillary oncotic pressure
  - Π_{bs} = Bowman's Space oncotic pressure (negligible)

Renal Edema


PROTEINURIA
Tubulointerstitium
Glomeruli
HYPOALBUMINEMIA
Infiltration T cells, Mϕ
↑ AII, ↓ NO
↓ K_f
↓ SNGFR
↑ Na reabsorption
↓ Filtered Na
Primary
Na retention
↓ U_{Na} V
Secondary
Na retention
+
Intravascular
volume
−
↑ P_C
Overwhelmed mechanisms
of edema removal
↓ P_{COP}
EDEMA

Clinical Manifestation

ETIOLOGY	PRESENTATION
• Cardiac	➢Ascending edema (diseased heart pumps more especially at the end of the day)
• Hepatic	➢Ascites (liver failure cause splanchnic vasodilation)
• Nutritional	➢Generalized edema/ anasarca (from hypoalbuminemia)
• Renal	➢Periorbital/facial edema (see next slides)

Why periorbital edema?

- P_i of the periorbital space is > 0 mmHg.
- The **periorbital tissue** has low interstitial hydrostatic pressure, making it more prone to fluid accumulation.
- So any small increase in volume (i.e. sodium retention from kidney disease), leads to edema

http://totalpict.com/images/65/651546517502d55cab9ea7.gif

Periorbital edema

- The skin and subcutaneous tissue around the eyes are particularly thin and elastic, with loosely arranged connective tissue. This structural characteristic allows even small amounts of fluid to accumulate and become visibly prominent.
- Very distensible

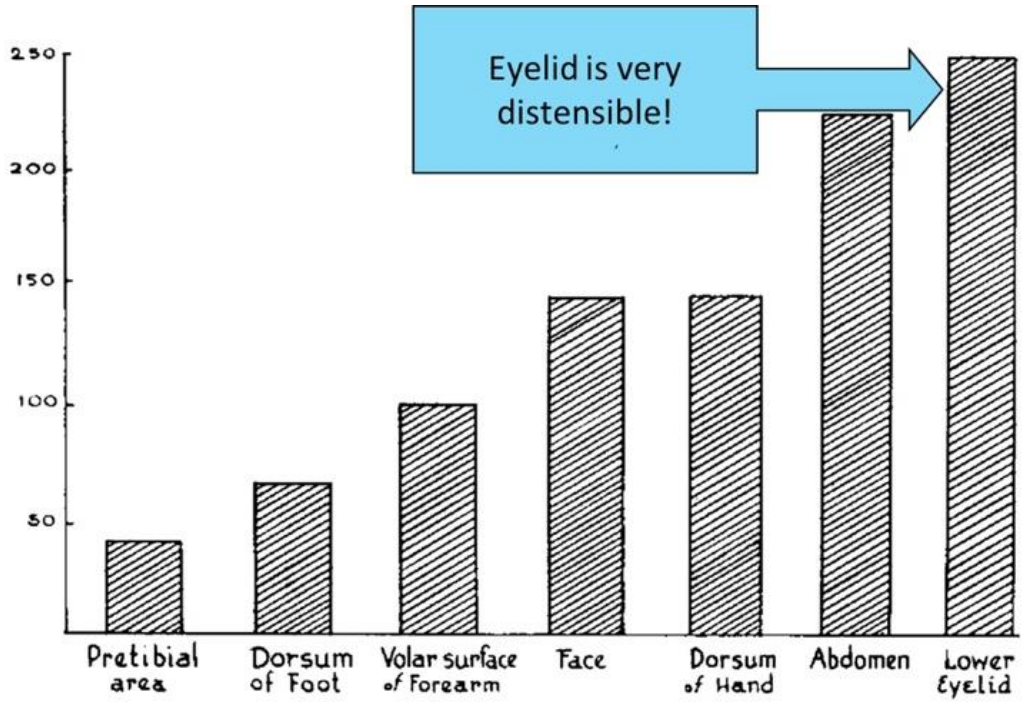
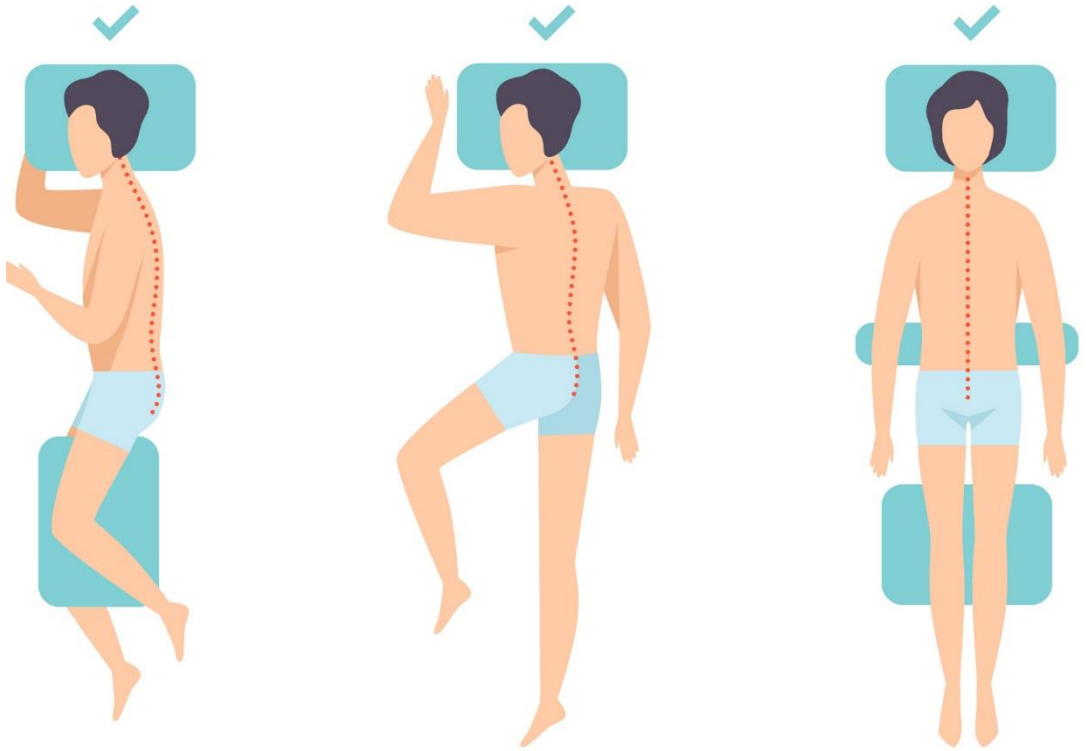


Fig. 4.—Comparison of the values for skin distensibility of seven areas of the body of normal subjects. The figure was constructed on the basis of the distensibility of the skin of the volar surface of the forearm, which was arbitrarily considered to be 100.

Burch, 1940

Periorbital edema

- The **periorbital region** is particularly susceptible to fluid pooling overnight, leading to noticeable edema upon waking.
- This is due to slow lymphatic flow during sleep and reduced influence of gravity.



Burch, 1940

Periorbital edema

- Persistent in renal edema
- Check for proteinuria (π_i).

How does proteinuria affect GFR?

- Proteinuria increases π_{bs} (Bowman's space oncotic pressure).
- This is usually negligible (equal to 0) normally.

$$GFR = Kf\,[(P_{gc} + \pi_{bs}) - (P_{bs} + \pi_{gc})]$$

Favor **Oppose**

How will you treat the edema then?

Knowing that the edema is due to increase hydrostatic pressure, treatment will include sodium limitation with or without water limitation; and diuretics.

Clinical Pearls:
Key Kidney Physiology Takeaways

1. **Hyponatremia = Water Problem** – Focus on fluid balance, not just sodium levels.
2. **In SIADH, prioritize fluid restriction**! -If IV fluids are necessary, ensure they are more concentrated than the urine osmolality.
3. **Concentrated Urine + Hyponatremia = ADH Disorder** – Consider ADH-related causes when you see this pattern.
4. **Check USG Before Interpreting** – Always assess urine specific gravity to avoid misinterpretation.
5. **Proteinuria + Hematuria = Glomerulonephritis** – Look out for signs of glomerular damage.
6. **Proteinuria = Filtration Problem** – Focus on kidney filtration when protein is present in urine.
7. **Hydrostatic Pressure in PSGN, Oncotic Pressure in Nephrotic Syndrome –** Know your pressures, know your edema.

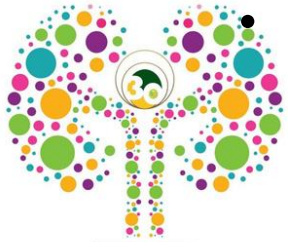
CASE 3

Case 3

- A 4-month-old male infant presents with a 3-day history of vomiting and watery diarrhea, along with lethargy, poor feeding, and no wet diapers for 8 hours.
- He was born full-term via normal delivery and is exclusively formula-fed.
- His physical examination reveals signs of dehydration, including lethargy, sunken eyes, dry mucous membranes, and a sunken fontanelle.
- Vital signs show mild tachycardia (HR 150 bpm), low-normal blood pressure (80/50 mmHg), and tachypnea (RR 40/min). The abdomen is soft with mild tenderness, and bowel sounds are hyperactive. There are no signs of edema, rash, or lymphadenopathy.

Case 3

- The resident on duty requests for a blood gas, serum and urine electrolytes.
- **Venous Blood Gas (VBG)**:
  - pH: 7.28
  - HCO_3^-: 15 mmol/L
  - pCO_2: 30 mmHg
- **Serum Electrolytes**:
  - Na^+: 134 mmol/L
  - K^+: 3.2 mmol/L
  - Cl^-: 110 mmol/L
  - HCO_3^-: 15 mmol/L
  - BUN: 25 mg/dL
  - Creatinine: 0.4 mg/dL



- **Urinalysis:**
  - Specific Gravity: 1.010
  - pH: 6.5
  - Protein: Negative
  - Glucose: Negative
  - Ketones: Trace
  - Microscopy: 0-2 WBCs/hpf, no casts.
- **Urine Electrolytes:**
  - Na^+: 60 mmol/L
  - K^+: 20 mmol/L
  - Cl^-: 70 mmol/L
- Fractional Excretion of Sodium (FENa): 1.8%

How would you approach the metabolic acidosis?

1. Compute for serum anion gap.
2. If normal, compute for urine anion gap.

Serum Anion Gap (AG)

Total Cations < Total Anions

$Na^+ + K^+ + UC < Cl^- + HCO_3^- +$ **UA**

$Na^+ + K^+ - (Cl^- + HCO_3^-) = UA - UC = AG$

$$Na^+ - (Cl^- + HCO_3^-) = AG$$

Normal Serum Anion Gap = 6-12 mmol/L

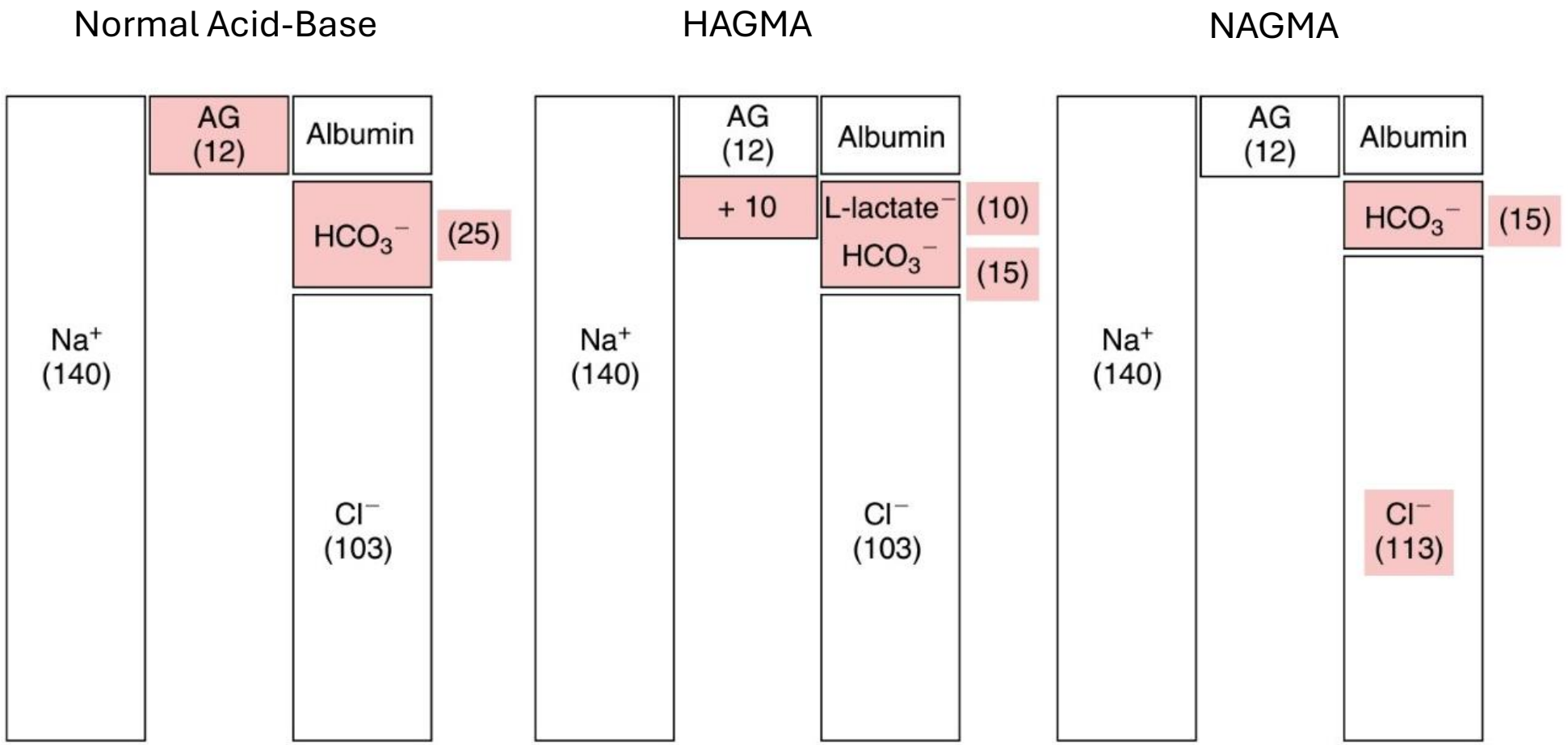
What is a serum anion gap?

- The anion gap is the measurement of the body's blood acid-base balance and the balance of electrolytes in the blood.
- It is the difference between measured cations (positively charged ions like Na+ and K+) and measured anions (negatively charged ions like Cl- and HCO3-)
- Normal serum anion gap is somewhere in the range of 6-12 mmol/L

When and why do we compute for it?

- Serum anion gap is usually used to identify the probable causes of metabolic acidosis in our patients.
- Metabolic acidosis may either be a normal anion gap metabolic acidosis (NAGMA) or high anion gap metabolic acidosis (HAGMA)

Serum Anion Gap (AG)



What is the difference between NAGMA and HAGMA?

- The human body is electrically neutral. Therefore, it does not have a true anion gap. However, there are only a limited ions that can be measured. Therefore, at face value, the subtraction between measured cations and measured anions will be around 6-12 mmol/L. In this slide, then difference between the measured cation (Na+) which is 140 and the measured anions (albumin, bicarbonate, and chloride) which is at 128 is 12.
- In clinical cases where there is an increased production of unmeasured acids (for example: albumin, lactate from lactic acidosis or ketones from ketoacidosis) like in diabetic ketoacidosis (DKA) or sepsis/shock (lactic acidosis), the anion gap is high. The normal response of the body to this increase in unmeasured acids is a decrease in the measured bicarbonate because it acts as a buffer and is therefore consumed. This then results in a high anion gap as seen in this slide. The bicarbonate is decreased to 15 because it acted as a buffer to the unmeasured L-lactate. So when you compute for the anion gap, sodium minus the total number of measured anion is 140 – 118 equals 22.
- On the other hand, in clinical cases where there is a loss in your bicarbonate through the GI tract (like in severe diarrhea, pancreatic fistula, or chronic laxative abuse) or kidneys (like in RTA or during prolonged use of carbonic anhydrase inhibitors [acetazolamide]), there is an extracellular shift of chloride resulting in hyperchloremia to maintain electroneutrality. This is why NAGMA is associated with hyperchloremia. As you can see in the previous slide, although the bicarbonate level decreased to 15, the anion gap is still 12 because the chloride level is increased to 113.

Anion Gap (AG)

High AG

- Acid involved: not only HCl
- HCO_3^- in exchange for phosphates, sulfates, organic acids
- Normochloremic
- “MUDPILES”
- **Management: removal**

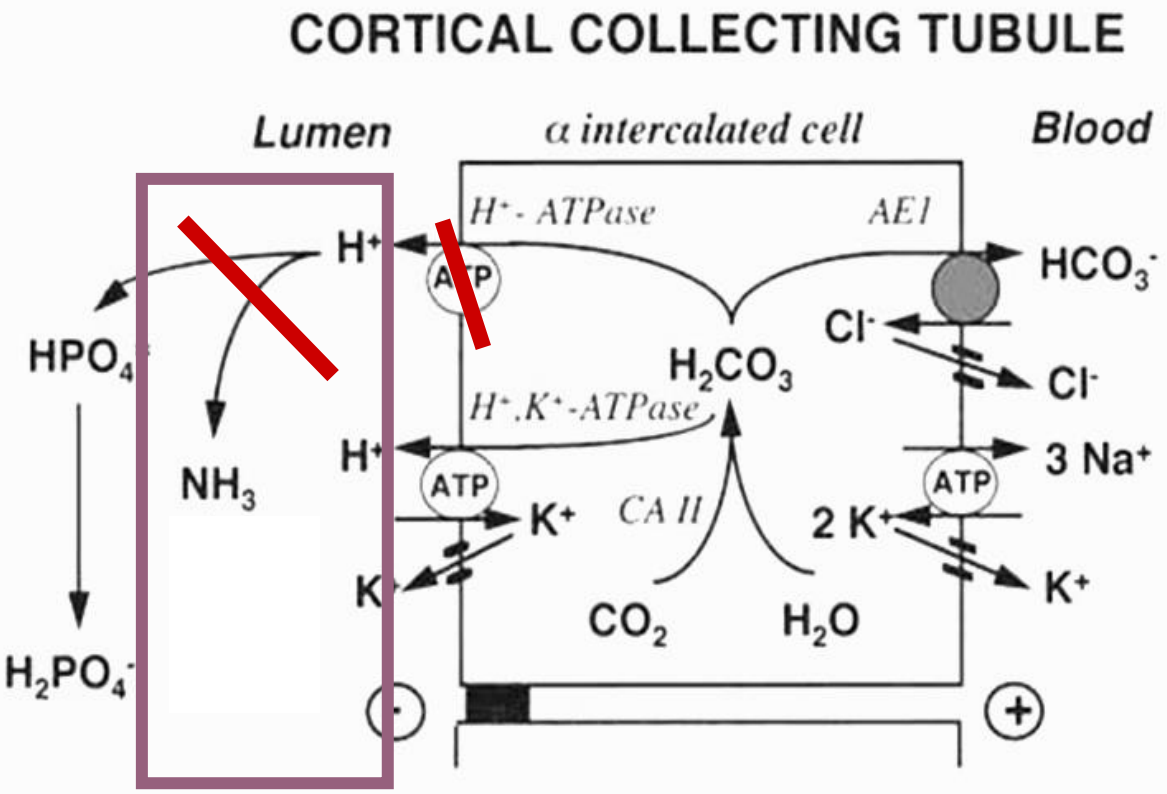
Normal AG

- Acid involved: HCl
- HCO_3^- in exchange for Cl^-
- Hyperchloremic
- “HARD UPS”
- **Management: bicarbonate therapy**

Urine Anion Gap

$$UAG = (U_{Na} + U_{K}) - U_{Cl}$$

- Surrogate measurement of urinary NH_4Cl excretion or distal acidification
- Recall that the proximal tubule reabsorbs bicarbonate while the distal tubule secretes hydrogen ions.
- Intact urinary NH_4Cl excretion:
  (-) UAG
- Decreased urinary NH_4Cl excretion :
  (+) UAG



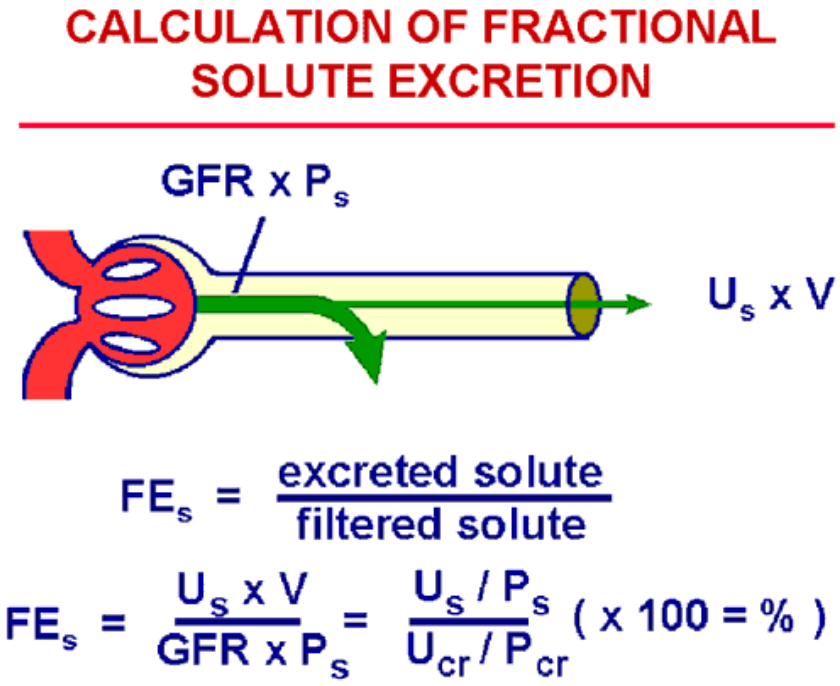
- In prerenal AKI where renal blood flow is decreased, the appropriate response is to reabsorb almost all of the filtered sodium to preserve the blood volume by decreasing the sodium and therefore fluid loss in the form of urine. Therefore, FENa will be less than 1%
- In intrinsic AKI, there is inappropriate sodium wasting due to tubular damage, FENa will be more likely greater than 2%.

What is the fractional excretion of sodium? How do you analyze that?

The **fractional excretion of sodium (FENa)** is a calculation used to assess the kidney's ability to conserve or excrete sodium. It helps differentiate between prerenal and intrinsic renal causes of acute kidney injury (AKI) by evaluating how much sodium is being excreted in the urine relative to the amount filtered by the kidneys.

Fractional Excretion of Sodium

*The **fractional excretion of sodium (FENa)** is a calculation used to assess the kidney's ability to conserve or excrete sodium. It helps differentiate between prerenal and intrinsic renal causes of acute kidney injury (AKI) by evaluating how much sodium is being excreted in the urine relative to the amount filtered by the kidneys.*



- FE_{Na} 1-2% (normal)
- FE_{Na} < 1%

  salt conservation in fluid loss; pre-kidney “success”
- FE_{Na} > 2%

  salt losing; intrinsic kidney injury

Limitations of using the FeNA

What do you think are limitations that can affect the accuracy and interpretation of the FeNa?

The fractional excretion of sodium (FENa) has several limitations that can affect its accuracy and interpretation

Limitations of using the FeNA

- **Dehydration and Volume Status**:
  FENa is less reliable in patients who are severely dehydrated or have fluctuating volume status. In these cases, the kidney may still be conserving sodium, but the FENa could be misleading due to extreme changes in serum or urine concentrations.

- **Diuretic Use**:
  Diuretics can significantly alter sodium and creatinine levels in both serum and urine. For example, diuretics may cause the kidneys to excrete sodium more freely, leading to falsely high FENa values, even in prerenal conditions. Conversely, diuretics can cause renal sodium retention, leading to falsely low FENa values in cases of intrinsic renal disease.

Limitations of using the FeNA

- **Chronic Kidney Disease (CKD)**:
  In patients with pre-existing kidney disease, the interpretation of FENa can be complicated. The kidneys may not respond to volume depletion as expected, and FENa may not accurately reflect the cause of acute kidney injury (AKI) due to impaired renal function.

- **Sepsis or Inflammatory States**:
  In sepsis or other inflammatory conditions, renal function may be altered, and sodium handling by the kidneys can be disrupted. This can lead to unpredictable FENa values, making it less reliable for determining the cause of AKI.

Limitations of using the FeNA

- **Urinary Creatinine Measurement**:
  FENa depends on accurate measurement of both urinary sodium and creatinine. Inaccurate urine collection or errors in laboratory analysis can lead to false calculations, affecting the interpretation of FENa

- **Age Considerations**:
  In pediatric populations, especially in infants and young children, kidney function and sodium handling may be immature, making FENa values less reliable or harder to interpret

- **Renal Obstruction**:
  FENa may be altered in patients with urinary tract obstruction. In this case, despite prerenal features, sodium retention may occur due to reduced renal perfusion, making FENa potentially misleading.

10 Clinical Pearls:
Key Kidney Physiology Takeaways

1. **Hyponatremia = Water Problem** – Focus on fluid balance, not just sodium levels.
2. **In SIADH, prioritize fluid restriction**! -If IV fluids are necessary, ensure they are more concentrated than the urine osmolality.
3. **Concentrated Urine + Hyponatremia = ADH Disorder** – Consider ADH-related causes when you see this pattern.
4. **Not All Pyuria Means UTI** – Don't jump to conclusions; further investigation is essential.
5. **Check USG Before Interpreting** – Always assess urine specific gravity to avoid misinterpretation.
6. **Proteinuria + Hematuria = Glomerulonephritis** – Look out for signs of glomerular damage.
7. **Proteinuria = Filtration Problem** – Focus on kidney filtration when protein is present in urine.
8. **Hydrostatic Pressure in PSGN, Oncotic Pressure in Nephrotic Syndrome** – Know your pressures, know your edema.
9. **Always Calculate Serum Anion Gap: Don't Forget Serum Chloride** – It guides your diagnosis and helps determine when to use bicarbonate.
10. **Urine Anion Gap = Distal Acidification: Remember Urine Chloride**– Use this test to assess for NAGMA.

Summary/ Conclusion/ Takeaways

- *As we wrap up, what's one concept from this session that truly stood out to you?*

10 Clinical Pearls:
Key Kidney Physiology Takeaways

1. **Hyponatremia = Water Problem** – Focus on fluid balance, not just sodium levels.
2. **In SIADH, prioritize fluid restriction**! -If IV fluids are necessary, ensure they are more concentrated than the urine osmolality.
3. **Concentrated Urine + Hyponatremia = ADH Disorder** – Consider ADH-related causes when you see this pattern.
4. **Not All Pyuria Means UTI** – Don’t jump to conclusions; further investigation is essential.
5. **Check USG Before Interpreting** – Always assess urine specific gravity to avoid misinterpretation.
6. **Proteinuria + Hematuria = Glomerulonephritis** – Look out for signs of glomerular damage.
7. **Proteinuria = Filtration Problem** – Focus on kidney filtration when protein is present in urine.
8. **Hydrostatic Pressure in PSGN, Oncotic Pressure in Nephrotic Syndrome** – Know your pressures, know your edema.
9. **Always Calculate Serum Anion Gap: Don’t Forget Serum Chloride** – It guides your diagnosis and helps determine when to use bicarbonate.
10. **Urine Anion Gap = Distal Acidification: Remember Urine Chloride**– Use this test to assess for NAGMA.