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Therapeutic Strategies for Interpreting Acid-Base Status with and Without Blood Gases

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DISCLOSURE

All planners, presenters, and reviewers of this session report no financial relationships relevant to this activity.

Learning Objectives

- Given a case scenario, determine acid-base status using blood gases and explain confounding factors to interpretation of the blood gases.
- Evaluate whether acetate should be added to parenteral nutrition in a given case scenario and determine the appropriate dose, if added, based on general guidelines.
- Discuss medications that are likely contributing to acid-base imbalance in a given case scenario.

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Therapeutic Strategies for Interpreting Acid-Base Status Blood Gases

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How often do you interpret ABGs in your current practice; how confident are you in interpreting ABGs?

A. None; I would like to learn more
B. Occasionally; I would like some more practice
C. Frequently; I'm pretty confident
D. All the time; I'm an expert

Outline

- ABGs in general
- One to practice, traditional
- Confounders type 1
- Same abg or different? As stewart
- Respiratory ABG?
- Confounders type 2?

Acid/Base Disorders

- Up to 90% of critically ill patients develop an acid/base disorder
  - May be self-limiting after management of the underlying condition
  - More commonly, they accompany complicated clinical conditions that obscure acid/base identification

- Correct identification is necessary to select the appropriate therapeutic intervention

Acid/Base Approaches

Co_2/HCO_3 (Boston)

- Entirely based on Henderson-Hasselbach equation
- Easy to use in stable patients

BE/deficit (Danish)

- BE is less influenced by changes in $PaCO_2$
- Allows better quantification of metabolic component

Anion gap

- Primary method for detecting unmeasured anions as the cause of metabolic acidosis
- Most critically ill patients have low albumin and phosphate, making AG less accurate

Stewart (Physio-chemical)

- Rejects Henderson-Hasselbach
- Independent variables are CO_2, SID, and total nonvolatile weak acids

Case: SZ

- SZ is a 46 y/o female admitted to MICU with sepsis
- Today is hospital day #3
- Acute renal failure, not yet on CRRT, not mechanically ventilated
- PMH: type 2 DM, hyperlipidemia, hypertension

Case: SZ

- SZ is currently receiving vancomycin, meropenem, NS, norepinephrine, and trophic enteral feeds.

- SZ's hemodynamic status has improved and the team anticipates discontinuation of pressors in the next 24 hours.

Case: SZ

	Today
Na	138
K	4.5
Cl	108
CO2	9
BUN	64
SCr	6.2
Glucose	209
Lactate	5.2
Albumin	2.1
WBC	16.7

	Today @ 0500
pH	7.142
pCO2	14.2
pO2	117
HCO3	4.9
BE	-24
O2SAT	97

How would you interpret SZ's ABG?

A. Metabolic acidosis
B. Respiratory acidosis
C. Metabolic acidosis with respiratory compensation
D. Respiratory acidosis with metabolic compensation

Arterial Blood Gases (ABGs)

- Laboratory assessment of acid-base status most frequently measured by blood gases

	pH	pCO_2	$P0_2$	HCO_3	Base Excess	$S0_2$
Arterial	**7.4 (7.35-7.45)**	**35-45**	**80-100**	**22-26**	**-2 to +2**	**95-100**
Venous	7.36 (7.33-7.43)	41-51	35-40	24-28	0 to +4	70-75

Acid/base –Traditional Approach

- Acid-base status is regulated by the
  - Lungs
  - Kidneys
  - Exogenous buffer system

Carbonic acid/bicarbonate buffer system

$$H_2O + CO_2 \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

Alkalosis

pH > 7.45 (> 7.4)

Respiratory alkalosis = ↓ PCO2

- Too much CO2 being removed from body (overbreathing)

Metabolic alkalosis = ↑ HCO3

- Too much base HCO30

pH < 7.35 (< 7.4)

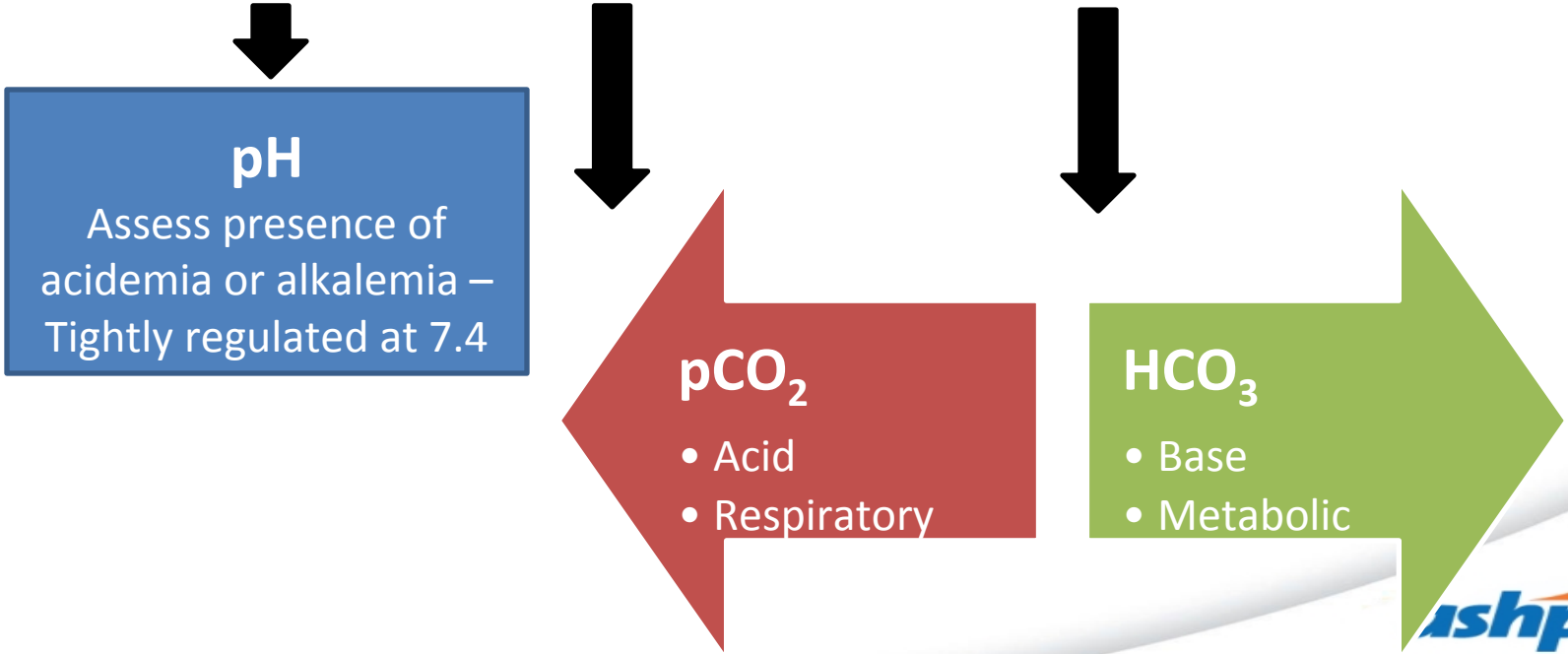
Respiratory acidosis = ↑ PCO2

- Lungs unable to remove CO2 from body

Metabolic acidosis = ↓ HCO3

- Consumption or loss of base (HCO3)

	pH	pCO_2	$P0_2$	HCO_3	Base Excess	$S0_2$
Arterial	7.4 (7.35-7.45)	35-45	80-100	22-26	-2 to +2	95-100



Interpreting ABGs – Traditional Approach

- 3 step approach:

1. Determine acidosis or alkalosis
2. Determine primary abnormality
3. Identify other abnormalities → compensation or mixed?

Interpret SZ's ABG

	Today @ 0500	Normal
pH	7.142	7.4
pCO2	14.2	35-45
pO2	117	
HCO3	4.9	22-26
BE	-24	
O2SAT	97	

How would you interpret SZ's ABG?

A. Metabolic acidosis
B. Respiratory acidosis
C. Metabolic acidosis with respiratory compensation
D. Respiratory acidosis with metabolic compensation

SZ's ABG

- 3 step approach:

1. Determine acidosis or alkalosis
   - pH 7.142 < 7.4, so **acidosis**
2. Determine primary abnormality
   - Is pCO_2 abnormal?
   - Would a low pCO_2 cause acidosis?
   - Is HCO_3 abnormal?
   - Would a low HCO_3 cause acidosis?
3. Identify other abnormalities → compensation or mixed?

SZ's ABG

- SZ has metabolic acidosis
  - The low CO2 would cause alkalosis, so the lungs are compensating for the primary disorder
- Compensated metabolic acidosis

- Remember the lungs (pCO_2) can compensate in minutes, but the kidneys (HCO_3) take days

Disorder	Compensatory response and equation
Metabolic acidosis	***Expected*** response is 1.2 ↓ for each 1 ↓ in HCO3 pCO2 = 40 – [1.2 x (24 – measured HCO3)]
Metabolic alkalosis	***Expected*** response is 0.7 ↑ for each 1 ↑ in HCO3 pCO2 = 40 + [0.7 x (measured HCO3 – 24)]
Respiratory acidosis	***Expected*** response is 1-3 ↑ for each 10 ↑ in pCO2 < 48 hours: pH ↓ = 0.08 x [(measured pCO2 – 40)/10] > 48 hours: pH ↓ = 0.03 x [(measured pCO2 – 40)/10]
Respiratory alkalosis	***Expected*** response is 2-4↓ for each 10 ↓ in pCO2 < 48 hours: pH ↑ = 0.08 x [(40 - measured pCO2)/10] > 48 hours: pH ↑ = 0.03 x [(40 - measured pCO2)/10]

Confounding Factors

- Not enough information from traditional approach
  - Evaluate whether the ABG matches what you would expect from the clinical picture
  - Mixed or underlying disorder

Stewart or Physiochemical Approach

- Controversial
- Rejects Henderson-Hasselbach
- HCO3 and H+ are dependent variables
  - Independent variables are CO_2, SID, and total nonvolatile weak acids
- Original equations too complicated to perform at bedside

- There are methods to incorporate this approach into bedside evaluation

Kaplan LJ,Frangos S. Critical Care 2005;9(2):198

Unifying Base Excess and Stewart Approaches

- One method involves calculating the albumin and lactate corrected anion gap
  - Then if elevated, the delta ratio; if normal, calculate the urinary anion gap
- Another method calculates the effect of the strong ion difference (SID) on the base excess
  - And the effect of weak acids on the base excess

Kishen R et al. International Journal of Nephrology and Renovascular Disease 2014:7 209–217.

Interpret ABG - DV

- DV, 56 y/o male, admitted to CVICU after cardiac arrest and ROSC, day #1
- Mechanically ventilated:

pH	7.122
pCO_2	75
pO_2	52
HCO_3	24.5
BE	-5
SO_2	73

How would you interpret DV's ABG?

A. Metabolic acidosis
B. Respiratory acidosis
C. Metabolic alkalosis
D. Respiratory alkalosis

ABG #2 - DV

- DV, 56 y/o male, admitted to CVICU after cardiac arrest and ROSC
- Mechanically ventilated:

		Normal
pH	7.122	7.4
pCO_2	75	35-45
pO_2	52	
HCO_3	24.5	22-26
BE	-5	
SO_2	73	

Confounding Factors

- Time when ABG measured
  - Respiratory vs metabolic time to compensation
  - Compared to past history or vent changes
- Nutrition/Medications

Respiratory Acid/Base Disorders and Time

- Respiratory changes to regulate acid/base status are rapid, in minutes to hours
  - Metabolic compensation takes 2-5 days to reach a new steady state
  - A respiratory disorder with a duration of less than 2-3 days is considered acute
- Review past ABGs, checking dates and times closely
  - When ventilator settings are being changed, there may be multiple ABGs per day

Respiratory Acid-Base Disorders

- Confounding factors
  - Respiratory acidosis may be caused by overfeeding
    - Consider obtaining RQ via indirect calorimetry or ventilator
    - May be caused by overfeeding, RQ > 1
      - RQ = VCO_2/VO_2
  - Salicylate toxicity may cause respiratory alkalosis (in addition to metabolic acidosis)
  - Caution needed if considering sodium bicarbonate to treat respiratory acidosis

Should buffers be used for respiratory acidosis?

TABLE 4

DIFFERENCES IN pHa, Pa_{CO_2} AND BASE EXCESS BEFORE AND AFTER ADMINISTRATION OF SODIUM BICARBONATE ($NaHCO_3$) AMONG PATIENTS WITH ALI

Case	pHa Pre-$NaHCO_3$	pHa Post-$NaHCO_3$	Pa_{CO_2} Pre-$NaHCO_3$ (*mm Hg*)	Pa_{CO_2} Post-$NaHCO_3$ (*mm Hg*)	Base Deficit Pre-$NaHCO_3$ (*mEq/L*)	Base Deficit Post-$NaHCO_3$ (*mEq/L*)	$NaHCO_3$ Dose (*mEq*)
1	7.17	7.10	58	64	−7.8	−11.0	50
2	7.23	7.18	93	108	11.6	12.5	50
3	7.14	7.08	33	48	−16.8	−15.6	200
5	7.27	7.05	38	40	−9.2	−18.7	115
9	7.27	7.09	46	52	−6.5	−14.4	50
10	7.17	7.11	50	59	−9.9	−10.9	30
Mean ± SD	7.21 ± 0.06	7.10 ± 0.04*	53 ± 19	62 ± 24*	−6.4 ± 9.5	−9.7 ± 11.3	82.5 ± 64.5

* $p < 0.05$ Wilcoxon signed rank test for comparison of pre- and post-$NaHCO_3$ values.

Carbonic acid/bicarbonate buffer system

$$H_2O + CO_2 \rightleftharpoons H_2CO_3 \rightleftharpoons H^+ + HCO_3^-$$

Kallet RH, et al. Am J Resp Crit Care. 2000;161: 1149-1153.

Sodium Bicarbonate to Treat Respiratory Acidosis

- Lack of evidence to suggest that sodium bicarbonate improves patient outcome in respiratory acidosis

- The ARDS Network trial allowed sodium bicarbonate infusions to maintain a pH greater than 7.30, but whether this had any effect on patient outcome is unclear

Gehlbach BK, Schmidt, GA. *Critical care*. 2004;8: 259-265.
Acute Respiratory Distress Syndrome Network,.N Engl J Med. 2000 May 4;342(18):1301-8.

Key Takeaways

- Key Takeaway #1: ABGs should always be interpreted in light of the clinical condition of the patient
- Key Takeaway #2: Simple acid/base disorders can be interpreted using a three step traditional approach
- Key Takeaway #3: Complex acid/base disorders require further investigation

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Therapeutic Strategies for Interpreting Acid-Base Status Without Blood Gases

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Are there times when ABG's would be helpful to you but they cannot be obtained?

A. YES

B. NO

Outline

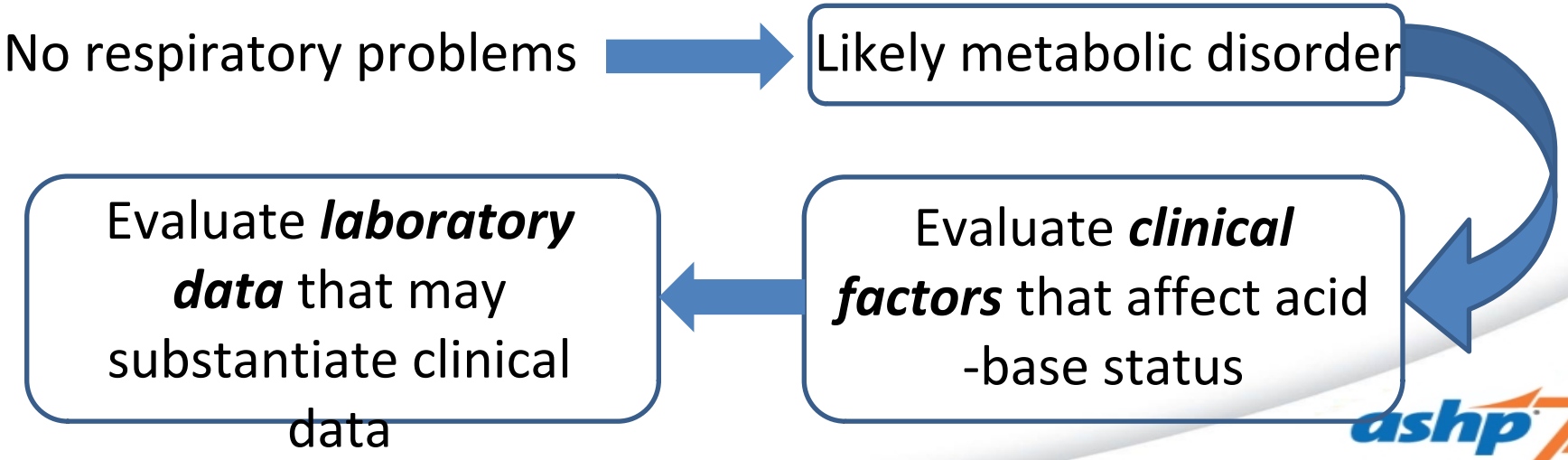
1. Algorithm for assessment
2. Cases
   a) Factors contributing to metabolic alkalosis
   b) Factors contributing to metabolic acidosis
   c) Physiology/anatomy considerations

Acid-Base Balance is Complex

- Blood gases give objective data
- Must make assumptions without blood gases
  - Less accurate than ABGs
  - Simplified view of the many factors contributing to acid-base status
  - Confounding factors may be missed

Algorithm for Acid-Base Assessment Without ABG's

Patient has respiratory problems → ABG's needed



Case 1

- 66 yo M admitted to the hospital with c/o abdominal pain, nausea, vomiting starting 12 hr PTA
- Diagnosed with SBO in ER
- Conservative therapy: fluids and NG suction
  - D5%/0.45% NaCl + 20 mEq KCl/L at 200 mL/h (60 mL/kg/d)
  - NG output of 4 L immediately in ER, then 2.5 – 3 L daily
- Day 6: Persistent SBO on imaging

Case 1: Labs

	Admit	Day 3	**Day 6**
Na^+	144	140	**145**
K^+	4.2	3.9	**3.3**
Chloride	107	105	**91**
Bicarb (CO_2)	23	26	**33**
Glucose	98	114	**99**
BUN	18	10	**22**
Creatinine	1.1	0.9	**0.7**

What is your assessment on day 6 based on the history and labs shown?

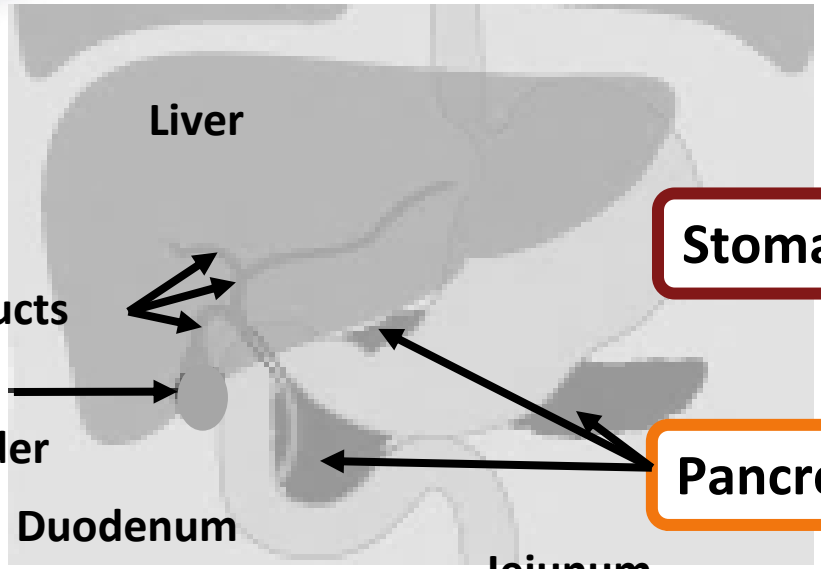
A. Labs are “contaminated” by IV fluids
B. Fluids at 60 mL/kg/d caused hypervolemia
C. Metabolic acidosis from Cl^- loss
D. Metabolic alkalosis from H^+ loss

Case 1: Interpreting Labs

	Day 6	
Na^+	145	High relative to Cl^-
K^+	3.3	Low
Chloride	91	Low
Bicarb (CO_2)	33	High
BUN	22	
Creatinine	0.7	

Lab interpretation MUST be supported by CLINICAL EVIDENCE

GI Physiology

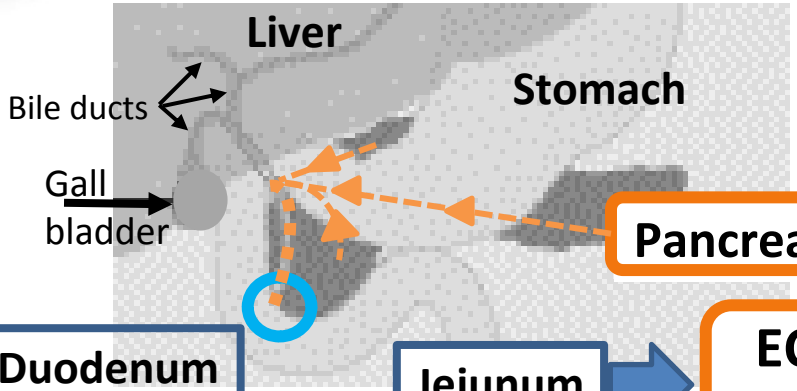


Stomach → Produces Hydrochloric Acid

Pancreas → Produces Bicarbonate

http://www.cancerresearchuk.org/about-cancer/type/gallbladder-cancer/treatment/the-stages-of-gallbladder-cancer

GI Physiology



Duodenum

Bicarbonate enters duodenum at common bile duct

Jejunum → EC Fistula, End Jejunostomy

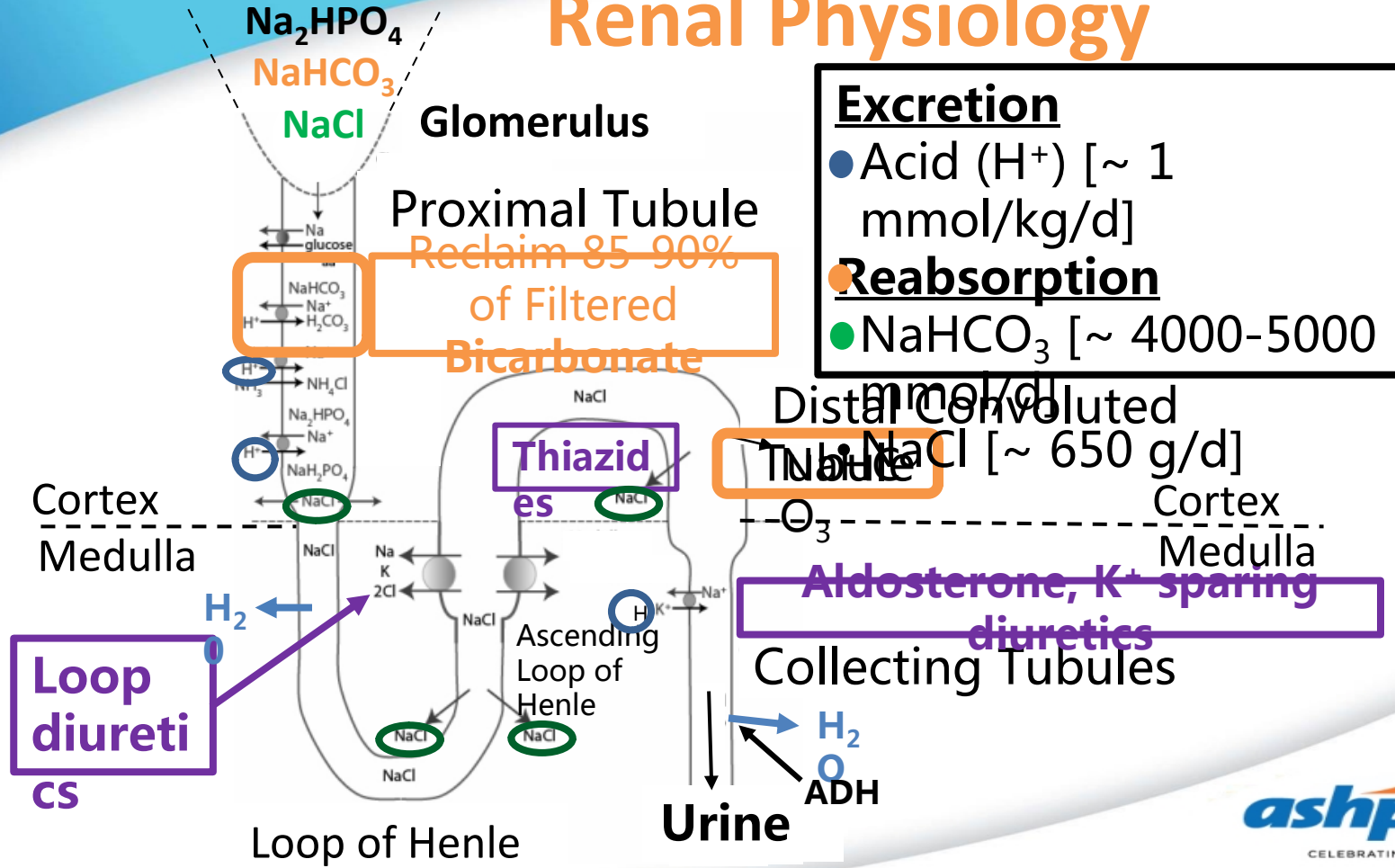
Ileum → EC Fistula, Ileostomy

Colon → Diarrhea

Bicarb Losses

http://www.cancerresearchuk.org

Renal Physiology



Excretion

- Acid (H^+) [~ 1 mmol/kg/d]

Reabsorption

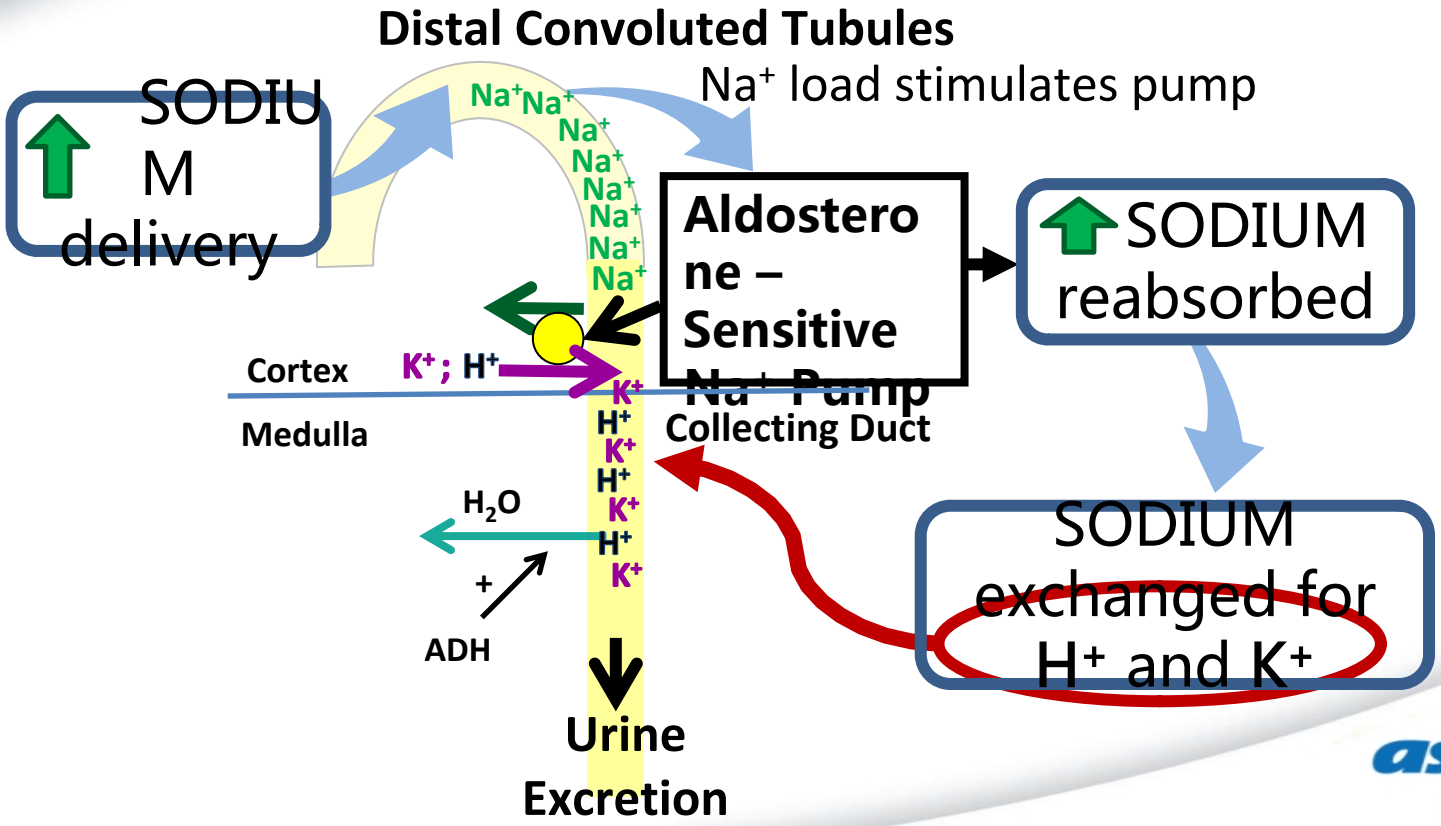
- $NaHCO_3$ [~ 4000-5000 mmol/d]
- NaCl [~ 650 g/d]

Medications Frequently Associated with Acid-Base Disturbance

Metabolic Acidosis	Mechanism
Spironolactone, Amiloride, Triampterene	Inhibit Na^+ reabsorption → hyperchloremia, normal AG
Metabolic Alkalosis	
Loop diuretics	Cl^- depletion, Na^+ to DCT
Penicillin	Cl^- depletion, Nonreabsorbable anion
Aminoglycosides	Cl^- depletion, stimulates calcium-sensing receptor

Kitterer D, et al. Drug-induced acid-base disorders. Pediatr Nephrol. 2015; 30(9):1407-23.

Renal Physiology



Metabolic Alkalosis

Lose Acid

- Gastric acid loss
- Renal H^+ loss
- Extracellular H^+ loss (intracellular shift)
- Decreased acid production (PPI, H_2RA)

Gain Bicarb

- Exogenous bicarb
- Bicarb precursor: Lactate, acetate, citrate
- Medications: Loop or thiazide diuretic, glucocorticoids, fludrocortisone, antacids

Metabolic Acidosis

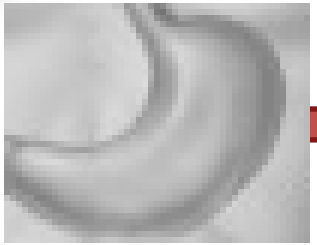
Gain Acid

- Reduce gastric acid loss
- Reduce renal H^+ loss (renal dysfunction)
- Increase production – infection, DKA

Lose Bicarb

- GI tract losses – enterocutaneous fistula, jejunostomy, ileostomy, diarrhea
- Stop/reduce administration of exogenous bicarb or bicarb precursor (lactate, acetate, citrate)

Case 1: Factors Affecting Acid-Base Status

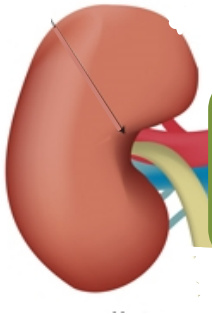


Hydrochloric Acid

Vomiting or NG suction

Acid (H^+) Loss

May have an affect:



↑ Na^+ to DCT

Aldosterone-sensitive Na+ pump stimulated

↑ Na^+ re-absorption

Exchange Na^+ for K^+ and H^+

Case 1: Factors Affecting Acid-Base Status

- Hypokalemia – when intracellular K^+ is low, more HCO_3 is reabsorbed in the kidney
- Chloride deficiency – HCO_3 is reabsorbed with Na^+ rather than chloride to maintain electroneutrality
- Volume depletion – Na^+ is retained, which increases HCO_3 reabsorption (contraction alkalosis)

What is your assessment on day 6 based on the history and labs shown?

A. Labs are “contaminated” by IV fluids
B. Fluids at 60 mL/kg/d caused hypervolemia
C. Metabolic acidosis from Cl^- loss
D. Metabolic alkalosis from H^+ loss

Metabolic Alkalosis Management

- What fluid issues should be considered?
  - Avoid bicarb, bicarb precursors
  - Lactate in LR, Acetate in PN, Citrate in CRRT
  - Adequate volume to prevent “contraction alkalosis”
- Any other therapies that impact alkalosis?
  - Adequate potassium to prevent hypokalemia
  - Reduce acid production in stomach (???)
    - PPI, H_2RA may reduce gastric acid removal with NG suction

Case 2

- 72 y.o. patient s/p colostomy after admission 12 days ago
- In ICU x 4 days; tube feeding advanced to goal on day 3
- Back to OR for end ileostomy hospital day 7
- Transferred from ICU to floor HD 9
- Ileostomy output HD 9 to 12: 1 → 1.7 → 3.6 → 4.7 liters/day
- Temperature to 38.4^{o} C last night, on broad spectrum abx

Case 2: Labs

	Day 7	Day 10	Day 12
Na+	139	135	**138**
K+	3.9	3.7	**5.5**
Chloride	106	108	**114**
Bicarb (CO_2)	25	20	**14**
Glucose	100	118	**136**
BUN	16	17	**32**
Creatinine	1	0.9	**1.5**

Case 2: Interpreting Labs

	Day 12	
Na^+	138	Low relative to Cl^-
K^+	5.5	High normal
Chloride	114	High
Bicarb (CO_2)	14	Low
BUN	32	
Creatinine	1.5	

Lab interpretation MUST be supported by CLINICAL EVIDENCE

What is your assessment on day 12 based on the history and labs shown?

A. Renal failure has resulted in hypervolemia
B. Increased ileostomy output is due to tube feeding
C. Metabolic acidosis
D. Metabolic alkalosis

Factors Affecting Acid-Base Status

GI tract losses or outputs

- Gastric losses? No
- Post-pancreas losses? Yes – Ileostomy with high output

Potassium reflects H^+ intra- and extra-cellular shifts

Gains or retention

- Renal H^+ gain? Yes - Renal impairment/failure
- Other acids? Possible
  - Lactic acid with sepsis
  - Organic acids (kidney)

What is your assessment on day 12 based on the history and labs shown?

A. Renal failure has resulted in hypervolemia
B. Increased ileostomy output is due to tube feeding
C. Metabolic acidosis
D. Metabolic alkalosis

Metabolic Acidosis Management

- Reduce chloride provision
  - Fluids with lower NaCl content
  - Use bicarb precursors: lactate (LR), acetate, citrate
- Removal of organic acids
  - Renal replacement therapy (CRRT, HD)
  - Treatment that improves renal function

Metabolic Acidosis Management

How much bicarb precursor is needed ?

- General rule: 1 mEq/kg/day of acetate for effect
  - If not excreting "usual" 1 mmol H^+/kg/day, must neutralize this
- Calculate bicarbonate deficit (mEq HCO_3)
  - Deficit = HCO_3 Vd x (desired HCO_3 – measured HCO_3)
    * HCO_3 Vd = (0.4 + 2.6/measured HCO_3) - ideal body wt
  - Deficit = 0.6 x wt (kg) x (desired HCO_3 - measured HCO_3)
  - HCO_3 end-point goal is typically 10-15

http://www.medscape.com/viewarticle/808418;
http://emedicine.medscape.com/article/242975-overview

Case 3

- 50 y.o. readmitted to the hospital for the 6th time in 5 months
- Tmax 39^{o} C, tachycardia, mild hypotension; abdominal pain and distention
- Home PN x 1.5 years after complications of vascular surgery; usual jejunostomy output of 3 – 4 Liters/day
- Hx multiple abdominal surgeries leading to EC fistula and end jejunostomy; multiple septic episodes/CRBSI
- Hx poor diet and medication adherence

Case 3

What type of acid-base problem is expected given the patient history?

A. Mixed acid-base d/o
B. Metabolic acidosis
C. Metabolic alkalosis
D. No acid-base d/o

Do labs support this?

Lab Test	3 days PTA	HD #1
Na^+	133	130
K^+	3.9	3.3
Chloride	103	112
Bicarb (CO_2)	20	17
Glucose	86	115
BUN	15	14

Case 3: Hospital Day #1

- Antibiotics continued from start in ED
  - Rapid improvement in HR, BP, temp by transfer to floor
- MIV D5%-0.45%NaCl + 20 mEq KCl/L @ 125 mL/hr
- Hold PN
- Abdominal CT: large abscess, dilated loops small bowel, no free air

Case 3: Hospital Day #2

- Blood cx: no growth
- Restart PN
  - Continuous infusion
- Stop MIV: D5%-0.45%NaCl + 20 mEq KCl/L @ 125 mL/hr

HPN Content

	Home PN			
	mEq/D	mEq/L	mEq/kg	
NaCl	230	77	4.4	
NaAcetate	240	80	4.6	5.4
K-Acetate	40	20	0.8	2.1
KCl	68	33	1.3	
Volume	3 L		58 mL/kg	

Case 3: Hospital Day #3

- Continued clinical response to antibiotics started in ED
- Abdomen more distended on am rounds
- Emesis x 8 starting mid afternoon → NG refused
- Fistula + jejunostomy output only 200 mL after noon
  - Was 1.5 – 1.8 L/day while npo in hospital
  - Usual output at home 3 – 4 liters/day
- Abdominal imaging indicates SBO
  - Likely transition point near Ligament of Trietz

Case 3: Hospital Day #4

What factors contributed to the rise in HCO_3 ?

- Acid loss: Emesis (x 8)
- Bicarb retention: 200 mL jejunostomy output
- Administration of acetate in PN

	3 days PTA	HD#1	HD#4
Na^+	133	130	**141**
K^+	3.9	3.4	**4.9**
Chloride	103	112	**107**
Bicarb (CO_2)	20	16	**29**
Glucose	86	115	**112**
BUN	15	14	**18**
Creatinine	1	0.9	**1**

Case 3: Hospital Day #5

- PN electrolyte content adjusted on HD #4 based on labs
- Patient admitted to ICU after emergent surgery
- Intubated → ABGs available to assess acid-base status

Key Takeaways

Key Takeaway #1: Acid-base status can be “simplistically” assessed using the clinical situation plus routine lab result when the patient’s respiratory status is not compromised

Key Takeaway #2: Sodium and chloride generally “move together” when fluid is responsible and move in opposite directions when a metabolic acid-base disturbance occurs

Key Takeaway #3: GI losses and renal function are keys to assessing metabolic acid-base status without ABGs

Key Takeaway #4: Things change, sometimes rapidly

Self-Assessment Questions: What is the Expected Effect on Acid-Base Status?

1. Patient with gastric fistula due to cancer. Output has been averaging 4 liters per day for over a week.

   A. Acidosis B. Alkalosis C. Need ABGs

2. The patient has taken only 2 liter 0.9% NaCl daily for the past 4 days due to increased edema.

   A. Worsen current acid-base disturbance
   B. Normalize acid-base balance
   C. Shift to the opposite acid-base status

Self-Assessment Question:
What is the Expected Effect on Acid-Base Status?

3. Patient with gastric fistula due to cancer. Continuing 2 L 0.9% NaCl daily, increased oral fluids to maintain hydration. Stopped H_2RA in PN a week ago.

   A. Worsen current acid-base disturbance

   B. Normalize acid-base balance

   C. Shift to the opposite acid-base status

   D. Need ABGs to assess acid-base status

Self-Assesment Questions: What is the Expected Effect on Acid-Base Status? The Preferred Therapeutic Option?

4. Patient with increased (> 3 L/d) jejunostomy output.

   A. Acidosis B. Alkalosis C. Need ABGs

5. Which “stock” fluid is most likely to help mitigate acid-base disruption in this patient?

   A. 0.9% NaCl

   B. D5W-0.45%NaCl

   C. Lactated Ringers

   D. All are equal if given in adequate volume

Self-Assesment Question:
What is the Preferred Therapeutic Option?

6. Patient with increased (> 3 L/d) jejunostomy output. PN to start. Wt 60 kg. Using the general guideline, how many mEq ACETATE would be added to PN?

   A. 154

   B. 77

   C. 60

   D. 30

Labs Results Today	
Na+	**135**
Chloride	**112**
Bicarb (CO_2)	**20**
Creatinine	**0.9**

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Self-Assessment Question:
What is the Expected Effect on Acid-Base Status?

7. A patient with large NG output for several days is most likely to develop what type of acid-base disorder?

   A. Metabolic acidosis
   B. Metabolic alkalosis
   C. No acid-base disorder
   D. Mixed acid-base disorder

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Therapeutic Strategies for Interpreting Acid-Base Status with and Without Blood Gases

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