Let's Talk About Gas:
ABG Interpretation Made Easy

Andrew Walker, PSM, MS, PA-C
Physician Assistant
Instructor of Medicine
Critical Care/Internal Medicine
Mayo Clinic Arizona

Stephanie Jalaba, MMS, PA-C
Physician Assistant
Assistant Professor, Department of PA
Medicine, Michigan State University
College of Osteopathic Medicine

Disclosures

- None.

Objectives

At the end of this presentation, the learner should be able to:

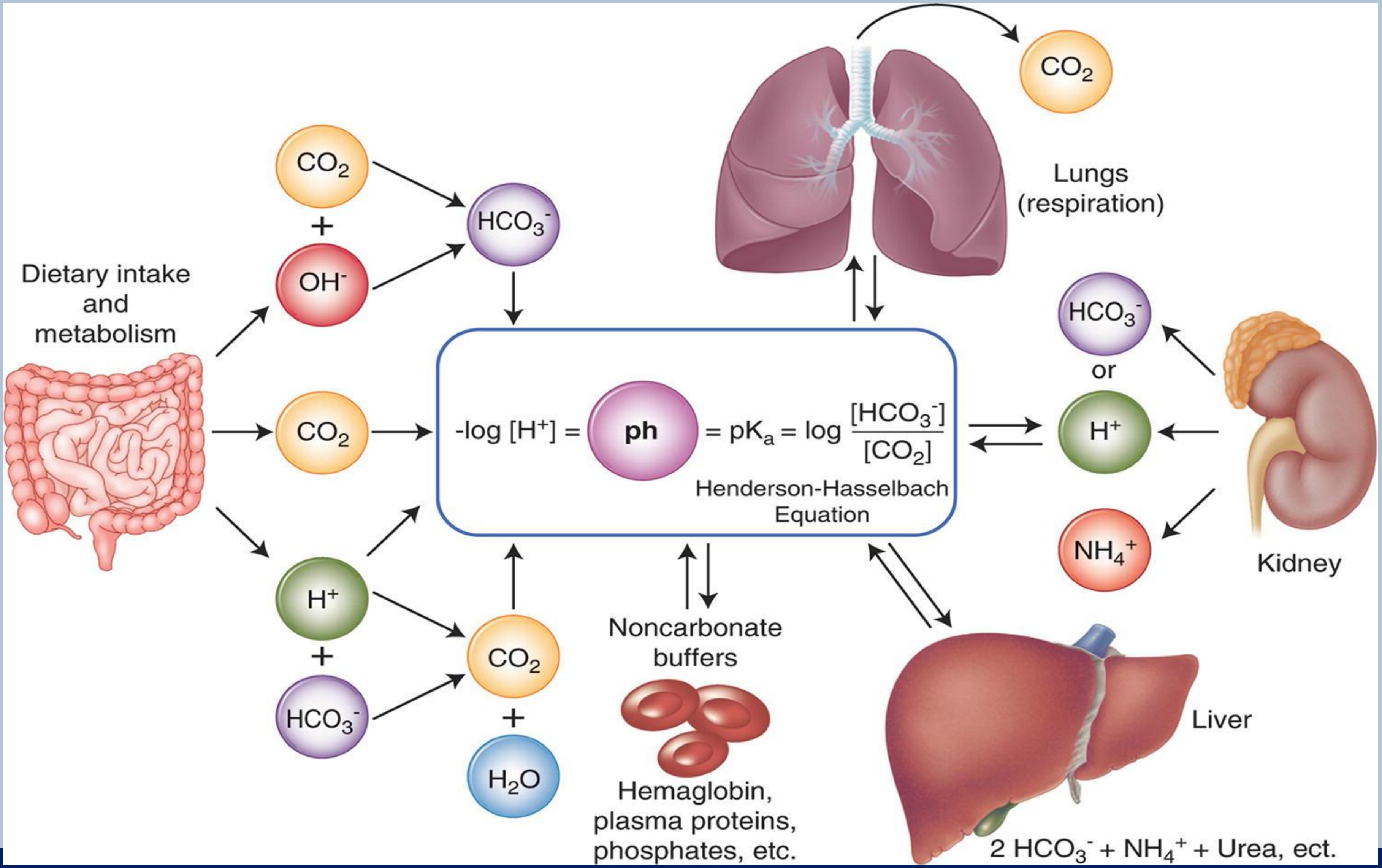
1. Analyze an ABG to determine the primary acid-base disorder.
2. Recognize compensation and mixed acid-base disorders.
3. Formulate differential diagnoses for the various acid-base disorders.
4. Apply ABGs to clinical scenarios and decision making.

Henderson-Hasselbalch Equation

$$pH = pK_a + \log_{10} \frac{\overset{\text{BASE}}{[A^-]}}{\underset{\text{ACID}}{[HA]}}$$

- The ratio of base-acid must stay relatively constant for the pH to stay constant
  - Because this is a logarithmic equation, it takes a fairly large change in acid or base to change the pH

Acid-Base Physiology



Components of the ABG

Component	Measure
pH	Acid-base balance
PaO_2	Partial pressure of oxygen
$PaCO_2$	State of alveolar ventilation
HCO_3^-	Reflects metabolic component of blood
Alveolar-arterial (A-a) gradient	Gradient between alveolar and arterial oxygen
Base excess*	The amount of acid or base it would take to return the pH back to 7.4

Indications for ABG

- Identify respiratory, metabolic, and mixed acid-base disorders
- Monitoring acid-base status in disorders such as DKA
- Quantification of oxyhemoglobin and oxygen carrying capacity of the patient
- Quantification of levels of dyshemoglobins (methemoglobin, carboxyhemoglobin)

Indications for ABG

- Measuring partial pressures of respiratory gases involved in ventilation and perfusion, e.g.…
  - COPD exacerbation
  - Asthma exacerbation
  - Pulmonary embolism
  - Pulmonary fibrosis
  - Pneumothorax
  - In these cases, can measure severity and progression of disease/exacerbation
- Assessment of response to mechanical ventilation

Some Clinical Scenarios Where an ABG is Useful in Acute Care

- Respiratory distress
- Hypoxia
- Airway obstruction
- Sepsis or shock
- DKA
- Renal failure
- Drug overdose or intoxication
- AMS or obtundation
- Monitoring response to invasive and non-invasive ventilation
- Code blue

Contraindications to ABG

- Local infection
- Distorted anatomy
- Abnormal Allen test
- AV fistula
- Severe PVD
- Relative contraindications:
  - Anticoagulation, tPA, severe coagulopathy

- Consider a-line if repeat ABGs will be necessary!

Before You Get an ABG

- Ask yourself…
  - What am I looking for/expecting?
  - Will this help guide or change my management?
    - How?
  - Will comparison be helpful after treatment/intervention?
    - Baseline ABG?

ABG Normal Values

Component	Low Normal	Normal Range	Estimated Normal	High Normal
pH	Acidosis	**7.35 – 7.45**	**7.4**	Alkalosis
PaO_2	Hypoxemia	**80 – 100 mmHg**	**100 – (0.3 x age)**	Hyperoxia
PCO_2	Respiratory alkalosis	**35 – 45 mmHg**	**40 mmHg**	Respiratory acidosis
HCO_3	Metabolic acidosis	**22 – 26 mEq/L**	**24 mEq/L**	Metabolic alkalosis
A-a gradient		< 10 mmHg	< 10 mmHg	
Base excess		-3 to +3 mEq/L		

ABG vs. VBG

- **Venous blood gases (VBG)** are widely used in the emergency setting
  - There is no data to confirm that this level of agreement is maintained in shock states or mixed acid-base disturbances

- **Why get a VBG over an ABG?**

How do ABG Values Compare to VBG

ABG	VBG
pH	pH + 0.035 units
pCO_2	pCO_2 + 5.7 mmHg *correlation dissociates in hypercapnia and shock
HCO_3	HCO_3 – 1.41 mmol/L
Base excess (BE)	BE + 0.089 mmol/L
Lactate	Does NOT correlate > 2mM
pO_2	Does NOT correlate (venous vs. arterial sample)

Clinical Application

Finally…something that makes sense!

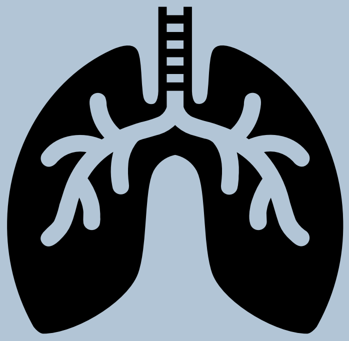
Why Do We Care?

- What do acidosis and alkalosis look like?
- What do they imply?
- How dangerous are they?
- What disturbances can they cause in the body?

Respiratory Acidosis

"I can't catch my breath!"

- Rapid, shallow breaths OR bradypnea
- Dyspnea
- Headache
- Disorientation
- Dizziness
- Drowsiness



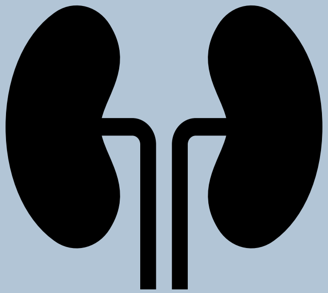
↓pH ↑pCO_2

- Hypoxia
- Hyperkalemia
- Dysrhythmias
- Hyperreflexia
- Muscle Weakness

<u>Retention of CO_2 by the lungs</u>

Metabolic Acidosis

- Confusion
- Drowsiness
- Headache
- Nausea, Vomiting
- Diarrhea



- Decreased BP
- Hyperkalemia
- Vasodilation
- Kussmaul respirations

↓pH ↓ HCO_3^-

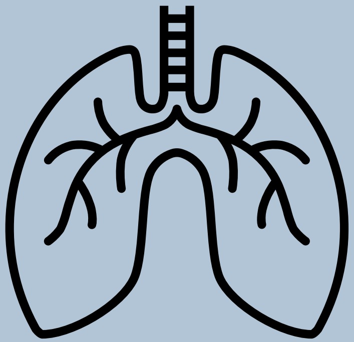
<u>Decreased ability of kidneys to excrete acid</u>

Clinical Consequences of Acidemia

Cardiovascular	• Impaired cardiac contractility • Decreased CO • Decreased systemic BP • Increased PVR • Decreased threshold for arrhythmia
Respiratory	• Hyperventilation (compensatory) with possible muscle fatigue
Neurologic	• Obtundation/coma
Metabolic	• Insulin resistance • Loss of bone and muscle • Protein degradation • Abnormalities in the release of many hormones • Hyperkalemia • Inhibition of anaerobic glycolysis • Reduction in ATP synthesis

Respiratory Alkalosis

- Rapid, deep breathing
- Lethargy
- Confusion
- Nausea, vomiting
- Numbness and tingling



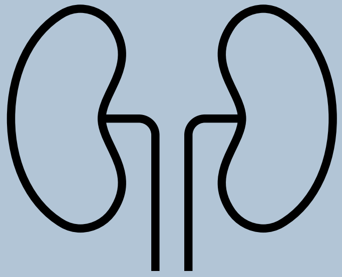
- Light headedness
- Seizure
- Tachycardia
- Hyperventilation
- Hyperkalemia

↑pH ↓pCO_2

Loss of CO_2 from the lungs

Metabolic Alkalosis

- Restlessness → lethargy
- Confusion
- Dizziness
- Irritability
- Nausea, vomiting
- Diarrhea



↑pH ↑HCO_3^-

- Tremors
- Muscle cramping
- Tingling in fingers/toes
- Hypokalemia
- Tachycardia

<u>A decrease in acid or an increase in base</u>

Clinical Consequences of Alkalemia

Cardiovascular	• Reduced coronary blood flow • Reduced threshold for angina • Decreased threshold for arrhythmia
Respiratory	• Hypoventilation (compensatory) with hypercapnia and hypoxemia
Neurologic	• Seizure • Tetany • Lethargy/delirium/stupor
Metabolic	• Hypokalemia • Hypomagnesemia • Hypophosphatemia • Stimulation of anaerobic glycolysis • Decreased oxyhemoglobin dissociation

Acid-base Differentials

Causes of Respiratory Acidosis

- ↓Respiratory stimuli
  - Drug overdose
  - Anesthesia
  - Intracranial issues (TBI, Stroke, SAH, etc.)
- Spinal or peripheral nerve issues (C-spine injury, myasthenia gravis, ALS, peripheral neuropathy, etc.)
- Pulmonary issues (ARDS, asthma, pneumonia, COPD, massive PE/Shock, atelectasis)
- Extrathoracic/abdominal distention (obesity, compartment syndrome)

Causes of Metabolic Acidosis

<u>MUDPILES</u>

- **M**ethanol
- **U**remia
- **D**KA
- **P**ropylene glycol (Ativan, Dilantin)
- **I**soniazid/Iron
- **L**actate
- **E**thanol/Ethylene glycol
- **S**alicylates / Seizures / Starvation

<u>USEDCRAP</u>

- **U**reteroenterostomy
- **S**mall bowel fistula
- **E**xcess chloride*
- **D**iarrhea
- **C**arbonic Anhydrase Inhibitors
- **R**enal Tubular Acidosis (RTA)
- **A**ddison's disease / Acetazolamide
- **P**ancreatoenterostomy

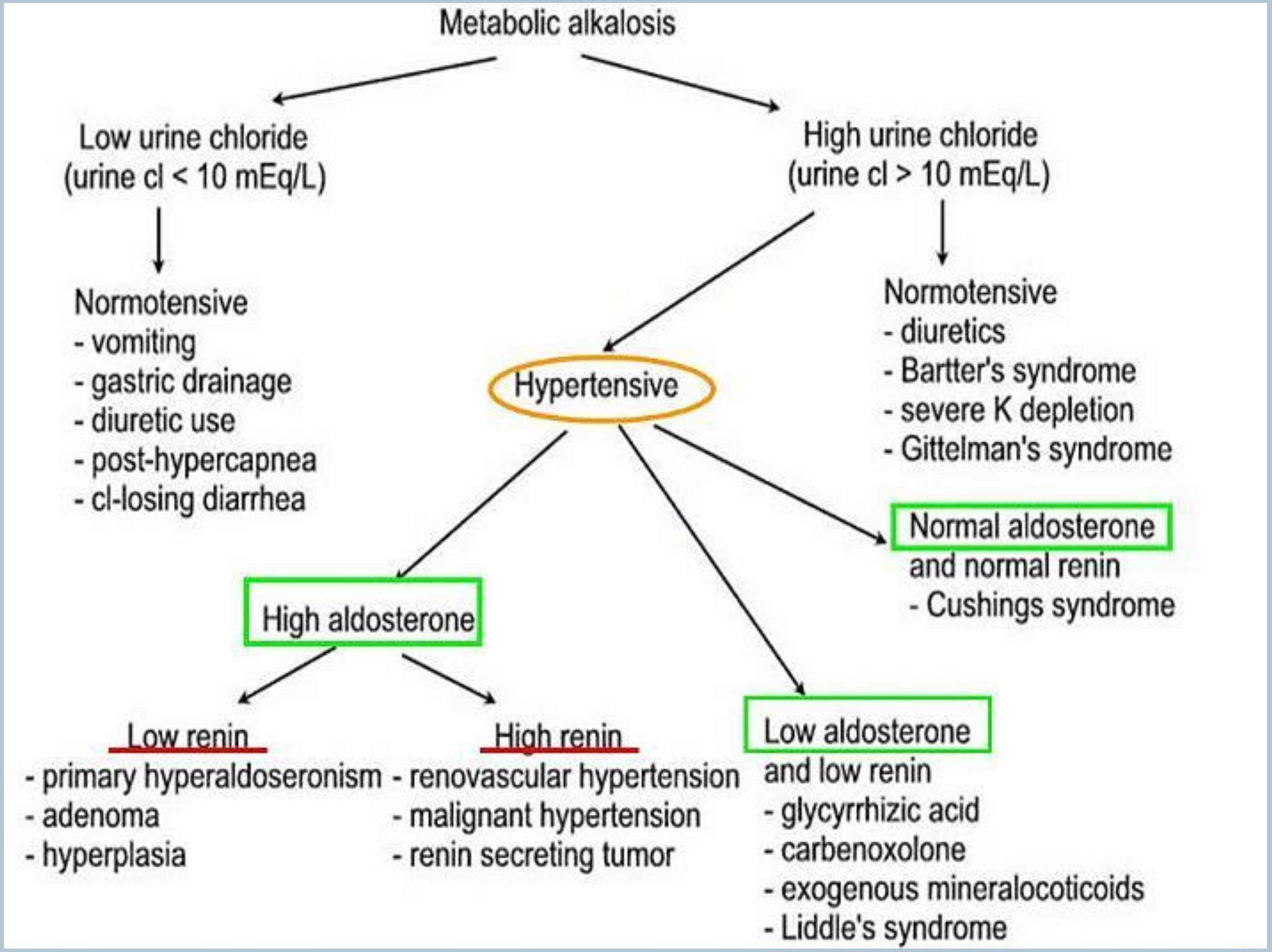
A Word About Chloride…

Fluid	Na	Cl	K	Mg	Ca	HCO3	Gluc	Ac	Gluc	Osm	pH
Plasma	140	104	4.5	1.25	2.5	24	0.08			290	7.4
0.9% Nacl	154	154								308	5.5
0.45% Nacl	77	77								406	
LR	130	109	4		1.5	28 (as Lac)				273	6.5
P-lyte	140	98	5	1.5				27	23	294	7.4
5% dex							5			278	

Causes of Respiratory Alkalosis

- Pain
- Trauma
- Sepsis
- Pulmonary embolism
- Shock
- Drugs
- Pregnancy
- Hyperventilation (think sick first!)
- Mechanical ventilation

Causes of Metabolic Alkalosis



Let’s do the math…

ABG Analysis/Calculations

ABG Analysis Big Picture

1. Look at the **pH** to determine primary disorder

2. Look at **PCO_2**, use in conjunction with pH to determine primary disorder
   - If pH and PCO_2 move in *opposite* directions, respiratory disorder is primary
   - If pH and PCO_2 move in the *same* direction, metabolic disorder is primary

ABG Analysis Big Picture

3. Look for **mixed disorder**
   - If both pCO_2 and HCO_3 are ↑ ↑ = respiratory acidosis OR metabolic alkalosis
   - If both pCO_2 and HCO_3 are ↓ ↓ = respiratory alkalosis OR metabolic acidosis
   - If **pCO_2 and HCO_3 move in opposite direction** ↑ ↓ **= mixed disorder is present**
4. Apply **Compensation rules**
   - Boston rule
   - Winter's formula
5. Look at **Calculated/Corrected Anion Gap** and **Delta Gap**
6. Clinical Application

**paO2 in ABG tells us about the partial pressure of oxygen in blood. It is not used in assessment of acid-base disorders.*

ABG Practice: Steps 1-2

- pH low (<7.4) = Acidosis, pH high (>7.4) = Alkalosis
- If pH and PCO_2 move in *opposite* directions, respiratory disorder is primary
- If pH and PCO_2 move in the *same* direction, metabolic disorder is primary

pH	PCO_2	HCO_3	Disorder
7.2	70	28	Respiratory Acidosis

pH	PCO_2	HCO_3	Disorder
7.2	30	16	Metabolic Acidosis

ABG Practice: Steps 1-2

pH	PCO_2	HCO_3	Disorder
7.5	40	31	Metabolic alkalosis

pH	PCO_2	HCO_3	Disorder
7.5	30	16	Respiratory alkalosis

Let’s add in the next layer of complexity…

Step 3: Look for mixed disorders

ABG Practice: Step 3

- If both pCO_2 and HCO_3 are ↑ ↑ = respiratory acidosis OR metabolic alkalosis
- If both pCO_2 and HCO_3 are ↓ ↓ = respiratory alkalosis OR metabolic acidosis
- If **pCO_2 and HCO_3 move in opposite direction** ↑ ↓ **= mixed disorder is present**

pH	PCO_2	HCO_3	Disorder
7.28	55	19	Primary respiratory acidosis, metabolic acidosis

1. Look at pH → acidosis
2. Look at pH and PCO_2 → opposite, respiratory disorder is primary
3. Look at pCO_2 and HCO_3 → opposite, mixed disorder present → HCO_3 low, implies metabolic acidosis also present

ABG Practice: Step 3

pH	PCO_2	HCO_3	Disorder
7.50	24	28	Respiratory alkalosis primary, metabolic alkalosis

1. Look at pH → alkalosis
2. Look at pH and PCO_2 → opposite, respiratory disorder is primary
3. Look at pCO_2 and HCO_3 → opposite, mixed disorder present → HCO_3 high, implies metabolic alkalosis also present

Let’s add another layer

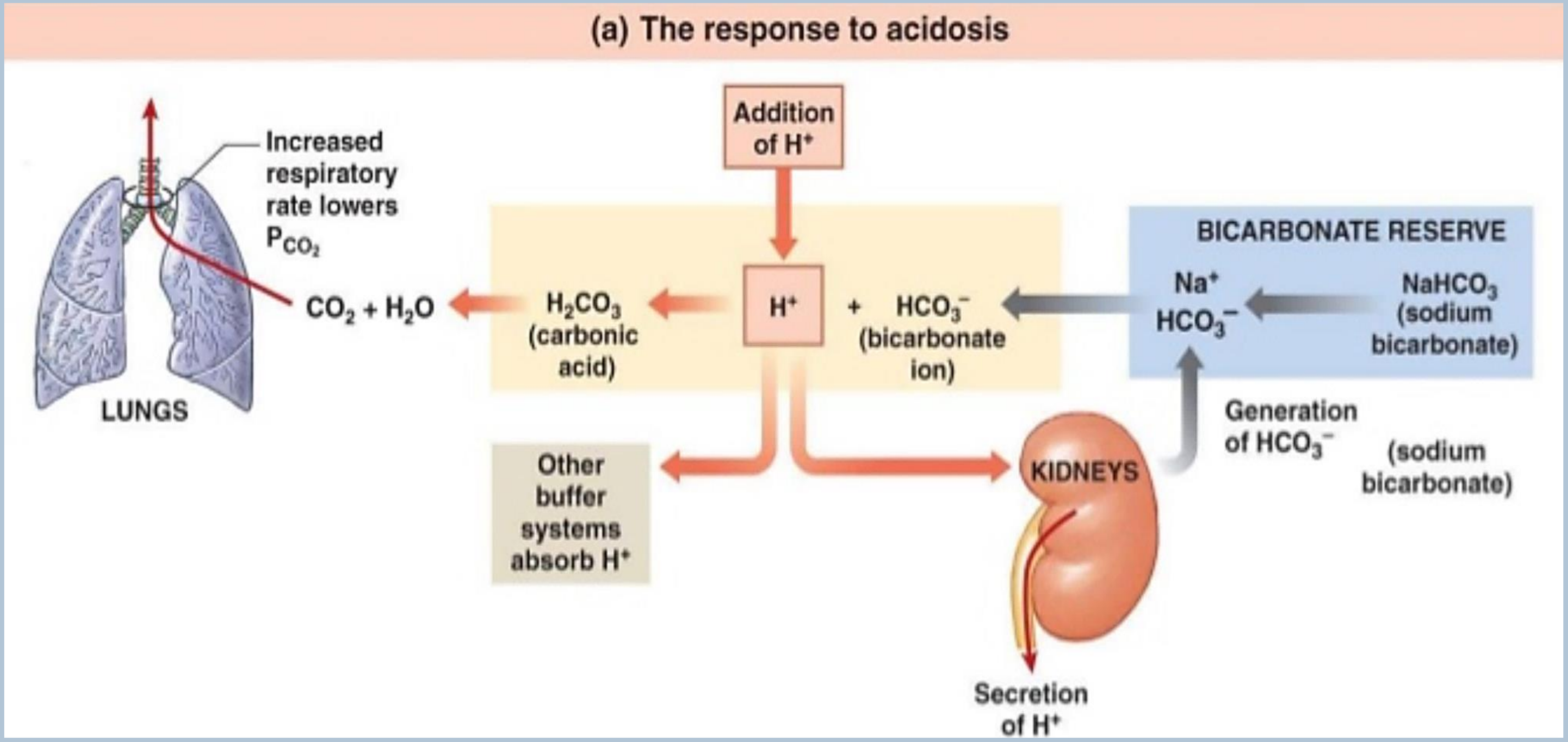
Step 4: Compensation

Compensation Physiology

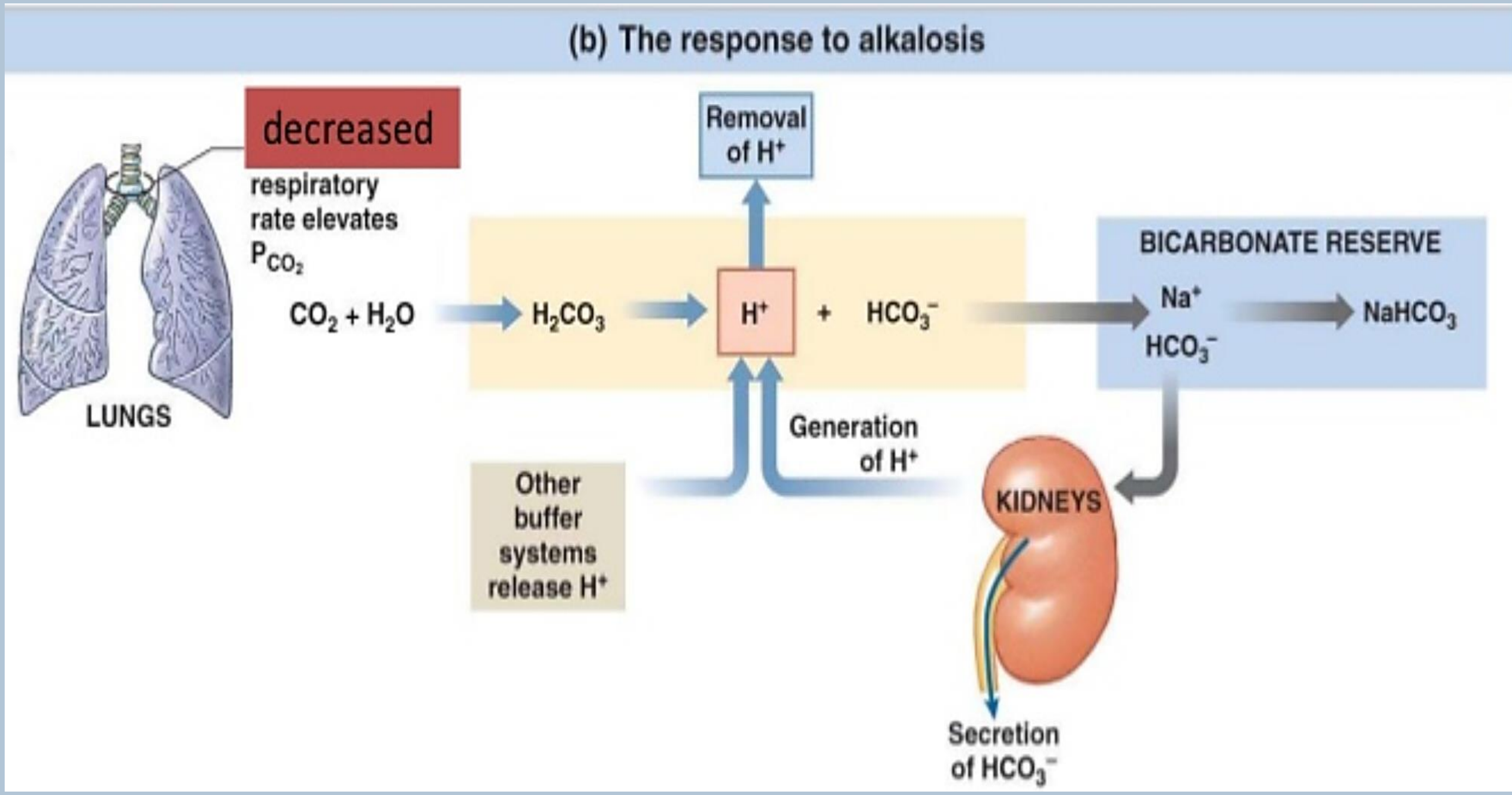
- **Respiratory system**: maintains pH by regulating CO_2
  - Can compensate quickly
- **Renal (metabolic) system**: regulates pH by excreting H^+ (acid) or reabsorbing HCO_3^- (base)
  - Several hours to days to compensate

- With the body's ACUTE compensation, the pH will NOT return to normal, but it may get close

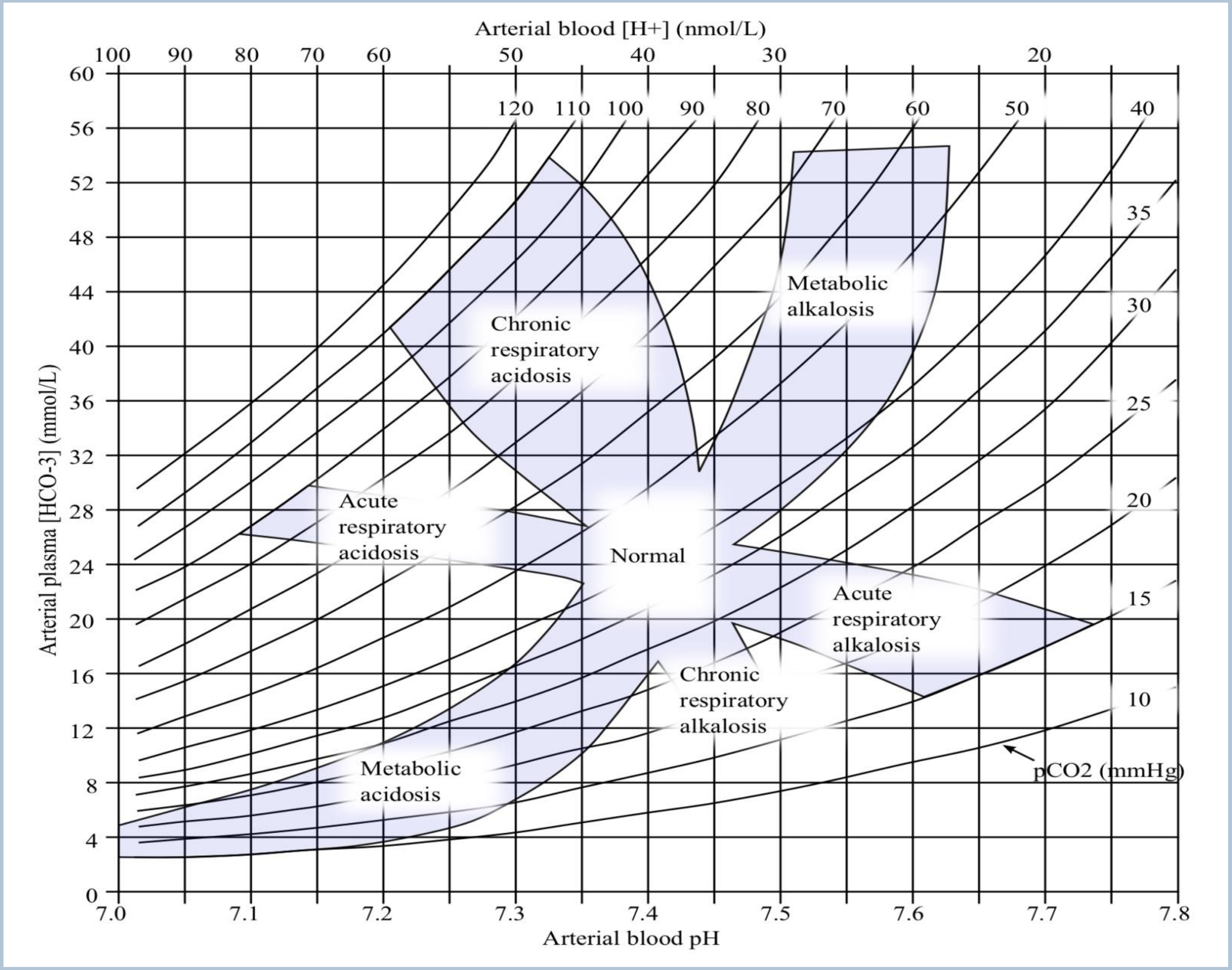
Buffering Systems/Compensation



Buffering Systems/Compensation



Acid-base Nomogram



Compensation

Acid-Base Disorder	Primary Change	Compensatory Change
Respiratory acidosis	PCO_2 ↑	HCO_3 ↑
Respiratory alkalosis	PCO_2 ↓	HCO_3 ↓
Metabolic acidosis	HCO_3 ↓	PCO_2 ↓
Metabolic alkalosis	HCO_3 ↑	PCO_2 ↑

ABG Practice: Step 4

Acid-Base Disorder	Primary Change	Compensatory Change
Respiratory acidosis	PCO_2 ↑	HCO_3 ↑
Respiratory alkalosis	PCO_2 ↓	HCO_3 ↓
Metabolic acidosis	HCO_3 ↓	PCO_2 ↓
Metabolic alkalosis	HCO_3 ↑	PCO_2 ↑

pH	PCO_2	HCO_3	Primary Disorder
7.28	60	35	Respiratory acidosis

1. pH: Acidosis
2. pCO_2 and pH move in opposite directions → primary respiratory disorder
3. pCO_2 and HCO_3 both high → respiratory acidosis
4. Expected change with respiratory acidosis → HCO_3 increases ☑

ABG Practice: Step 4

Acid-Base Disorder	Primary Change	Compensatory Change
Respiratory acidosis	PCO_2 ↑	HCO_3 ↑
Respiratory alkalosis	PCO_2 ↓	HCO_3 ↓
Metabolic acidosis	HCO_3 ↓	PCO_2 ↓
Metabolic alkalosis	HCO_3 ↑	PCO_2 ↑

pH	PCO_2	HCO_3	Primary Disorder
7.5	55	36	Metabolic alkalosis

1. pH: Alkalosis
2. pCO_2 and pH move in same direction → primary metabolic disorder
3. pCO_2 and HCO_3 are high = metabolic alkalosis
4. Expected change with metabolic alkalosis → PCO_2 increases ☑

You’re experts! Let’s add on…

Calculating for compensation

Calculating for Compensation

- **Respiratory Rule #1:** pH changes INVERSELY by 0.08 for 10 mm CO_2 in **ACUTE** cases
    - If CO_2= 50, pH will be 7.32 (0.08 below)
    - If CO_2=30, pH will be 7.48 (0.08 above)

*DO NOT USE IN CHRONIC CASES…pH usually corrects/compensates to normal (7.4).

Calculating for Compensation

Boston Rules (to predict changes in HCO_3^- from $PaCO_2$ – respiratory disorders):

Change in CO2	Change in HCO3	Condition	Example
10	1	Acute Resp Acidosis	If CO2=50, HCO3=25
10	2	Acute Resp Alkalosis	If CO2=30, HCO3=22
10	4	Chronic Resp Acidosis	If CO2=50, HCO3=28
10	5	Chronic Resp Alkalosis	If CO2=30, HCO3=19

Calculating for Compensation

- Winter's Formula (metabolic acidosis):
  - Expected $pCO_2 = 1.5 \times HCO_3 + 8 \pm 2$

- Metabolic Alkalosis
  - Expected $pCO_2 = 0.7 \times [HCO_3^-] + 20\ (\pm 5)$

One last concept(s)…

Step 5: Look at **Calculated/Corrected Anion Gap** and **Delta Gap**

Anion Gap (Calculated)

- **Anion Gap** = the difference between measured cations (Na+, K+) and measured anions (Cl- and HCO_3^-)

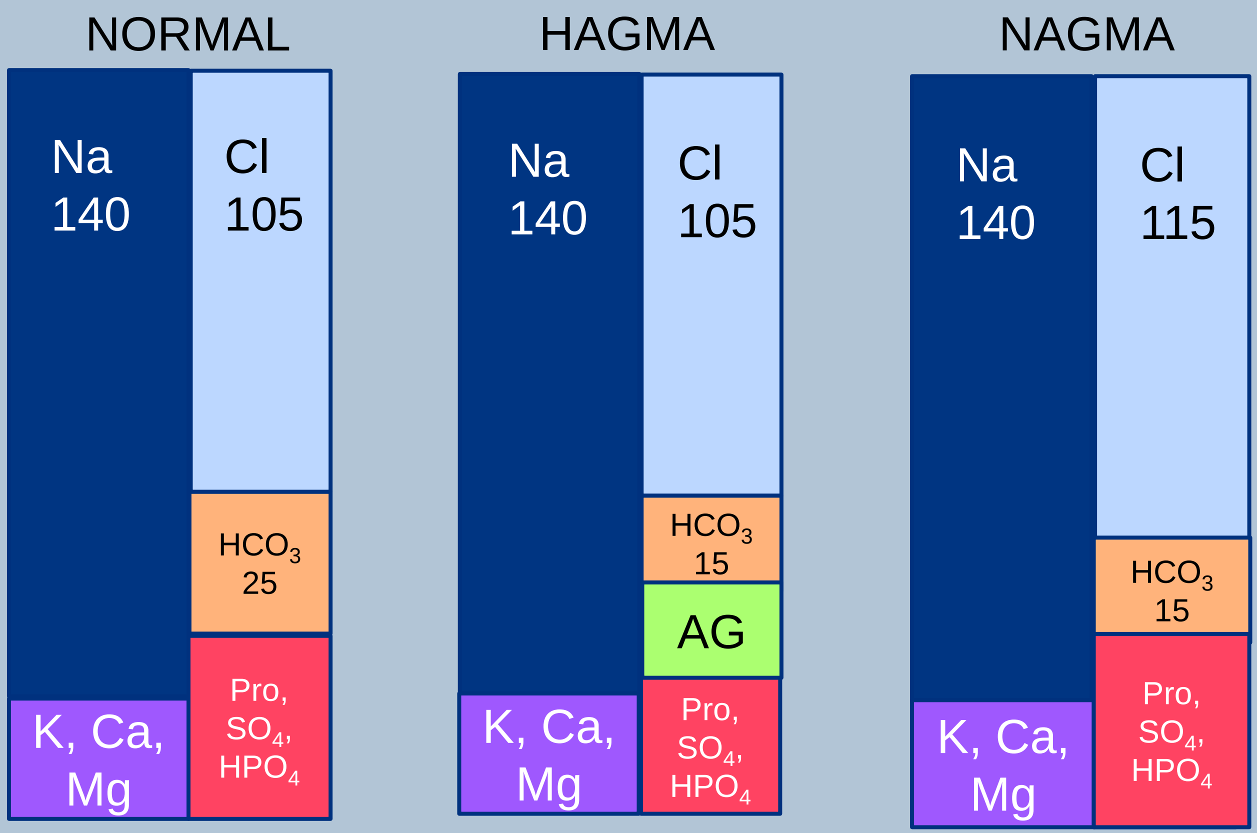
  $$= \mathbf{Na - [Cl + HCO_3]}$$

  Normal range = 4 – 12 mmol/L

  *We call this the calculated anion gap (cAG), because it is calculated from the CMP

- Significance?
  - The law of electrochemical neutrality
  - Provides foresight into managing acidosis

High vs. Normal Anion Gap Metabolic Acidosis



High Anion Gap Metabolic Acidosis (HAGMA)

- Accumulation of or impairment of excretion of acids
- **MUDPILES:**
  - Methanol
  - Uremia
  - DKA
  - Propylene glycol (Ativan, Dilantin)
  - Isoniazid/Iron
  - Lactate
  - Ethanol/Ethylene glycol
  - Salicylates/Seizures/Starvation

Normal Anion Gap Metabolic Acidosis (NAGMA)

- Mainly from losses of bicarb (HCO_3^-)
- **USEDCRAP (earlier slide) OR**
- **ABCDE**
  - **A**ddison's
  - **B**icarbonate loss (GI or renal – think v/d, fistula, ostomy)
  - **C**hloride excess*
  - **D**iuretics (acetazolamide)*
  - **E**xtra – Renal tubular acidosis (RTA**)***

Anion Gap (corrected)

- Corrected Anion Gap (corrAG) = takes into account the unmeasured anions (albumin, sulfate, phos), which may change with acute or chronic illness

= [2 x Albumin] + [0.5 Phosphate] (+/- 2) OR **[3 x Albumin]**

Normal range = 8 – 12 mmol/L

- Significance?
  - If **calcAG > corrAG**, there is **high gap metabolic acidosis** present

Delta Gap

- *Check in the presence of HAGMA to determine if pure HAGMA or coexisting disorder present*

- **Delta Gap = [calcAG – corrAG] + HCO_3**
  - **Net sum = 24**
    - HAGMA only
  - **Net sum < 24**
    - NAGMA (non-anion gap acidosis) is present
  - **Net sum > 24**
    - METABOLIC ALKALOSIS is present

Practice: Minding the Gaps

<u>ABG:</u>

pH 7.18

PCO_2 34 mmHg

HCO_3 12 mEq/L

<u>BMP:</u>

Na= 138, K=3.8,

Cl=115 Albumin=2.3,

Phos=1

calcAG = 138 – [115+12] = **11**

corrAG = 2 x 2.3 + [0.5 x1] = **5.1 (+/- 2)**

calcAG > corrAG → HAGMA present

Delta Gap = [calcAG- corrAG] + HCO_3 = 6 + 12 **= 18**

18 < 24…therefore**, NAGMA present also**

Let’s practice…

Case 1

A 26 YO M with asthma presents to the ED with difficulty breathing x 3 days. It is getting progressively worse. He has tried his regular and rescue inhalers; nothing seems to help. He looks pale and is taking rapid, shallow breaths. On exam, he has diffuse wheezing in all lung fields.

Vitals:
- HR – 120
- BP – 113/76
- RR – 28
- SpO_2 – 92%
- Temp – 37.8C

ABG results:
- pH – 7.08
- pCO_2 – 80 mmHg
- HCO_3^- – 28 mEq/L

Acute Respiratory Acidosis

Other results (pertinent):
- Na – 138 mEq/L (135-145)
- K – 4.0 mmol/L (3.6-5.2)
- Cl – 106 mEq/L (96-106)
- Albumin – 3.8 g/dL (3.5-5.5)
- Phos – 3.0 mg/dL (2.8 – 4.5)

Case 1

ABG results:
- pH – 7.08
- pCO_2 – 80 mmHg
- HCO_3^- – 28 mEq/L

Compensation rules…

- **Respiratory rule #1**
- pCO_2 ↑ by 40 (10x4)
- Expected pH change = 0.08 X 4 = 0.32
- Actual pH change = 7.4 - 7.08 = 0.32

Case 1

ABG results:
- pH – 7.08
- pCO_2 – 80 mmHg
- HCO_3^- – 28 mEq/L

Other results (pertinent):
- Na – 138 mEq/L (135-145)
- K – 4.0 mmol/L (3.6-5.2)
- Cl – 106 mEq/L (96-106)
- Albumin – 3.8 g/dL (3.5-5.5)
- Phos – 3.0 mg/dL (2.8 – 4.5)

- calcAG = 138- [106+28] = 4
- corrAG = [2x3.8]+[0.5x3] = 9.1
- calcAG < corrAG = no additional HAGMA
- Delta gap = not needed

Case 1 DDx + Management

- Asthma exacerbation → rapid, shallow breaths → retaining CO_2 → Acute Respiratory Acidosis

- Management
  - Reverse the respiratory acidosis, augment breathing to help improve gas exchange until the patient improves and can do so on their own
  - How?
  - When would you get a follow-up ABG?

Case 2

A 35 YO M presents to the ED with gun shot wound (GSW) to the abdomen. He was found down by a civilian about 20 minutes after the shooting who called 911. Upon arrival, he appears pale, diaphoretic, and is experiencing severe abdominal pain. He is slightly altered and cannot tell you where he is. He has no past medical history.

Vitals:

- HR – 116
- BP – 86/68
- RR – 10
- SpO_2 – 96%
- Temp – 37.6C

ABG results:

- pH – 7.18
- pCO_2 – 34 mmHg
- HCO_3^- – 12 mEq/L

Acute Metabolic Acidosis

Other results (pertinent):

- Na – 132 mEq/L (135-145)
- K – 3.6 mmol/L (3.6-5.2)
- Cl – 92 mEq/L (96-106)
- Albumin – 3.2 g/dL (3.5-5.5)
- Phos – 2.1 mg/dL (2.8 – 4.5)

Case 2

ABG results:
- pH – 7.18
- pCO_2 – 34 mmHg
- HCO_3^- – 12 mEq/L

Compensation rules…

- Winter's formula (metabolic acidosis)
  - Expected PCO_2 in metabolic acidosis:

  $= 1.5 \times HCO_3 + 8$ = **26 (+/- 2)**

- **Expected pCO_2 is lower than our actual… what does this mean?**

Case 2

- The expected degree of respiratory compensation is not present…
- There is also a respiratory acidosis

- Respiratory depression/AMS → slower, possibly more shallow breaths → retention of CO_2 → respiratory acidosis
  - Normally, the body would begin breathing more quickly to blow off CO_2 and help compensate for the metabolic acidosis…

Case 2

ABG results:

- pH – 7.18
- pCO_2 – 34 mmHg
- HCO_3^- – 12 mEq/L

Other results (pertinent):

- Na – 132 mEq/L (135-145)
- K – 3.6 mmol/L (3.6-5.2)
- Cl – 92 mEq/L (96-106)
- Albumin – 3.2 g/dL (3.5-5.5)
- Phos – 2.1 mg/dL (2.8 – 4.5)

- calcAG = 132 – [92+12] = 28
- corrAG = [2x3.2]+[0.5x2.1] = 7.45
- calcAG>corrAG = HAGMA present (we already know this)
- Delta gap = [28-7.45]+12 = 32.55
- Delta gap > 24 → metabolic alkalosis also present

Case 2 DDx

HAGMA

- MUDPILES:
  - Methanol
  - Uremia
  - DKA
  - Propylene glycol (Ativan, Dilantin)
  - Isoniazid/Iron
  - Lactate
  - Ethanol/Ethylene glycol
  - Salicylates/Seizures/Starvation

Respiratory Acidosis

- ↓respiratory stimulus
- Atelectasis
- Additional injuries?

Metabolic Alkalosis

- GI acid loss
  - Vomiting/NG drainage
  - Diarrhea
  - Ostomy
  - Dehydration
- Renal acid loss
  - Bartter, Gitelman syndrome
  - Diuretics
- Added base
  - Laxatives, milk-alkali syndrome, antacid OD
  - Iatrogenic bicarb administration
- Endocrine
  - Cushing
  - Steroid excess
  - Hyperaldosteronism

Case 2 Management

- You obtain a lactate level to confirm your suspicion… = 6.2 mmol/L (<2.3).
- His renal function is normal…for now.
- His H/H is 8 g/dL / 26% (13.2-16.6 / 38.3-48.6%).

Case 2 Management

- Acute blood loss → hypovolemic shock → decreased organ perfusion and O2 delivery → interference with aerobic metabolism → increased anaerobic metabolism → production of lactic acid and metabolic acidosis

- How are you going to manage this patient?
- How did the ABG help us in this scenario?
- Will you get a repeat ABG? If so, when?

Case 3

A 64 YO M with ESRD s/p kidney transplant, Type 2 DM, and chronic HFrEF presents to the ED with 3 days of fatigue, abdominal pain, and shortness of breath. He is unsure, but he may have had a fever. On exam, he appears unwell and has crackles in the L lung base. He missed dialysis today. CXR shows a L basilar infiltrate.

Vitals:
- HR 100
- BP 92/78
- RR 20
- SpO_2 84%
- Temp 38.0

ABG results:
- pH 7.29
- pCO_2 23.3
- HCO_3^- 11.1
- pO_2 52.9

Acute Metabolic Acidosis

Other results (pertinent):
- Na – 136
- K – 5.2
- Cl – 106
- AG – 18
- Glucose - 260
- Albumin – 2.8
- Phos – 3.0
- BUN – 89.1
- Cr – 4.3
- Lactate – 1.3
- Beta-hydroxybutyrate – 2.7

Case 3

ABG results:

- pH 7.29
- pCO_2 23.3
- HCO_3^- 11.1
- pO_2 52.9

Compensation rules…

Winters formula

Expected pCO_2= 1.5 x 11 + 8 = 24.5 (+/- 2)

Actual pCO_2 = 23.3 ☑

Case 3

ABG results:
- pH 7.29
- pCO_2 23.3
- HCO_3^- 11.1
- pO_2 52.9

Other results (pertinent):
- Na – 136
- K – 5.2
- Cl – 106
- Glucose – 260
- Albumin – 2.8
- Phos – 3.0
- BUN – 89.1
- Cr – 4.3
- Lactate – 1.3
- Beta-hydroxybutyrate – 2.7

- calcAG = 136 – [106+11] = 19
- corrAG = [2x2.8] + [0.5x3.0] = 7.1
- calcAG>corrAG = HAGMA present (we already know this)
- Delta gap = [19-7] + 11 = 23
- Delta gap < 24 = NAGMA also present

Case 3 DDx

<u>MUDPILES = HAGMA</u>

- **M**ethanol
- **U**remia
- **D**KA
- **P**ropylene glycol (Ativan, Dilantin)
- **I**soniazid/Iron
- **L**actate
- **E**thanol/Ethylene glycol
- **S**alicylates / Seizures / Starvation

<u>ABCDE = NAGMA</u>

- **A**ddison's
- **B**icarbonate loss (GI or renal – think v/d, fistula, ostomy)
- **C**hloride excess
- **D**iuretics (acetazolamide)
- **E**xtra – Renal tubular acidosis (RTA**)**

Case 3 Management

- How are we going to correct this patient's HAGMA and NAGMA ?

1. Treat DKA
2. Dialysis…add bicarb
3. Treat underlying cause of DKA…what was going on with this patient?

- Are you going to get another ABG? If so, when?

Case 4

A 23 YO F returns from a volunteer humanitarian trip to Haiti. She presents to her PA with nausea & vomiting x2 days. The vomiting is becoming more frequent, and she can't keep down any liquids. On exam, she appears significantly dehydrated and her respirations are frequent but shallow.

Vitals:
- HR 102
- BP 90/64
- RR 20
- SpO_2 99%
- Temp 37.6 C

ABG results:
- pH 7.56
- pCO_2 40 mmHg
- HCO_3^- 32 mEq/L

Acute Metabolic Alkalosis

Other results (pertinent):
Na – 132
K – 3.4
Cl – 86
Albumin – 2.3
Phos – 2.0
Glucose – 86
BUN – 32
Cr – 1.0
Lactate – 1.8

Case 4

ABG results:

- pH 7.56
- pCO_2 40 mmHg
- HCO_3^- 32 mEq/L

- Compensation rules…
- Expected $pCO_2 = 0.7 \times [32] + 20 = 42.4\ (+/-5)$
- Actual $pCO_2 = 40$ ✓

Case 4

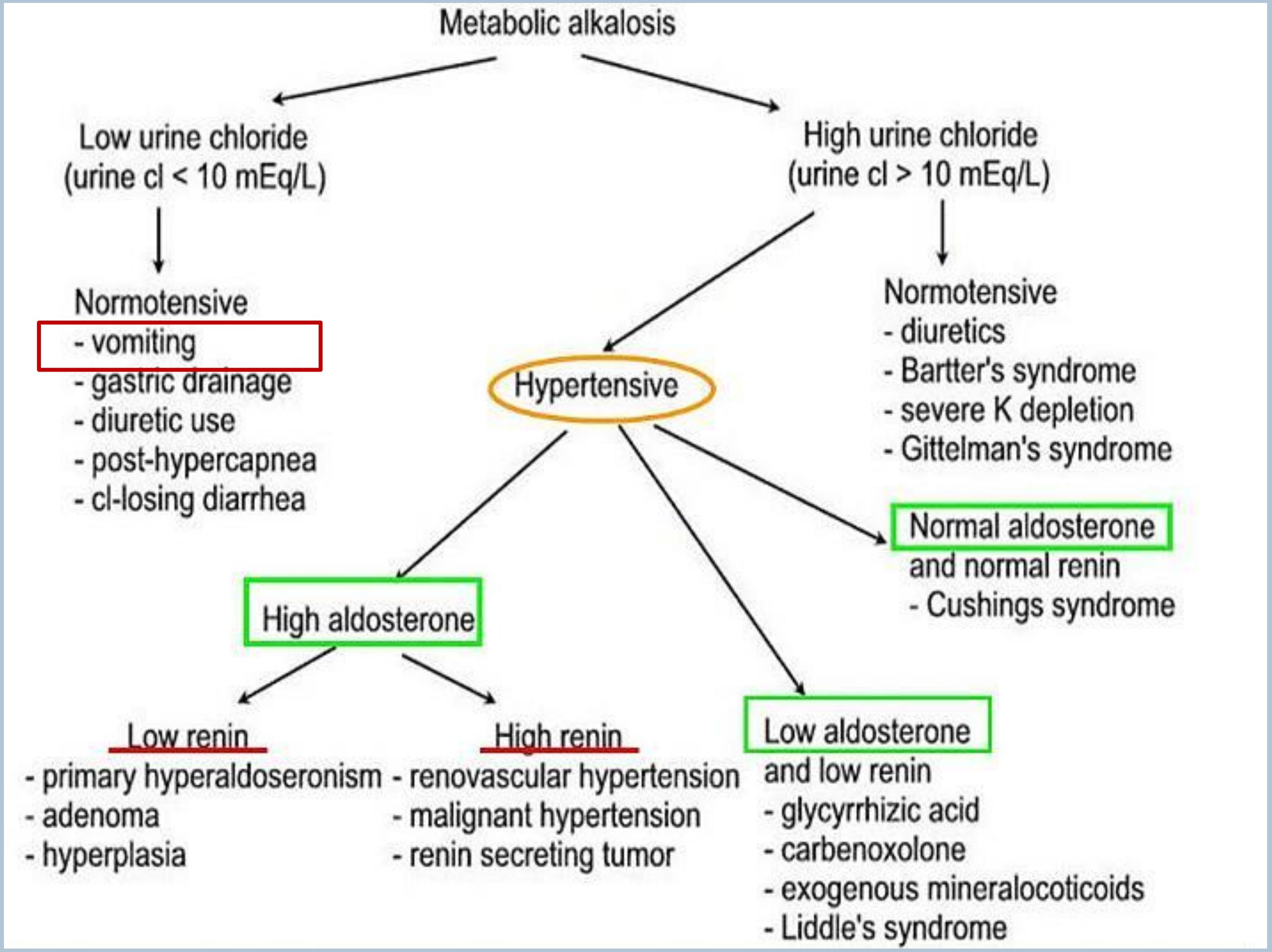
ABG results:
- pH 7.56
- pCO_2 40 mmHg
- HCO_3^- 32 mEq/L

Other results (pertinent):
Na - 132
K – 3.4
Cl – 86
Albumin – 2.3
Phos – 2.0
Glucose – 86
BUN – 32
Cr – 1.0
Lactate – 1.8

- calcAG = 132 – [86+32] = 14
- corrAG = [2x2.3]+[0.5x2.0] = 5.6
- calcAG>corrAG = HAGMA present
- Delta gap = [14-5.6] + 32 = 40.4
- Delta gap > 24 = Metabolic Alkalosis (we already know this)

Acute Metabolic Alkalosis + HAGMA

Case 4 DDx



Metabolic alkalosis
Low urine chloride
(urine cl < 10 mEq/L)
High urine chloride
(urine cl > 10 mEq/L)
Normotensive
- vomiting
- gastric drainage
- diuretic use
- post-hypercapnea
- cl-losing diarrhea
Hypertensive
Normotensive
- diuretics
- Bartter's syndrome
- severe K depletion
- Gittelman's syndrome
Normal aldosterone
and normal renin
- Cushings syndrome
High aldosterone
Low renin
- primary hyperaldoseronism
- adenoma
- hyperplasia
High renin
- renovascular hypertension
- malignant hypertension
- renin secreting tumor
Low aldosterone
and low renin
- glycyrrhizic acid
- carbenoxolone
- exogenous mineralocoticoids
- Liddle's syndrome

Case 4 DDx

MUDPILES = HAGMA

- **M**ethanol
- **U**remia
- **D**KA
- **P**ropylene glycol (Ativan, Dilantin)
- **I**soniazid/Iron
- **L**actate
- **E**thanol/Ethylene glycol
- **S**alicylates / Seizures / Starvation

Beta-hydroxybutyrate = 2.3 (↑)

Case 4 Management

- How are we going to manage this patient?
  - Fluids
  - Antiemetics → nutrition
  - Look for infectious causes

- Does the ABG change our course of action?

- Will you get a follow up ABG? If so, when?

Case 5

A 68 YO F presents to the ED with 3 days of progressively worsening cough and shortness of breath. She has been experiencing intermittent fevers. Her appetite is diminished, and she is fatigued. On exam, she has scattered crackles. CXR reveals a multifocal pneumonia.

Vitals:
HR 96
BP 112/82
RR 26
SpO_2 84%
Temp 37.9 C

ABG Results:
- pH 7.55
- pCO_2 22 mmHg
- HCO_3^- 16 mEq/L

Acute Respiratory Alkalosis

Other results (pertinent):
Na – 134
K – 4.2
Cl – 100
BUN – 32
Cr – 1.2
Albumin – 3.8
Phos – 3.0
Lactate – 3.2

Case 5

ABG Results:
- pH 7.55
- pCO_2 22 mmHg
- HCO_3^- 16 mEq/L

Compensation rules…Boston Approach for Resp Disorders

Change in CO2	Change in HCO3	Condition	Example
10	1	Acute Resp Acidosis	If CO2=50, HCO3=25
10	2	Acute Resp Alkalosis	If CO2=30, HCO3=22
10	4	Chronic Resp Acidosis	If CO2=50, HCO3=28
10	5	Chronic Resp Alkalosis	If CO2=30, HCO3=19

pCO_2 went from 40 → 22 (roughly 20)

We expect HCO_3 to decrease by 4 (using 24 baseline), therefore expecting HCO_3 to be 20 → there is compensation, and potentially another unaccounted-for process

Is there a mixed disorder present?

Case 5

ABG Results:
- pH 7.55
- pCO_2 22 mmHg
- HCO_3^- 16 mEq/L

Other results (pertinent):
Na – 134
K – 4.2
Cl – 100
BUN – 32
Cr – 1.2
Albumin – 3.8
Phos – 3.0
Lactate – 3.2

- calcAG = 134– [100+16] = 18
- corrAG = [2x3.8] + [0.5x3.0] = 9.1
- calcAG>corrAG = HAGMA present
- Delta gap = [18-9.1] +16 = 32.9
- Delta gap = 24.9 …close enough! Only HAGMA present

Acute Respiratory Alkalosis + HAGMA

Case 5 DDx

- Hypoxic stimulation 2/2 pneumonia leads to hyperventilation to try to correct the hypoxia, at the expense of CO_2 loss → acute respiratory alkalosis

- **MUDPILES = HAGMA**
  - **M**ethanol
  - **U**remia
  - **D**KA
  - **P**ropylene glycol (Ativan, Dilantin)
  - **I**soniazid/Iron
  - **L**actate
  - **E**thanol/Ethylene glycol
  - **S**alicylates / Seizures / Starvation

Case 5 Management

- What is an appropriate management strategy to correct the respiratory alkalosis?
  - Supplemental oxygen
- How are we going to correct the metabolic acidosis?
  - Treat the infection!
  - Clear the lactate…ensure perfusion (? fluids)
- How did the ABG help us here?
- Would you get a repeat ABG? If so, when?

In Summary...

- Look at the pH first
  - Do pH and PCO_2 change in the same direction?
- Use all components/calculations of the ABG
  - *Don't skip the calculations…it may change your management!*
- Apply clinically to your patient; don't just treat the numbers
  - Use your differentials…MUDPILES, USEDCRAP, etc.
- Consider what will happen if you start treatment
  - Will treating help or hurt?

Questions? walker.andrew@mayo.edu; jalabast@msu.edu

References

- Brandis K. Acid Base Book. https://www.anaesthesiamcq.com/AcidBaseBook/. Accessed August 1, 2021.
- Simple and mixed acid-base disorders. UpToDate.com. https://www.uptodate.com/contents/simple-and-mixed-acid-base-disorders?search=simple%20and%20mixed%20acid%20base%20disorders&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1. Updated September 9, 2020. Accessed August 1, 2021.
- Story DA. Stewart Acid-Base: A Simplified Bedside Approach. *Anesthesia & Analgesia.* 2016;123(2):511-515. doi: 10.1213/ANE.0000000000001261
- VBG versus ABG. litfl.com. https://litfl.com/vbg-versus-abg/. Published Nov 3, 2020. Accessed August 1, 2021.
- Reddi AS. *Acid-Base Disorders: Clinical Evaluation and Management*. Cham, Switzerland: Springer Nature Switzerland AG; 2020. https://link-springer-com.proxy2.cl.msu.edu/book/10.1007%2F978-3-030-28895-2#toc. Accessed August 1, 2021.