

Physiological Causes of Abnormal ABG's

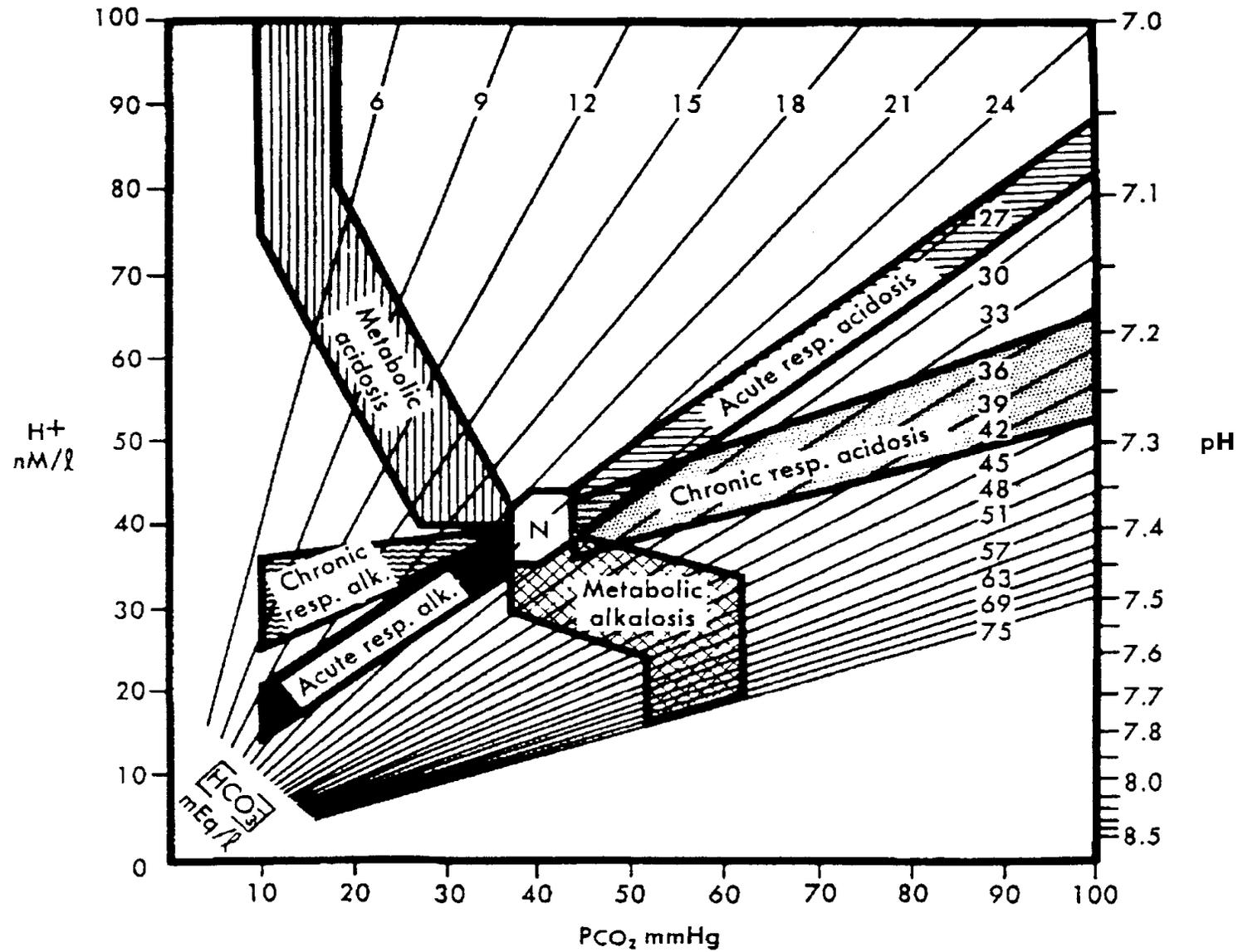


Fig. 10-7 Acid-base map. (See text for description.) (Reprinted from Goldberg M et al: JAMA 223:1973. Copyright 1973, American Medical Association.)

Major Student Performance Objective 1

1. The student will be able to discuss causes for various types of blood gas results.
2. They will also be required to discuss VA effects and compensatory mechanisms for acid base disturbances.

Supporting Student Performance Objective:

- Discuss disease cause for respiratory acidemia, respiratory alkalemia, metabolic acidemia, and metabolic alkalemia.
- Discuss effects of VA on respiratory alkalemia and acidemia.
- Recognize increased VA effect to compensate for respiratory acidemia.
- Give possible causes, given ABG results.
- Discuss electrolytes role in ABG changes and/or results.

ABG

$\text{CO}_2 > 45$

* Calculate
Golden Rule

Measured $>$ **Respiratory Acidosis
with Compensation**
Calculated

Measured = **Pure Respiratory
Acidosis**
Calculated

Measured $<$ **Respiratory and
Metabolic Acidosis**
Calculated

CO_2 WNL

pH

<7.35 Normal >7.45

Metabolic
Acidosis

Normal ABG

Metabolic
Alkalosis

$\text{CO}_2 < 35$

pH

<7.35 Normal >7.45

Metabolic Acidosis
with Compensation

Respiratory
Alkalosis with
Compensation

Respiratory
Alkalosis

Understanding ABG Results

This topic is very important and comes up a lot on exams on in the Ward round, so I made study notes -

Interpreting an arterial blood gas result:

1. Determine whether the sample is arterial or venous
Saturation $> 88\%$ = Arterial
 $< 88\%$ = Mixed, Venous, or arterial sample with significant pulmonary disease.
2. Determine is there respiratory failure occurring (low pO_2), and if this is type 1 (low pO_2 with normal or low pCO_2) or type 2 (low pO_2 with raised pCO_2)
3. Determine the acid/base status of the patient:
 - Determine whether the sample is acidotic, alkalotic, or normal (pH)
 - Determine whether the pCO_2 is normal or abnormal
 - Determine whether the HCO_3 is normal or abnormal
 - Determine whether the primary problem is metabolic or respiratory
 - Determine whether compensation is occurring via the table below:

Condition	Primary Disturbance	Compensatory Factor
Metabolic Acidosis	Decreased HCO_3^-	Decreased PaCO_2
Metabolic Alkalosis	Increased HCO_3^-	Increased PaCO_2
Respiratory Acidosis	Increased PaCO_2	Increased HCO_3^-
Respiratory Alkalosis	Decreased PaCO_2	Decreased HCO_3^-

The Respiratory Buffer Response

A normal by-product of cellular metabolism is carbon dioxide (CO₂). CO₂ is carried in the blood to the lungs, where excess CO₂ combines with water (H₂O) to form carbonic acid (H₂CO₃). The blood pH will change according to the level of carbonic acid present. This triggers the lungs to either increase or decrease the rate and depth of ventilation until the appropriate amount of CO₂ has been re-established. Activation of the lungs to compensate for an imbalance starts to occur within 1 to 3 minutes.

Respiratory Acidemia

\uparrow PaCO₂ & \downarrow pH

- Always caused by alveolar hypoventilation (decreased VA)
 1. $VA = (VT - VD)f$
 2. If VA is decreased then either one of three is present:
 - a. Decreased VT
 - b. Decreased f
 - c. Increased VD

Acute Respiratory Acidemia

(no metabolic compensation and HCO_3 normal)

Short-term alveolar hypoventilation

- Drug OD – CNS Depression
- Neuromuscular Disease
- Trauma
- Acute Bronchospasm

Chronic Respiratory Acidemia

(metabolic compensation by increased HCO₃)

Long-term alveolar hypoventilation

- Increased VD - COPD
- Decreased VT and/or Rate –
chronic neuromuscular disease
- Decreased VT – extreme obesity,
Pickwickian syndrome

Common Causes of Respiratory Acidosis

- **WITH NORMAL LUNGS**

- **CNS depression**
 - **Anesthesia**
 - **Sedative drugs**
 - **Narcotic analgesics**
- **Neuromuscular disease**
 - **Poliomyelitis**
 - **Myasthenia gravis**
 - **Guillain-Barre syndrome**
- **Trauma**
 - **Spinal cord**
 - **Brain**
 - **Chest wall**
- **Severe restrictive disorders**
 - **Obesity (Pickwickian syndrome)**
 - **Kyphoscoliosis**

- **WITH ABNORMAL LUNGS**

Disease of lungs and/or upper airway

- **Chronic obstructive pulmonary disease**
- **Acute airway obstruction or severe asthma attack (late phases)**
- **Severe pulmonary edema**

Respiratory Alkalemia

\downarrow PaCO₂ & \uparrow pH

- Always caused by alveolar hyperventilation (increased VA)
 1. $VA = (VT - VD)f$
 2. If VA is increased then either one of three is present:
 - a. Increased VT
 - b. Increased f
 - c. Decreased VD

Common Causes of Respiratory Alkalosis

\downarrow PaCO₂ & \uparrow pH

- **WITH NORMAL LUNGS**

- Anxiety
- Fear
- Stimulant drugs
- CNS lesions
- Pain
- Sepsis
- Hypobarism (high altitude)

Any acute pulmonary insult

- Pneumonia
- mild asthma attack
- early pulmonary edema
- pulmonary embolism

- **WITH ABNORMAL LUNGS**

- Hypoxemia-causing conditions
 - Acute asthma
 - Pneumonia
- Stimulation of vagal lung receptors
 - Pulmonary edema
 - Pulmonary vascular disease

- **WITH NORMAL OR ABNORMAL LUNGS**

- Iatrogenic (physician induced) hyperventilation

Bicarbonate (HCO_3^-)

- An alkaline electrolyte (main anion in the extracellular fluid) whose major function is the regulation of acid-alkaline balance. It acts as a buffer to neutralize acids and maintain the 20:1 bicarbonate/carbonic acid ratio needed to keep the body in homeostasis. The kidneys selectively regulate the amount of bicarbonate retained or excreted.

The Renal Buffer Response

In an effort to maintain the pH of the blood within its normal range, the kidneys excrete or retain bicarbonate (HCO_3^-). As the blood pH decreases, the kidneys will compensate by retaining HCO_3^- and as the pH rises, the kidneys excrete HCO_3^- through the urine. Although the kidneys provide an excellent means of regulating acid-base balance, the system may take from hours to days to correct the imbalance. When the respiratory and renal systems are working together, they are able to keep the blood pH balanced by maintaining 1 part acid to 20 parts base.

Metabolic Acidemia

Decreased pH, Decreased HCO₃

Common Causes of Metabolic Acidosis

- **Lactic Acidemia, due to anaerobic metabolism**
 - Decreased PaO₂
 - Decreased circulation
 - Decreased Hgb
- **Ketoacidosis**
 - Diabetes
 - Starvation – Fasting, diet irregularities
 - Alcoholic Ketoacidosis
- **Renal Failure**
- **Aspirin OD or other acid-type drugs**
 - Salicylate intoxication
 - Methanol (formic acid)
 - Ethylene glycol (oxalic acid)
 - Paraaldehyde (acetic acid)

Metabolic Alkalemia

Increase pH, Increased HCO_3

Common Causes of Metabolic Alkalosis

- **INCREASE IN BASE**
 - Administration/ingestion of HCO_3
 - Hypochloremia
 - Diuretic therapy
- **LOSS OF FIXED ACID**
 - Severe vomiting
 - Nasogastric suction
 - Hypokalemia
 - Potassium deficiency
 - Corticosteroids

Mixed Acid-base disorders are common

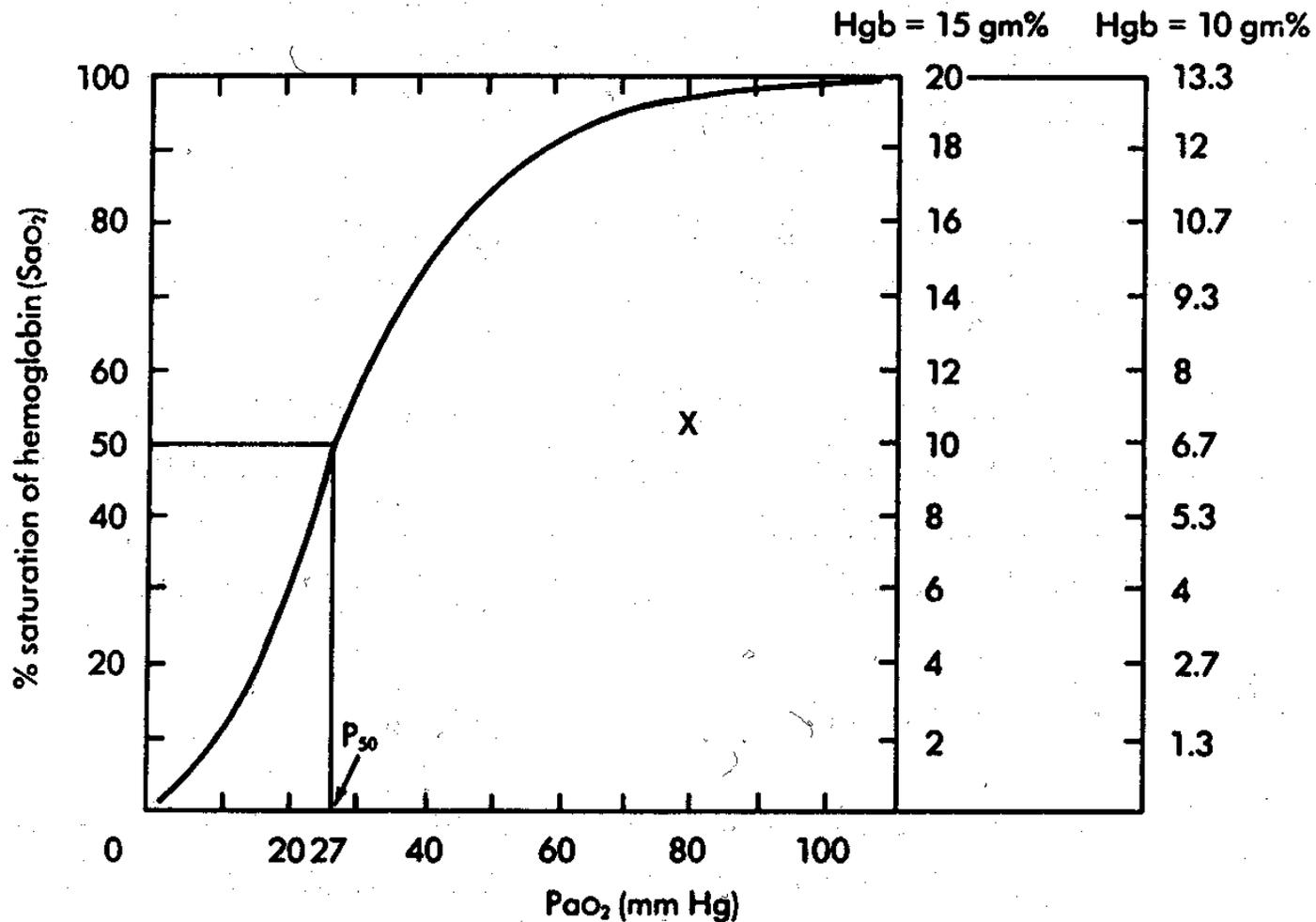
- In chronically ill respiratory patients, mixed disorders are probably more common than single disorders, e.g., RAc + MAlk, RAc + Mac, Ralk + MAlk.
- In renal failure (and other patients) combined MAlk + MAc is also encountered.
- Always be on lookout for mixed acid-base disorders. They can be missed!

Oxygen dissociation curve: SaO_2 vs. PaO_2

Also shown are CaO_2 vs. PaO_2 for two different hemoglobin contents: 15 gm% and 10 gm%.

CaO_2 units are ml O_2 /dl. P_{50} is the PaO_2 at which SaO_2 is 50%.

Point 'X' is discussed on later slide.



Anion Gap

Metabolic acidosis is conveniently divided into elevated and normal anion gap (AG) acidosis. AG is calculated as

$$AG = Na^+ - (Cl^- + CO_2)$$

Note: CO_2 in this equation is the “total CO_2 ” measured in the chemistry lab as part of routine serum electrolytes, and consists mostly of bicarbonate. Normal AG is typically 12 ± 4 mEq/L. If AG is calculated using K^+ , the normal AG is 16 ± 4 mEq/L. Normal values for AG may vary among labs, so one should always refer to local normal values before making clinical decisions based on the AG.