

# Acid-Base & CO<sub>2</sub> Balance

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This Presentation is Approved for  
1 CRCE Credit Hour

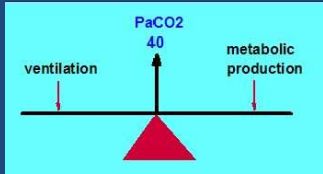
## Learning Objectives

- > Explain the physiology & pathophysiology of carbon dioxide exchange & acid-base balance
- > Determine the ventilatory & acid-base status from blood-gas values

## CO<sub>2</sub> Transportation & Regulation

## CO<sub>2</sub> Production & Excretion

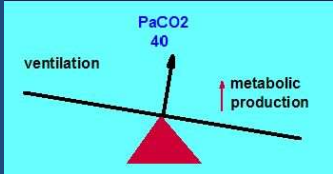
- > Produced with H<sub>2</sub>O as part of glucose metabolism
- > Excreted by ventilation



The diagram shows a horizontal beam balanced on a central red triangular fulcrum. On the left side, a downward arrow is labeled 'ventilation'. On the right side, a downward arrow is labeled 'metabolic production'. In the center, above the fulcrum, an upward arrow is labeled 'PaCO2 40'.

## CO<sub>2</sub> Production & Excretion

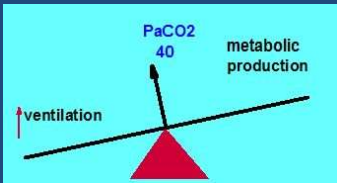
- > Increased metabolism



The diagram shows a seesaw tilted upwards on the right side. The fulcrum is a red triangle. On the left side, a downward arrow is labeled 'ventilation'. On the right side, a downward arrow is labeled 'metabolic production' with a small upward-pointing arrow next to it, indicating an increase. In the center, above the fulcrum, an upward arrow is labeled 'PaCO2 40'.

## CO<sub>2</sub> Production & Excretion

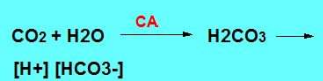
- > Increased ventilation



The diagram shows a seesaw tilted downwards on the left side. The fulcrum is a red triangle. On the left side, a downward arrow is labeled 'ventilation' with a small upward-pointing arrow next to it, indicating an increase. On the right side, a downward arrow is labeled 'metabolic production'. In the center, above the fulcrum, an upward arrow is labeled 'PaCO2 40'.

## CO<sub>2</sub> Transport

- > Mechanisms
  - ❖ Dissolved in plasma ( $PCO_2 \times 0.03$ )
  - ❖ Converted to  $HCO_3^-$ 
    - Greatest amount of  $CO_2$
    - Requires carbonic anhydrase (CA) as catalyst



## CO<sub>2</sub> Transport

- > Mechanisms
  - ❖ Combined with hemoglobin
    - Greatest amount of **exchanged**  $CO_2$
    - $CO_2$ -Hb dissociation increased by increased  $O_2$ -Haldane shift

## CO<sub>2</sub> Transport

- > Total  $CO_2 = HCO_3 + \text{dissolved } CO_2$
- Total  $CO_2$  (mEq/L) =  $24 + (0.03)(40) = 25.2$  mEq/L

## CO<sub>2</sub> Abnormalities

- > Decreased: hyperventilation
  - ❖ Neurogenic hyperventilation
    - Increased intracranial pressure (ICP)
    - Anxiety
  - ❖ Hypoxemia
  - ❖ Compensation for metabolic acidemia

## CO<sub>2</sub> Abnormalities

- > Increased: hypoventilation
  - ❖ Depressed ventilation
    - Ventilatory fatigue
    - Depressant drugs, e.g. anesthetics
    - Neuromuscular dysfunction

## CO<sub>2</sub> Abnormalities

- > Increased: hypoventilation
  - ❖ Depressed ventilation
    - Ventilatory fatigue
    - Depressant drugs, e.g. anesthetics
    - Neuromuscular dysfunction
  - ❖ Increased dead space ventilation
    - Emphysema: loss of alveolar capillaries
    - Tachypnea: decreased tidal volume
    - Massive pulmonary embolism

## CO<sub>2</sub> Abnormalities

- Increased CO<sub>2</sub> production
  - ❖ Increased metabolism
    - Fever
    - Shivering: recovery from hypothermia
    - Seizures: may also cause hypoventilation

## CO<sub>2</sub> Abnormalities

- Increased CO<sub>2</sub>
  - ❖ Excessive glucose intake, e.g. I.V. fluids
  - ❖ Compensation for metabolic alkalemia

## Acid-Base Balance

## Regulators of Acid-Base Balance

- Buffers
- Ventilation
- Renal function

## Regulators of Acid-Base Balance

- Buffers
  - ❖ First to act
  - ❖ No pH change until they are depleted
  - ❖ HCO<sub>3</sub><sup>-</sup> is the most important one
  - ❖ Hemoglobin: second most important

## Regulators of Acid-Base Balance

- Ventilation
  - ❖ Responds almost immediately to pH change
  - ❖ Excretes/retains CO<sub>2</sub> - volatile acid

## Regulators of Acid-Base Balance

- > Kidney
  - ❖ Requires time to respond
  - ❖ Excretes/retains  $\text{HCO}_3^-$  or  $\text{H}^+$  (fixed base, acid)

## Acid-Base Balance

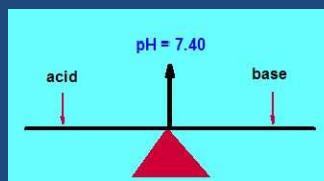
- > Parameters used to interpret
  - ❖ pH - normal = 7.4
  - ❖  $\text{PCO}_2$  - normal = 40
  - ❖  $\text{HCO}_3^-$  - normal = 24

## Acid-Base Balance

- > Parameters used to interpret
  - ❖ Base change - also known as base excess
    - Defined: the quantity of base, in mEq/L, required to normalize the pH, with  $\text{PCO}_2$  adjusted to 40
    - Normal value = 0 (zero)
    - Estimate by subtracting normal  $\text{HCO}_3^-$  (24) from measured  $\text{HCO}_3^-$
    - Used to calculate dosage of bicarbonate to treat acidemia

## Acid-Base Balance

- > Depends on maintaining 20:1 ratio of base:acid (Henderson-Hasselbalch equation)



## Acid-Base Balance

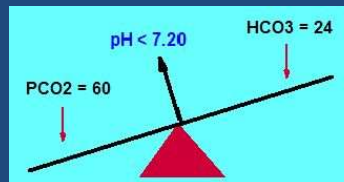
- > Depends on maintaining 20:1 ratio of base:acid (Henderson-Hasselbalch equation)

$$\text{pH} = \text{pKa} + \log (\text{Base}/\text{Acid})$$

FYI see links below to view calculation of pH using the H-H equation

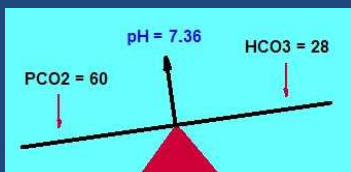
## Respiratory Acidemia

- > Hypoventilation - addition of volatile acid -  $\text{CO}_2$
- > Acute, uncompensated



## Compensated Respiratory Acidemia

- > Chronic hypoventilation
- > Renal retention of  $\text{HCO}_3^-$
- > Occurs over hours-days
- > Rarely fully compensated



## Respiratory Acidemia

- > Management
  - ❖ Increase alveolar ventilation - caution with chronic hypercapnia
    - Rapid reversal is hazardous - alkalemia
    - Complete reversal will delay ventilator weaning
  - ❖ If ventilation cannot be increased, e.g. permissive hypercapnia - Tromethamine (THAM™)

## Metabolic Acidemia

- > Fixed acid excess OR
- > Base deficiency
- > pH does not change until buffers are neutralized
- > Ventilation compensates immediately, unless compromised or controlled
- > Associated with hyperkalemia

## Metabolic Acidemia

- > Causes
  - ❖ Diabetes - ketones
  - ❖ Renal Failure
    - Non-production of  $\text{HCO}_3^-$
    - Failure to excrete acid ions
  - ❖ Hepatic failure: failure to catabolize lactic acid

## Metabolic Acidemia

- > Causes
  - ❖ Diarrhea -  $\text{HCO}_3^-$  loss
  - ❖ Ingestion of acid
  - ❖ Congenital metabolic disease, e.g. maple syrup urine disease (MSUD)

FYI see links below for article on MSUD

## Metabolic Acidemia

- > Causes
  - ❖ Tissue hypoxia - lactic acidemia
    - Severe hypoxemia
    - Shock, e.g. septic shock
    - Nucleoside analogues (HIV meds)
    - Diagnosed with serum lactate measurement

FYI see links below for article on lactic acidemia & nucleoside analogues

## Metabolic Acidemia

- > Anion Gap =  $[Na^+] - ([Cl^-] + [HCO_3^-])$
- > Normal =  $[140 - (100 + 24)] = 16$
- > So what? If the source of acidemia is unclear, the anion gap can narrow the choices.

FYI see links below for more information on anion gap

## Metabolic Acidemia

- > Elevated anion gap acidemia causes
  - ❖ Methanol, metformin (diabetic agents)
  - ❖ Uremia
  - ❖ Diabetic ketoacidosis
  - ❖ Paraldehyde - rarely used
  - ❖ Iron, isoniazid, inhalants (abuse)
  - ❖ Lactic acid
  - ❖ Ethylene glycol, ethanol (alcoholic ketoacidosis)
  - ❖ Salicylates, solvents, starvation

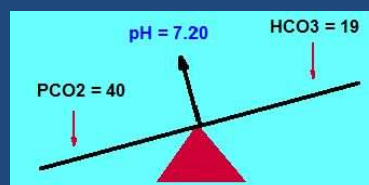
## Metabolic Acidemia

- > Non-anion gap acidemia sources
  - ❖ Gastrointestinal  $HCO_3^-$  loss - diarrhea
  - ❖ Renal failure - renal tubular acidosis
  - ❖ Hyperalimentation
  - ❖ Post-hypocapnea, e.g. normal postnatal maternal condition

FYI see links below for more information on renal acidosis

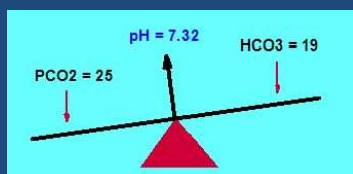
## Uncompensated Metabolic Acidemia

- > Negative base change - estimated =  $(19 - 24) = -5$  mEq/L



## Partly Compensated Metabolic Acidemia

- > Full compensation is rare



## Metabolic Acidemia - Management

- > Do not treat  $pH \geq 7.20$
- > Treat underlying cause
  - ❖ Insulin - ketoacid
  - ❖ Restore oxygenation
  - ❖ Restore perfusion
  - ❖ Restore hemoglobin
  - ❖ Dialysis - renal failure
  - ❖ Withdraw any causative agents
- > Buffer therapy (later)

### Respiratory Alkalemia

- > Acute, uncompensated
- > Hyperventilation

PCO<sub>2</sub> = 20      pH = 7.60      HCO<sub>3</sub> = 23

### Respiratory Alkalemia

- > Chronic hyperventilation: common in late pregnancy
- > Renal compensation: excrete HCO<sub>3</sub><sup>-</sup>
- > Management: treat underlying cause

PCO<sub>2</sub> = 20      pH = 7.47      HCO<sub>3</sub> = 19

### Metabolic Alkalemia - Base Excess

- > Causes
  - ❖ Administration of HCO<sub>3</sub>
  - ❖ Vomiting, nasogastric suctioning
  - ❖ Diuretic therapy

FYI see links below for article on metabolic alkalemia

### Metabolic Alkalemia - Base Excess

- > Causes
  - ❖ Administration of HCO<sub>3</sub>
  - ❖ Vomiting, nasogastric suctioning
  - ❖ Diuretic therapy
- > Consequences
  - ❖ Hypokalemia - tachydysrhythmias
  - ❖ Leftward shift in HbO<sub>2</sub> curve: aggravates hypoxia

### Metabolic Alkalemia

- > Acute, uncompensated

PCO<sub>2</sub> = 40      pH = 7.60      HCO<sub>3</sub> = 31

### Metabolic Alkalemia

- > Partly compensated
- > Hypoventilation to retain CO<sub>2</sub>
- > Results in base excess: estimated (31 - 24) = 7

PCO<sub>2</sub> = 47      pH = 7.48      HCO<sub>3</sub> = 31

## Metabolic Alkalemia

- > Management
  - ❖ Treat underlying cause
  - ❖ Acetazolamide (Diamox)
    - For pH = 7.48 & HCO<sub>3</sub> = 28 mmol/l
    - Single dose = 500 mg

FYI see links below for article on acetazolamide & alkalemia

## Arterial vs. Venous Samples

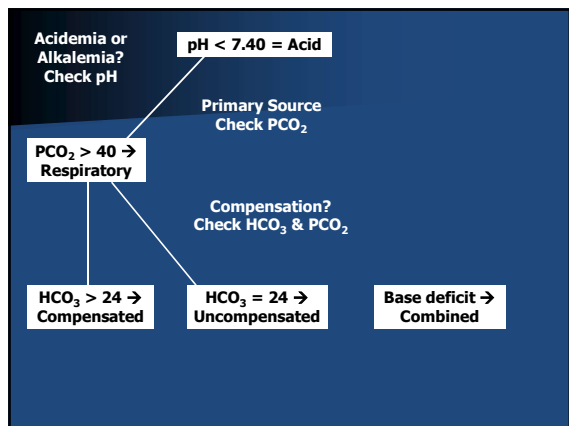
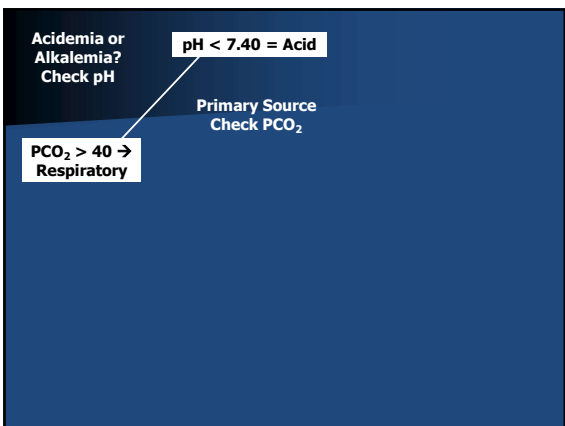
- > Mixed venous samples
  - ❖ Superior to arterial samples in determining
    - Acid-base status, especially during resuscitation
    - Lactate levels
  - ❖ Sites
    - Pulmonary artery
    - Central veins
    - Peripheral vein - emergency

## Arterial vs. Venous Samples

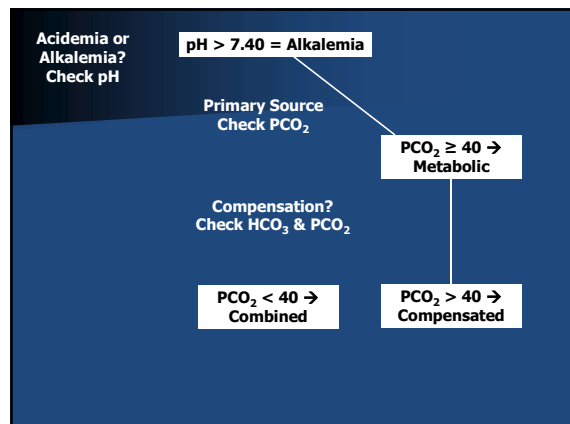
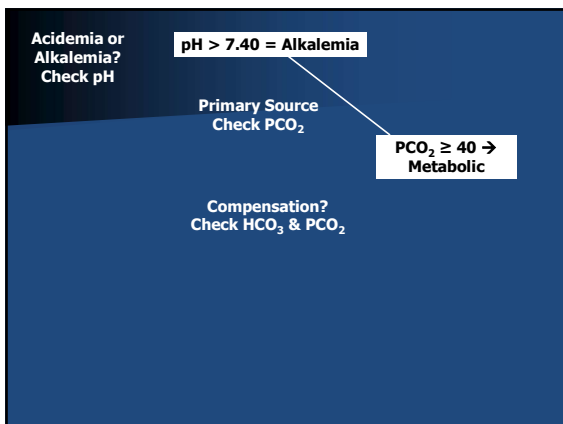
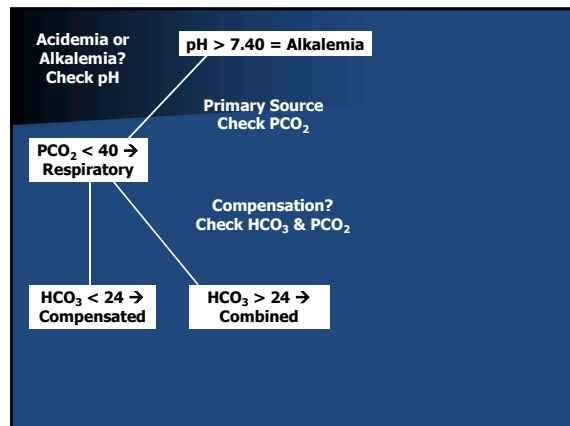
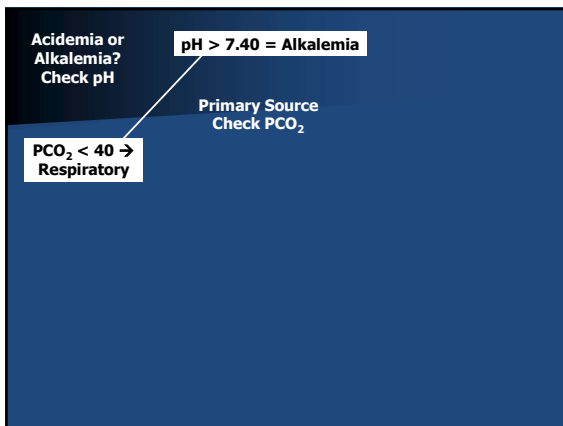
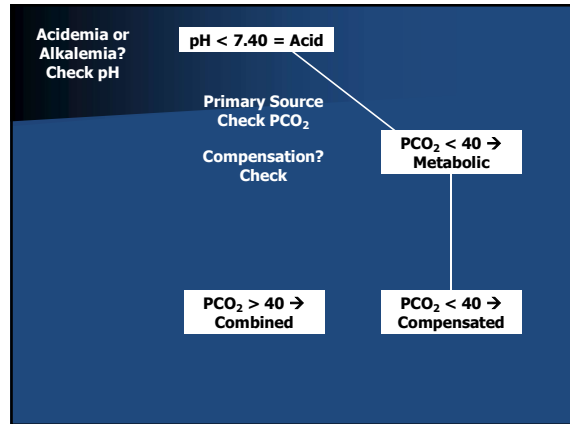
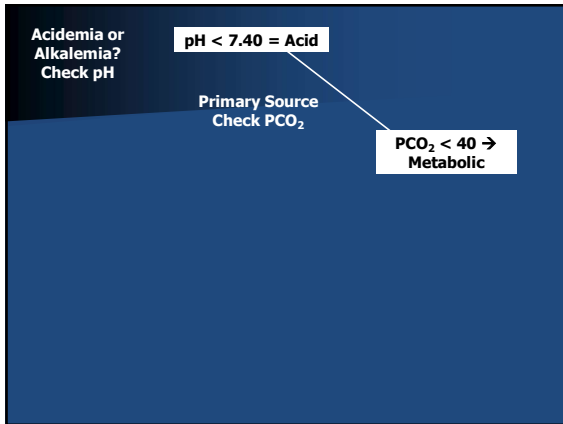
- > Mixed venous normal values

pH	7.36
PCO <sub>2</sub>	44
HCO <sub>3</sub> <sup>-</sup>	28
lactate	1mmol/L

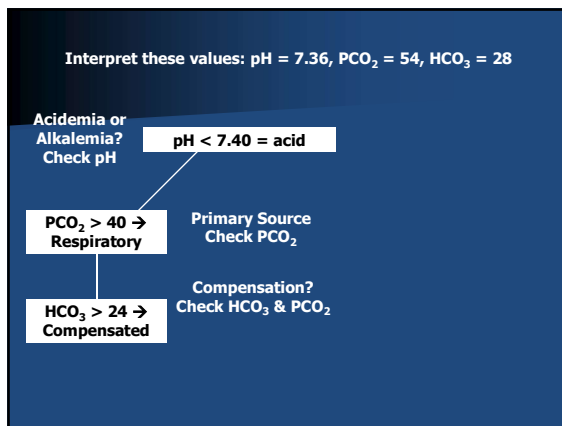
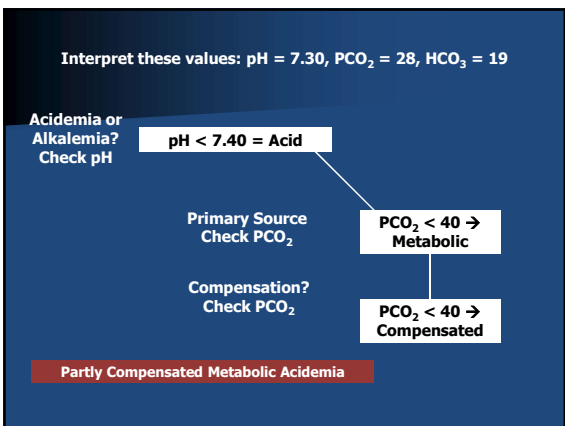
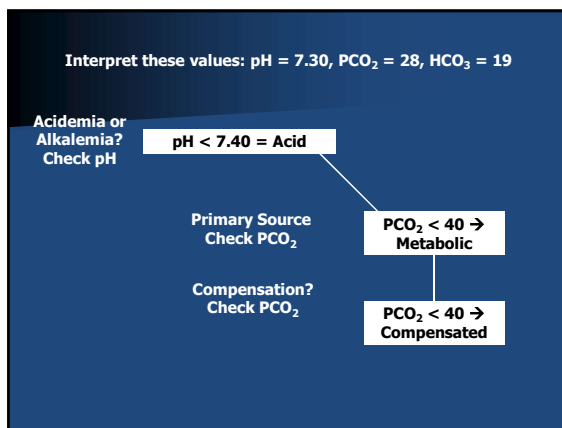
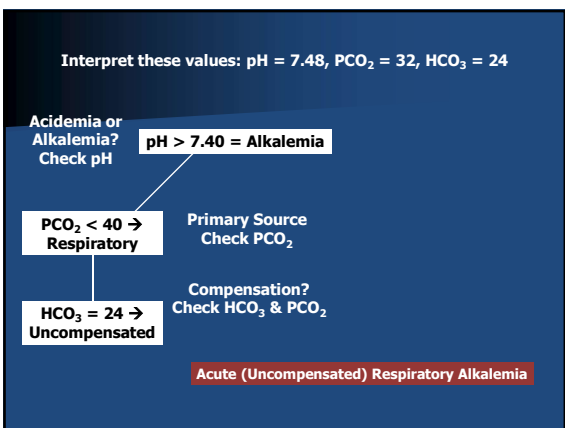
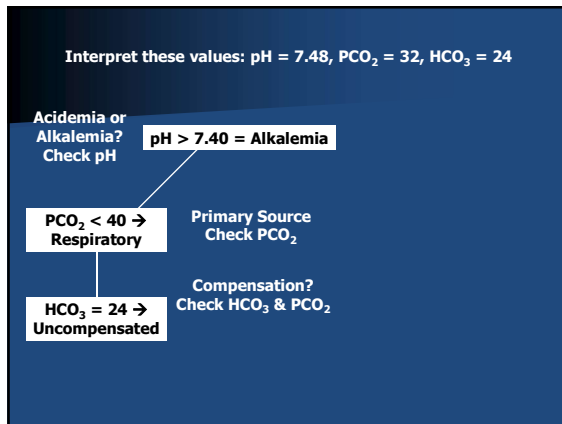
## Acid-Base Balance Algorithm

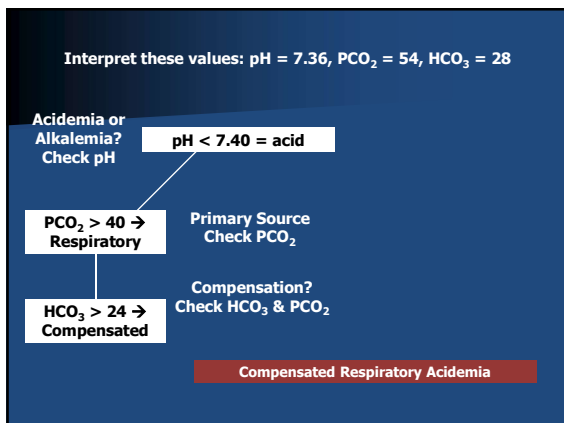






# Interpretation Practice





## Buffer Therapy

### Buffer Therapy

- > Purpose: to reverse acid-base imbalance, usually acidemia
- > NaHCO<sub>3</sub>: action - provides HCO<sub>3</sub><sup>-</sup> → [H<sup>+</sup>] + [HCO<sub>3</sub><sup>-</sup>] → H<sub>2</sub>O + CO<sub>2</sub> → depends on ventilation to excrete CO<sub>2</sub>

### NaHCO<sub>3</sub>- Complications

- > Respiratory acidemia if CO<sub>2</sub> not excreted
- > Metabolic alkalemia (overdose)
- > Hyponatremia
- > Cerebral edema

### NaHCO<sub>3</sub>- Contraindications

- > pH > 7.20
- > Severe hyponatremia

### NaHCO<sub>3</sub>

- > Administration titrated with blood pH
- > Formula for dosage

$$\text{HCO}_3 \text{ (mEq)} = \text{kg} \times (15 - \text{observed HCO}_3^-) \times 0.5$$

## Carbicarb

- > Mixture of  $\text{NaHCO}_3$  &  $\text{NaCO}_3$
- > Buffers without net generation of  $\text{CO}_2$
- > No human trials have been conducted

FYI see links below for article on lactic acidemia management (includes Carbicarb)

## Buffer Therapy

- > Tris-hydroxymethylaminomethane - Tromethamine (THAM™)
- > Reverses acidemia without excretion of  $\text{CO}_2$
- > Action: organic proton acceptor (eats  $\text{H}^+$ )

## Buffer

- > THAM™
  - ❖ Indications
    - Metabolic acidemia with hypernatremia
    - Acidemia in conjunction with limitations in ventilation-permissive hypercapnia

FYI see links below for article on THAM™ & permissive hypercapnia

## Buffer

- > THAM™
  - ❖ Complications
    - Apnea
    - Hypoglycemia
    - Hypokalemia
    - Alkalemia
    - Tissue necrosis from infiltration

## Buffer

- > THAM™
  - ❖ Dosage: ml's of THAM™ of 0.3 M solution = body weight in kg x base deficit in mEq/l

## Buffer

- > Trometamol (Tribonat™)
  - ❖ Currently used in Europe
  - ❖ Ingredients
    - $\text{NaHCO}_3$
    - THAM™
    - Acetate
    - $\text{PO}_4$

## Buffer

- > Trometamol (Tribonat™) advantages
  - ❖ Minimal effect on  $PCO_2$
  - ❖ Minimal overcorrection risk
  - ❖ Less Na than  $NaHCO_3$
  - ❖ No tissue irritability

## O<sub>2</sub> Induced Hypercapnia

- > COPD patients who are CO<sub>2</sub> retainers
- > During exacerbations
- > Underlying causes
  - ❖ VQ Mismatch: increased VDA
  - ❖ Haldane effect: increased release of CO<sub>2</sub> from Hb
- > Maintain  $SPO_2 < 92\%$

See links below for abstract on O<sub>2</sub>-induced hypercapnia

## Summary & Review

- > CO<sub>2</sub> transport & balance
  - ❖ Balance: production vs. excretion
  - ❖ Transport forms & mechanisms
  - ❖ Causes of abnormal  $PCO_2$

## Summary & Review

- > Acid-base balance
  - ❖ Regulators
  - ❖ Parameters & normal values
  - ❖ Abnormalities
    - Values
    - Causes
    - Management
  - ❖ Acid-base algorithm

## Summary & Review

- > Buffer therapy
  - ❖  $NaHCO_3$  - Metabolic acidemia
  - ❖ Carbicarb
  - ❖ THAM™ - metabolic & respiratory acidemia
  - ❖ Trometamol (Tribonat™)
    - Best of both
    - Not available in USA

## References

- > Kallet RH, Jasmer RM, Luce JM, Lin LH, Marks JD. The treatment of acidosis in acute lung injury with tris-hydroxymethyl aminomethane (THAM). *American Journal of Respiratory & Critical Care Medicine*. 161(4 Pt 1):1149-53, 2000.
- > Wilkins RL, Krider SJ, Sheldon RL. *Clinical Assessment in Respiratory Care* (3rd ed.) Chap 6. 1995; Mosby, St. Louis.
- > Scanlon CL, Wilkins RL, Stoller JK. *Egan's Fundamentals of Respiratory Care*, 7th ed. Chap. 12. 1998. Mosby, St. Louis.
- > American Heart. *Association Advanced Life Support* 1997. p 7.15.

## References

- Shapiro BA et al. Clinical application of blood gases (4th ed.). 1989 Year Book, Chicago. Chap. 5.
- Verma A, Schein RM, Jayaweera DT, Kett DH. Fulminant neuropathy & lactic acidosis associated with nucleoside analog therapy. *Neurology*. 1999;53(6):1365-7.
- Luft FC. Lactic acidosis update for critical care clinicians. *J Am Soc Nephrol*. 2001 Feb;12 Suppl 17:S15-9.
- Weil MH, Rackow EC, Trevino R, Grundler W, Falk JL, Griffel MI. Difference in acid-base state between venous & arterial blood during cardiopulmonary resuscitation. *N Engl J Med*. 1986 Jul 17;315(3):153-6.

## References

- Weil MH, Ruiz CE, Michaels S, Rackow EC. Acid-base determinants of survival after cardiopulmonary resuscitation. *Crit Care Med*. 1985 Nov;13(11):888-92.
- Jansen TC, van Bommel J, Schoonderbeek FJ, Sleswijk Visser SJ, van der Klooster JM, Lima AP, Willemsen SP, Bakker J. Early lactate-guided therapy in intensive care unit patients: a multicenter, open-label, randomized controlled trial. *Am J Respir Crit Care Med*. 2010 Sep 15;182(6):752-61.
- Abdo WF, Heunks LM. Oxygen-induced hypercapnia in COPD: myths & facts. *Crit Care*. 2012 Oct 29;16(5):323.