Acid Base Disorders

Introduction

- Acid –base abnormalities are common in the intensive car unit . Most metabolic and physiologic processes in the body require the pH to be within a narrow range of 7.35-7.45 .
- To achieve a normal acid-base balance , the interaction between the respiratory , renal and buffering system should be intact .
- When there is an acid-base abnormality, the buffer system will be the first to respond by altering hydrogen ions.
- The lungs will remove the carbon dioxide and the kidneys will excrete acidic urine .

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Goals

- Review basics of acid base physiology & pathophysiology
- Distinguish between respiratory and metabolic acidosis and alkalosis
- Understand the significance of the “anion gap”
- **MAKE ACID BASE DISORDERS STRAIGHT FORWARD!**

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Definitions

- Acid*emia*: Blood pH < 7.35
- Alkal*emia*: Blood pH > 7.45
- Acidosis:is a process that will result in acidemia if left unopposed.
- Alkalosis:is a process that will result in alkalemia if left unopposed
- Metabolic refers to a disorder that results from a primary alteration in [H+] or [HCO3-].
- Respiratory refers to a disorder that results from a primary alteration in PCO2 due to altered CO2 elimination.
- Normal HCO3- 24 meq/L; Normal PCO2 40 mm Hg ; Normal pH 7.35-7.45

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Definitions (Continued)

PH: - is a negative logarithm of Hydrogen ion concentration; and it is the initials of these two wards (puissance Hydrogen) that mean the power of hydrogen

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Definitions (Continued)

An acid: - is a hydrogen ion or proton donor, and a substance which causes a rise in H+ concentration on being added to water.

A base: - is a hydrogen ion or proton acceptor, and a substance which causes a rise in OH- concentration when added to water.

Strength of acids or bases refers to their ability to donate and accept H+ ions respectively.

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Importance of acid-base balance

- The hydrogen ion (H+)concentration must be precisely maintained within a narrow physiological range

- ***Small changes from normal can produce marked changes in enzyme activity & chemical reactions within the body***

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- Acidosis - CNS depression, coma (pH ~ 6.9)
- Alkalosis - CNS excitability, tetany, siezures
- Hydrogen ion concentration is most commonly expressed as pH (= negative logarithm of the H+ concentration)

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ACID-BASE CALCULATIONS

- The Henderson equation is easier to use, but only applies when pH is between 7.2 and 7.6. For this equation, one must calculate [H+] from pH. [H+] = 40 nEq/L when pH is 7.4. The [H+] increases 10 nEq/L for a 0.1 unit drop in pH.
- Henderson Eq.
  $[H^+] = 24 \times PCO_2 / [HCO_3^-]$

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ph	H+ (nmol/l)
7	100
7.1	80
7.2	63
7.3	50
7.36	44
7.4	40
7.44	36
7.5	32
7.6	25
7.7	20

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Normal pH:

- **Arterial** blood: 7.35 - 7.45
- **Venous** blood, interstitial fluid: 7.35
- **Intracellular**: 6.0-7.4 (average 7.0)

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Regulation of pH

- *Buffer systems - very rapid (seconds), incomplete
- *Respiratory responses - rapid (minutes), incomplete
- *Renal responses - slow (hours to days), complete

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Background

$H^+ + H^+CO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow H_2O + CO_2$

⦿-Metabolic Disorders:

Affect HCO_3^-: (Normal 22-26 meq/L)

-Metabolic Acidosis

– Acid neutralizes $HCO_3^- \Rightarrow \downarrow H^+CO_3^-, \downarrow$ pH

-Metabolic Alkalosis

- $\uparrow H^+CO_3^-$ Production Drives Rxn to Right $\Rightarrow$
- $\downarrow H^+ \Rightarrow \uparrow$ pH

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Background

⦿ $H^+ + H^+CO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow H_2O + CO_2$

⦿ Respiratory Disorders:

- Disorders that affect pCO_2. Normal 35-45 mm Hg

- Respiratory Acidosis

  $\uparrow pCO_2 \Rightarrow$ Drives Rxn to Left $\Rightarrow \uparrow H^+ \Rightarrow \downarrow$ pH

- Respiratory Alkalosis

  $\downarrow pCO_2 \Rightarrow$ Drives Rxn to Right $\Rightarrow \downarrow H^+ \Rightarrow \uparrow$ pH

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Primary Abnormality in Acid Base Disorders

	Acidosis	Alkalosis
Respiratory	↑ pCO_2	
Metabolic		

$$H^+ + H^+CO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow H_2O + CO_2$$

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Primary Abnormality in Acid Base Disorders

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Metabolic		

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Primary Abnormality in Acid Base Disorders

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Metabolic	$\downarrow H^+CO_3^-$	

$$H^+ + H^+CO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow H_2O + CO_2$$

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Primary Abnormality in Acid Base Disorders

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Respiratory	$\uparrow pCO_2$	$\downarrow pCO_2$
Metabolic	$\downarrow H^+CO_3^-$	$\uparrow H^+CO_3^-$

$$H^+ + H^+CO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow H_2O + CO_2$$

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Simple Acid-Base Disorders:

Type of Disorder	pH	$PaCO_2$	$[HCO_3]$
Metabolic Acidosis	↓	↓	↓
Metabolic Alkalosis			
Acute Respiratory Acidosis			
Chronic Respiratory Acidosis			
Acute Respiratory Alkalosis			
Chronic Respiratory Alkalosis			

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Metabolic Alkalosis	↑		↑
Acute Respiratory Acidosis			
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Acute Respiratory Alkalosis			
Chronic Respiratory Alkalosis			

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Acute Respiratory Alkalosis			
Chronic Respiratory Alkalosis			

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Simple Acid-Base Disorders:

Type of Disorder	**pH**	**$PaCO_2$**	**$[HCO_3]$**
Metabolic Acidosis			
Metabolic Alkalosis			
Acute Respiratory Acidosis	↓	↑	
Chronic Respiratory Acidosis	↓	↑	
Acute Respiratory Alkalosis			
Chronic Respiratory Alkalosis			

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Simple Acid-Base Disorders:

Type of Disorder	**pH**	**$PaCO_2$**	**$[HCO_3]$**
Metabolic Acidosis			
Metabolic Alkalosis			
Acute Respiratory Acidosis	↓	↑	↑
Chronic Respiratory Acidosis	↓	↑	↑↑
Acute Respiratory Alkalosis			
Chronic Respiratory Alkalosis			

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Simple Acid-Base Disorders:

Type of Disorder	**pH**	**$PaCO_2$**	**$[HCO_3]$**
Metabolic Acidosis			
Metabolic Alkalosis			
Acute Respiratory Acidosis			
Chronic Respiratory Acidosis			
Acute Respiratory Alkalosis	↑	↓	
Chronic Respiratory Alkalosis	↑	↓	

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Simple Acid-Base Disorders:

Type of Disorder	**pH**	**$PaCO_2$**	**$[HCO_3]$**
Metabolic Acidosis			
Metabolic Alkalosis			
Acute Respiratory Acidosis			
Chronic Respiratory Acidosis			
Acute Respiratory Alkalosis	↑	↓	↓
Chronic Respiratory Alkalosis	↑	↓	↓↓

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Simple Acid-Base Disorders:

Type of Disorder	**pH**	**$PaCO_2$**	**$[HCO_3]$**
Metabolic Acidosis	↓	↓	↓
Metabolic Alkalosis	↑	↑	↑
Acute Respiratory Acidosis	↓	↑	↑
Chronic Respiratory Acidosis	↓	↑	↑↑
Acute Respiratory Alkalosis	↑	↓	↓
Chronic Respiratory Alkalosis	↑	↓	↓↓

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Compensation

- For each acid-base disorder , there is a compensatory response mediated by the kidneys or the lungs that tends to bring the pH back towards normal.

- Compensation is never complete (i.e. pH never returns to 7.4). Therefore if the pH < 7.4, the primary process is an acidosis. If the pH > 7.4 the primary process is an alkalosis

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Compensated Abnormality in Acid Base Disorders

	Acidosis	Alkalosis
Respiratory Compensation	↑ pCO_2 ↑ $H^+CO_3^-$	↓ pCO_2 ↓ $H^+CO_3^-$
Metabolic Compensation	↓ $H^+CO_3^-$ ↓ pCO_2	↑ $H^+CO_3^-$ ↑ pCO_2

$$H^+ + H^+CO_3^- \Leftrightarrow H_2CO_3 \Leftrightarrow H_2O + CO_2$$

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Compensation (Continued)

- Formulas predict normal compensation in both acute and chronic conditions.
- Inadequate compensation tells you that something else is wrong!
- Metabolic compensation takes time and is more complete in chronic conditions than acutely

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Buffer Systems

- A substance that can prevent major changes in the pH of body fluids by removed or releasing hydrogen ions ,they can act quickly to prevent excessive changes in hydrogen ion concentration

- Bicarbonate, phosphate and protein buffering systems are the three major buffering systems

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Bicarbonate buffer system

*Primary extracellular buffer system (>50% of extracellular buffering)

*Accurate assessment - readily calculated from *PCO*2 and pH using available blood gas machines

*Consists of carbonic acid (weak acid) and bicarbonate

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*CO2 regulated by the lungs - rapidly

*HCO3- is regulated by the kidneys – slowly

*Not powerful

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Protein buffer system

*Most powerful

*75 % of all intracellular buffering

*Hemoglobin

-important extracellular buffer due to large concentration of hemoglobin in blood

-buffering capacity varies with oxygenation

-reduced hemoglobin is a weaker acid than oxyhemoglobin

-dissociation of oxyhemoglobin results in more base available to combine w/ H+

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Plasma protein

*acid buffer

*important intracellular buffer system

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Phosphate buffer system

*H2PO4- and HPO42-

*important renal buffering system

*extracellular concentration, 1/12 that of bicarbonate

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Respiratory Responses

- occurs within minutes of alteration in pH due to stimulation/depression of respiratory centers in the CNS

- H+ acts directly on respiratory center in Medulla Oblongata

- alveolar ventilation increases/decreases in response to changes in CO_2

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Renal Responses

- the kidneys regulate pH by either acidification or alkalinization of the urine
- complex response that occurs primarily in the proximal renal tubules
- with acidosis, rate of H+ secretion exceeds HCO3- filtration
- with alkalosis, rate of HCO3- filtration exceeds H+ secretion
- occurs over hours/days, and is capable of nearly complete restoration of acid/base balance

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Renal & Respiratory Compensation

Primary Disorder	Primary change	Predicted Compensatory Response
Metabolic acidosis	↓ HCO3	1.2 ↓ PaCO2 per 1 meg ↓ HCO3
Metabolic Alkalosis	↑ HCO3	.7 ↑ PaCO2 per 1meq ↑ HCO3
Respiratory acidosis: Acute	↑PaCO2	1 meq ↑ HCO3 per 10 mm ↑PaCO2
Respiratory acidosis: Chronic	↑PaCO2	3.5 meq ↑ HCO3 per 10 mm ↑PaCO2
Respiratory alkalosis: Acute	↓PaCO2	2 meq ↓HCO3 per 10mm ↓ PaCO2
Respiratory alkalosis: Chronic	↓PaCO2	4 meq ↓HCO3 per 10mm ↓ PaCO2

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SUMMARY OF SIMPLE ACID-BASE DISORDERS AND COMPENSATION

Primary Acid-Base Disorder	Primary Defect	Effect on pH	Compensatory Response	Expected Range of Compensation	Limits of Compensation
Respiratory Acidosis	Hypoventilation (↑PCO2)	↓	HCO3- Generation	↑ [HCO3-] = 1-4 mEq/L for each 10 mm Hg ↑ PCO2	[HCO3-] = 45 mEq/L
Respiratory Alkalosis	Hyperventilation (↓PCO2)	↑	HCO3- Consumption	↓ [HCO3-] = 2-5 mEq/L for each 10 mm Hg ↓ PCO2	[HCO3-] = 12-15 mEq/L
Metabolic Acidosis	Loss of HCO3- or gain of H+(↑ HCO3-)	↓	Increase in Ventilation (↓PCO2)	PCO2 = 1.5[HCO3-] + 8	PCO2 = 12-14 mm Hg
Metabolic Alkalosis	Gain of HCO3- or loss of H+ (↓ HCO3-)	↑	Decrease in Ventilation (↑PCO2)	↑ PCO2 = 0.6 mm Hg for each 1 mEq/L ↑ [HCO3-]	PCO2 = 55 mm Hg

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GENERAL ASPECTS OF ACID-BASE DISORDERS

- A primary alteration in [H+], [HCO3-] or PCO2 results in abnormal pH.
- The body has several mechanisms to correct pH towards the normal range.

-In the acute phase (minutes to hours), the extra- and intra-cellular buffer systems (most importantly the bicarbonate system) minimize the pH changes.

- In the chronic phase (hours to days), renal or respiratory compensation partially or completely restore pH towards normal.

- There are limits to both types of compensation.
- Compensation does not result in over correction of pH.

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DATA REQUIRED TO DIAGNOSE ACID-BASE DISORDERS

- An arterial blood gas shows the blood pH, PCO2 and [HCO3-].
- A chemistry panel shows the [total CO2], [Cl-], [K+] and [Na+], [glucose], [BUN] and [creatinine].
- The [total CO2] is the sum of the measured [CO2] + [HCO3-]. Thus the [HCO3-] from the blood gas and the [total CO2] from the electrolyte panel usually are within 2 mEq/L. Otherwise the measurements are in error or were taken at different times.

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NORMAL LABORATORY VALUES

Arterial Blood Gas:

pH	7.35-7.45
[H+]	35-45 nmol/L or neq/L
PCO2	35-45 mm Hg
[HCO3-]	22-26 mmol/L or mEq/L

Plasma Electrolytes

[Na+]	135-145 mEq/L
[K+]	3.5-5.0 mEq/L
[Cl-]	96-109 mEq/L
[total CO2]	24-30 mEq/L

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SIMPLE ACID-BASE DISORDERS

- Simple acid-base disorders have one primary abnormality.

- The four primary disorders are respiratory acidosis, respiratory alkalosis, metabolic acidosis and metabolic alkalosis.

- Mixed acid-base disorders have more than one abnormality. Two to three primary disorders can be combined together to result in a mixed disorder.

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Metabolic Acidosis

- Secondary to
  - ↑ Acid production or
  - ↑ $H^+CO_3^-$ loss
- Characterized by low serum $H^+CO_3^-$

(by hyperventilation ⇔ ↓ PCO2 ⇒ ↓ HCO3-)

- Divided into two categories:
  - Anion gap metabolic acidosis (High anion gap)
  - NonAnion gap metabolic acidosis (Normal anion gap)

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Anion Gap

- The anion gap is the difference between primary measured cations (sodium $Na^+$and potassium K^+) and the primary measured anions (chloride Cl^- and bicarbonate $HCO3^-$) in serum.
- This test is most commonly performed in patients who present with altered mental status, unknown exposures, acute renal failure, and acute illnesses

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Anion Gap

Serum Anion Gap = $[Na^+]$ - ($[H^+CO_3^-]$ + $[Cl^-]$)

- The normal serum anion gap is 8-16 mEq/L
- The anion gap can also be used in urine using the following equation:
- Urine anion gap = $Na^+ + K^+ - Cl^-$

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- For the urine anion gap, the most prominently unmeasured anion is ammonia.
- Healthy subjects typically have a gap of 0 to slightly normal (< 10 mEq/L).
- A urine anion gap of more than 20 mEq/L is seen in metabolic acidosis when the kidneys are unable to excrete ammonia (such as in renal tubular acidosis).
- If the urine anion gap is zero or negative but the serum AG is positive, the source is most likely gastrointestinal (diarrhea or vomiting).

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A decreased anion gap (< 6 mEq/L)

- May suggest the following:
- Hypoalbuminemia
- Plasma cell dyscrasia
- Monoclonal protein
- Bromide intoxication
- Normal variant

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A normal anion gap (6-12 mEq/L)

- May indicate the following
- Loss of bicarbonate (ie, diarrhea)
- Recovery from diabetic ketoacidosis
- Ileostomy fluid loss
- Carbonic anhydrase inhibitors (acetazolamide, dorzolamide, topiramate)
- Renal tubular acidosis
- Arginine and lysine in parenteral nutrition
- Normal variant

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An elevated anion gap (>12 mEq/L; “mud pilers”)

- May indicate the following:
- Methanol
- Uremia
- Diabetic ketoacidosis
- Propylene glycol
- Isoniazid intoxication
- Lactic acidosis
- Ethanol ethylene glycol
- Rhabdomyolysis/renal failure
- Salicylates

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Unmeasured anions which accumulate ➔ Anion Gap Acidosis

- Lactate
- Ketones
- Sulfates and phosphates
- Other organic acids

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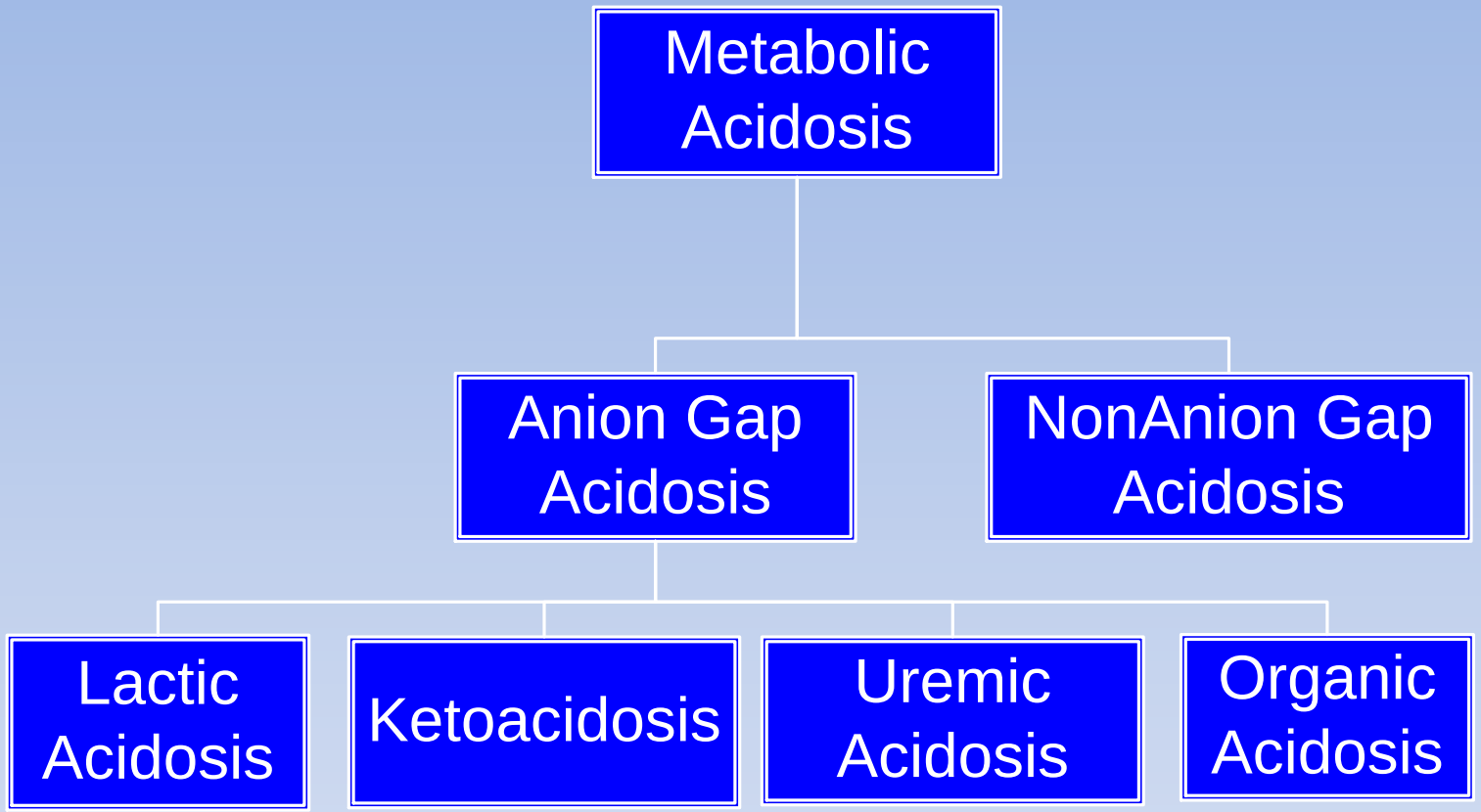

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Lactic Acidosis

- Fundamentally what causes a lactic acidosis?
- Answer: Anaerobic metabolism

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The differential diagnosis of Lactic Acidosis

- Lactic acidosis occurs whenever the cells are unable to utilize aerobic respiration: i.e. whenever the cells are unable to obtain or utilize oxygen
- Consider Murphy's law: "Whatever can go wrong will go wrong!" (i.e. take each step in oxygen absorption and distribution—any one of them can go away and cause lactic acidosis.)

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The differential diagnosis of Lactic Acidosis

- Low environmental O2
- Inability to absorb O2
- O2 unable to bind Hg
- Unable to pump O2
- Tissues unable to utilize O2
- High altitude
- Lung Disease
- CO poisoning
- Shock (cardiogenic)
- Septic shock
- Focal vascular obstruction
- Cyanide poisoning

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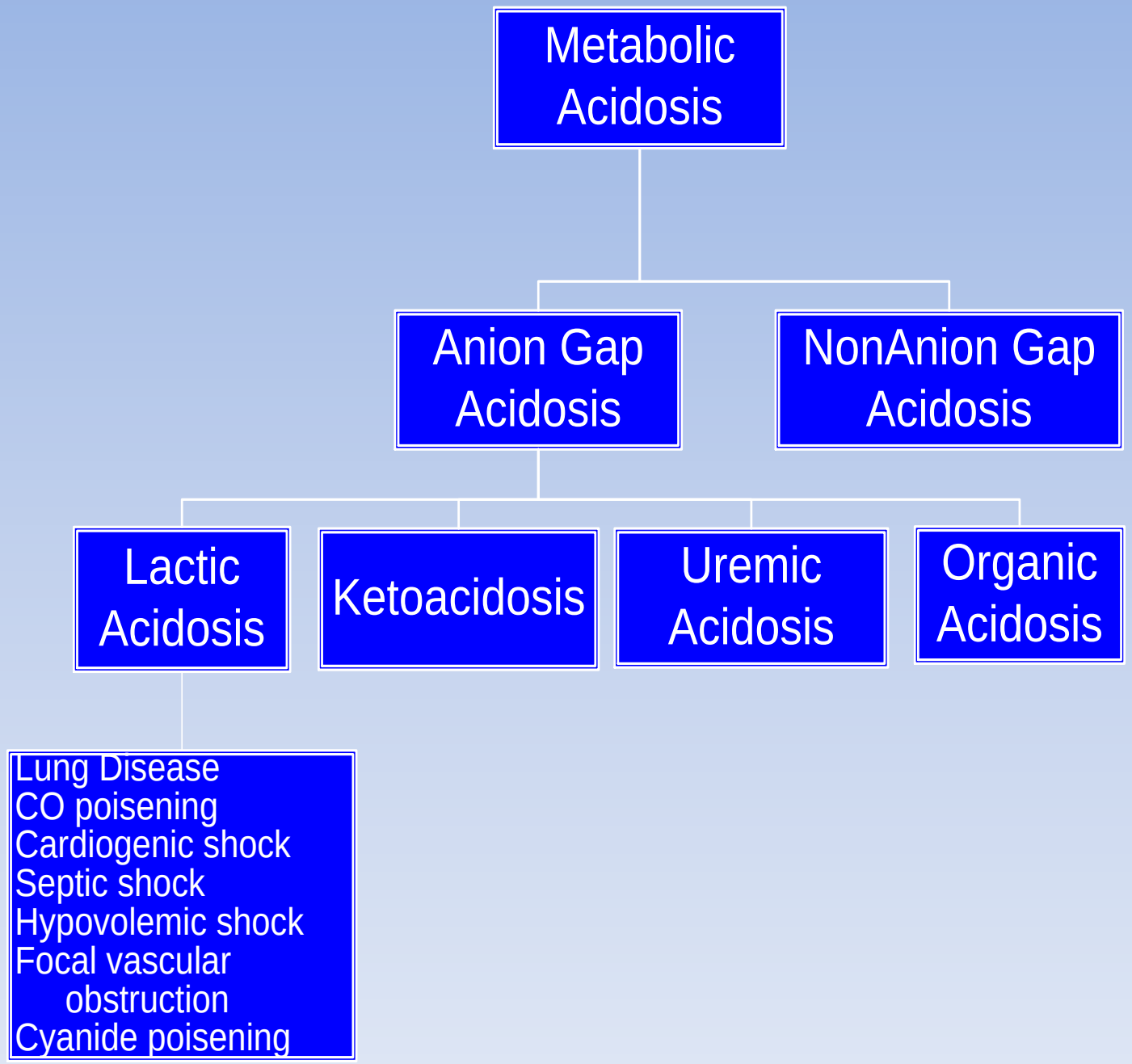

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KetoAcidosis

- Occurs whenever the cells are unable to utilize glucose

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KetoAcidosis

- Three etiologies
  - Diabetic Ketoacidosis
    - Primarily in type 1 diabetes mellitus
    - Severe, life threatening
    - Often associated with precipitating illness
  - Starvation ketoacidosis
    - Mild acidosis
  - Alcoholic ketoacidosis
    - Mild acidosis

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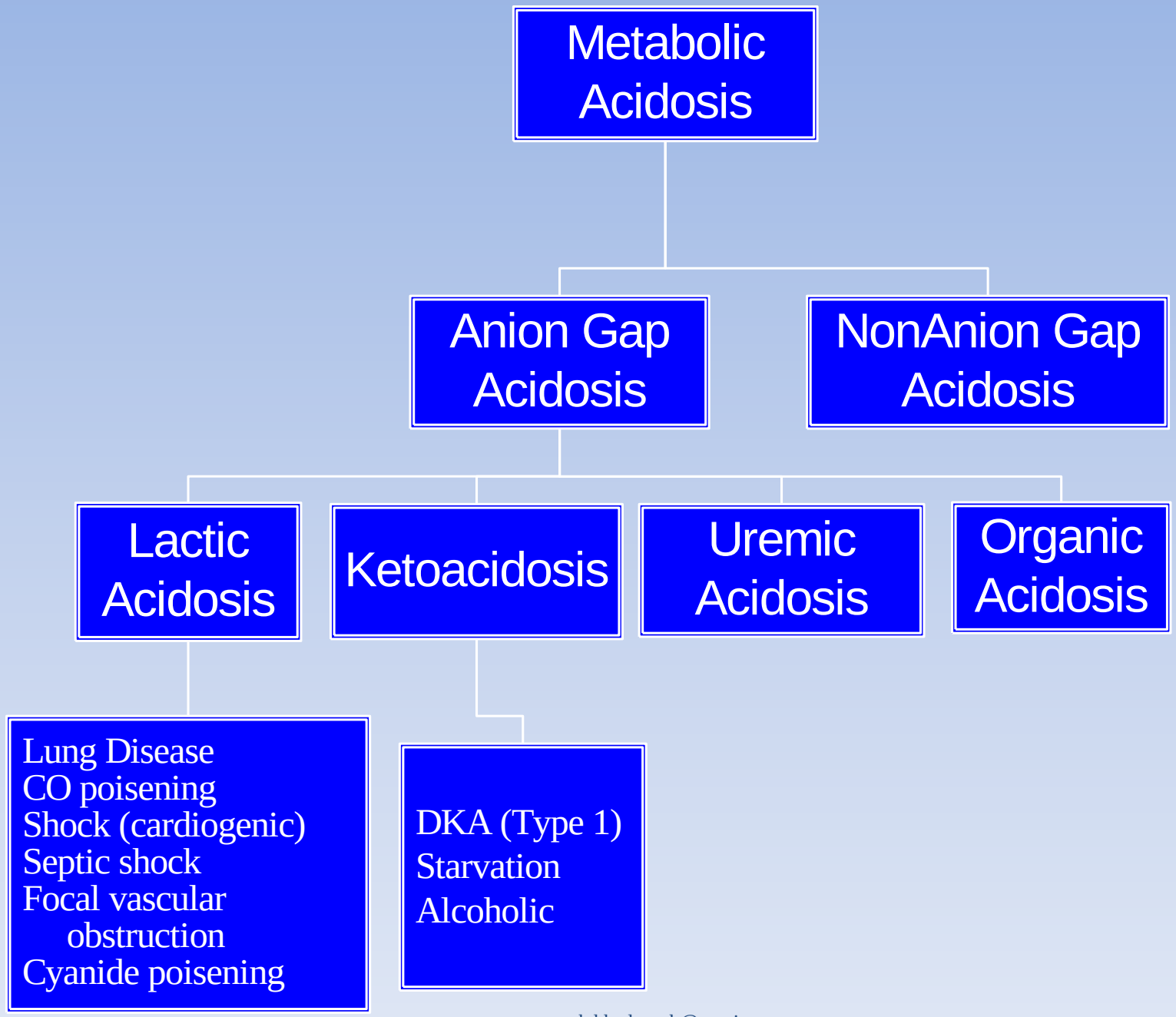

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Other anion gap acidosis

- Uremia
  - Failure to excrete daily metabolic acid load
  - Accumulation of phosphates and sulfates
- Organic acidosis
  - Methanol
  - Ethylene Glycol
  - Salicylates

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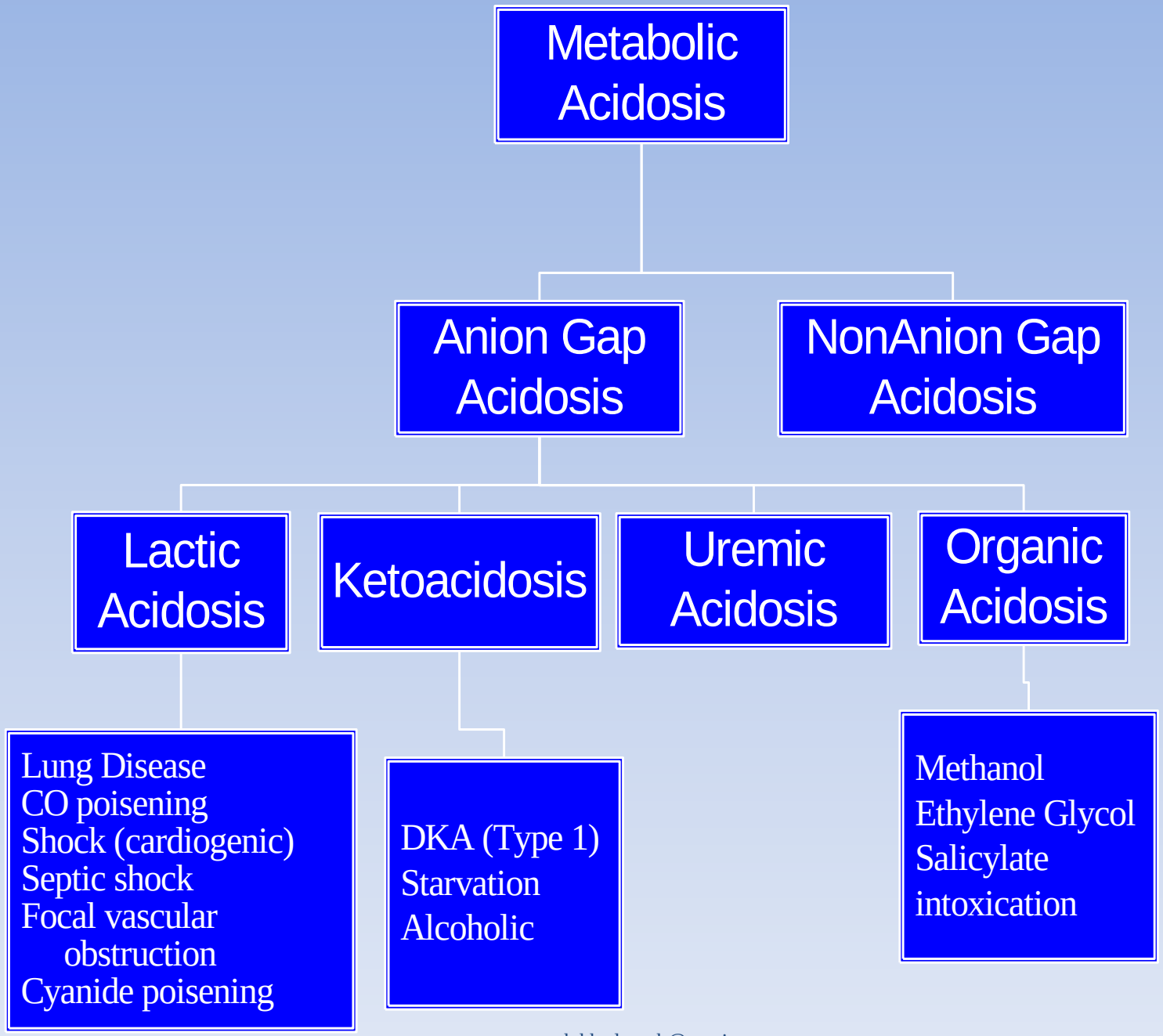

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Non-anion gap metabolic acidosis

- ↑ Bicarbonate loss
  - Diarrhea
  - Severe Burns
  - Urinary loss (renal tubular acidosis)

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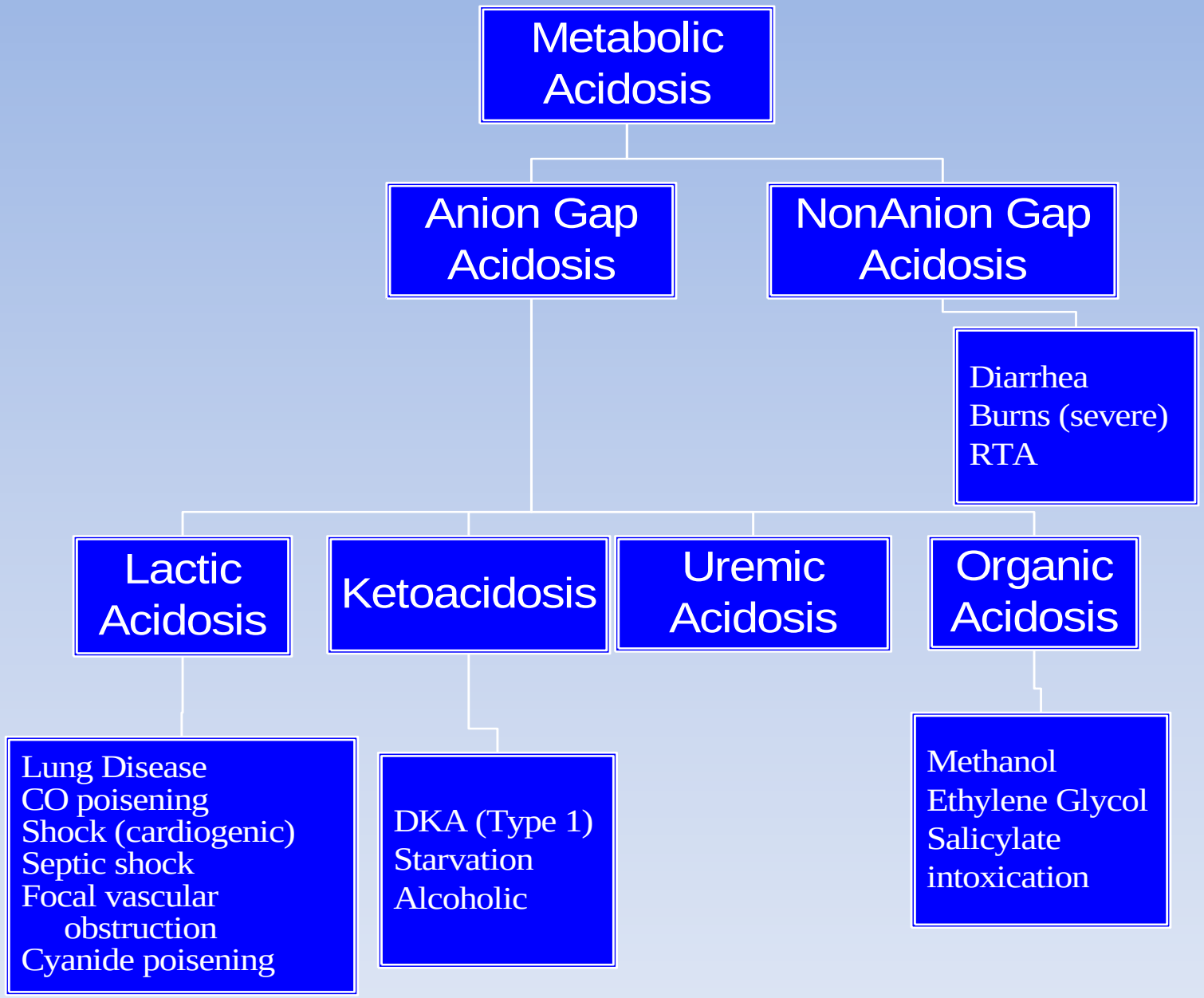

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Clinical Manifestation

- Headache
- Confusion
- Drowsiness
- ↑ RR and depth
- Nausea and vomiting
- Peripheral vasodilation and decreased Cardiac output (pH ↓7)
- ↓BP
- Hyperkalemias

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Metabolic Acidosis: Treatment

- Treat underlying cause
- Alkali replacement
  - Acute metabolic acidosis
    - indicated when is pH less than ~7.15
    - goal is to raise serum [HCO_3] to ~15mmol/L
    - bicarbonate dose =

      $0.5 \times BW\ (kg) \times \{[HCO_3]_{desired} - [HCO_3]_{actual}\}$
  - Chronic metabolic acidosis
    - goal of treatment is to prevent long term sequelae
    - serum [HCO_3] should be normalized

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Metabolic Alkalosis

Generation

Maintenance

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Metabolic Alkalosis: Generation

- Acid loss
  - renal acid losses
    - diuretic therapy
    - mineralocorticoid excess
    - Cushing's syndrome
    - Severe potassium depletion
  - gastrointestinal losses
    - gastric acid loss
    - chloride diarrhea

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Metabolic Alkalosis: Maintenance

- Decreased GFR
  - renal failure
- Increased proximal HCO_3^- reabsorption
  - chloride depletion
- Increased distal tubular H^+ secretion
  - hypokalemia

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Metabolic Alkalosis: Treatment

- Saline responsive
  - intravascular volume expansion with normal saline
  - potassium repletion
- Saline resistant
  - potassium repletion
  - mineralocorticoid antagonists
  - acetazolamide

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Respiratory Acidosis

- Think “Murphy’s Law” again
- From Brain to alveolus, many problems can cause hypoventilation ➔ ↑ $PaCO_2$ ➔ ↓pH (Respiratory acidosis)

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Respiratory Acidosis

- Brain
- Spinal Cord
- Peripheral Nerve
- NeuroMuscular Junction
- Lung and Pleural disease

- Stroke
- Drug Intoxication
- C spine injury,
- Guillan Barre
- Myasthenia Gravis
- Asthma, COPD, ARDS, etc

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Clinical Manifestation

- Hypyrcapnia
- ↑Pulse
- ↑RR
- ↑BP
- Mental cloudiness
- Feeling of fullness in the head
- ↑ICP
- Headache
- Hyperkalemia

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Respiratory Alkalosis

- Hyperventilation ➔ ↓PaCO2 ➔ ↑ pH
- Etiologies
  - Fever
  - Pain
  - Anxiety
  - Pulmonary disease
  - Sepsis
  - Salicylate intoxication
  - Neurologic disorders

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MIXED ACID-BASE DISORDERS

- Mixed acid-base disorders include all combinations of 2-3 simple acid base disorders.

- One must be able to recognize mixed acid-base disorders. This can be accomplished by examining the degree of compensation and calculating an anion gap.

- If the pH, PCO2 and [HCO3-] do not fit the rules of compensation for a simple disorder, one must hypothesize that there is a mixed acid-base disorder (or hypothesize that there is an error in the data).

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- If there is extreme acidemia or alkalemia, one could hypothesize multiple acid-base disorders that that are additive.

- If there is a mild acidemia or alkalemia, or pH is normal, particularly with an anion gap one could hypothesize multiple acid-base disorders that cancel each other out.

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Summary of the Approach to ABGs

1. Check the pH
2. Check the pCO_2
3. Select the appropriate compensation formula
4. Determine if compensation is appropriate
5. Check the anion gap
6. If the anion gap is elevated, check the delta-delta
7. If a metabolic acidosis is present, check urine pH
8. Generate a differential diagnosis

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Putting it Together

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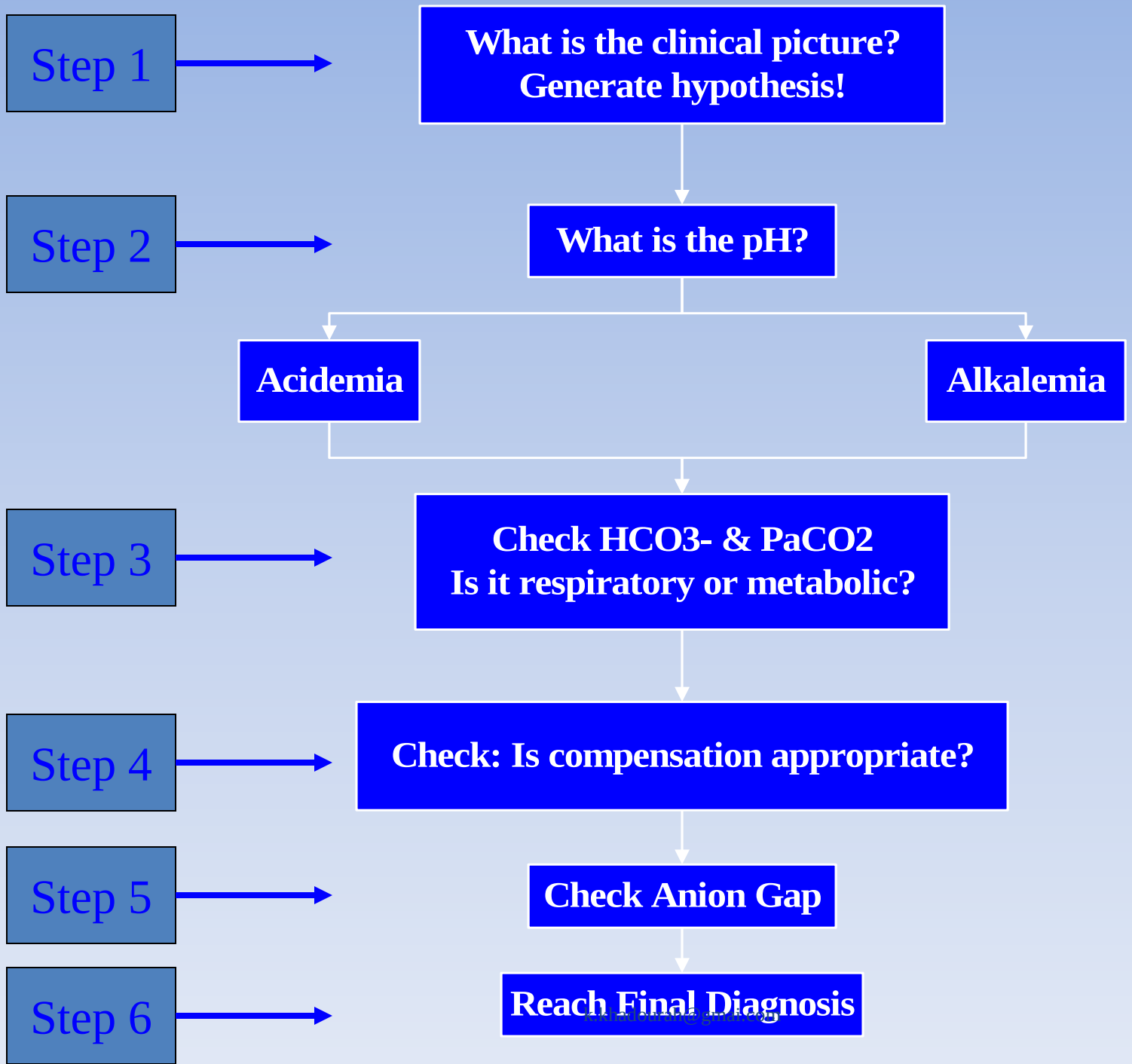
Step 1
What is the clinical picture?
Generate hypothesis!
Step 2
What is the pH?
Acidemia
Alkalemia
Step 3
Check HCO3- & PaCO2
Is it respiratory or metabolic?
Step 4
Check: Is compensation appropriate?
Step 5
Check Anion Gap
Step 6
Reach Final Diagnosis

pH7.34, PaCO2 60 , HCO3- 31

Primary Disorder	Primary change	Predicted Compensatory Response
Metabolic acidosis	↓ HCO3	1.2 ↓ PaCO2 per 1 meg ↓ HCO3
Metabolic Alkalosis	↑ HCO3	.7 ↑ PaCO2 per 1meq ↑ HCO3
Respiratory acidosis: Acute	↑PaCO2	1 meq ↑ HCO3 per 10 mm ↑PaCO2
Respiratory acidosis: Chronic	↑PaCO2	3.5 meq ↑ HCO3 per 10 mm ↑PaCO2
Respiratory alkalosis: Acute	↓PaCO2	2 meq ↓HCO3 per 10mm ↓ PaCO2
Respiratory alkalosis: Chronic	↓PaCO2	4 meq ↓HCO3 per 10mm ↓ PaCO2

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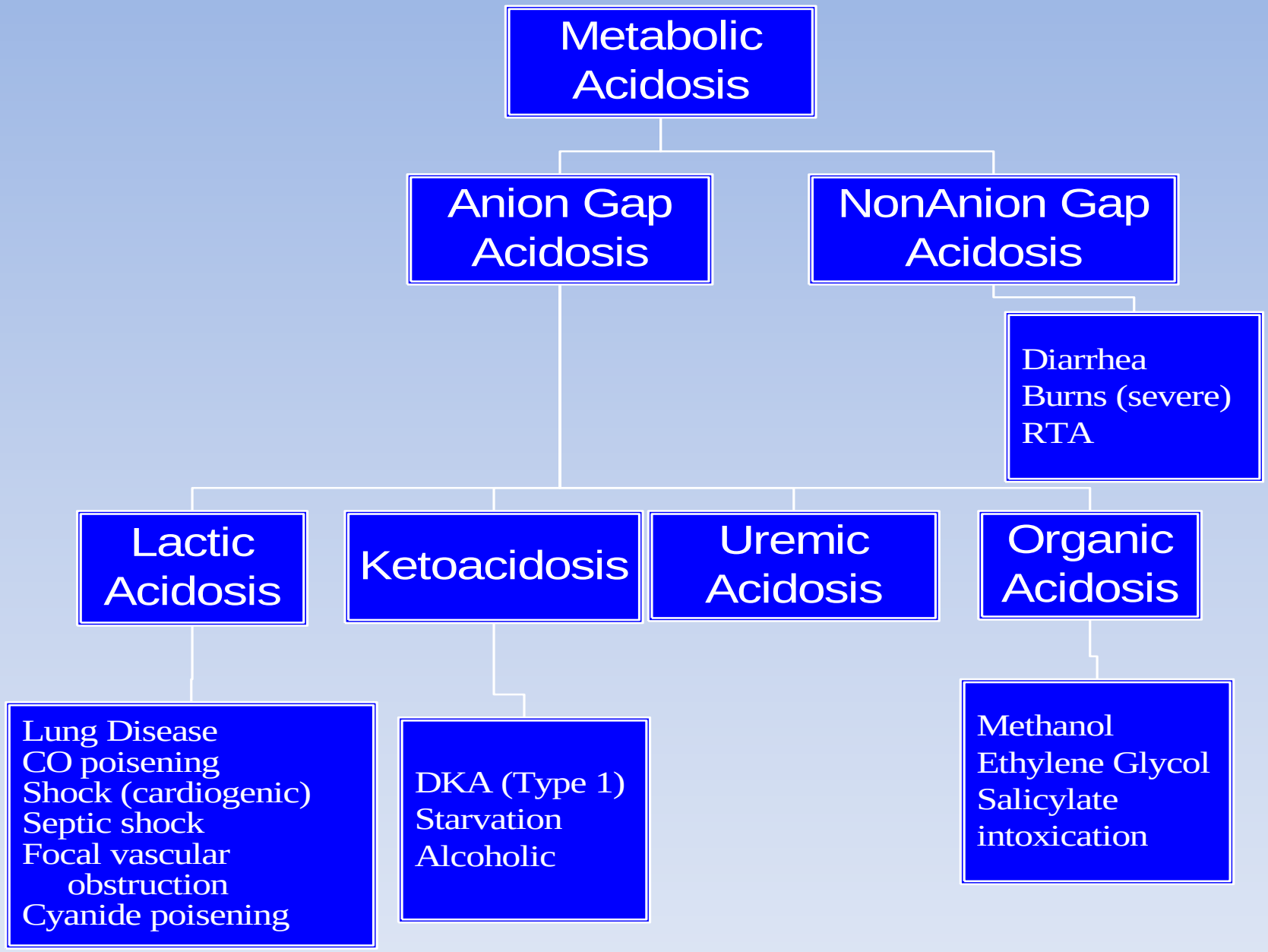

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Who gets your last ICU bed?

- ♦♦ 75 y.o. WF with COPD with CC cough & SOB
  - R.A. ABG ➔ 7.35, PaC02 60, Pa02 48.

- ♦♦ 70 y.o. WM with COPD with CC purulent sputum, SOB.

  ABG on 4L ➔ 7.2, PaC02 60, Pa02 of 70

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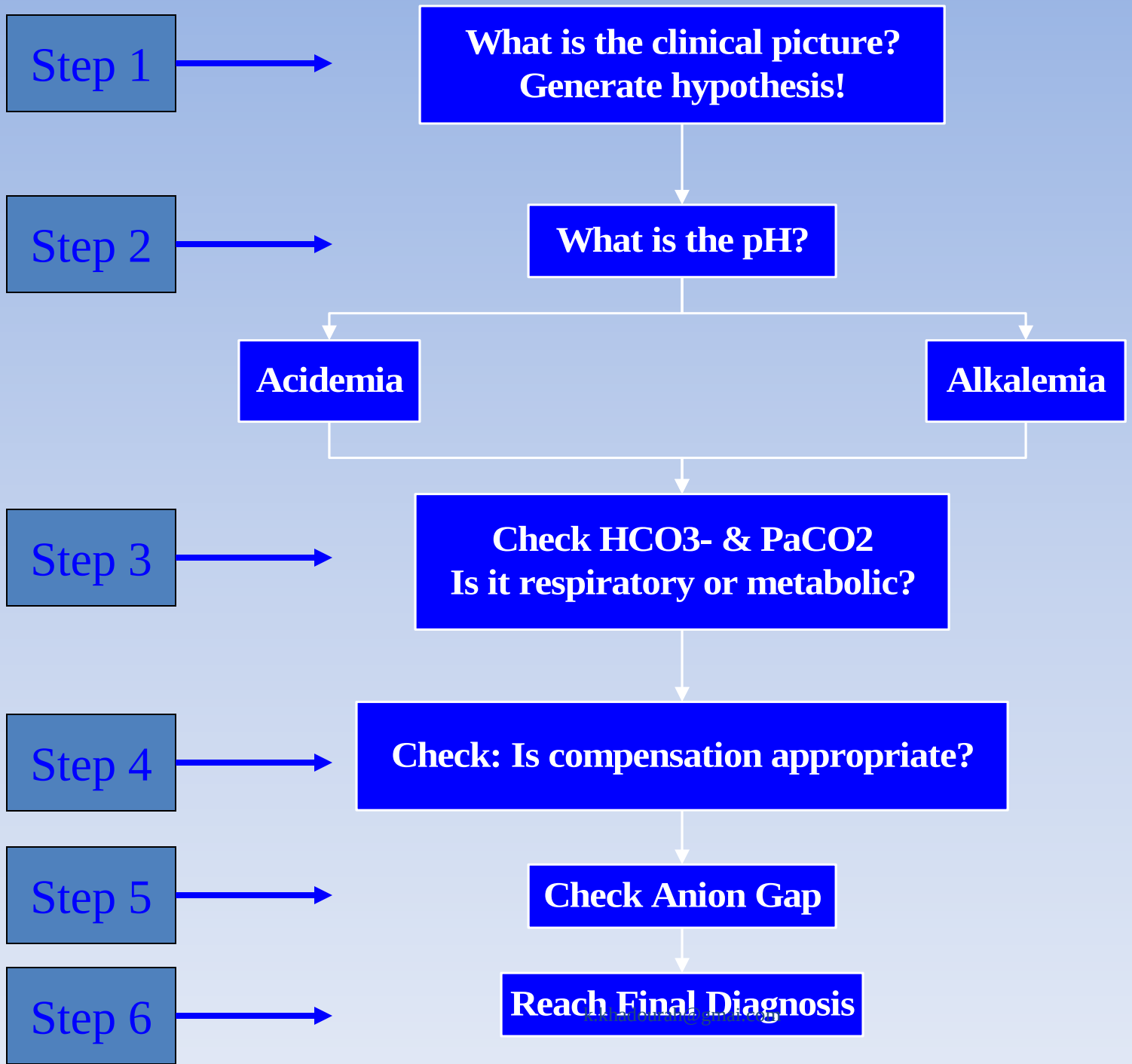
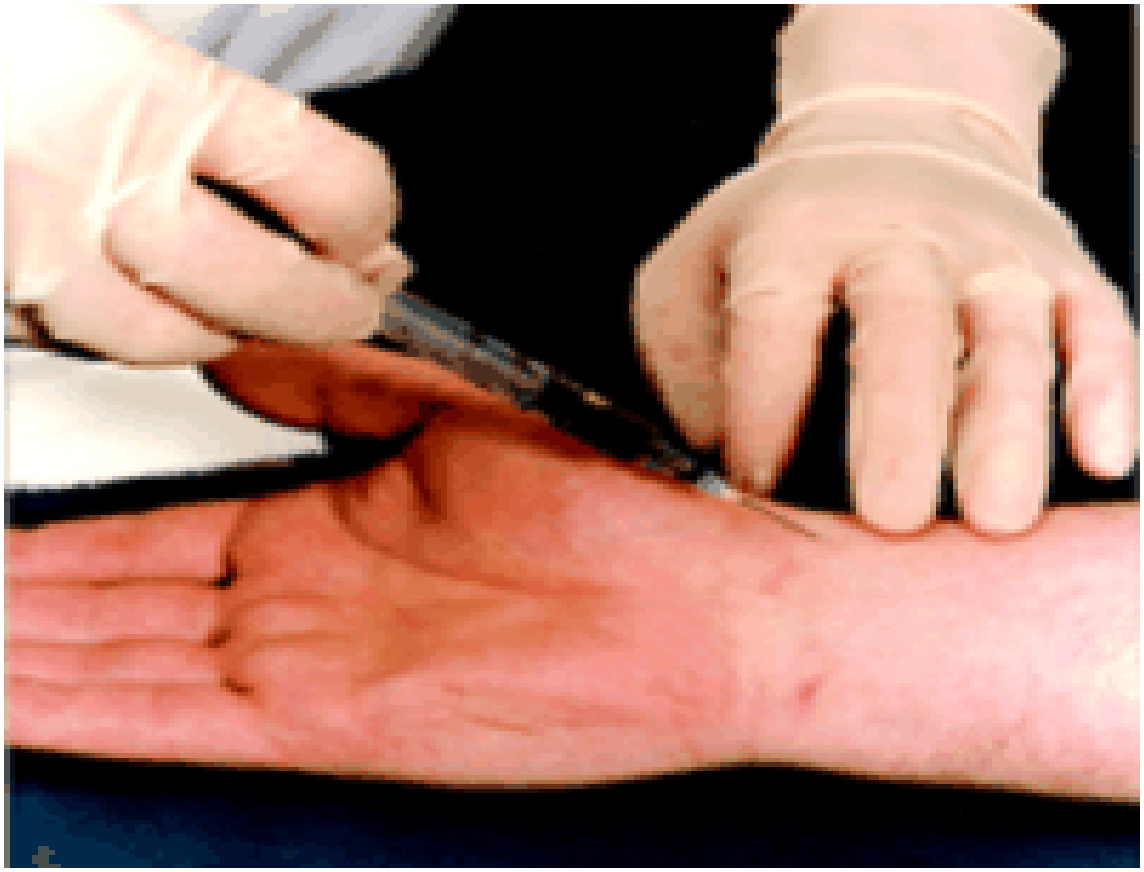

Step 1
What is the clinical picture?
Generate hypothesis!
Step 2
What is the pH?
Acidemia
Alkalemia
Step 3
Check HCO3- & PaCO2
Is it respiratory or metabolic?
Step 4
Check: Is compensation appropriate?
Step 5
Check Anion Gap
Step 6
Reach Final Diagnosis

Arterial puncture



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Problems of taking arterial blood samples

- Bleeding
- Vessel obstruction
- Infection

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Allen's test

- The radial and ulnar arteries are occluded by firm pressure while the fist is clenched.
- The hand is opened and the arteries released one at a time to check their ability to return blood flow to the hand.

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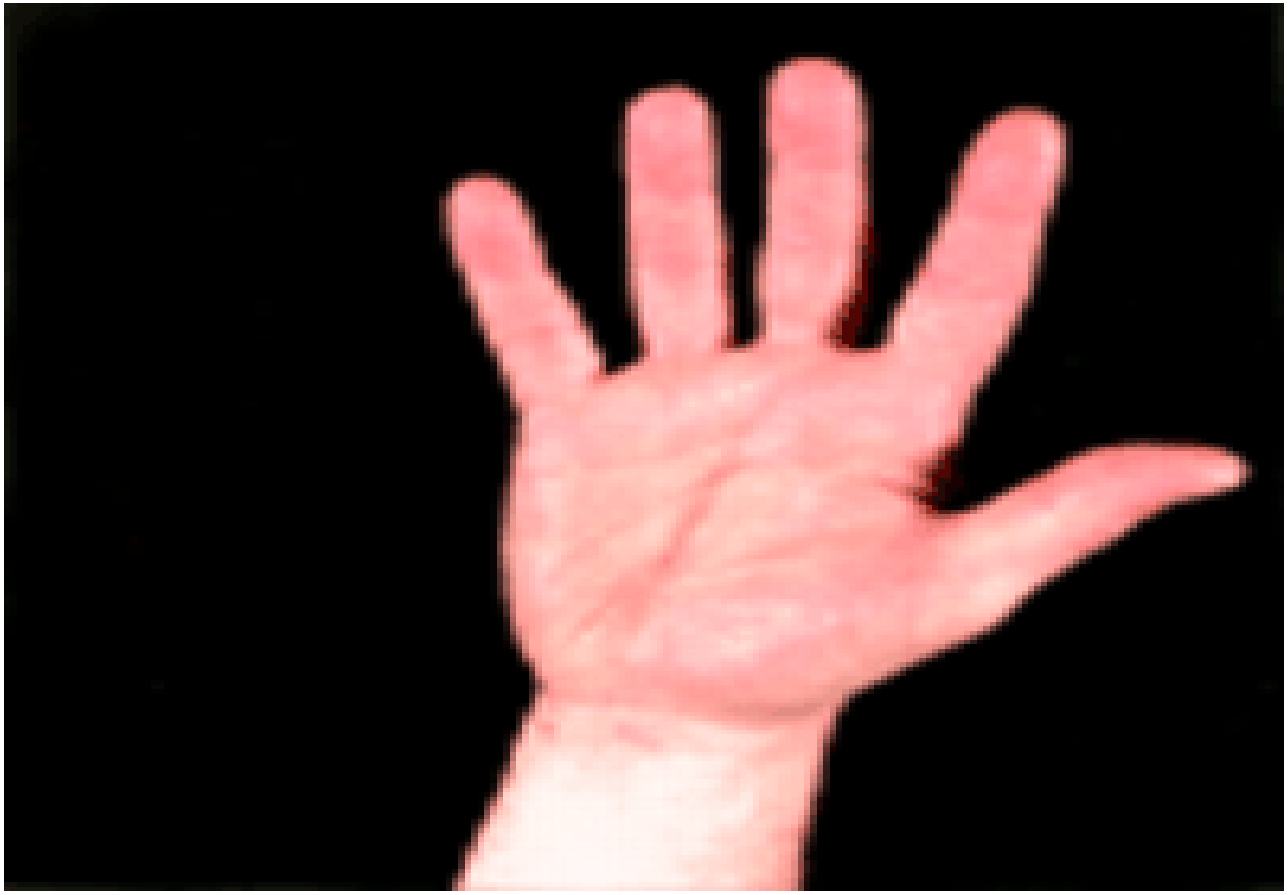

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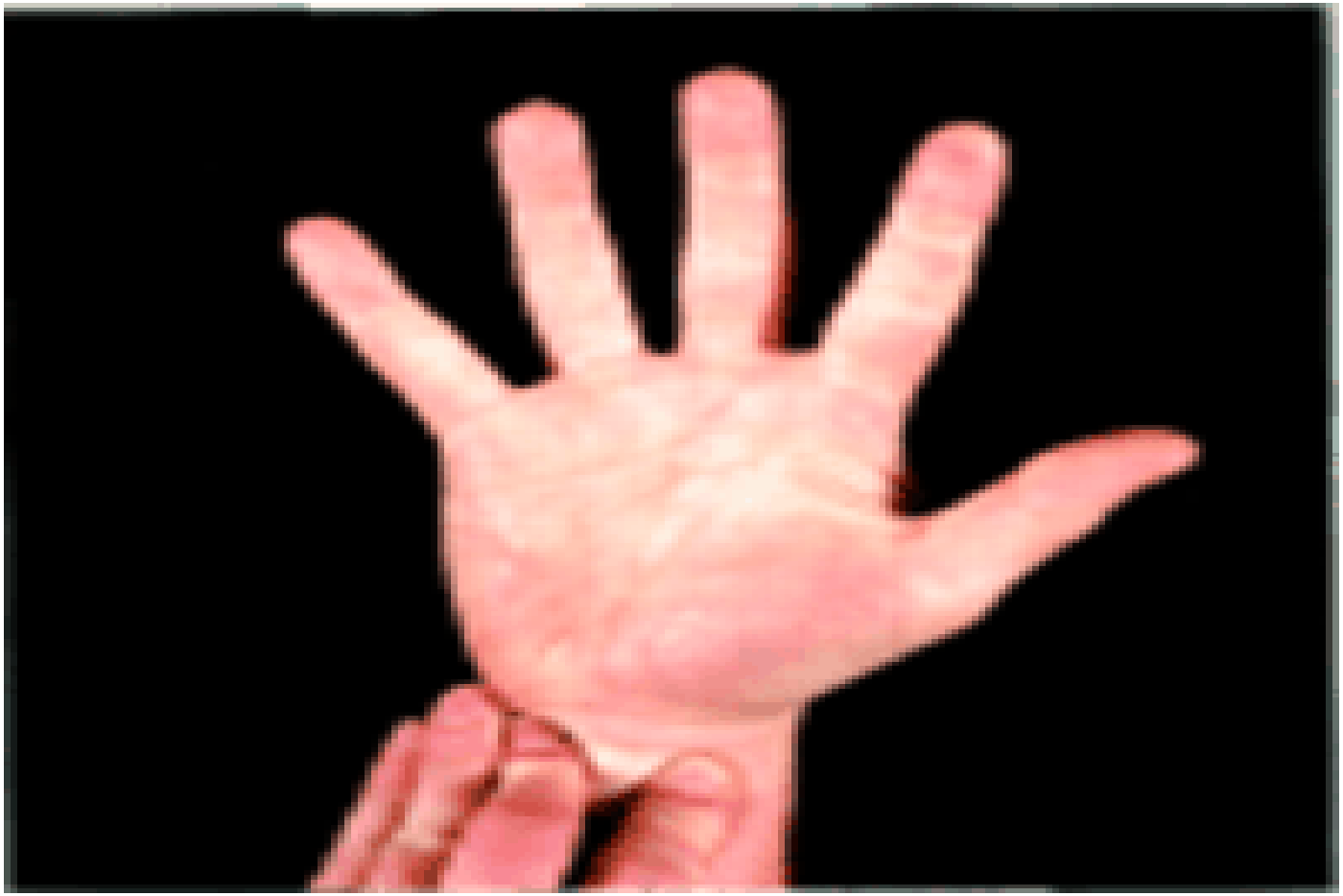

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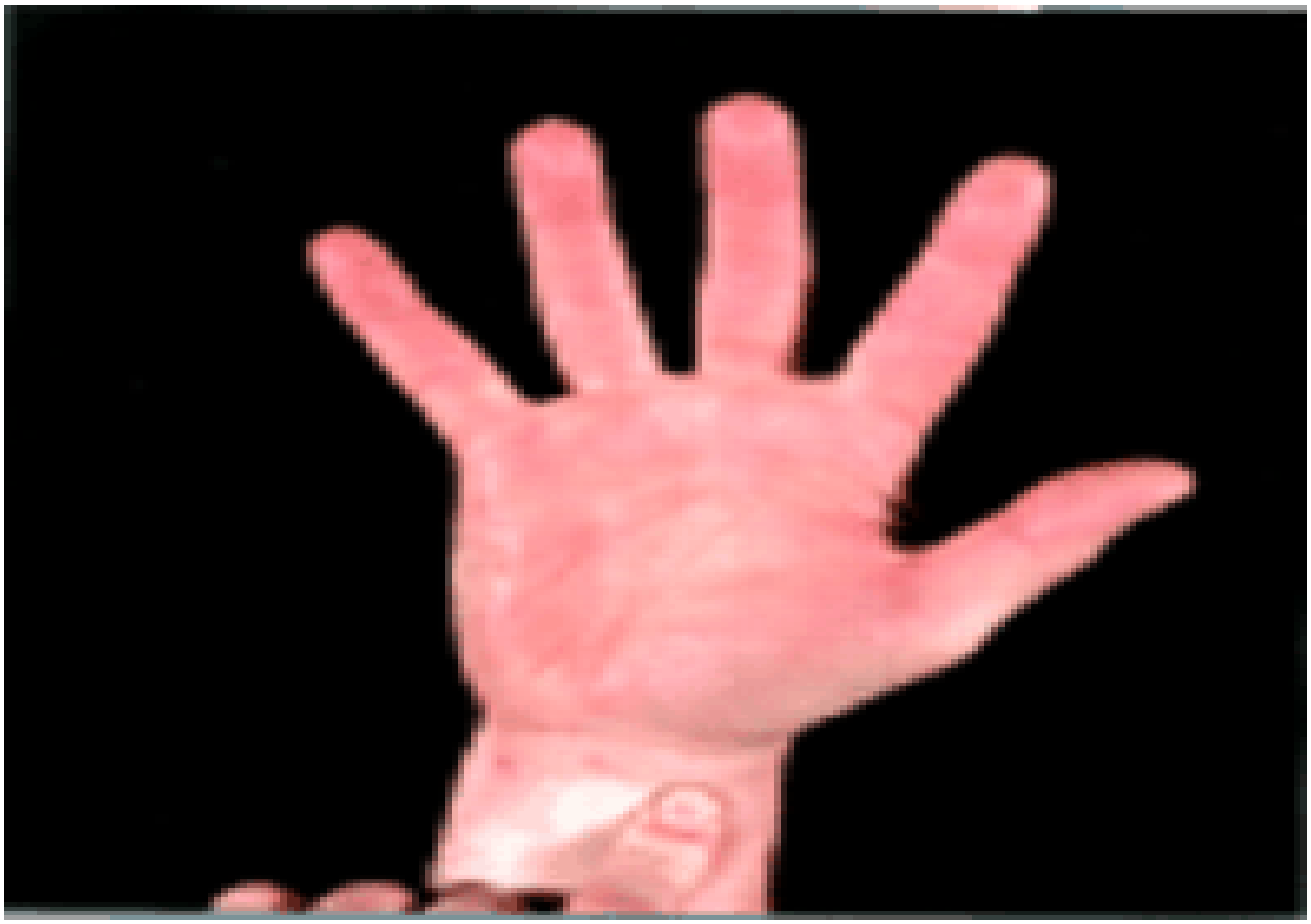

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