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Blood Gas Interpretation

Advanced Care Paramedicine

Module: 12
Section: 02b

Arterial Blood Gases

- An ABG is a blood test that measures the levels of many different gases in the blood
- Some people find this method of drawing blood to be more painful than the more standard method
- Because the test requires oxygen-rich blood, the blood sample must be taken from an artery
  - radial, brachial, femoral
  - Caution should be taken with patient on anticoagulants

- Used to
  - Assess oxygenation status
  - Acid-base balance
  - Response to ventilatory assistance and oxygen therapy

ABG

- Information obtained
  - pH — $[H^+]$
  - pCO_2 — Partial pressure CO_2
  - pO_2 — Partial pressure O_2
  - HCO_3^- — Bicarbonate
  - BE — Base excess (or deficit)
    - Excess usually indicates metabolic alkalosis
    - Deficit usually indicates metabolic acidosis
  - SaO_2 — Oxygen Saturation (from monitor)
  - Anion Gap — Calculated value

Base Excess

- Base excess is a surplus amount of base (alkali) within the blood
- Normal range: –2 to +2 mEq/L
  - Positive BE (greater than +2) this indicates there has been a gain of a base (or a loss of an acid) due to non-respiratory causes.
  - Negative BE (less than -2) indicates a loss of base (or a gain of acid) due to a non-respiratory cause.

Anion Gap

- Other obtainable information from the ABG in conjunction with normal blood work
- Difference between Na^+ and Cl^- and HCO_3^- in the extracellular fluid
  - $[Na^+]+[\text{other cations}] = [Cl^-] + [HCO_3^-]+[\text{other anions}]$
  - $[\text{other anions}]-[\text{other cations}]=[Na^+]-([Cl^-]+[HCO_3^-])$
  - Anion Gap = $Na^+-(Cl^- + HCO_3^-)$
- Useful particularly in the differential diagnosis of acid/base disorders

Anion Gap

- Mnemonic MUDPILES identifies most common causes (metabolic acidosis)
  - M: Methanol
  - U: Uremia
  - D: Diabetic ketoacidosis (DKA)
  - P: Paraldehyde
  - I: Infection
  - L: Lactic acidosis
  - E: Ethylene glycol
  - S: Salicylates

Methanol

- CNS depressant
  - Also known as methyl alcohol
  - 90 to 95 percent of methanol in blood metabolized
  - Metabolized by hepatic enzymes
  - Alcohol dehydrogenase
  - Forms formaldehyde and formic acid
  - Formic acid primary toxin

Methanol

- Organic solvent found in:
  - Cleaning materials
  - Paints
  - Varnishes
  - Sterno fuel
  - Formaldehyde
  - Antifreeze
  - Gasohol
  - "Moonshine" alcohol
  - Windshield wiper fluid

Methanol

- Toxicity commonly result of:
  - Accidental or intentional ingestion
  - Skin exposure
  - Inhalation exposure
  - Industrial setting
  - “Huffing”

Methanol

- Signs and symptoms
  - CNS disturbances
  - Depression, headache, vertigo
  - Cardiovascular collapse
  - Visual disturbances
  - Abdominal pain, N/V

Methanol

- Diagnostic evaluation
  - Blood glucose
    - May be low
  - BUN/creatinine
    - May be elevated
    - Late finding
  - Anion gap
    - Elevated

Methanol

- Diagnostic evaluation
  - Arterial blood gas
    - Marked metabolic acidosis
    - Decreased pH
    - decreased HCO_3^-
  - Complete blood count
    - May show anemia

Methanol

- Diagnostic evaluation
  - Urinalysis
    - Formaldehyde odor
    - Methanol concentrations of blood alcohol content (BAC)
    - 0–20 mg/dL
      - Usually asymptomatic
    - 20–150 mg/dL
      - Toxic level, treatment required
    - 150+ mg/dL
      - Potentially fatal if untreated

Methanol

- Treatment
  - Hemodialysis definitive treatment
  - Supportive care
  - IV or oral ethanol administration
    - Administer to BAC of 100mg/dL
  - Typical infusion
    - 7.6–10 mL/kg over 30 min
    - 1.4 mL/kg/hr maintenance infusion

Methanol

- Treatment
  - NG tube insertion
    - Gastric lavage if ingestion <1 hour old
  - Sodium bicarbonate
    - If:
      - pH levels <7.25
      - Serum bicarbonate <15 mEq/L
      - 50 mEq of 8.4 percent sodium bicarbonate every 30–60 minutes as needed
      - Fomepizole (Antizol)

Uremia

- Syndrome of metabolic and clinical abnormalities associated with hormonal, electrolyte, and fluid imbalances
  - Develop with renal failure
  - Associated with CRF and ARF
- Literal translation means “urine in the blood”

Uremia

- Common clinical derangements
  - Anemia
  - Acidosis
  - Hyperkalemia
  - Malnutrition
  - Hypertension
- Signs and symptoms
  - N/V
  - Weight loss
  - Muscle cramps
  - Change in mental status

Uremia

- Diagnostic evaluation
  - BUN
    - Elevated
  - Serum creatinine
    - Elevated
  - Creatinine clearance
  - Decreased if renal failure (RF) present

Uremia

- Diagnostic evaluation
  - Arterial blood gas
    - Metabolic acidosis
      - Decreased pH
      - Decreased HCO_3^-
  - Urinalysis
    - Positive for protein, ketones, hemoglobin, glucose, and myoglobin
  - Other data
    - Increases in potassium, phosphorus, and parathyroid hormone
    - Decreases in calcium, magnesium, serum bicarbonate, and hemoglobin

Uremia

- Treatment
  - Management of hyperkalemia
  - Correction of acidosis

Diabetic Ketoacidosis (DKA)

- Potentially life-threatening condition of diabetes
  - Characterized by hyperglycemia, acidosis, and ketonuria in the insulin-dependent diabetic
  - A relative or an absolute insulin deficiency decreases glucose movement across cell membranes
    - Causes intracellular hypoglycemia
  - Release of counter-regulatory hormones increases serum glucose levels and forms ketone bodies
  - Acidosis results from depletion of extracellular bicarbonate
  - Polyuria, polyphagia, polydypsia

Diabetic Ketoacidosis (DKA)

- Diagnostic evaluation
  - Serum glucose
    - Often >300 mg/dL
  - Arterial blood gas
    - Metabolic acidosis
    - Decreased pH
    - Decreased HCO_3^-
    - Decreased $PaCO_2$

Diabetic Ketoacidosis (DKA)

- Diagnostic evaluation
  - BUN
    - Elevated
  - Creatinine
    - Elevated
  - Urinalysis
    - Ketonuria
  - Hyperkalemia

Diabetic Ketoacidosis (DKA)

- Treatment
  - Fluid volume replacement
  - Insulin
  - Hypokalemia, if it develops
  - Supportive care as needed

Paraldehyde

- Sedative/hypnotic
  - Used to treat:
    - Acute delirium tremors associated with alcohol withdrawal
    - Seizures
  - When metabolized, paraldehyde turns into acetic acid and acetaldehyde
    - Effects similar to ethyl alcohol
    - Doses of 120 ml can cause:
      - CNS depression
      - Coma
      - Death

Paraldehyde

- Diagnostic evaluation
  - Potassium
    - Elevated
  - Arterial blood gas
    - Metabolic acidosis
    - Decreased pH
    - Decreased HCO_3^-
  - Urinalysis
    - Deceased pH
  - Elevated acetic acid

Paraldehyde

- Treatment
  - Supportive care
  - Vasopressors if hypotension presents

Infection

- Systemic inflammatory response syndrome (SIRS)
  - Precursor to sepsis and end-organ failure
  - Ischemia at infection site leads to anaerobic metabolism
  - Buildup of lactic acid
  - Endotoxin release promotes vasodilation
  - Hypotension
  - Further ischemia
  - Extravasation of vascular volume

Infection

- Diagnostic evaluation
  - White blood cells
    - Elevated
    - Decreased if sepsis has developed
  - Arterial blood gas
    - Metabolic acidosis
    - Decreased pH
    - Decreased HCO_3^-

Infection

- Diagnostic evaluation
  - BUN
    - Elevated
  - Creatinine
    - Elevated
  - Serum lactate
    - Elevated

- Treatment
  - Supportive care
  - Vasopressors
  - Fluid volume replacement
  - Broad-spectrum antibiotics

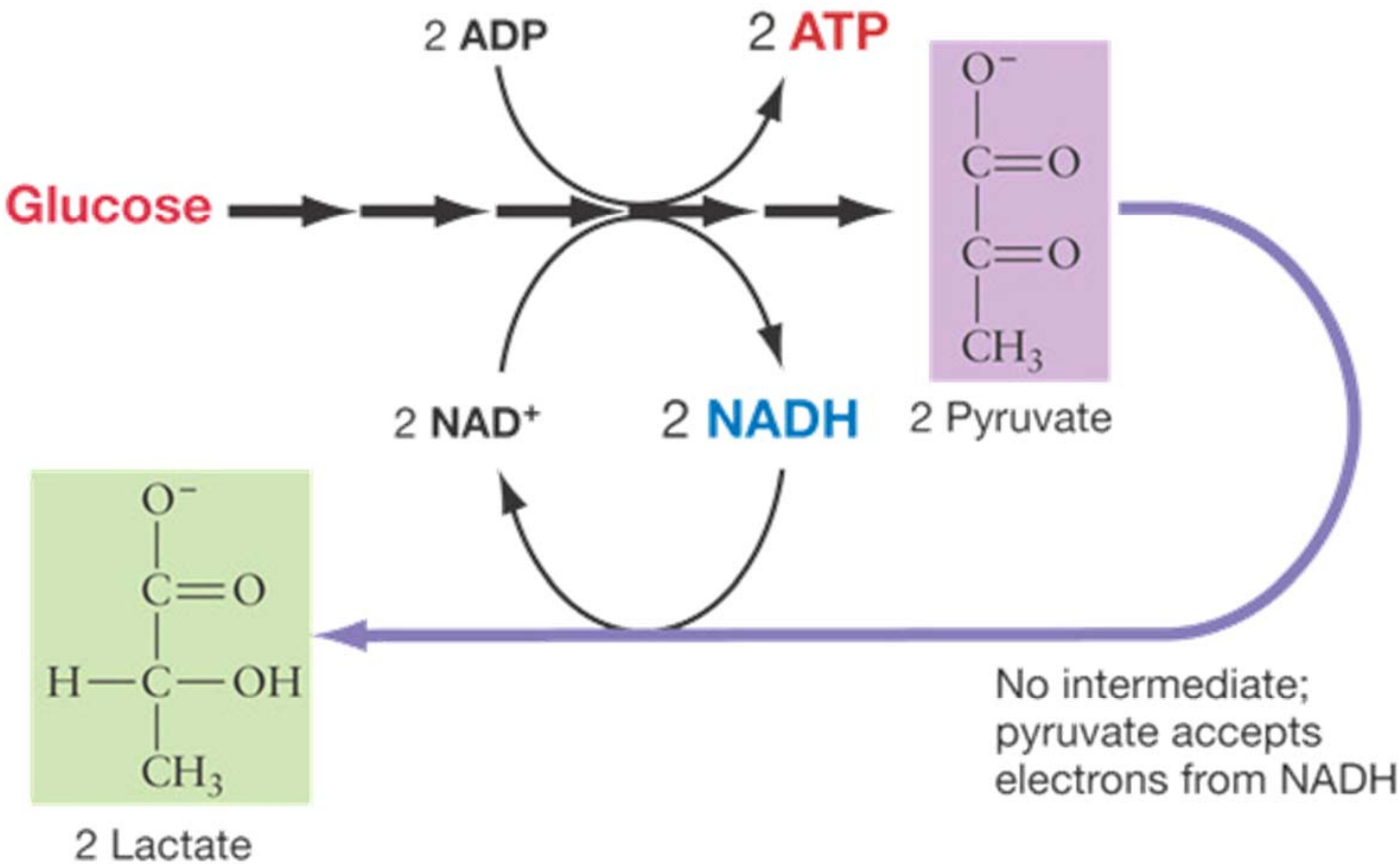
Lactic Acidosis

- Elevations in lactic acid occur as a result of abnormal conversion of pyruvate (pyruvic acid) to lactate
  - Lactemia develops when the bicarbonate buffer system is overwhelmed
  - Lactic acidosis said to be present when:
    - Lactic acid exceeds 5 mmol/L
    - pH levels drop below 7.25

Lactic Acidosis

- Two types of lactic acidosis
  - A
    - Associated with clinical evidence of poor tissue perfusion or oxygenation
  - B
    - B1: Associated with underlying disease
    - B2: Associated with drug and toxins
    - B3: Associated with inborn errors in metabolism

Lactic Acidosis



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Lactic Acidosis

- Diagnostic evaluation
  - Potassium
    - Elevated
  - Serum lactate
    - Elevated
  - Arterial blood gas
    - Metabolic acidosis
    - Decreased pH
    - Decreased HCO_3^-

Lactic Acidosis

- Treatment
  - Focused on improving perfusion status
  - Supportive care as needed
  - IV fluid volume replacement
  - Vasopressors
  - Sodium bicarbonate
    - If toxic ingestion caused condition

Ethylene Glycol

- Clear, colorless, odorless, viscous liquid solvent found in common household and industrial materials
  - Also found in antifreeze and hydraulic brake fluid
- Common cause of accidental ingestion in pediatric population due to its sweet taste
  - Similar to methanol in presentation
  - Skin exposure does not cause toxicity
  - Ethylene glycol converted to glycoaldehyde via alcohol dehydrogenase

Ethylene Glycol

- Glycoaldehyde metabolized to glycolic acid
  - Greatest contributor to metabolic acidosis
- Can form a precipitate with calcium that may cause widespread injury to kidney, brain, and liver tissues
- Clinical presentation of toxicity typically presents in three phases

Ethylene Glycol Toxicity

- Phase I
  - 0–12 hours postingestion
  - Signs and symptoms include:
    - CNS depression phase
    - N/V
    - Ataxia, nystagmus, myoclonic jerking
    - Seizures, coma

Ethylene Glycol Toxicity

- Phase II
  - Cardiopulmonary toxic phase
  - 12–72 hours postingestion
  - Accumulation of oxalate crystal in lung tissue, vascular tree, myocardium
  - Signs and symptoms include:
    - Tachycardia, tachypnea
    - Hypertension

Ethylene Glycol Toxicity

- Phase III
  - Renal toxicity phase
  - Acute tubular necrosis
  - Signs and symptoms include:
    - Flank, abdominal pain
    - Oliguric renal failure

Ethylene Glycol

- Diagnostic evaluation
  - – Lab data used similar to methanol
  - – Urinalysis may reveal:
    - Presence of calcium oxalate crystals
  - – Fluorescence under Wood’s lamp
  - – Serum calcium decreased
  - – Secondary to calcium oxalate binding

Ethylene Glycol

- Treatment
  - Similar to that for methanol
  - Hemodialysis definitive treatment
  - Supportive care
  - IV crystalloid
  - 250–500 mL/hr
  - Increase GFR, aid in renal clearance of toxins
  - IV sodium bicarbonate
  - For pH <7.25
  - IV ethanol infusion
  - IV fomepizole (Antizol)

Salicylate Poisoning

- Common medication used for its anti-inflammatory, analgesic, and antipyretic properties
- Poisoning can occur with intentional overdose or chronic use

Salicylate Poisoning

- Toxicity
  - Cyclic-ATP production decreases
  - Oxygen consumption increases
  - Carbon dioxide production increases
  - Heat production increases
  - Krebs cycle changes and carbohydrate metabolism leads to the accumulation of pyruvate, lactate, and acetoacetate, with resultant metabolic acidosis

Salicylate Poisoning

- Signs and symptoms
  - Tachycardia, tachypnea
  - Fever, increased metabolic rate
  - Abdominal pain, N/V, dehydration
  - Tinnitus, deafness
  - CNS disturbances
  - Mild confusion to coma

Salicylate Poisoning

- Diagnostic evaluation
  - Arterial blood gas
    - Respiratory alkalosis, metabolic acidosis
  - Coagulation studies
    - Prolonged prothromin time
    - Decreased platelet count
  - Electrolytes
    - Hypokalemia

Salicylate Poisoning

- Diagnostic evaluation
  - Urinalysis
    - Low pH
  - Salicylate levels
    - Peak levels at 4–6 hours
    - Normal salicylate: 15–30 mg/dL
    - Patients typically symptomatic: 40–50 mg/dL
    - Potentially life threatening: >100 mg/dL

Salicylate Poisoning

- Treatment
  - Supportive care
  - GI decontamination
  - Gastric lavage
  - Activated charcoal
  - Fluid resuscitation as needed

Salicylate Poisoning

- Treatment
  - Urine alkalization
  - IV sodium bicarbonate
  - 100–150 mEq per liter of IV solution
  - Potassium replacement
  - If serum levels <3.5 mEq/L
  - KCl 20–40 mEq/L of IV solution
  - Goal is serum K 4.5–5.0 mg/dL

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ABG

- Normal Ranges
  - pH 7.35 – 7.45
  - pCO_2 35 – 45 mmHg
  - pO_2 80 – 100 mmHg
  - HCO_3^- 22 – 26 mmol/L
  - BE -2 to +2 mmol/L
  - SaO_2 > 95 %
  - Anion Gap 8 - 16 mEq/L

Acid-Base Balancing

Acidosis

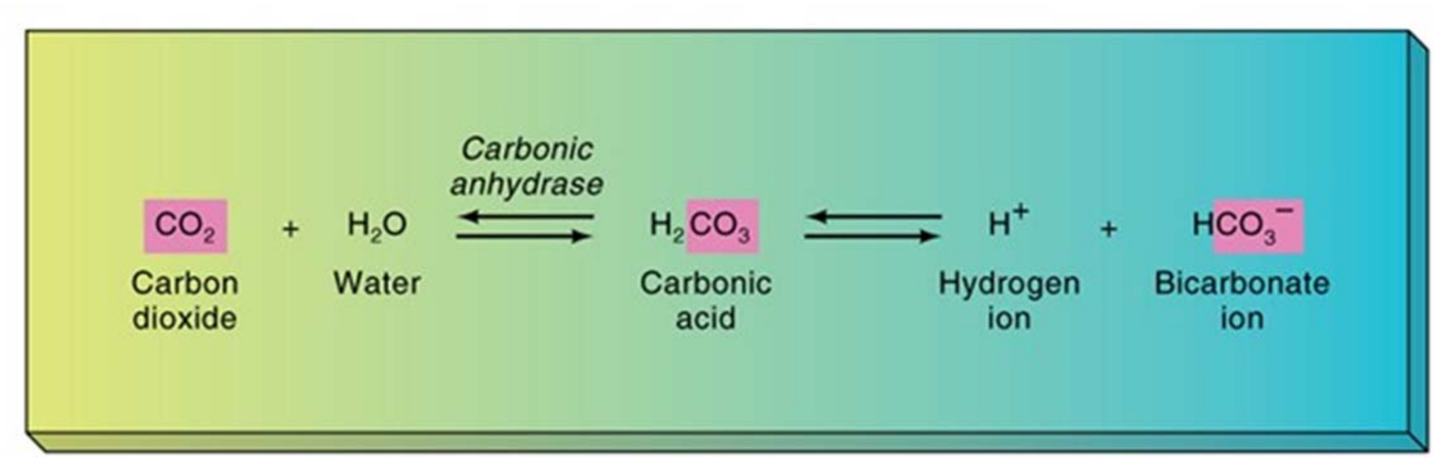
- Metabolic Acidosis
  - ↓ HCO_3^-
- Respiratory Acidosis
  - ↑ CO_2

Alkalosis

- Metabolic Alkalosis
  - ↑ HCO_3^-
- Respiratory Alkalosis
  - ↓ CO_2

ABG

- Acidosis
  - pH < 7.35
  - pCO_2 > 45 mmHg
  - HCO_3^- < 22 mmol/L
- Alkalosis
  - pH > 7.45
  - pCO_2 < 35 mmHg
  - HCO_3^- > 26 mmol/L



ABG Interpretation

- Step 1
  - Examine pO_2 and SaO_2
  - Determine oxygen status
  - Low pO_2 (< 80 mmHg) and SaO_2

	Low	Normal	High
pO_2	< 80 mmHg	80 – 100 mmHg	> 100 mmHg
SaO_2	< 95 %	95 – 100 %	100%
	Hypoxia	Adequate	Adequate

ABG Interpretation

- Step 2
  - Examine pH

	Low	Normal	High
pH	< 7.35	7.35 – 7.45	> 7.45
	Acidosis	Normal	Alkalosis

ABG Interpretation

- Step 3
  - Examine pCO_2 and HCO_3^-
  - Is the pCO_2 abnormal with a normal HCO_3^-
  - Is the HCO_3^- abnormal with a normal pCO_2

	Acidosis		Alkalosis	
	Respiratory	Metabolic	Respiratory	Metabolic
pCO_2	> 45 mmHg	35 – 45 mmHg	< 35 mmHg	35 – 45 mmHg
HCO_3^-	22 – 26 mmol/L	< 22 mmol/L	22 – 26 mmol/L	> 26 mmol/L

- Step 4
  - Determine if there is a compensatory mechanism in place to try and correct the pH

	Acidosis		Alkalosis	
	Respiratory	Metabolic	Respiratory	Metabolic
pCO_2	> 45 mmHg	< 35 mmHg	< 35 mmHg	> 45 mmHg
HCO_3^-	> 26 mmol/L	< 22 mmol/L	< 22 mmol/L	> 26 mmol/L
	Compensated	Compensated	Compensated	Compensated

What are the compensations?

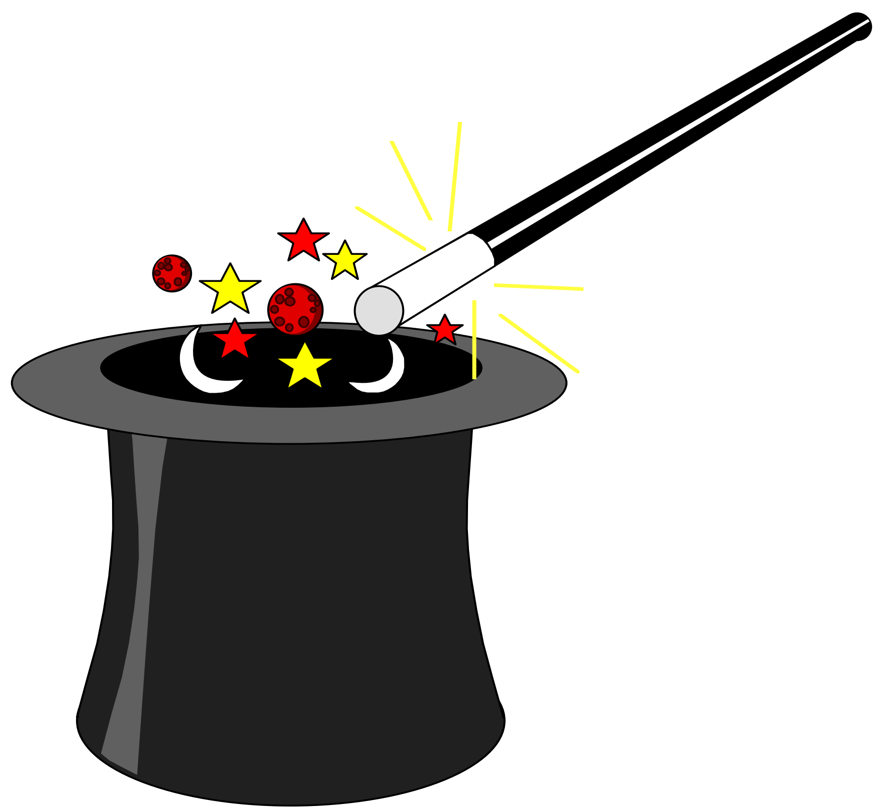
Respiratory acidosis ⇆ Metabolic alkalosis

Respiratory alkalosis ⇆ Metabolic acidosis

- In respiratory conditions the kidneys will attempt to compensate and visa versa
- Ex:
  - In chronic respiratory acidosis (COPD) the kidneys increase the elimination of H^+ and absorb more HCO_3^-
  - The ABG will show normal pH, ↑CO_2 and ↑ HCO_3^-
- Buffers kick in within minutes
  - Respiratory compensation is rapid and starts within minutes and complete within 24 hours
  - Kidney compensation takes hours and up to 5 days.

It’s not magic

- It just takes some practice…



Practice ABG's

- Number 1
  - pH 7.48 – High
  - pCO_2 32 mmHg – Low
  - pO_2 90 mmHg – Normal
  - HCO_3^- 24 mmol/L – Normal
  - S_aO_2 95 % – Normal

Respiratory Alkalosis

Practice ABG's

- Number 2

– pH	7.32	– Low
– pCO_2	48 mmHg	– High
– pO_2	60 mmHg	– Low
– HCO_3^-	25 mmol/L	– Normal
– S_aO_2	90 %	– Low

Respiratory Acidosis

Practice ABG's

- Number 3
  - pH 7.30 – Low
  - pCO_2 40 mmHg – Normal
  - pO_2 95 mmHg – Normal
  - HCO_3^- 18 mmol/L – Low
  - S_aO_2 100 % – Normal

Metabolic Acidosis

Practice ABG's

- Number 4

– pH	7.38	– Normal
– pCO_2	48 mmHg	– High
– pO_2	87 mmHg	– Normal
– HCO_3^-	28 mmol/L	– High
– S_aO_2	94 %	– Low

Respiratory Acidosis with Metabolic Compensation

Practice ABG's

- Number 5

– pH	7.49	– High
– pCO_2	40 mmHg	– Normal
– pO_2	94 mmHg	– Normal
– HCO_3^-	30 mmol/L	– High
– S_aO_2	99 %	– Normal

Metabolic Alkalosis

Practice ABG's

- Number 6

– pH	7.35	– Normal
– pCO_2	48 mmHg	– High
– pO_2	62 mmHg	– Low
– HCO_3^-	27 mmol/L	– High
– S_aO_2	91 %	– Low

Respiratory acidosis with metabolic compensation

Practice ABG's

- Number 7
  - pH 7.45 – Normal
  - pCO_2 47 mmHg – High
  - pO_2 93 mmHg – Normal
  - HCO_3^- 29 mmol/L – High
  - S_aO_2 97 % – Normal

Metabolic Alkalosis with respiratory compensation

Practice ABG's

- Number 8
  - pH 7.31 – Low
  - pCO_2 38 mmHg – Normal
  - pO_2 95 mmHg – Normal
  - HCO_3^- 15 mmol/L – Low
  - S_aO_2 99 % – Normal

Metabolic Acidosis

Practice ABG's

- Number 9
  - pH 7.30 – Low
  - pCO_2 50 mmHg – High
  - pO_2 65 mmHg – Low
  - HCO_3^- 24 mmol/L – Normal
  - S_aO_2 89 % – Low

Respiratory Acidosis

Practice ABG's

- Number 10

– pH	7.48	– High
– pCO_2	40 mmHg	– Normal
– pO_2	110 mmHg	– High
– HCO_3^-	30 mmol/L	– High
– S_aO_2	100 %	– Normal

Metabolic Alkalosis

Obtaining an ABG

Procedure

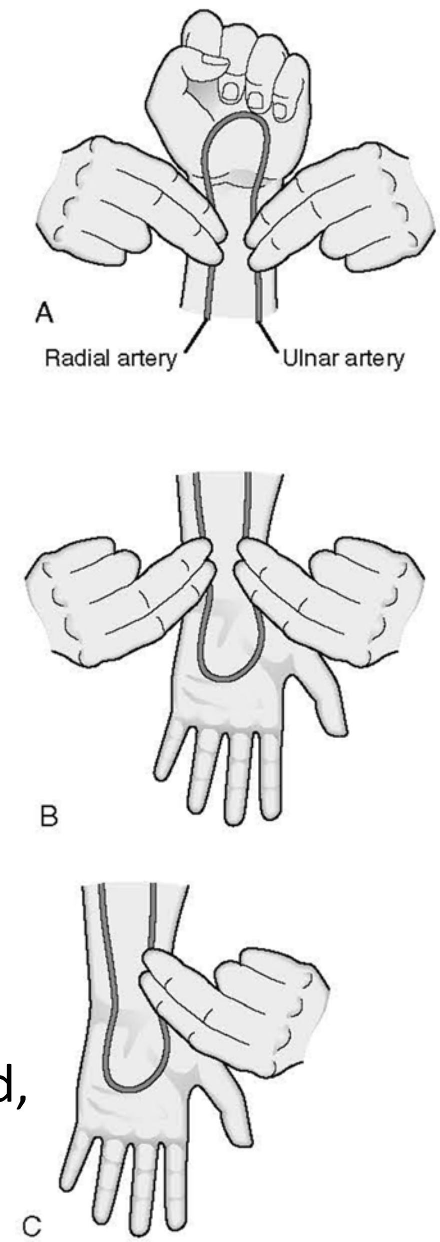
- Equipment required
  - One 20-gauge to 25-gauge, 1-inch to 1.5-inch hypodermic needle (note: longer needles are needed for brachial and femoral artery puncture)
  - One 1 to 5 mL pre-heparinized syringe with a rubber stopper or cap
  - Two 2 X 2 gauze pads
  - Antiseptic solution/Alcohol Swabs
  - One plastic bag (for transport of sample to laboratory)
  - One adhesive bandage
  - PPE
  - Container with crushed ice or cold water

Procedure

- If the radial artery is to be used, perform the modified Allen's Test
  - Recommended before a radial artery puncture to assess the patency of the ulnar artery and an intact superficial palmar arch

Procedure

- Have patient raise hand over their head and make a fist several times
- With the hand clenched, apply direct pressure on the radial and ulnar arteries (A)
- Instruct patient to lower arm then open fist (B)
- Release pressure over ulnar artery and observe for return of color (C)
  - < 7 seconds
    - Indicates positive result (ulnar artery and superficial palmar arch intact)
  - 8 to 14 seconds
    - equivocal
  - > 14 seconds
    - Indicates negative result (ulnar artery may be occluded, do not perform radial puncture)



Procedure

- Palpate selected artery and stabilize artery
  - If radial
    - Confirm positive modified Allen Test
    - Stabilize artery by hyperextending wrist slightly
- Clean area with alcohol swab or antiseptic
- Palpate pulse proximal to insertion site
  - Hold alcohol swab with same fingers used to palpate artery
- Insert needle bevel up
  - Radial 45° (30° – 60°)angle
  - Femoral 60° - 90° angle
- Stop advancing when blood is noted
- Collect 2 – 3 ml of blood into syringe
- Hold alcohol swab over puncture site and withdraw needle
- Apply pressure over injection site for 5 minutes
  - 10 if on anticoagulants
  - Reassess pulse
- Remove air from syringe, remove needle and place cap on syringe
- Dispose of sharps
- Place in bag, label
- Place on ice and ship