ABSTRACT
Arterial Blood gas interpretation is essential part of diagnosing and managing patient’s physiological condition. Arterial blood gases are an invaluable tool in assessing ventilation, acid-base balance and oxygenation. PH designates acid–base balance of arterial blood. If one of the variables forces PH too far from normal range then cells of the body will be unable to function adequately To counteract this there are buffer systems in the body, 2 main namely respiratory and renal system.

Two system acts through formation of H2CO3.

\[
\begin{align*}
H_2O + CO_2 &= H_2CO_3 \\
H_2CO_3-H+ &= CO2-
\end{align*}
\]

Balance work in both direction. Compensation occurs in predictable manner. Inadequate compensation suggests another acid base disorder. PH determines primary disorder. There can never be overcompensation

Resp system balances PH by producing HCO3 Or eliminating H+.
Renal system will reflect metabolic activity within body in the form of low HCO3

Various parameter of ABG are used for Assessing such as Ventilation by PCO2, Gas exchange & Oxygenation by PO2 & SaO2, Acid Base Disorder by PH, Befc,HCO3

ABG is very imp tool in managing mechanical ventilator. It is used right from beginning in initiating ventilator, setting, Monitoring, managing complication and liberating patient from ventilator

INTRODUCTION
Respiratory Acid base disorder

Respiratory Acidosis \( pH < 7.35 \) (Normal = 7.35-7.45)\( CO2 > 45 \) (Normal = 35-45)

Respiratory acidosis (alveolar hypoventilation) - acute, chronic

Acute response is independent of renal HCO3 wasting. The chronic compensation is governed by renal HCO3 wasting.

CK Jani, Mumbai

PH designates acid–base balance of arterial blood, Ideal PH- 7.4 Many variables affect PH of the blood If one of the variables forces PH too far from normal range then cells of the body will be unable to function adequately

To bring back PH to normal there are buffer systems in the body, mainly respiratory and renal system

Balancing component of respiratory system is dissolved CO2, .PCO2 which is produced by cellular process and removed by lungs

Balancing component of renal system is dissolved HCO3 produced by kidney. Kidney helps to control PH by eliminating H+.

Two system acts through formation of H2CO3.

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\begin{align*}
H_2O + CO_2 &= H_2CO_3 \\
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\]

Balance work in both direction. Resp system balances PH by producing HCO3 Or eliminating H+.

Renal system will reflect metabolic activity within body in the form of low HCO3

Our body regulates PH by using opposite system to balance PH. If PH is out of normal range due to resp system, it will be renal system that makes compensation. Compensation may not be complete suggesting another acid base disorder

Complete compensation returns PH normal. There is never overcompensation

Respiratory Acidosis

Acute Respiratory Acidosis \( pH < 7.35 \) (Normal = 7.35-7.45)
CO2 > 45 (Normal = 35-45)

Respiratory acidosis (alveolar hypoventilation) - acute, chronic

Acute response is independent of renal HCO3 wasting. The chronic compensation is governed by renal HCO3 wasting

PaCO2 is elevated and pH is acidic. The decrease in pH is accounted for entirely by the increase in PaCO2. Bicarbonate and base excess will be in the normal range because the kidneys have not had adequate time to establish effective compensatory mechanisms. The blood pH will change according to the level of H2CO3 present. High level of H2CO3 triggers the lungs to either increase or decrease the rate and depth of ventilation

Activation of the lungs to compensate for an imbalance starts within 1-3 minutes; H ion is titrated by non HCO3 organic tissue buffers. Hb is an example

The kidney has little involvement in this phase

CAUSES

Drugs -Narcotics, sedatives, or anesthetics
Neuromuscular diseases, or neuromuscular blocking drugs
Impaired respiratory muscle function related to spinal cord injury,
Pulmonary disorders such as atelectasis, pneumonia, pneumothorax, pulmonary edema, or bronchial obstruction, Massive pulmonal embolism
Ventilatory restriction & Hypoventilation due to pain, chest wall injury/deformity, or abdominal distension
Increased CO2 production: shivering, rigors, seizures, malignant hyperthermia, hypermetabolism, increased intake of carbohydrates
Improper ventilator setting

Signs and Symptoms
are centered within the pulmonary, nervous, and cardiovascular systems.

Pulmonary - dyspnea, respiratory distress, and/or shallow respirations
Nervous system - headache, restlessness, and confusion If PCO2 levels become extremely high, drowsiness and unresponsiveness may be noted
Cardiovascular - tachycardia and dysrhythmias

Compensation Acute HCO3 will increase HCO3 increase of 2 mmol/L per 10 mmHg increase in PaCO2 (↓pH by 0.08/10 mm Hg)

Chronic HCO3 will increase 4 mmol/L HCO3 increase of 4mmol per 10 mmHg increase in PaCO2 (↓pH by 0.03/10 mm Hg ↓PaCO2)

Treatment
1. If pt unstable connect to MV
2. Increase ventilation by increasing respiratory rate / or TV
3. If patient is connected to Mechanical ventilator
4. Address correctable cause rapidly which includes pneumothorax, pain, and CNS depression related medications
5. If the cause cannot be readily resolved, support the patient with mechanical ventilation while Treatment is rendered

Chronic Respiratory Acidosis
PaCO2 is elevated with a pH in the acceptable range
Renal mechanisms increase the excretion of H+ within 24 hours and may correct the resulting acidosis caused by chronic retention of CO2 to a certain extent

Causes
chronic lung disease (BPD, COPD)
Neuro muscular disease
Extreme obesity
Chest wall deformity

Respiratory Alkalosis
PH > 7.45, CO2 <45 , pH, CO2, Ventilation
pH, CO2, Ventilation.
Acute response is independent of renal HCO3 wasting. The chronic compensation is governed by renal HCO3 wasting. The increase in pH is accounted for entirely by the decrease in paCO2.
Bicarbonate and base excess will be in the normal range because the kidneys have not had sufficient time to establish effective compensatory mechanisms

Compensation
CO2 ⇐ HCO3 (↑Cl to balance charges ⇐ hyperchloremia

Acute vs. Chronic

Acute - CO2 by 2 mEq/L for every 10mmHg ↓ in PCO2

Chronic - 4 mEq/L of HCO3 for every 10mmHg ↓ in PCO2

Causes
Cerebral edema, brain trauma, brain tumor, CNS infection
CNS stimulation: fever, pain, Fear, Anxiety, CVA

Hypoxemia or hypoxia: lung disease, profound anemia, low FiO2
Stimulation of chest receptors: pulmonary edema, pleural effusion, pneumonia, pneumothorax, pulmonary embolus

Drugs, hormones: salicylates, catecholamines, medroxyprogesterone, progestins

Pregnancy, liver disease, sepsis, hyperthyroidism
Incorrect mechanical ventilation settings
Associated with Alveolar Hyperventilation, Psychogenic, Hypermetabolic states (fever, thyrotoxicosis
Drug use : salicylates and progesteron

Signs and Symptoms
Headache, Vertigo, Parenthesion, Numb fingers/toes/circumoral, carpal pedal spasms and tetany, Tinnitus (ringing in the ears

Treatment
Treat the underlying cause
Sedatives or analgesics
Correction of hypoxia, connect mechanical ventilator to decrease respiratory rate and to decrease the tidal volume
Antipyretics for fever
Treat hyperthyroidism.
Breathe into a paper bag for hyperventilation
Resolve the underlying cause
Monitor for respiratory muscle fatigue
When the respiratory muscle become exhausted, acute respiratory failure may precipitate

ABG for evaluation of Oxygenation
ABG measures arterial PO2, Sao2 ie Arterial PO2 associated with haemoglobin

While interpreting it is essential to know at what FIO2 patient is breathing?
Partial pressure of oxygen (PaO2). This measures the pressure of oxygen dissolved in the blood and how well oxygen is able to move from the airspace of the lungs into the blood.

Sao2 measures how much of the hemoglobin in the red blood cells is carrying oxygen

Assessment of Gas Exchange
1. Alveolar-arterial O2 tension difference
   - A-a gradient
   - PAO2-PaO2
   - PAO2 = FIO2(PB - PH2O) - PaCO2/RQ*
2. PaO2/FIO2,P/F ratio
   Convenient & widely used bedside index of O2 exchange that attempts to adjust for FIO2
   Simple to calculate but it is affected by change in SVCO2
   PaO2 / FIO2 (Normal = 500, ARDS < 200), ALI <300 where FIO2 range is 0.21 to 1.0

The O2 number has nothing to do with your acid-base ABG interpretation!

What does the PaO2 mean?
The PO2 tells us if the patient has HYPOXEMIA (decreased oxygen in the blood).
Normal PaO2 = 80-100. (Hypoxemia = PaO2<80)
PaO2 assesses Perfusion (gas exchange).

What is SaO2 saturation?
SaO2 (oxygen saturation) measures the percent of oxygen bound to hemoglobin. This tells weather the patient has HYPOXIA (decreased O2 in the tissues).
Normal SaO2 = Greater that 95%
PaO2 dramatically drops when it is less that 92%.
SaO2, or oxygen saturation, measures the degree to which oxygen is bound to hemoglobin.

Each hemoglobin molecule has four oxygen binding sites. When those sites are occupied, the hemoglobin molecule cannot hold any more oxygen. Hemoglobin binding sites can hold molecules other than oxygen.

Carboxyhemoglobin (HbCO) is a hemoglobin molecule that has carbon monoxide where the oxygen should attach. The blood will have a cherry red color.

Methemoglobin (MetHb) is produced when certain poisons or a genetic condition affect the iron portion of the hemoglobin subunit. It changes blood to a brownish color.

The presence of either carbon monoxide or methemoglobin changes the affinity of oxygen for haemoglobin. Oxygen will be less available to be carried on the hemoglobin molecule

The hemoglobin molecule in these conditions is unusable. If enough hemoglobin is inactivated like this, it can cause tissue hypoxia.

Oxygen Saturation will fall if:
Inspired oxygen level is diminished, such as at increased altitudes, Upper or middle airway obstruction (such as during an acute asthmatic attack)
The percentage of all the available heme-binding sites saturated with oxygen is the haemoglobin oxygen saturation (in arterial blood, the SaO2). Note that SaO2 alone doesn’t reveal how much oxygen is in the blood; for that we also need to know the hemoglobin content.

Oxygen’s affinity for hemoglobin changes depending on pH and temperature
Oxyhemoglobin Dissociation Curve
The oxyhemoglobin dissociation curve is a tool used to show the relationship between oxygen saturation and the PaO2.

The oxyhemoglobin dissociation curve can be used to estimate the PaO2 if the oxygen saturation is known. The illustration demonstrates that if the curve is not shifted (A waveform), an
Oxygen saturation of 88% is equivalent to a PaO2 of about 60 mm Hg. With a left shift, the same saturation is equivalent to a much lower PaO2.

Hemoglobin levels need to be considered in determining the adequacy of available O2. A patient may have an O2 saturation of 100% but have a hemoglobin level of 2 Gm. In this patient, the actual amount of O2 delivered at the cellular level will still be deficient due to the very low number of hemoglobin molecules available to carry O2.

**Oxyhemoglobin dissociation curve:** The main factor in determining how much oxygen hemoglobin is carrying at a given time is the pO2 to which the hemoglobin is exposed. A high pO2 means hemoglobin is carrying more oxygen. A low pO2 means less oxygen is carried by hemoglobin. Without sufficient partial pressure, oxygen is unable to make the journey from a dissolved state in the blood to the hemoglobin binding sites.

The oxyhemoglobin dissociation curve reflects this exact relationship. The correlation is not linear, meaning a given rise or fall in pO2 is not always reflected in the same amount of rise or fall in oxygen saturation. If the pO2 is within normal range or higher, a rise in pO2 will result in a very small increase in O2 saturation.

**Shifts to the curve:** When conditions are other than normal, i.e., changes in body temperature or in pH, this curve will shift to the left or right. A shift does not affect the uptake of oxygen by hemoglobin in the lungs, but does affect the release of oxygen at the cellular level.

a. **Left shift:** Occurs in the presence of alkalosis and makes hemoglobin less willing to give up oxygen to the cells.

b. **Right shift:** Occurs in the presence of acidosis and fever and is physiologically advantageous as hemoglobin is much more willing to release oxygen to the cells.

**Hypoxia** – inadequate amount of oxygen available to or used by tissues for metabolic needs

**Causes**

- Inadequate inspiratory partial pressure of oxygen
- Hypoventilation
- Right to left shunt
- Ventilation-perfusion mismatch
- Incomplete diffusion equilibrium
- How and when do body tissues become hypoxic?
  - Problem Too little oxygen reaches the alveoli – bronchospasm, Pneumonia
  - Circulatory (perfusion) Mechanism Too little blood reaches the alveoleia
  - Cardiogenic shock, Pulm embolism
- **Shunt**
  - The term shunt is used to describe a situation where there is ventilation without oxygenation
  - Circulatary problems that result in a shunt are called “V-Q shunt
  - V and Q can be compared directly by nuclear medicine procedure called a “V-Q scan” or a “lung scan.
  - It is normal for a small percentage of air in the lungs not to reach blood. This is “dead space”. It’s an anatomical necessity. Air in the nasopharynx, trachea and bronchi does not reach the alveoli before exhalation. More than this amount of “dead space” can lead to hypoxia
  - When dealing with shunts and mismatches, you consider value known as “A-a gradient.”

This value has been used to compare oxygenation of the alveoli and that

\[ \text{P(A-a)O2} = \text{PaO2} - \text{PaO2} \text{ of the arteries} \]

“\( A/a \) ratio” can give a more accurate prediction of V-Q mismatch.

**Alveolar Gas Equation**

\[ \text{PAO2} = \text{FiO2} \times (\text{Patm} - \text{Pwater}) - \frac{\text{PaCO2}}{R} \]

Simplify to: \[ \text{PAO2} = 150 - \frac{(\text{PaCO2}) \times 1.2}{R} \]

Except in a temporary unsteady state, alveolar PO2 (PAO2) is always higher than arterial PO2 (PaO2).

\( P(A-a)O2 \) is the alveolar-arterial difference in partial pressure of oxygen, though it does not actually result from an O2 pressure gradient in the lungs. Instead, it results from gravity-related blood flow changes within the lungs (normal ventilation-perfusion imbalance).
PAO2 is always calculated based on FIO2, PaCO2, and barometric pressure.

Normal P(A-a)O2 ranges from 5 to 25 mm Hg breathing room air (it increases with age).

A higher than normal P(A-a)O2 means the lungs are not transferring oxygen properly from alveoli into the pulmonary capillaries. Except for right to left cardiac shunts, an elevated P(A-a)O2 signifies some sort of problem within the lungs.

Assessment of Gas Exchange

Alveolar-arterial O2 tension difference
A-a gradient
PAO2-PaO2
PAO2 = FIO2(PB - PH2O) - PaCO2/RQ*
PaO2/FIO2, P/F ratio

Points to remember:
PaO2 is oxygen dissolved in plasma, not total O2.
SaO2 is saturated hemoglobin minus HbCO and MetHb.
Ventilation does not equal oxygenation.
A shunt is normal alveolar O2, but low blood O2.

Using ABG for Mechanical Ventilation Care

Arterial blood gas analysis is a very important life saving investigation. It includes pH, pCO2 and pO2 as measured value, and bicarbonate as a calculated value. Patient on ventilator is not under control of respiratory centre, ventilator acts as artificial respiratory centre and it needs constant adjustment of parameters like tidal volume, oxygen percentage and peak flow rate according to patients ABG’s status. In brief it is described how ventilator is managed and at every stage it is assessed by ABG.

Initial ventilator settings

The following guide is an example of the steps involved in operation a mechanical ventilator;
1. Set the machine to deliver the tidal volume required. 7-8ml/kg
2. Adjust the machine to delivery lowest of concentration of oxygen to maintain normal PaO2 (80-100mgHg), this setting may be high initially but will gradually be reduced
3. Record peak inspiratory pressure
4. Set mode and rate according to physician’s order.
5. Adjust sensitivity so that the patient can trigger
6. Record minute volume and measure carbon dioxide partial pressure (PCO2), PH, PO2, after 20 minutes of continuous mechanical ventilation.
7. Adjust setting according to arterial blood gas (ABG) results

to provide normal values or those set by physician.

8. If patient suddenly becomes confused or agitated or begins to bucking the ventilator for some unexplained reason, assess for hypoxia and manually ventilate 100% O2 with resuscitation bag. Ensure that the patient is in sync with ventilator when thoracic expansion coincides with the inspiratory phase of the machine and exhalation occurs passively, the patient is said to fight or buck the ventilator when out of phase with the machine.

The following factors contribute to this:
Anxiety, hypoxia, increase secretions, hypercapnia, inadequate minute volume, and pulmonary edema.

These problems must be corrected before restoring to the use of paralyzing agents to reduce bucking, otherwise, the underlying problem is easy to be masked, and the patient’s condition will continue to deteriorate.

Troubleshooting ventilator problems

A. Increase in peak airway pressure, causes are: coughing or airway tube blocked, (solution are : suction airway , empty condensation fluid from circuit)
B. Fighting ventilator- decreasing lung compliance- (solution are: adjust sensitivity, manually ventilate patient, assess for hypoxia, or bronchospasm ; check ABG,
C. Tubing kinked -(solution are :check tubing , reposition patient, insert oral airway if necessary);
Pneumothorax or atelectasis (solution are manually ventilate patient and notify physician, clear secretions
D. decrease in pressure or loss of volume causes are: increase patient’s compliance, leak in ventilator or tubing, cuff on tube /humidifier not tight (solutions are: check entire ventilator circuit for patency, and correct leak
E. Cardiovascular compromise ,causes are :decrease in venous return due to application of positive pressure to lungs( solutions are : assess adequate volume status by measure V/S , CVP, PCWP and urine output , notify physician if the values are abnormal
Barotraumas /pneumothorax, causes are: application of positive pressure to lungs
High mean airway pressures lead to alveolar rupture (solutions are: notify physician, prepare patient for chest tube insertion, avoid high pressure settings for patient with COPD ARDS , or history of pneumothorax
F. pulmonary infection causes are: bypass of normal defense mechanisms , frequent breaks in ventilator circuit , decreased mobility , impaired cough reflex( solutions are : use meticulous aseptic technique , provide frequent mouth care , optimize nutritional status.
G. Weaning off ventilator maximum inspiratory pressure (PIP) at least 20 cm H₂O, tidal volume 7-5-7 ml/kg.

Monitor activity level, assess dietary intake, and monitor results of laboratory tests of nutritional status.

Terminate the weaning process if adverse reactions occur, including: heart rate an BP increase, SaO₂ saturation decreases, RR increase or decrease, ventricular dysrhythmias, fatigue, panic, cyanosis, erratic or labored breathing, paradoxical chest movement.

Postpone weaning

If the weaning process continues, measure tidal volume, and minute ventilation every 20-30 minute, compare with the patient’s desired and check ABG.

Ventricular Management

Indication for Mechanical Ventilation

Hypeoxemia Hypercapnia Clinical

| PO₂ < 55 torr or | PCO₂ > 44 torr | Respiratory distress accompanied by shock or somnolence. |
| SO₂ < 92% | acutely or chronically with pH > 7.25 | |

Despite supplement inspired oxygen

PCO₂ elevated chronically with pH > 7.25 despite non-invasive ventilation assist devices.

Intubate patient: Oral ET tube size > 7.0 (in adults) to avoided high airway resistance, suctioning difficulty, and possible occlusion from mucus and blood.

Initial Ventilator settings:

<table>
<thead>
<tr>
<th>Mode</th>
<th>Assist/Control (A/C*) or Intermittent Mandatory Ventilation (IMV*):</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspired Oxygen (FI0₂)</td>
<td>Select FI0₂ based on PaO₂ from previous ABG or empirically start at FI0₂ = 1.0 and decrease until SO₂ &gt; 94% (paO₂ approximately 75 torr).</td>
</tr>
<tr>
<td>PEEP (cmH₂O)</td>
<td>Begin at 5 to provide physiologic backpressure lost by the ET tube bypassing glottal muscles. Increase PEEP in increments of 2.5 to PEEP maximum if the FI0₂ &gt; 0.6.</td>
</tr>
<tr>
<td>Respiratory Rate (RR)</td>
<td>Begin at 8-12/min. Increase the RR if the patients spontaneous RR &gt; 5 above the set RR.</td>
</tr>
<tr>
<td>Tidal Volume (VT)</td>
<td>Patients without ARDS: Begin at 8 or 10 mL/KG and round off to the nearest 50 mL. Patients with ARDS: Use 6 ml/kg of ideal body weight.</td>
</tr>
<tr>
<td>Nebulizer treatments</td>
<td>Albuterol, ipratropium bromide when indicated frequency: at least q4h, See COPD acute respiratory failure and Acute exacerbation of asthma. Consider 2% bicarbonate solutions as mucolytic based on sputum viscosity.</td>
</tr>
</tbody>
</table>

Oxygenation (PO₂)

Adjust FI0₂ and PEEP to alter SaO₂.

The SaO₂ varies directly with the FI0₂ and PEEP.

For hypoxemia (SaO₂ < 94%) required FI0₂ > 0.6 first

Increase PEEP from 0 to 5 cm H₂O in steps of 2.5 to a PEEP maximum.

If hypoxemia persists, then increase the FI0₂ in steps of 1.0 until 1.0 is reached or SO₂ > 93%.

For SO₂ > 95% at PEEP maximum

FI0₂ is first reduced in steps of 0.10 until < 0.6 then PEEP is reduced in steps of 2.5 to a minimum of 5 before further reduction of FI0₂.

Ventilation (PCO₂)

Adjust RR VT alter pCO₂ and pH.

The pCO₂ varies inversely with the VE* (RR x VT).

If pH < 7.35, increase VE (to lower pCO₂) by increasing RR by 2/min to a maximum of 30; if academia persists.

Consider increasing VT in steps of 50 ml to a maximum of 15 ml/kg with the following cautions:

- In ARDS, high VT causes alveolar damage; Limit VT (< 6 mL/kg ideal body weight) to keep plateau pressure < 30. May allow permissive CO₂ retention and lower pH.
- In COPD or asthma, high VE may cause autpeep.

Autopeep should be measured before increasing VE.

Aim for pH <7.35; not for normal CO₂; minimize autpeep; keep plateau pressure < 30 and allow permissive CO₂ retention.

If pH >7.45, decrease VE (to raise pCO₂) by decreasing RR by 2 until < 8, then decrease VT in steps of 50 ml.

If patients RR remains elevated despite the ventilator RR reduction, consider sedation.

Pulmonary consultation should be considered for any patient on a ventilator and should be obtained for patients with ARDS or ventilatory failure due to any primary pulmonary diseases state.

Take home message

Arterial blood gas analysis is an essential part of diagnosing and managing a patient’s oxygenation status and acid-base balance. The usefulness of this diagnostic tool is dependent on being able to correctly interpret the results.

Understanding arterial blood gases can sometimes be confusing. A logical and systematic approach using steps makes interpretation much easier. Applying the concepts of acid base balance will help the healthcare provider to follow the progress of treatment of a patient.

ABG assesses the patient’s physiological condition. Remember that ABG analysis is only part of the patient assessment. Valuable information can be gained from clinical and homodynamic parameters.

The most important part of analyzing lab values is to remember to treat the patient, not the numbers. Lab results should always be...
correlated with good clinical data. Accurate history and physical examinations are a clinician’s best resources.

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