Respiratory monitoring in Pediatric Intensive Care Unit (PICU) is an essence of critical care. Be it clinical, invasive or noninvasive, monitoring remains crucial in overall assessment of a critically ill child with cardio-respiratory problems. A functioning knowledge of the various tools of monitoring is essential in applying their use to patient care. This chapter discusses traditional methods of evaluation of respiratory system and newly established gold standard techniques as well. Attention is also given to newer modalities, including those that are investigational or currently limited to bench application, that give promise for future application in PICU clinical practice. Pulse oximetry and Capnography are the most commonly employed monitoring modalities, which have transformed the practice of critical care in last 10 years. Arterial blood gases and calculated oxygen indices have been most commonly used and form essential part of monitoring in PICU. However may be the excellent information provided by respiratory monitors it cannot replace careful bedside clinical examination.

Essentially respiratory monitoring consists of:
1. Physical examination
2. Non-invasive monitoring
3. Invasive monitoring

Physical Examination
Measuring the respiratory rate (Table 1) is easy and has got good accuracy in prediction of lower respiratory tract infection. Presence of increased work of breathing is suggested by flaring of alae nasi, suprasternal, intercostal and substernal retractions, use of accessory muscles of respiration and paradoxical breathing.

<table>
<thead>
<tr>
<th>Age</th>
<th>Respiratory rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infant (birth−1 year)</td>
<td>30−60</td>
</tr>
<tr>
<td>Toddler (1−3 years)</td>
<td>24−40</td>
</tr>
<tr>
<td>Preschooler (3−6 years)</td>
<td>22−34</td>
</tr>
<tr>
<td>School-age (6−12 years)</td>
<td>18−30</td>
</tr>
<tr>
<td>Adolescent (12−18 years)</td>
<td>12−16</td>
</tr>
</tbody>
</table>

Cyanosis of tongue and oral mucosa indicate oxygen saturation (SaO₂) of less than 80 percent. However, there is significant inter-observer variability and difficulty in SaO₂ interpretation.

Let’s take a moment to review the Silverman-Anderson Index related to the assessment of the neonates with suspected or diagnosed RDS. When a neonate is premature, or has underlying pathology, then expiratory grunting, retraction of the chest wall muscles and other signs of respiratory distress may be readily seen. The Silverman – Anderson Index, commonly referred to as the Silverman retraction score, was developed as a systematic means of assessing newborn respiratory status, particularly when respiratory distress is suspected.

Silverman- Anderson Index (Table 2)

<table>
<thead>
<tr>
<th>Feature</th>
<th>Score 0</th>
<th>Score 1</th>
<th>Score 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest movement</td>
<td>Equal</td>
<td>Respiratory Lag</td>
<td>See-saw respiration</td>
</tr>
<tr>
<td>Intercostal retractions</td>
<td>None</td>
<td>Minimal</td>
<td>Marked</td>
</tr>
<tr>
<td>Xiphoid retraction</td>
<td>None</td>
<td>Minimal</td>
<td>Marked</td>
</tr>
<tr>
<td>Nasal Flaring</td>
<td>None</td>
<td>Minimal</td>
<td>Marked</td>
</tr>
<tr>
<td>Expiratory Grunt</td>
<td>None</td>
<td>Audible wheeze by stethoscope</td>
<td>Audible</td>
</tr>
</tbody>
</table>

The parameters assessed by inspection and auscultation of the upper and lower chest and nares on a scale of 0, 1, or 2. As it is observed in the table 2, the higher the score, the more severe is the respiratory distress.
Non-Invasive Respiratory monitoring

History
Oximetry measures the percentage of hemoglobin saturated with oxygen by passing specific wavelengths of light through the arterial blood. In 1875 a German physiologist named Karl von Vierofdt demonstrated that the oxygen in his hand was consumed when a tourniquet was applied. This was done utilizing transmitted light waves, but the development of the pulse oximeter was still a long way off. In 1936 Karl Matthes developed the first ear saturation meter that used two wavelengths of light. This compensated for the variations in tissue absorption. This idea was improved upon in 1940 when Glen Millikin developed a lightweight oximeter to help the military to solve their aviation hypoxia problem. The modern pulse oximeter was developed in 1972 by Takuo Aoyagi while he was working in Tokyo developing a noninvasive cardiac output measurement, using dye dilution and an ear densitometer. He noticed a correlation in the difference between unabsorbed infrared and red light and the oxygen saturation. This led to the clinical application of the pulse oximeter. It was not until 1980 that Nellcor produced the first commercial pulse oximeter that was reliable, robust, and affordable. In 1988 the use of a pulse oximeter during anesthesia and recovery room became mandatory in Australia. Since then, its use has become mandated in many areas from pre-hospital treatment to intensive care units.

Pulse oximetry is now an integral part of PICU monitoring which helps in the assessment of the patient’s cardio-respiratory (oxygenation) status. It is a simple, non-invasive and continuous method of monitoring the oxygen saturation of arterial blood (SaO₂) and now widely accepted as the fifth vital sign. The pulse oximeter is a convenient, cost-effective way to monitor the patient’s oxygenation status (and thereby O₂ content) and determine the changes before they are clinically apparent. It is important to know how oximeters work in order to maximize their performance and avoid errors in the interpretation of results.

Pulse oximetry is based on principles of spectrophotometry governed by Beer-Lambert law. The mandatory condition for interpretation of SaO₂ is the presence of a pulsatile arterioles blood flow.

How pulse oximeter works? Interpretation of SaO₂ is based on the fact that oxygenated hemoglobin (HbO₂) and deoxygenated hemoglobin (Hb) have different absorption spectra. Currently available pulse oximeters use two light-emitting diodes (LEDs) that emit light at the 660 nm (red) and the 940 nm (infrared) wavelengths. These two wavelengths are used because HbO₂ and Hb have different absorption spectra at these particular wavelengths. In the red region, HbO₂ absorbs less light than Hb, while the reverse occurs in the infrared region. The ratio of absorbencies at these two wavelengths is calibrated empirically against direct measurements of SaO₂ in volunteers, and the resulting calibration algorithm is stored in a digital microprocessor within the pulse oximeter. During subsequent use, the calibration curve is used to generate the pulse oximeter’s estimate of arterial saturation (SpO₂). In addition to the digital readout of O₂ saturation and pulse rate, most pulse oximeters display a plethysmographic waveform which can help clinicians to distinguish an artifactual signal from the true signal.

There are two techniques of measuring SaO₂: transmission and reflectance. In the transmission method the emitter and photodetector are opposite of each other with the measuring site in-between. The light can then pass through the site. In the reflectance method, the emitter and photodetector, is next to each other on top the measuring site. The light bounces from the emitter to the detector across the site. The transmission method is the most common type of method of choice in use.

The normal SpO₂ value for adolescents and elders is greater than 95%, and for children, a level greater than 90-92% is normal. SpO₂ can be misleading as other factors must be considered when determining whether this SpO₂ is normal for the particular patient.

Critical discussion on Pulse oximetry (SpO₂ = SaO₂)

- SaO₂ gives fairly good idea of not only saturation but also of oxygen content (CaO₂) provided Carboxyhemoglobin (COHb) and methemoglobin (MetHb) are expected in normal amounts. Since 98% of CaO₂ is contributed by saturated hemoglobin, hence it is a good idea that one should always calculate CaO₂, every time, after observing SpO₂ since CaO₂ is the better indicator of oxygenation.

\[
CaO₂ = SaO₂(98\%) + PaO₂(2\%)
\]

\[
[CaO₂ = 1.34[Hb]SaO₂ + PaO₂[0.003]
\]

Interpretation SpO₂ should always be done in context of ODC. Since conditions causing Left shift can have normal saturation but patient may be hypoxic (low PaO₂). Similarly conditions causing Right shift may have low SaO₂ but patient may not be hypoxic.
Limitations of Pulse oximetry Oximeters have a number of limitations which may lead to inaccurate readings. Shape of oxygen dissociation curve, Carboxyhemoglobin, Methemoglobin Anemia, Dyes, Nail polish, Ambient light, motion artifact, Skin pigmentation and Low perfusion states are other causes as well.

Pulse oximeters measure SpO\textsubscript{2} that is physiologically related to arterial oxygen tension (PaO\textsubscript{2}) according to the oxyhemoglobin dissociation curve (ODC). Because the ODC has a sigmoid shape, oximetry is relatively insensitive in detecting the development of hypoxemia in patients with high baseline levels of PaO\textsubscript{2} (upper flat portion of ODC curve).

Since pulse oximeters use only two wavelengths of light and, thus, it can distinguish only two substances, Hb and HbO\textsubscript{2}. When COHb and MetHb are also present, four wavelengths are required to determine the ‘fractional SaO\textsubscript{2}’; i.e., \((\text{HbO}_2 \times 100)/(\text{Hb} + \text{HbO}_2 + \text{COHb} + \text{MetHb})\) and this can be measured by Co-oximetry. In the presence of elevated COHb levels, oximetry consistently over-estimates the true SaO\textsubscript{2} by the amount of COHb present since it has got same absorption spectrum as of HbO\textsubscript{2}. Elevated MetHb levels also may cause inaccurate oximetry readings. Anemia does not appear to affect the accuracy of pulse oximetry even in non-hypoxemic patients with acute anemia; pulse oximetry was accurate in measuring O\textsubscript{2} saturation. Severe hyperbilirubinemia (mean bilirubin, 30.6 mg/dl) does not affect the accuracy of pulse oximetry.

Intravenous dyes such as methylene blue, indocyaninegreen, and indigocarmine can cause falsely low SpO\textsubscript{2} readings. Nail polish, if blue, green or black, causes inaccurate SpO\textsubscript{2} readings, whereas acrylic nails do not interfere with pulse oximetry readings. Falsely low and high SpO\textsubscript{2} readings occur with fluorescent and xenon arc surgical lamps. Motion artifact continues to be a significant source of error and false alarms. In a recent, prospective study in an intensive care unit setting, SpO\textsubscript{2} signals accounted for almost half of a total of 2525 false alarms.

Inaccurate oximetry readings have been observed in pigmented patients, but not by all investigators. Low perfusion states, such as low cardiac output, vasoconstriction and hypothermia may impair peripheral perfusion and may make it difficult for a sensor to distinguish a true signal from background layers.

An under-recognized and worrisome problem with pulse oximetry is that many users have a limited understanding of how it functions and the
implications of its measurements. In a recent survey, 30% of physicians and 93% of nurses thought that the oximeter measured \( \text{PaO}_2 \). Some clinicians also have a limited knowledge of the ODC, and they do not recognize that \( \text{SpO}_2 \) values in the high 80s represent seriously low values of \( \text{PaO}_2 \). In the above survey, some doctors and nurses were not especially worried about patients with \( \text{SpO}_2 \) values as low as 80% (equivalent to \( \text{PaO}_2 \leq 45 \text{ mm of Hg} \)).

Conventional pulse oximetry has problems during ambient light, abnormal hemoglobin, pulse rate and rhythm, vasoconstriction and cardiac function, physical motion and low perfusion and that has great impact on when making critical decisions. Arterial blood gas tests have been used to supplement or validate pulse oximeter readings. The advent of “Next Generation” pulse oximetry technology has demonstrated significant improvement in the ability to read through motion and low perfusion; thus making pulse oximetry more dependable to take decisions during critical period.

It is important to remember that pulse oximeters assess oxygen saturation only and thereby Oxygenation status and gives no indication of the level of \( \text{CO}_2 \) and thereby Ventilation status. For this reason they have a limited benefit in patients developing respiratory failure due to \( \text{CO}_2 \) retention.

The pulse oximeter may be used in a variety of situations that require monitoring of oxygen status and may be used either continuously or intermittently. It is not a substitute for an ABG, but can give clinicians an early warning of decreasing arterial oxyhemoglobin saturation prior to the patient exhibiting clinical signs of hypoxia. The pulse oximeter is a useful tool but the patient must be treated—not the numbers. As with all monitoring equipment, the reading should be interpreted in association with the patient’s clinical condition. If a patient is short of breath and bluish with a saturation reading of 100%, check for possible causes due to artifact. Never withhold therapeutic oxygen from a patient in distress while waiting to get a reading. If the patient appears to be in perfect health and the saturation is reading 70%, this should alert you to the possibility of interference. Never ignore a reading which suggests the patient is becoming hypoxic. The main disadvantage of pulse oximeter is its inability to use in cases of hyperoxia at saturations between 90-100%.

**Masimo pulse oximetry - a new promising way of measuring \( \text{SpO}_2 \)!!**

What makes Masimo pulse oximetry different from conventional pulse oximetry?

Conventional pulse oximetry assumes that arterial blood is the only blood moving (pulsating) in the measurement site. During patient motion, the venous blood also moves, which causes conventional pulse oximetry to under-read because it cannot distinguish between the arterial and venous blood. Masimo signal technology identifies the venous blood signal, isolates it, and cancels the noise and extracts the arterial signal, and then reports the true arterial oxygen saturation and pulse rate.

Following setbacks of Conventional Pulse Oximetry for inaccurate monitoring or signal dropout during the reading are rectified by Masimo technology:

- Patient Motion or Movement
- Low Perfusion (low signal amplitude)
- Intense Ambient Light (lighting or sunlight)
- Electrosurgical Instrument Interference

**Capnography**

End-tidal \( \text{CO}_2 \) (Et\( \text{CO}_2 \)) monitoring is an exciting non-invasive technology that is more commonly used in the emergency department, intensive care units and in the pre-hospital settings. Its main use has been in verifying endotracheal tube position, during mechanical ventilation and cardio-pulmonary resuscitation, but it is being studied and used for other purposes as well. The American Heart Association new guidelines states the secondary confirmation of proper endotracheal tube placement in all patients by exhaled \( \text{CO}_2 \) immediately after intubation and during transport is essential.

Et\( \text{CO}_2 \) monitoring is an exciting new technology that measures \( \text{CO}_2 \) in the exhaled breath continuously and non-invasively. \( \text{CO}_2 \) is produced during cellular metabolism, transported to the heart and exhaled via the lung and so Et\( \text{CO}_2 \) reflects ventilation, metabolism and circulation. If any two systems are kept constant then changes in the third system reflect
changes in EtCO₂. This was first studied clinically by Smallhout and Kalenda in the 1970’s, and in the late 1980’s – 1990’s this methodology has been studied extensively in various clinical settings. The most common use of EtCO₂ is to verify endotracheal tube (ETT) position. It is being increasingly studied and used during cardiopulmonary resuscitation (CPR) and other clinical settings.

What is Capnography?
It is a graphical representation of noninvasive, continuous measurement of exhaled carbon dioxide (EtCO₂) concentration over time accompanied by digital display that provides EtCO₂ value and distinct waveform (tracing) for each respiratory cycle.

Some definitions: Capnometry
• Capnometer: Provides only a numerical measurement of carbon dioxide
• Capnogram: Is a waveform display of carbon dioxide over time
• Capnography: A numerical value of the EtCO₂ and a waveform of the concentration of CO₂ present in the airway. And Respiratory rate detected from the actual airflow

Normal Capnogram (Fig)

The Capnogram is divided into four distinct phases:
1. Phase I (A-B) is the beginning of exhalation. It represents most of the anatomical dead space. CO₂ is almost zero.
2. Phase II (B-C) is where the alveolar gas begins to mix with the dead space gas and the CO₂ begins to rapidly rise.
3. Phase III (C-D) represents the alveolar gas, usually has a slight increase in the slope as “slow” alveoli empty. The “slow” alveoli have a lower V/Q ratio and therefore have higher CO₂ concentrations. In addition, diffusion of CO₂ into the alveoli is greater during expiration. This is more pronounced in infants. EtCO₂ is measured at the maximal point of Phase III (D)
4. Phase IV (D-E) is the inspiritional phase

Note that the presence of the alveolar plateau confirms that the measurement is End-tidal. Without a Capnography you cannot be sure that a measured CO₂ value is really end-tidal.

A normal value for ETCO₂ is approximately 38-40 mm Hg.

Types of CO₂ Monitors
There are two types of CO₂ monitors: 1) Mainstream and 2) Sidestream.

Mainstream…….salient features are……..
• The infrared sensor is located in the airway adapter, between the ET tube and the breathing circuit tubing.
• Response time is faster and may be as little as 40msec
• Water cannot be drawn-in to disrupt sensor function, and since no mixing of gases in the sample tube it is nearly a very accurate one.
• Difficult to calibrate without disconnecting (makes it hard to detect rebreathing)
• More prone to the reading being affected by moisture.
• Sensor device is larger in size hence can kink the tube.
• Adds dead space to the airway.
• Bigger chance of being damaged by mishandling.

Sidestream…… salient features are…..
• Can be used with in intubated or non-intubated patients thus have wider applications.
• The airway adapter is positioned at the airway (whether or not the patient is intubated) to allow aspiration of gas from the patient’s airway back to the sensor, which lies either within or close to the monitor, thus gas is sampled through a small tube
• Analysis is performed in a separate chamber
Clinical Applications of CO₂ Monitoring

The EtCO₂ level read on the display of the monitor depends upon the proper functioning of the following:

- Lungs and airways
- Patient ventilation system
- Respiratory mechanism
- Patient’s metabolism and circulation

Malfunctions of the lungs and airway OR the patient’s ventilation system can be depicted as follows:

- Upper airway obstruction – reflected by an increased EtCO₂
- Apnea – reflected by a sudden cessation of EtCO₂ readings
- Improper ventilator operation – reflected by either high or low EtCO₂ readings
- Hyperventilation – reflected by a decreased EtCO₂
- Hypoventilation – reflected by an increase in EtCO₂
- A faulty one-way valve – reflected by an increased inspired CO₂ and increased EtCO₂
- Esophageal intubation – reflected by no EtCO₂ reading
- Respiratory depression (from anesthesia) – reflected by a decreased EtCO₂
- Increased level of muscle relaxation – reflected by a decreased EtCO₂
- Reversal of muscle relaxant and resulting improvement in muscle tone – reflected by an increased EtCO₂
- Malignant hyperthermia – reflected by an increased EtCO₂

PaCO₂-EtCO₂ gradient

- It is usually < 6 mm Hg
- EtCO₂ is usually less
- Difference depends on the number of underperfused alveoli
- Tend to mirror each other if the slope of Phase III is horizontal or has a minimal slope

- Decreased cardiac output will increase the gradient
- The gradient can be negative when healthy lungs are ventilated with high tidal volume and low rate
- Decreased functional residual capacity also gives a negative gradient by increasing the number of slow alveoli

LIMITATIONS

1. Critically ill patients often have rapidly changing dead space and V/Q mismatch
2. Higher rates and smaller tidal volumes can increase the amount of dead space ventilation
3. High mean airway pressures and PEEP restrict alveolar perfusion, leading to falsely decreased readings
4. Low cardiac output will decrease the reading.

Indications for Capnography are:

1. Confirm and verify tracheal intubation placement.
2. Evaluate ventilator settings and circuit integrity.
3. Assess cardiopulmonary status and changes in pulmonary blood flow.
4. Assess airway management and changes in airway resistance.
5. Monitor effectiveness of CPR.
8. Monitor the effectiveness of ventilator weaning process, and response to changes in ventilator settings (i.e., respiratory rate, flow and/or volume).
9. Reduce the number and/or frequency of arterial blood gas drawings.
10. Aids in the treatment of neurological patients and the possibility of increasing intracranial pressures.

Other uses……

- Metabolic
  - Assess energy expenditure
- Cardiovascular
  - Monitor trend in cardiac output
  - Can use as an indirect Fick method, but actual numbers are hard to quantify
  - Measure of effectiveness in CPR
  - Diagnosis of pulmonary embolism by measuring measure gradient
### Differential Diagnosis of Abnormal Capnogram

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Possible Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sudden drop of EtCO₂ to zero</td>
<td>Esophageal intubation&lt;br&gt;Ventilator disconnection or malfunction&lt;br&gt;Defect in CO₂ analyzer&lt;br&gt;Dislodged or obstructed endotracheal tube</td>
</tr>
<tr>
<td>Sudden fall of EtCO₂ (not to 0)</td>
<td>Leak in ventilator system, obstruction&lt;br&gt;Partial disconnect in ventilator circuit&lt;br&gt;Partial airway obstruction (secretions)</td>
</tr>
<tr>
<td>Exponential fall of EtCO₂</td>
<td>Cardiac Arrest&lt;br&gt;Hypotension (sudden)&lt;br&gt;Severe hyperventilation&lt;br&gt;Cardiopulmonary bypass&lt;br&gt;Pulmonary Embolism</td>
</tr>
<tr>
<td>Change in CO₂ Baseline</td>
<td>CO₂ absorber saturation (anesthesia)&lt;br&gt;Calibration error&lt;br&gt;Water droplet in analyzer&lt;br&gt;Mechanical failure (ventilator)</td>
</tr>
<tr>
<td>Sudden increase of EtCO₂</td>
<td>Accessing an area of lung previously obstructed&lt;br&gt;Release of tourniquet&lt;br&gt;Sudden increase in blood pressure</td>
</tr>
<tr>
<td>Gradual lowering of EtCO₂</td>
<td>Hypovolemia&lt;br&gt;Decreasing Cardiac Output&lt;br&gt;Decreasing body temperature, hypothermia, drop in metabolism</td>
</tr>
<tr>
<td>Gradual increase in EtCO₂</td>
<td>Rising body temperature&lt;br&gt;Hypoventilation&lt;br&gt;CO₂ absorption&lt;br&gt;Partial airway obstruction (foreign body), reactive airway disease</td>
</tr>
<tr>
<td>Constantly high EtCO₂</td>
<td>Respiratory depression due to drugs&lt;br&gt;Metabolic alkalosis (respiratory compensation)&lt;br&gt;Insufficient minute ventilation</td>
</tr>
</tbody>
</table>

### Microstream Technology

It is 3<sup>rd</sup> generation technology which can be used with intubated or non-intubated patients and requires low sample flow rate - 50 ml/min. It allows its use in neonate & pediatric patients. In this technology sampling lines not flooded with moisture. Microstream improves upon conventional Sidestream sampling based upon the principle that CO₂ molecules absorb IR radiation at specific wavelengths.

**Advantages**

1. No sensor at airway
2. Intubated and non-intubated patients (neonatal through adult)
3. No routine calibration
4. Automatic zeroing
5. Accurate at small tidal volumes and high respiratory rates
6. Superior moisture handling

**Pulmonary Function Tests**

Few of the numerous pulmonary function tests currently available have an impact upon clinical management of the critically ill child, particularly if the patient has to be moved to a laboratory. A number of other tests require highly specialized equipment and fulfill a predominant research role.
Clinical relevant tests

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Tests</th>
<th>Common clinical use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pa02, S02, PaCO2</td>
<td>Arterial blood gases</td>
<td>Oxygenation, Ventilation status</td>
</tr>
<tr>
<td>SpO2</td>
<td>Pulse oximetry</td>
<td>Oxygen saturation, content status</td>
</tr>
<tr>
<td>End-tidal PCO2</td>
<td>Capnography</td>
<td>Ventilation status</td>
</tr>
<tr>
<td>Vital capacity, tidal volume</td>
<td>Spirometry, electronic flowmetry.</td>
<td>Serial measurement of borderline function (VC &lt; 10-15ml/kg) e.g. Gullain–Barré syndrome</td>
</tr>
<tr>
<td>Peak expiratory flow rate</td>
<td>Wright peak flow meter,</td>
<td>(Spontaneous ventilation) asthma</td>
</tr>
<tr>
<td>FEV1, FVC</td>
<td>Spirometry, electronic flowmetry.</td>
<td>(Spontaneous ventilation) asthma, obstructive / restrictive disease.</td>
</tr>
<tr>
<td>Lung/chest wall compliance</td>
<td>Pressure-volume curve</td>
<td>Ventilator adjustments, monitoring disease progression.</td>
</tr>
<tr>
<td>Flow volume loop, pressure volume loop</td>
<td>Pneumotachograph* manometry</td>
<td>Ventilator adjustment</td>
</tr>
</tbody>
</table>

*(Pneumotachograph: an apparatus for recording the rate of airflow to and from lungs)*

Research tests (examples)

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Tests</th>
<th>Research use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diaphragmatic strength</td>
<td>Gastric and esophageal manometry</td>
<td>Respiratory muscle functions, weaning.</td>
</tr>
<tr>
<td>(transdiaphragmatic pressure)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pleural(intrathoracic) pressure</td>
<td>Esophageal manometry</td>
<td>Ventilator trauma, work of breathing, weaning.</td>
</tr>
<tr>
<td>Functional residual capacity</td>
<td>Closed circuit helium dilution (bag-in-box)</td>
<td>Lung volumes, compliance</td>
</tr>
<tr>
<td></td>
<td>open circuit N2 washout.</td>
<td></td>
</tr>
<tr>
<td>Ventilation-perfusion relationship</td>
<td>Multiple inert gas elimination technique,</td>
<td>Regional Lung ventilation-perfusion, pulmonary gas exchange.</td>
</tr>
<tr>
<td></td>
<td>isotope technique</td>
<td></td>
</tr>
<tr>
<td>Pulmonary diffusing capacity</td>
<td>Carbon monoxide uptake</td>
<td>Pulmonary gas exchange.</td>
</tr>
</tbody>
</table>

Notes…
- Compliance equals the change in pressure during a linear increase in volume above FRC.
- The Bohr equation calculates physiological deadspace (Vd/Vt); normally it is less than 30%.
- The shunt equations estimates the proportion of blood shunted past poorly ventilated alveoli (Qs) compared to total lung blood flow (Qt).

These useful equations are supplement to assess pulmonary function, and ventilation/perfusion mismatch…
- V/Q = 1, Ventilation and perfusion are well matched.
- V/Q>1, increased deadspace (where alveoli are poorly perfused but well ventilated)
- V/Q<1, increased venous admixture or shunt (where alveoli are well perfused but poorly ventilated)

1. Alveolar gas equation: \( \text{PAO}_2 = \text{FiO}_2 \times (\text{PB} - \text{PH}_2\text{O}) - (\text{PaCO}_2 / \text{RQ}) \) [RQ=0.8]

2. Calculating the alveolar: arterial oxygen gradient: \( \text{(A-a) DO}_2 \), normal is 10-15 mm of Hg.
3. Bohr equation: \( V_d/V_t = (\text{PaCO}_2 - \text{expired PCO}_2)/\text{PaCO}_2 \)
4. Shunt equation: \( Q_s/Q_t = (C_{i,O}_2-CaCO_2) / (C_{v,O}_2-CvO_2) \) where \( CCO_2 = \) end capillary O\(_2\) content, \( a = \) arterial, \( v = \) mixed venous.
5. Expected \( \text{PaO}_2 = \text{FiO}_2 \times 5 \). A very useful equation with limitations.
1. P-V curve be obtained in fully relaxed and ventilated patient.
2. Both static (chest) and dynamic (lung) respiratory system compliance can be determined.
3. The lower inflexion point represents appropriate setting for external Positive End Expiratory Pressure (PEEP).
4. The upper inflexion point represents the maximum setting for PEAK AIRWAY PRESSURE (PAP).

X ray
A very commonly ordered investigation in PICU which has diagnostic, therapeutic and prognostic value is x-ray chest. (This has been discussed detailed in other chapter in this book)

Invasive monitoring
Arterial blood gas analysis
The term arterial blood refers to a specific set of tests performed on arterial blood sample. It provides four key point information: pH, \( \text{PO}_2 \), \( \text{HCO}_3^- \), and \( \text{PCO}_2 \). The name blood gas is really a partial misnomer since \( \text{H}^+ \) and \( \text{HCO}_3^- \) are not gases. It is a gold standard investigation to assess pulmonary functions and cardiac as well.

Basic Concepts
- Arterial Blood Gas
- Gas Exchange
- Acid-Base Disturbances

Systematic Analysis of Arterial Blood Gases
1. Oxygenation
2. Stepwise approach to Acid-Base Disorders

Basic Introduction of Arterial Blood Gases
The term hypoxia refers to reduced \( \text{O}_2 \) delivery to tissues. The term hypoxemia refers to reduced \( \text{O}_2 \) content in arterial blood. A normal arterial pressure of \( \text{O}_2 \) is dependent on the atmospheric pressure, temperature, inspired \( \text{O}_2 \) content, and the patient’s age.

Hypoxemia can be for two basic reasons; oxygen may not be delivered to the alveolar air sacs (hypoventilation) or oxygen in the alveoli may not enter into the blood stream. A patient can be hypercarbic (high levels of \( \text{CO}_2 \)) or hypocarbic (low level of \( \text{CO}_2 \)) which is due to an inability to normally exchange gas in the lungs.

The terms acidemia and alkalemia refer to alterations in blood pH, and are the result of underlying disturbance(s) (metabolic and/or respiratory). The terms acidosis and alkalosis refer to the processes that alter the acid-base status. There can be (and often are) more than one of these processes simultaneously in a patient

Diseases that alter the acid-base status of a patient can be divided….
1. Metabolic
2. Respiratory

Metabolic processes are those that primarily alter the \( \text{HCO}_3^- \) concentration in the blood. A decrease in serum \( \text{HCO}_3^- \) (an alkali or base) leads to a metabolic acidosis, while an increase in serum \( \text{HCO}_3^- \) leads to a metabolic alkalosis.

Respiratory processes alter the pH by changing the \( \text{CO}_2 \) levels. \( \text{CO}_2 \) accumulation causes an acid state in the blood (through carbonic acid), and as respirations (respiratory rate and/or tidal volume) increase, the body eliminates more \( \text{CO}_2 \) (acid) and is left with a respiratory alkalosis. In other words, a decrease in ventilation leads to retention and increased levels of \( \text{CO}_2 \), and thus a respiratory acidosis.

In conclusion, pH altering processes can be one of four types:
1. Metabolic acidosis,
2. metabolic alkalosis,
3. Respiratory acidosis,
4. Respiratory alkalosis.

Again, one or more of these processes may be present in a patient with an abnormal acid-base status.

Systematic Analysis of Arterial Blood Gases
Arterial blood gases are obtained for two basic purposes:
1. To determine oxygenation and
2. To determine acid-base status.

Let’s elaborate now, how to determine oxygenation, and then evaluate the acid-base status systematically.

Determining Oxygenation i.e. Alveolar: arterial oxygen gradient: \( (A-a) \text{DO}_2 \) (Age and FiO2 dependent derivative)
An important part of interpreting blood gases is to assess oxygenation. An arterial oxygen concentration
(\(P_{A,O_2}\)) of less than 60 mm Hg, associated with an oxygenation (\(S_{A,O_2}\)) of less than 90%, is poorly tolerated in humans; therefore a \(P_{A,O_2}\) of less than 60 is termed hypoxemic. However, “normal” oxygenation decreases with age as the lungs become less efficient at diffusing oxygen from the alveolus to the blood. Again, normal oxygenation for age can be estimated as…\(P_{A,O_2} = 104.2 - (0.27 \times \text{age})\) Or more crudely, normal oxygenation for age is roughly 1/3 of the patient’s age subtracted from 100. Using this estimation for example a 60-year-old patient should have a \(P_{A,O_2}\) of 80 and a 15-year-old patient should have a \(P_{A,O_2}\) of 95. Values less than this would be considered hypoxemic for age.

Calculating the alveolar: arterial oxygen gradient: (A-a) \(D_{O_2}\) can determine if hypoxia is a reflection of hypoventilation (in other words, decreased because of a rise in \(P_{A,CO_2}\)) or due to deficiency in oxygenation. Unlike oxygen (for which alveolar concentrations are higher than arterial concentrations) \(CO_2\) freely diffuses across the lung such that the arterial and alveolar concentrations are identical. As a patient hypoventilates, \(CO_2\) will accumulate in the body (more \(CO_2\) is produced through metabolism than can be eliminated) and thus in the blood (where we measure it as \(P_{A,CO_2}\)). The carbon dioxide displaces the oxygen in the alveolus. This reciprocal relationship between oxygen and carbon dioxide in the alveolus is described by the alveolar gas equation: \(P_{A,O_2}\) (partial pressure of oxygen in the alveolus) = \(150 - 1.25 \times (P_{A,CO_2})\)

\[PA = \text{partial pressure of a gas in the alveolus}.
\]
\[Pa = \text{partial pressure of a gas in the arterial blood}.
\]
This equation assumes that the patient is breathing room air (21% \(O_2\)) at atmospheric pressure.

Where do 150 come from? :
- (Atmospheric P - water vapor P) x FIO\(_2\). At room temperature, at sea level,
- Atmospheric pressure = 760 mm Hg;
- In the lung, the air is fully saturated with water, giving a water vapor pressure of about 47.
- Room Air is about 21%, thus at room air, the \(P_{A,O_2}\) = 0.21(760-47) = 149.7, or about 150.

AND…Where does 1.25 come from?
This is a fudge factor which is derived from the respiratory quotient. The formula actually requires that the \(P_{A,CO_2}\) be divided by the respiratory quotient, which is defined as the ratio of \(CO_2\) produced to \(O_2\) consumed (and which depends on diet and metabolism). We estimate the RQ to be 0.8, and the reciprocal of 0.8 is 1.25.
This value is the partial pressure of \(O_2\) within the alveolus. Because the \(CO_2\) freely diffuse from arterial blood to alveolar airspaces, the \(P_{A,CO_2}\) is equal to the \(P_{A,O_2}\), which is measured in the arterial blood gas. The above equation can then be rewritten as \(P_{A,O_2} = 150 - 1.25 \times (P_{A,CO_2})\)

Thus….A-a \(D_{O_2} = \frac{P_{A,O_2} - Pa_{O_2}}{\text{Pa}}\) Or

\(A-a \ D_{O_2} = [150 - 1.25 \times (P_{A,CO_2})] - P_{O_2}\)

A normal A-a gradient is 10-20 mm Hg, with the normal gradient increasing within this range as the patient ages. An increased A-a gradient identifies decreased \(O_2\) in the arterial blood compared to the \(O_2\) in the alveolus. This suggests a process that interferes with gas transfer, or in general terms, suggests ventilation-perfusion mismatch. A normal A-a gradient in the face of hypoxemia suggests the hypoxemia is due to hypoventilation and not due to underlying lung disorders.

When the patient is not breathing room air then…

\(A-a \ G_{O_2} = \{(FIO\(_2\) \times 760 - 47) - (1.25 \times (P_{A,CO_2})) - P_{O_2}\)
Stepwise Approach to Diagnosing Acid-Base Disorders

In order to understand the various processes that can co-exist in a patient, one must systematically evaluate blood gases and serum electrolytes. The simple method of six steps to analyze the acid-base status of the patient is presented here.

| pH and HCO₃ | Moves in same direction |
| pH and PCO₂ | Moves in opposite direction |
| HCO₃ and PCO₂ | Moves in same direction |
| HCO₃ and PCO₂ | Moves in opposite directions | Simple disorder |
| HCO₃ and PCO₂ | Moves in opposite directions | Mixed disorder |

Normal values of ABG

Steps in Acid-Base Analysis

- Step 1. Consider the clinical settings! Anticipate the disorder!
- Step 2. Look at pH?
- Step 3. Who is the culprit for changing pH?... Metabolic / Respiratory process
- Step 4. If respiratory...... acute and /or chronic And Is metabolic compensation appropriate?
- Step 5. If metabolic acidosis, Is respiratory compensation appropriate? Anion gap'ed and/or normal or both?
- Step 6. Is more than one disorder present? Mixed one?

STEP 1: Clinical assessment based on clinical settings is an essential first step. From the history, examination and initial investigations make a clinical decision as to what is the most likely acid-base disorder(s).

This is very important but be aware that in some situations, the history may be inadequate, misleading or the range of possible diagnoses large. Mixed disorders are often difficult: the history and examination alone are usually insufficient in sorting these out.

1. Vomiting.......... Metabolic alkalosis
2. Diarrhoea .......... Metabolic acidosis

3. Septicemia .......... Lactic acidosis
4. Hypotension, Hypoxemia, Shock .......... Lactic acidosis
5. Diabetes mellitus... Ketoacidosis
6. Pneumonia .......... Respiratory alkalosis/acidosis
7. Bronchial asthma .......... Respiratory alkalosis/acidosis
8. Hepatic failure .......... Respiratory alkalosis, Metabolic alkalosis
9. CNS disorders .......... Respiratory alkalosis
10. Renal disorders .......... Metabolic acidosis

*KEY POINT: Metabolic alkalosis and acidosis can exist together with any respiratory either acidosis or alkalosis. Both two respiratory disorders can’t occur simultaneously

STEP 2: Look at the pH

The pH of the arterial blood gas measurement identifies the disorder as alkalemic or acidemic. pH >7.4 ....... Alkalosis, pH < 7.4 ............... Acidosis, pH = 7.4 ............... Normal or mixed disorder

(Only Chronic Respiratory alkalosis can have normal value of pH)

STEP 3: Who is responsible for this change in pH? Who is the CULPRIT?

HCO₃...... METABOLIC  PCO₂ ...... Respiratory > 26 ...... Met. Alkalosis > 45 ...... Resp. Acidosis
< 22 ...... Met. Acidosis < 35 ...... Resp. Alkalosis

It is essential to determine whether the disturbance affects primarily the arterial PaCO₂ or the serum HCO₃.

- ...... Respiratory disturbances alter the arterial PaCO₂ (normal value 35-45)
- ...... Metabolic disturbances alter the serum HCO₃ (normal value 22-26)

If the pH is low (i.e., the primary and controlling disturbance is acidity causing acidemia) either the PaCO₂ is high or the HCO₃ is low. (These are the only ways in which the pH can be low). A high PaCO₂ defines a primary respiratory acidity and a low HCO₃ defines a primary metabolic acidosis. Conversely, if the pH is high (i.e., the primary and controlling disturbance is alkalosis causing alkalemia) either the PaCO₂ is low or the HCO₃ is high. (These are the only ways in which the pH can be high). A low
PaCO₂ defines a primary respiratory alkalosis and a high HCO₃⁻ defines a primary metabolic alkalosis.

**STEP 4**: If it is a primary respiratory disturbance, Is it acute? And/OR Chronic.

*For 10 mm change in pCO₂*

- **Acidosis** (↑CO₂) → pH ↓ → acute ...... by 0.08, chronic ...... by 0.03
- **Alkalosis** (↓CO₂) → pH ↑ → acute ...... by 0.08, chronic ...... by 0.03

**HCO₃⁻** Compensates as ....

- **Acidosis** (↑CO₂) → HCO₃⁻↑ ....... Acute ...... by 1, Chronic ...... by 3
- **Alkalosis** (↓CO₂) → HCO₃⁻↓ ....... Acute ...... by 2, Chronic ...... by 5

For example,

- In an acute respiratory acidosis, if the PCO₂ rises from 40 to 50, you would expect the pH to decline from 7.40 to 7.32.
- In an acute respiratory alkalosis, if the PCO₂ falls from 40 to 30, you would expect the pH to rise from 7.40 to 7.48.

In chronic respiratory disturbances, there are renal mediated shifts of bicarbonate that alter and partially compensate for the pH shift for a change in the PaCO₂.

- In a chronic respiratory acidosis, if the PCO₂ rises from 40 to 50, you would expect the pH to decline from 7.40 to 7.37.
- In a chronic respiratory alkalosis, if the PCO₂ falls from 40 to 30, you would expect the pH to rise from 7.40 to 7.43.

Remember: to suspect if
- compensated HCO₃⁻ is > expected: additional metabolic alkalosis is there
- compensated HCO₃⁻ is < expected: additional metabolic acidosis is there

**STEP 5**:

If it is a primary metabolic disturbance, whether respiratory compensation appropriate?

For metabolic acidosis: Expected PCO₂ = (1.5 x [HCO₃⁻]) + 8 + 2 ........ Winter’s formula

For metabolic alkalosis: Expected PCO₂ = 6 mm for 10 mEq. rise in Bicarb.

.........UNCERTAIN COMPENSATION

*Remember*: to suspect if
- Compensated PCO₂ is > expected: additional respiratory acidosis is there.
- Compensated PCO₂ is < expected: additional respiratory alkalosis is there.

Processes that lead to a metabolic acidosis can be divided into

1) Increased anion gap and 2) Normal anion gap.

The anion gap is the difference between the measured serum cations (positive) and the measured serum anions (negative). *(Of course, there is no real gap; in the body the numbers of positive and negative charges are balanced. The gap refers to the difference in positive and negative charges among cations and anions which are commonly measured.)*

The commonly measured cation is sodium. (Some people also use potassium to calculate the gap; that results in a different range of normal values.) The measured anions include chloride and bicarbonate. Thus the anion gap can be summarized as: \( AG = [Na^+] - ([Cl^-] + [HCO₃^-]) \).

The normal anion gap is 12. An anion gap of > than 12 is increased. Anion gap > 25 has got distinct value having significant ACIDOSIS. This is important, because it helps to significantly limit the differential diagnosis of a metabolic acidosis. The most common etiologies of a metabolic acidosis with an increased anion gap include:

- Commonest pediatric causes are Lactic acidosis, diabetic ketoacidosis and renal failure.
- Aspirin, Ketones (starvation, alcoholic and diabetic ketoacidosis)
- Uremia (renal failure), Lactic acidosis, Ethanol, Paraldehyde and other drugs
- Methanol other alcohols, and ethylene glycol intoxication

*Key point*: The true anion gap is underestimated in hypoalbuminemia (fall in unmeasured anions); \( AG \) must be adjusted. Remember to adjust AG: For every 1.0 fall in albumin, increase the AG by 2.5

**STEP 6**: Is more than one DISORDER present?

- Proper Clinical history
- pH normal, and PCO₂ and HCO₃ out of range
- PCO₂ and HCO₃ moving in opposite directions
- Degree of compensation for primary disorder is inappropriate.

**Key messages**

1. Respiratory monitoring helps in the early diagnosis of change in a physiological parameter of oxygenation and ventilation, and provides guidelines towards institution of appropriate therapy.
2. Basic knowledge of the principles of monitoring tools and correct interpretation of data is important since failure to do so can result in misdirected therapy.
3. Pulse oximetry and Capnography are the essential monitors in PICU which need clinical correlation.
4. Arterial blood gas analysis is an integral part of respiratory monitoring in PICU.
5. No amount of monitoring, though excellent information provided by monitors, however, can replace careful bedside clinical signs.

**Reference**

4. Lawrence Martin. In : All you really need to know to interpret arterial Blood gases 1992