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Symposium: Monitoring in Pediatric Critical Care

Respiratory Monitoring in PICU

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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 a 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 subcostal retractions, use of accessory muscles of respiration and paradoxical breathing.

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Normal Respiratory Rates…….. (Table1)

Age	Respiratory rate
Infant (birth–1 year)	30-60
Toddler (1–3 years)	24-40
Preschooler (3–6 years)	22-34
School-age (6–12 years)	18-30
Adolescent (12–18 years)	12-16

Cyanosis of tongue and oral mucosa indicate oxygen saturation (SaO_2) of less than 80 percent. However, there is significant inter-observer variability and difficulty in SaO_2 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 a 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)

Feature	Score 0	Score 1	Score 2
Chest movement	Equal	Respiratory Lag	See-saw respiration
Intercostal retractions	None	Minimal	Marked
Xiphoid retraction	None	Minimal	Marked
Nasal Flaring	None	Minimal	Marked
Expiratory Grunt	None	Audible wheeze by stethoscope	Audible

The parameters assessed by inspection and auscultation of the upper and lower chest and nares on a scale of 0,1or 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 (SaO_2) 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 O_2 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 SaO_2 is the presence of a pulsatile arteriolar blood flow.*

How pulse oximeter works? Interpretation of SaO_2 is based on the fact that oxygenated hemoglobin (HbO_2) 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 HbO_2 and Hb have different absorption spectra at these particular wavelengths. In the red region, HbO_2 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 SaO_2 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 (SpO_2). In addition to the digital readout of O_2 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 SaO_2: 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.

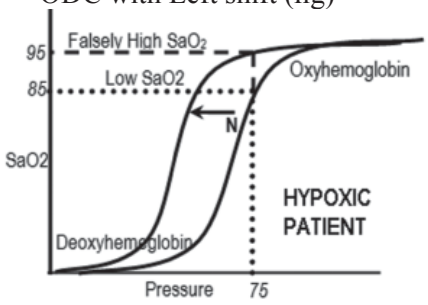
The normal SpO_2 value for adolescents and elders is greater than 95%, and for children, a level greater than 90-92% is normal. SpO_2 can be *misleading* as other factors must be considered when determining whether this SpO_2 is normal for the particular patient.

Critical discussion on Pulse oximetry ($SpO_2 = SaO_2$)

- SaO_2 gives fairly good idea of not only saturation but also of *oxygen content* (CaO_2) provided Carboxyhemoglobin (COHb) and methemoglobin (MetHb) are expected in normal amounts. Since 98% of CaO_2 is contributed by saturated hemoglobin, hence it is a good idea that one should always calculate CaO_2, every time, after observing SpO_2 since CaO_2 is the better indicator of oxygenation.
  $CaO_2 = SaO_2$ **(98%)** + PaO_2 (2%).
  [$CaO_2 = 1.34ÏHbÏSaO_2 + PaO_2Ï0.003$]

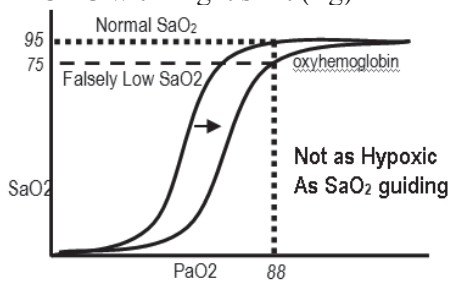
Interpretation SpO_2 should always be done in context of ODC. Since conditions causing Left shift can have normal saturation but patient may be hypoxic (low PaO_2). Similarly conditions causing Right shift may have low SaO_2 but patient may not be hypoxic.

- ODC with Left shift (fig)



Shift to L…(a *L* kalosis)
1. *Higher affinity of O_2 towards Hb (tight binding).*
2. *Easy on loading of O_2 from lungs*
3. *High SaO_2 for given PaO_2 (Falsely high)*
4. *CaO_2 is relatively high still difficult and less DO_2 to tissues*

- ODC with Right shift (fig)



Shift to R
1. *Lower affinity of O_2 towards Hb (loose binding).*
2. *Easy off loading O_2 to tissues*
3. *Low SaO_2 for given PaO_2 (Falsely low)*
4. *CaO_2 relatively less still, easy and more DO_2 to tissues*

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_2 that is physiologically related to arterial oxygen tension (PaO_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_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_2. When COHb and MetHb are also present, four wavelengths are required to determine the 'fractional SaO_2': i.e., ($HbO_2 \times 100$)/ (Hb + HbO_2 + COHb + MetHb) and this can be measured by Co-oximetry. In the presence of elevated COHb levels, oximetry consistently over- estimates the true SaO_2 by the amount of COHb present since it has got same absorption spectrum as of HbO_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_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_2 readings. Nail polish, if blue, green or black, causes inaccurate SpO_2 readings, whereas acrylic nails do not interfere with pulse oximetry readings. Falsely low and high SpO_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_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 PaO_2. Some clinicians also have a limited knowledge of the ODC, and they do not recognize that SpO_2 values in the high 80s represent seriously low values of PaO_2. In the above survey, some doctors and nurses were not especially worried about patients with SpO_2 values as low as 80% (equivalent to $PaO_2 \leq 45$ 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 CO_2 and thereby Ventilation status. For this reason they have a limited benefit in patients developing respiratory failure due to 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 SpO2 !!

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 CO_2 ($EtCO_2$) 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 CO_2 immediately after intubation and during transport is essential.

$EtCO_2$ monitoring is an exciting new technology that measures CO_2 in the exhaled breath continuously and non-invasively. CO_2 is produced during cellular metabolism, transported to the heart and exhaled via the lung and so $EtCO_2$ 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 $EtCO_2$ is to verify endotracheal tube (ETT) position. It is being increasingly studied and used during cardiopulmonary resuscitation (CPR) and other clinical settings.

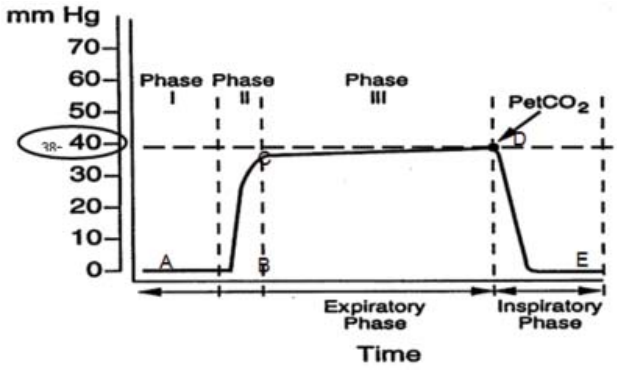
What is Capnography?

It is a graphical representation of noninvasive, continuous measurement of exhaled carbon dioxide ($EtCO_2$) concentration over time accompanied by digital display that provides $EtCO_2$ 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 $EtCO_2$ and A waveform of the concentration of CO_2 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. CO_2 is almost zero.
2. Phase II (B-C) is where the alveolar gas begins to mix with the dead space gas and the CO_2 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 CO_2 concentrations. In addition, diffusion of CO2 into the alveoli is greater during expiration. *This is more pronounced in infants.* $EtCO_2$ is measured at the maximal point of Phase III (D)
4. Phase IV (D-E) is the inspirational 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 CO_2 value is really end-tidal.

A normal value for $ETCO_2$ is approximately 38-40 mm Hg.

Types of CO_2 Monitors

There are two types of CO_2 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

- Very reliable
- Time delay of 1-60 seconds
- Less accurate at higher respiratory rates
- Prone to plugging by water and secretions
- Ambient air leaks are common.

Clinical Applications of CO_2 Monitoring

The $EtCO_2$ 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 $EtCO_2$
- Apnea – reflected by a sudden cessation of $EtCO_2$ readings
- Improper ventilator operation – reflected by either high or low $EtCO_2$ readings
- Hyperventilation – reflected by a decreased $EtCO_2$
- Hypoventilation – reflected by an increase in $EtCO_2$
- A faulty one-way valve – reflected by an increased inspired CO_2 and increased $EtCO_2$
- Esophageal intubation – reflected by no $EtCO_2$ reading
- Respiratory depression (from anesthesia) – reflected by a decreased $EtCO_2$
- Increased level of muscle relaxation – reflected by a decreased $EtCO_2$
- Reversal of muscle relaxant and resulting improvement in muscle tone – reflected by an increased $EtCO_2$
- Malignant hyperthermia – reflected by an increased $EtCO_2$

$PaCO_2$-$EtCO_2$ gradient

- It is usually < 6 mm Hg
- $EtCO_2$ 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.

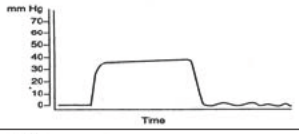
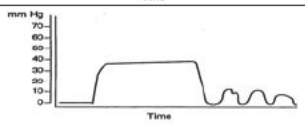
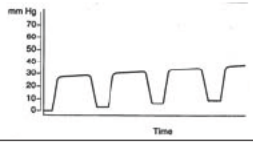
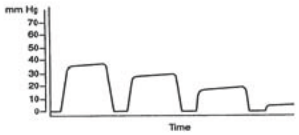
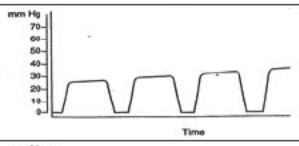
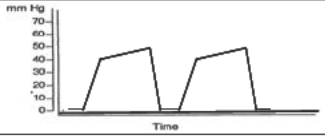
Indications for Capnography are:

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.
6. Monitor ventilatory status of the respiratory impaired patient.
7. Monitor ventilation of a nonintubated patient during sedation/analgesia.
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)

Symptom	Possible Cause	
Sudden drop of $EtCO_2$ to zero	Esophageal intubation Ventilator disconnection or malfunction Defect in CO2 analyzer Dislodged OR obstructed endotracheal tube	
Sudden fall of $EtCO_2$ (not to 0)	Leak in ventilator system, obstruction Partial disconnect in ventilator circuit Partial airway obstruction (secretions)	
Exponential fall of $EtCO_2$	Cardiac Arrest Hypotension (sudden) Severe hyperventilation Cardiopulmonary bypass Pulmonary Embolism	
Change in CO_2 Baseline	CO2 absorber saturation (anesthesia) Calibration error Water droplet in analyzer Mechanical failure (ventilator)	
Sudden increase of $EtCO_2$	Accessing an area of lung previously obstructed Release of tourniquet Sudden increase in blood pressure	
Gradual lowering of $EtCO_2$	Hypovolemia Decreasing Cardiac Output Decreasing body temperature, hypothermia, drop in metabolism	
Gradual increase in $EtCO_2$	Rising body temperature Hypoventilation CO_2 absorption Partial airway obstruction (foreign body), reactive airway disease	
Constantly high $EtCO_2$	Respiratory depression duo to drugs Metabolic alkalosis (respiratory compensation) Insufficient minute ventilation	

Microstream technology

It is 3rd 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 CO_2 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

Measurement	Tests	Common clinical use
PaO_2,SaO_2,$PaCO_2$	Arterial blood gases	Oxygenation, Ventilation status
SpO_2	Pulse oximetry	Oxygen saturation, content status
End-tidal PCO_2	Capnography	Ventilation status
Vital capacity, tidal volume	Spirometry, electronic flowmetry.	Serial measurement of borderline function (VC < 10-15ml/kg) e.g. Gullain –Barré syndrome
Peak expiratory flow rate	Wright peak flow meter,	(Spontaneous ventilation) asthma
FEV_1, FVC	Spirometry, electronic flowmetry.	(Spontaneous ventilation) asthma, obstructive / restrictive disease.
Lung/chest wall compliance	Pressure- volume curve	Ventilator adjustments, monitoring disease progression.
Flow volume loop, pressure volume loop	Pneumotachograph* manometry	Ventilator adjustment

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

Research tests (examples)

Measurement	Tests	Research use
Diaphragmatic strength (transdiaphragmatic pressure)	Gastric and esophageal manometry	Respiratory muscle functions, weaning.
Pleural(intrathoracic) pressure	Esophageal manometry	Ventilator trauma, work of breathing, weaning
Functional residual capacity	Closed circuit helium dilution (bag-in-box) open circuit N_2 washout.	Lung volumes, compliance
Ventilation-perfusion relationship	Multiple inert gas elimination technique, isotope technique	Regional Lung ventilation-perfusion, pulmonary gas exchange.
Pulmonary diffusing capacity	Carbon monoxide uptake	Pulmonary gas exchange.

Notes…

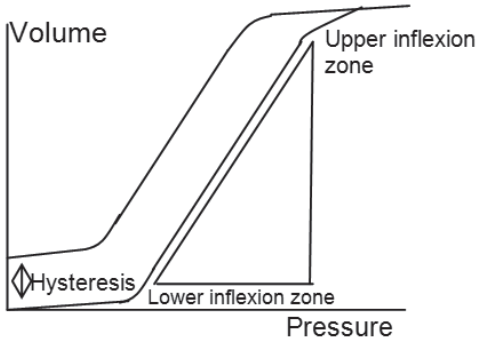
- Compliance equals the change in pressure during a linear increase in volume above FRC.
- The Bohr equation calculates physiological deadspace (V_D); normally it is less than 30%.
- The shunt equations estimates the proportion of blood shunted past poorly ventilated alveoli (Qs) compared to total lung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

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: $PAO_2 = FiO_2 (PB - PH_2O) - (PaCO_2 / RQ)$ [RQ=0.8]
2. Calculating the alveolar: arterial oxygen gradient**:** (A-a) DO_2, normal is 10-15 mm of Hg.
3. Bohr equation: $V_D/V_T = (PaCO_2\text{-expired } PCO_2)/ PaCO_2$
4. Shunt equation: $Qs/Q_T = (C_CO_2\text{-}CaCO_2)/ (C_CO_2\text{-}CvO_2)$ where CCO_2= end capillary O_2 content, a = arterial, v= mixed venous.
5. Expected $PaO_2 = FiO_2$Ï5. A very useful equation with limitations.

Pressure-volume curve relationship



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, PO_2, [HCO_3], and PCO_2. The name *blood gas* is really a partial misnomer since H^+ and 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 O_2 delivery to tissues. The term **hypoxemia** refers to reduced O_2 content in arterial blood. A normal arterial pressure of O_2 is dependent on the atmospheric pressure, temperature, inspired 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 CO_2) Or hypocarbic (low level of 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 HCO_3 concentration in the blood. A decrease in serum HCO_3 (an alkali or base) leads to a metabolic acidosis, while an increase in serum HCO_3 leads to a metabolic alkalosis.

Respiratory processes alter the pH by changing the CO_2 levels. 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 CO_2 (acid) and is left with a respiratory alkalosis. In other words, a decrease in ventilation leads to retention and increased levels of 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.

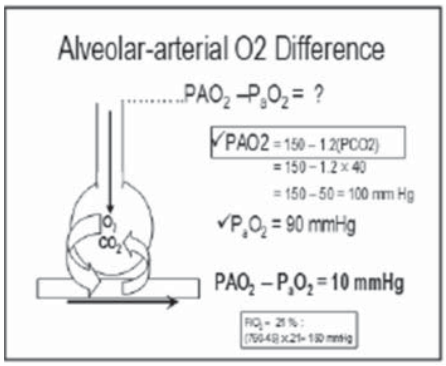
Determining Oxygenation i.e. Alveolar: arterial oxygen gradient: (A-a) DO_2

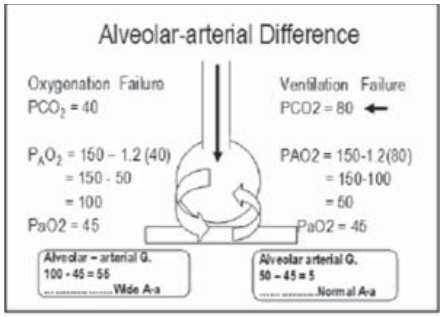
(Age and FiO2 dependent derivative)

An important part of interpreting blood gases is to assess oxygenation. An arterial oxygen concentration

(PaO_2) of less than 60 mm Hg, associated with an oxygenation (SaO_2) of less than 90%, is poorly tolerated in humans; therefore a PaO_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…PaO_2 = 104.2 - (0.27 x 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 PaO_2 of 80 and 15-year-old patient should have a PaO_2 of 95. Values less than this would be considered hypoxemic for age.

Calculating the alveolar: arterial oxygen gradient:
(A-a) DO_2 can determine if hypoxia is a reflection of hypoventilation (in other words, decreased because of a rise in $PaCO_2$) 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 $PaCO_2$). 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: PAO_2 (partial pressure of oxygen in the alveolus) = 150-1.25 ($PACO_2$)
PA = partial pressure of a gas in the alveolus.
Pa = 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 PAO_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 $PACO_2$ 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 $PACO_2$ is equal to the $PaCO_2$, which is measured in the arterial blood gas. The above equation can then be rewritten as
PAO2 = 150-1.25 ($PaCO_2$)
Thus….A-a DO_2 = PAO_2 - PaO_2 Or
A-a DO_2 = [150-1.25 ($PaCO_2$)] - PaO_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 gradient = {(FIO_2) (760-47) - (1.25) ($PaCO_2$)} - PaO_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 HCO_3 …Moves in same direction pH and PCO_2 ….Moves in opposite direction HCO_3 and PCO_2 …Moves in same direction …Simple disorder HCO_3 and PCO_2 … Moves in opposite directions …mixed disorder

pH : 7.35-7.45, PCO_2 : 35-45 HCO_3 : 22-26, PaO_2 : 90-99

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 PCO_2 …… 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 $PaCO_2$ or the serum HCO_3.

- ……Respiratory disturbances alter the arterial $PaCO_2$ (normal value 35-45)
- ……Metabolic disturbances alter the serum HCO_3 (normal value 22-26)

If the pH is low (i.e., the primary and controlling disturbance is acidosis causing acidemia) either the $PaCO_2$ is high or the HCO_3 is low. (These are the only ways in which the pH can be low). A high $PaCO_2$ defines a primary respiratory acidosis and a low HCO_3 defines a primary metabolic acidosis.

Conversely, if the pH is high (i.e., the primary and controlling disturbance is alkalosis causing alkalemia) either the $PaCO_2$ is low or the HCO_3 is high. (These are the only ways in which the pH can be high). A low

$PaCO_2$ defines a primary respiratory alkalosis and a high HCO_3 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 pCO_2
pH….changes….as
Acidosis (↑CO_2).....pH ↓ … acute……by 0.08, chronic…by 0.03
Alkalosis (↓CO_2).... pH ↑… acute…… by 0.08, chronic…by 0.03
HCO3…. Compensates as….
Acidosis (↑CO_2).....HCO_3↑…….. Acute……by 1, Chronic…by 3
Alkalosis (↓CO_2) … HCO_3↓……Acute……by 2, Chronic…by 5
For example,
In an acute respiratory acidosis, if the PCO_2 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 PCO_2 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 $PaCO_2$.
In a chronic respiratory acidosis, if the PCO_2 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 PCO_2 falls from 40 to 30, you would expect the pH to rise from 7.40 to 7.43.

Remember: to suspect if
- compensated HCO_3 is > expected: additional metabolic alkalosis is there
- compensated HCO_3 is < expected: additional metabolic acidosis is there

STEP 5 :
If it is a primary metabolic disturbance, whether respiratory compensation appropriate?
For metabolic acidosis: Expected PCO_2 = (1.5 x [HCO_3]) + 8 + 2 …….. Winter's formula
OR Expected CO_2 is equal to Last two digits of pH (important & easy to remember.)
For metabolic alkalosis: Expected PCO_2 = 6 mm for 10 mEq. rise in Bicarb.
……UNCERTAIN COMPENSATION
Remember : to suspect if
- Compensated PCO_2 is > expected : additional respiratory acidosis is there .
- Compensated PCO_2 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] + [HCO_3-]).
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 PCO_2 and HCO_3 out of range

- PCO_2 and HCO_3 moving in opposite directions
- Degree of compensation for primary disorder is inappropriate.

Conflict of Interest: None **Source of Funding**: None

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.

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