The Oxygen Goes In... and What To Do When It Doesn’t

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DISCLOSURES

None of the planners or presenters of this session have disclosed any conflict or commercial interest.
The Oxygen Goes In...and What To Do When It Doesn’t

OBJECTIVES:

1. Understand oxygenation, assessment of hypoxemia, and refractory hypoxemia.
2. Describe the various basic and advanced oxygen delivery systems, including non-invasive and invasive mechanical ventilation strategies.
3. Explain management of hypoxemia, and hypoxemic respiratory failure.
Pop Quiz...

If we breathe oxygen during the day, what do we breathe at night?

Nitrogen
Goals and Objectives (For You)

• Understand the physiologic mechanisms required for oxygen delivery.
• Understand the differences between Hypoxemia and Hypoxia.
• Understand the differences between and clinical implications of PaO2, SaO2, SpO2, and FiO2.
• Understand Oxygen Dissociation and the O2 Dissociation Curve.
• Understand effects of pathophysiology on the O2 Dissociation Curve and Tissue Oxygenation.
Goals and Objectives
(For Me)

• Discuss components of oxygenation.
• Discussion on prescribing oxygen therapy.
• Discuss monitoring oxygen therapy.
• Cover myths and truths about oxygen therapy.
• Discuss non-invasive and invasive treatments.
The “Air” We Breathe

- Nitrogen: 78.08%
- Oxygen: 20.95%
- Argon: 0.93%
- Xenon: 0.000009%
- Neon: 0.0018%
- Hydrogen: 0.00005%
- Helium: 0.0005%
- Krypton: 0.0001%
- Carbon dioxide: 0.038%
The Clinical Oxygenation Puzzle

Rocket Science?
Keep Them Straight

• **Hypoxemia** – a decreased partial pressure (PO2) in the blood plasma.

• **Hypoxia** – a decreased level of tissue oxygenation.
  o Not “measured” in a clinical sense.
  o PO2 and factors affecting oxyhemoglobin dissociation allow us to make a clinical judgment of the degree of hypoxia.
  o Other lab values can lead us to diagnosing hypoxia:
    o HHb
    o Lactate
Degrees of Hypoxemia

- **Mild** – PaO2 65 – 80 mmHg
- **Moderate** – PaO2 50 – 64.9 mmHg
- **Severe** – PaO2 < 50 mmHg
Oxygen Delivery

Four Major Components Must Be Intact

1. Bulk flow from the environment to a highly vascularized surface (ventilation).
2. Diffusion into the blood.
3. Bulk flow to body tissues (cardiac output).
4. Diffusion into mitochondria of cell.
4 = 3

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1. External Respiration (O2 loading).
2. Oxygen Delivery (O2 transport).
3. Internal Respiration (O2 unloading).
External Respiration (Oxygen Loading)
The Act of a Spontaneous Breath

• Contraction of diaphragm and intercostals:
  • Diaphragmatic contraction – increases vertical diameter of thoracic cavity:
    • Air flows primarily to lung bases.
    • Intercostals – increase the A-P diameter of the thoracic cavity.

• During exercise and disease states:
  • Accessory muscles:
    • Sternocleidomastoid muscles (elevate sternum).
    • Scalene muscles (elevate first 2 ribs).
    • Pectoralis muscles (elevate ribs 3-5).
Factors Affecting External Respiration (Loading)

- Distribution of Pulmonary Ventilation.
- Distribution of Pulmonary Circulation.
Factors Affecting External Respiration (Loading)
Factors affecting External Respiration: Disturbances of Ventilation

- Increased Airway Resistance
  - Pulmonary secretions
  - Bronchospasm
  - Mucosal edema
  - Artificial airways
  - Extrinsic airway compression

- Abnormal FRC
  - Increased (COPD)
  - Decreased (Atelectasis)

- Positive Pressure Ventilation
- Airway Closure
Factors affecting External Respiration: Disturbances of Perfusion

• Primary Disturbances
  • Pulmonary Embolus
  • Vascular Tumors
  • Medications:
    • Isoproteronol
    • Nitroglycerin

• Compensatory Disturbances
  • HPV (Hypoxic Pulmonary Vasoconstriction)
Factors affecting External Respiration: Deadspace

• Deadspace – Ventilation that does not participate in gas exchange (wasted ventilation):
  • Alveolar – No true alveolar deadspace in healthy individuals:
    • Pulmonary embolus.
    • Decreased Cardiac Output (CO).
  • Anatomic – Conducting Airways (≈150ml).
  • Physiologic – Sum of Alveolar + Anatomic.
Dead Space vs. Alveolar Ventilation—Some Definitions

- **Dead Space Ventilation** (VD) – that volume of VE that does not participate in gas exchange, made up of (1) gas filling the conducting airways and (2) gas reaching the alveoli but not participating in gas exchange.

- **Alveolar Ventilation** (VA) – the volume of gas in one minute that (1) reaches the alveoli AND (2) participates in gas exchange.

- *Difficult to measure.*
“Norm, this ‘dead space’ talk is confusing, give me an example”.

Okay, here we go...

Two patients have a minute ventilation of 10 l/min:

    Patient A is taking 50 breaths of 200ml tidal volume.
    Patient B is taking 20 breaths of 500ml tidal volume.

Remember that anatomic dead space is approximately 150ml in an adult.

The dead space to tidal volume ratio (VD/Vt) for each patient:

    Patient A VD/Vt = 75%
    (meaning 75% of minute ventilation is wasted).
    Patient B VD/Vt = 30% (normal = 30%).

Patient A will suffer muscle fatigue and become acidemic (hypercarbia).
Factors affecting External Respiration: Shunt

• **Shunt** – Blood passing through the lungs and not participating in gas exchange:
  
  • **Anatomic** – blood bypasses pulmonary capillaries and returns to heart through some other anatomic channel:
    • Bronchial circulation.
    • ASD/VSD.
  
  • **Physiologic** – blood passing normally through pulmonary circulation but not coming into contact with alveolar gas (absent in healthy individuals):
    • Pulmonary Edema.
    • ARDS.
    • Pneumonia.
    • Lobar Atelectasis.
Shunt vs. Deadspace
Diffusion: The Last Link in External Respiration

- Two Requirements:
  - Sufficient time for complete equilibration of gases.
  - Sufficient number of AC units (surface area) to participate in gas exchange.
Alveolocapillary (AC) Membrane

- Normal thickness: 1 micron (1/1000 mm).
- Comprised of:
  - Alveolar membrane
  - Interstitial fluid
  - Capillary membrane
Pulmonary capillary Transit Time

• Time available for complete gas equilibration at the alveolocapillary membrane:
  • Normal: 1 second (at rest).
    • Gas Equilibration: 0.25 seconds (at rest).
    • 3/4 of total transit time acts as a reserve.
Answer The Following:

1. Hypoxemia is a ______ condition.
   a. Blood
   b. Tissue

2. Most pulmonary perfusion occurs in zone _____.
   a. One
   b. Two
   c. Three

3. Most gas inhaled during normal tidal breathing enters the lung ______.
   a. Apices
   b. Bases
Oxygen Delivery
(O2 Transport)
Oxygen Delivery (Transport)

• O2 carried in two forms:
  • Dissolved in plasma (PO2) – PO2 provides the driving pressure for oxygen diffusion into the cells and combining with hemoglobin.
  • Combined with Hemoglobin (Oxyhemoglobin) – each hemoglobin molecule is capable of combining with four oxygen molecules.
Dissolved Oxygen Transport

- Normal = 15 ml O2/min
- Assuming one had no hemoglobin and could carry O2 only in the dissolved state, this would fall far short of normal average O2 consumption of 250 ml/min.
- Calculated:
  \[ \text{Calculated} = (\text{Cardiac Output}) \times (\text{Volume of Dissolved O2}) \]
  \[ = (5000 \text{ ml blood/min}) \times (0.3 \text{ ml O2/100 ml blood}) \]
Imagine... for a second...
If we had to rely solely on dissolved O2 to meet basic metabolic needs...

- Cardiac output would need to be $= 83 \text{ l/min}$ at rest and $166 \text{ l/min}$ with exercise!!!!
- PO2 would need to be $2000 \text{ mmHg}$ at rest!!!!
Combined Oxygen Transport

- Each molecule of Hb can carry four molecules of oxygen.
- Oxyhemoglobin affinity increases with each molecule added (opposite effect occurs with unloading).
Combined Oxygen Transport

• Oxygen bound to hemoglobin.
• Normal = 985 ml O2/min.
• Calculated in 2 steps:
  1. (Hb) x SaO2 x 1.34 ml O2/g HbO2 = volume of combined O2
  2. (Cardiac Output) x (volume of combined O2) = Combined O2 Transport
     = (5000 ml blood/min) x (19.7 ml O2/100 ml blood) = 985 ml O2/min
Total Oxygen Transport

• Dissolved + Combined = Total
  • Dissolved = 0.3 ml O2/100 ml blood (plasma).
  • Combined = 19.7 ml O2/100 ml blood (bound to Hb).
  \[= (\text{Cardiac Output}) \times (20 \text{ ml O2/100 ml blood})\]
  \[= (5000 \text{ ml blood}) \times (20 \text{ ml O2/100 ml blood}) = 1000 \text{ ml O2/min}.\]

*Remember normal O2 consumption at rest is 250ml/min so there is quite a reserve available for times of increased O2 demands.
Answer The Following:

1. The two forms of oxygen transport are:
   a. Loading and unloading.
   b. Dissolved and combined.
Internal Respiration
(O2 Unloading)
Cellular Oxygen Supply

- All arteries carry virtually identical concentrations of oxygen.
- Not all cells are supplied with equal amounts of oxygen:
  - A result of some cells not being exposed to the same amount of blood.
  - Cells furthest away from capillaries are most susceptible to hypoxia.
- Arterioles control blood supply to capillary networks:
  - Local influence – increased metabolism.
  - Central influence – epinephrine release.
Cellular Oxygen Utilization

• On average, approximately 5 ml O2 is taken up and used by the tissues for every 100 ml perfusion.
• Cellular utilization varies by organ system.
• Some tissues are able to extract more O2 when additional oxygen is needed:
  • Skeletal muscle at peak exercise may extract all of blood O2 supply.
  • Cardiac muscle has high resting extraction, but cannot increase extraction further.
Mixed Venous SaO2
Biochemical Respiration

(What goes on in the cell while we’re not looking)
(“The good kind”)

• Simplified, pyruvic acid is oxidized within the mitochondria of the cell producing ATP.*
  • Oxygen availability is crucial to ATP production!
  • We know this process as oxidative phosphorylation.

*Krebs cycle.
Biochemical Respiration
(The “bad kind”)

• In the absence of oxygen, a small amount of ATP is generated by metabolizing glucose:
  • We know this process as anaerobic glycolysis.
  • Results in production of lactic acid.
Okay, let’s get a little more clinical.
PO2
Partial Pressure of Oxygen (mmHg)

• PO2 is the measurement of the partial pressure of oxygen in the plasma (only a piece of the oxygenation puzzle, and can be basically thought of as “available” oxygen).

 idade dependent decline in baseline PO2.
  Mean PO2 95 ± 2 mmHg at age 20.
  Mean PO2 73 ± 5 mmHg at age 75.
  Mean PO2 73 at age >75.
SaO2
Arterial Oxygen Saturation (%)

- SaO2, or O2 saturation, is the measurement of the percentage of oxygen bound to (being carried by) the red blood cell’s hemoglobin molecule:
  - Hb is either saturated with four O2 molecules or desaturated (carrying no O2):
    - HHb
  - In normal situations, a PO2 of 60mmHg will yield a SaO2 of 90%.
  - Another helpful rule of thumb (PO2/SaO2):
    - 40, 50, 60mmHg = 75, 80, 90%.
PO2

PO2

PO2

PO2

PO2

SaO2

SaO2

SaO2

SaO2

Serves Red Cell

PO2

SaO2

SaO2

SaO2

Red cell releases O2 at tissues
Oxyhemoglobin Dissociation Curve

• Several factors influence how much hemoglobin is saturated with oxygen.
• PO2 is the major influence.
• This PO2/SaO2 relationship is direct but not linear:
  • At low PO2 values, small changes in PO2 have large changes in SaO2.
• The dissociation curve will help you evaluate tissue (end organ) oxygenation.
Oxyhemoglobin Dissociation Curve

A. P50 = 26 mmHg

B. Mixed Venous PO2 = 40 mmHg

C. PO2 60 mmHg = SaO2 90%
The “Two Straight Lines” of the O2 Dissociation Curve

1. The steep lower portion (dissociation “unloading” portion).
2. The flat upper portion (association “loading” portion)
We Need To Think Differently...

• Along with thinking that we must always “improve” patients’ SaO2; we should also be assuring that O2 is unloading properly.

• Think from the top of the curve downward or “loading” to “unloading”.
Other Factors Affecting O2 Dissociation

• Body Temperature:
  • Hypothermia.
  • Hyperthermia (fever).

• Blood pH:
  • Alkalosis.
  • Acidosis.

• PCO2:
  • Hypercapnea.
  • Hypocapnea.

• 2,3 Diphosphoglycerate
**Left shift**
- Decreased temp
- Decreased 2-3 DPG
- Decreased [H+]
- CO

**Right shift** (reduced affinity)
- Increased temp
- Increased 2-3 DPG
- Increased [H+]
What Should I See in My Patient?

<table>
<thead>
<tr>
<th>Condition</th>
<th>Expectation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever/Hyperthermia</td>
<td>Decreased SaO2/SpO2</td>
</tr>
<tr>
<td>Alkalemia</td>
<td>Increased SaO2/SpO2</td>
</tr>
<tr>
<td>Acidemia</td>
<td>Decreased SaO2/SpO2</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Increased SaO2/SpO2</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Decreased SaO2/SpO2</td>
</tr>
</tbody>
</table>
FiO2

• FiO2 – fraction of inspired oxygen:
  • Concentration of oxygen being administered to the patient:
    • Expressed as a decimal:
      • i.e. 0.5 = 50% concentration.
Answer the following:

1. All cells receive equal amounts of oxygen.
   a. True.
   b. False.

2. Most cellular energy is produced through:
   a. Glycolysis.
   b. The Krebs cycle.

3. Actual utilization of oxygen occurs in the ______ of the cells.
   a. Nucleus.
   b. Mitochondria.
   c. Golgi Bodies.

4. The relationship between PaO2 and SaO2 is:
   a. Direct and linear.
   b. Indirect and linear.
   c. Direct and nonlinear.

5. In a human with a normal PO2, increased Hb-O2 affinity usually has a net ______ effect.
   a. Beneficial.
   b. Detrimental.

6. A right-shifted O2Hb dissociation curve enhances oxygen:
   a. Loading.
   b. Unloading.
Oxygen Therapy

Plan A

"This is our most portable tank... unfortunately it only lasts 3 minutes."
Oxygen Therapy

• Remember, oxygen is a drug requiring a prescription (order).
• Oxygen Therapy carries risks and benefits as with any other drug:
  • Oxidizer.
  • Often not weaned or misused.
Goals Of Oxygen Therapy

• Correct Hypoxemia.
• Decrease Work Of Breathing (WOB).
• Decrease myocardial work.
Oxygen Delivery

Reduced Oxygen Delivery

- Decreased environmental oxygen level.
- Impaired diffusion at alveolo-capillary membrane.
- Reduced Cardiac Output.
- Impaired diffusion at blood-tissue interface.
Oxygen Therapy Devices

1. Nasal Cannula
2. Simple Mask
3. Venturi Mask
4. Aerosol Mask
5. Nonrebreather
6. High Flow Mask
7. Mask CPAP
8. Ventilator
How To Choose A Device

- How sick is the patient?
- Acute or Chronic illness?
- How long is therapy needed?
- How cooperative is the patient?
“Low Flow” vs. “High Flow” Devices

- Low Flow (variable performance) – Does not meet the patients peak inspiratory flow demands:
  - Requires the patient to entrain air within or around the device to produce delivered FIO2. FIO2 values are theoretical and can vary with changes in RR and Vt.

- High Flow (fixed performance) – Meets patient’s peak inspiratory flow demands for FIO2 delivery.
Low Flow (variable performance) Device Explained

Atmospheric room air oxygen concentration of 21%

100% O2 source mixes with 21% room air to create delivered concentration

100% O2 source runs through device
High Flow (fixed performance) Device Explained

Set oxygen concentration on device is delivered at the airway consistently without fluctuation.

21% room air oxygen is entrained into the device by the O2 flow.

100% O2 source flows into device at a set flow rate.
Nasal Cannula

- Low Flow (Variable Performance) Device
- Functional liter flow range: 1 – 6 LPM.
- Theoretical FIO2 range: 24% - 44%.
- Can be used in the acute or chronic setting.
- Should be humidified at ≥4 LPM.
Simple Mask

- Low Flow (Variable Performance) Device.
- Functional liter flow: 6 – 12 LPM.
- Theoretical FIO2 delivery: 40 – 60%.
- NEVER run at less than 6 LPM.
- Best used for short term O2 therapy (i.e. Post Anesthesia).
Venturi Mask

- High Flow (Fixed Performance) Device.
- Provides exacting FIO2 delivery range of 24 - 50%.
- Best used in acute setting.
- Can readily assess the patient’s FIO2 requirements and titrate easily.
Aerosol Mask

- High Flow (Fixed Performance) Device.
- Functions on same premise as Venturi Mask.
- FiO2 range: 28 – 100%.
- Should use dual system at FiO2 >50%.
- Can be used in acute or chronic setting.
Non-Rebreather

- Low Flow (Variable Performance) Device.
- Functional liter flow: 10 – 30 LPM.
- Theoretical FiO2 delivery: 100% as long as reservoir bag remains inflated during patient’s inspiratory phase.
- Used in acute setting.
High Flow Mask

- High Flow (Fixed Performance) Device.
- Provides exacting FiO2 range from 21–100% at flow rates > 30 LPM.
- Used in acute setting.
- Can be cool or warm humidified.
Mask CPAP/Ventilator

- Ultimate High Flow (Fixed Performance) Devices.
- Used in acute setting.
Hypoxic Drive
Hypoxic Drive

- Triggered by stimulation of the peripheral chemoreceptors on the aortic arch and bifurcations of the internal and external carotid arteries.
- **Functional in ALL of us.**
- Highly sensitive to a decreased “oxygen supply”:  
  - Low PO2  
  - Decreased blood flow  
  - Decreased hemoglobin  
  - Markedly increased PCO2  
  - Increased H+ concentration  
  - Methemoglobin
Hypoxic Pulmonary Vasoconstriction (HPV)

- **Hypoxic Pulmonary Vasoconstriction is a physiologic protective mechanism which prevents right to left shunting of blood, enhancing the efficiency of pulmonary gas exchange in two ways:**
  1. Reduction of venous admixture (physiologic shunt) by redirection of blood flow from poorly ventilated to better ventilated compartments.
  2. Reduction of alveolar deadspace.

- **Compensatory mechanism in COPD.**
Hypoxic Pulmonary Vasoconstriction (HPVC)
Can Severe Hypoxemia be Survived?

<table>
<thead>
<tr>
<th>Site</th>
<th>Altitude</th>
<th>Pb</th>
<th>PiO2</th>
<th>pH</th>
<th>PCO2</th>
<th>PO2</th>
<th>SaO2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Balcony</td>
<td>27,559 ft</td>
<td>253 mmHg</td>
<td>43 mmHg</td>
<td>7.53</td>
<td>13.3 mmHg</td>
<td>24.6 mmHg</td>
<td>54%</td>
</tr>
<tr>
<td>Everest</td>
<td>27,559 ft</td>
<td>253 mmHg</td>
<td>43 mmHg</td>
<td>7.53</td>
<td>13.3 mmHg</td>
<td>24.6 mmHg</td>
<td>54%</td>
</tr>
<tr>
<td>Sea Level</td>
<td>0 ft</td>
<td>760 mmHg</td>
<td>140 mmHg</td>
<td>7.40</td>
<td>40 mmHg</td>
<td>&gt;80 mmHg</td>
<td>98%</td>
</tr>
</tbody>
</table>

Clinical "Acclimatization" to Hypoxemia and Cellular Hypoxia

• “Oxygen Conformance – Reduction in VO2 by down-regulation of “non-essential” cellular processes:
  • Reversible on re-exposure to normoxia.
  • Not associated with long term cellular harm.
  • Similar mechanisms have been demonstrated in critically ill patients during multi-organ failure.

Effects of Excessive Oxygen and Hyperoxemia

- Considerable at FiO2 > 0.5 (50%).
- Damage similar to ARDS.
- Exacerbation of existing lung injury in ARDS.
- Several cardiovascular responses to “supranormal” arterial oxygenation:
  - Reduced stroke volume and CO.
  - Increased PVR.
  - Coronary artery vasoconstriction.
  - Reduced coronary blood flow.

And Now...Some Controversial Stuff

Giving that COPD patient lots of oxygen is cool...

No its not, you'll knock out his hypoxic drive...heh...heh!
Are We “Oxy-Morons”?

They forgot one…

“No more than 2 l/min for COPD patients”.
Are *all* COPD patients with CO2 retention liable to retain more CO2 while receiving O2 Therapy during an exacerbation?

✓ Yes.
✓ This situation unnecessarily alarms the healthcare team.
✓ This phenomenon is believed to be a result of the effect of oxygen therapy on HPVC, and the resultant increase in dead-space ventilation that occurs.
✓ Treatment of concomitant illness (pneumonia) and exacerbations with bronchodilators may counteract compensatory HPVC.
Do *all* COPD patients with CO2 retention breathe in response to a *hypoxic drive*?

✓ NO!
✓ The degree or type of COPD may be enough to cause CO2 retention but not severe hypoxemia.
✓ Remember that while PCO2 is considered the primary stimulus to breathe, it is actually the resultant increase in H+ ions and decreased pH of the CSF.
✓ It is only a small percentage of COPD patients (~2%) that have severe enough disease to create levels of hypoxemia to trigger a “hypoxic” drive.
Do we limit O2 therapy to “2 l/min” for all COPD patients with CO2 retention?

✓ NO!
✓ As discussed in the previous slide, approximately 2% of COPD patients have a hypoxic stimulus to breathe.
✓ Application of ANY amount of oxygen in this patient can cause cessation of respiratory effort.
✓ As long as the patient has a normal respiratory rate and is arousable, it is unlikely that they are “hypoxic drivers”.
Bronchodilators & O2 Saturation

- Truth: Bronchodilation can actually temporarily DECREASE SpO2 by creating “dead space” units post-bronchodilation. OXYGEN remains the only treatment for hypoxemia or decreased SpO2.
- Truth: Physiology is such that pulmonary blood flow is shunted to functional air spaces in clinical illness (Pneumonia i.e.). Reversing this process by creating more dead space can be detrimental to the patient.
Monitoring Oxygen Therapy
Monitoring Oxygen Therapy

• ABGs:
  • Costly.
  • Invasive.
  • Not covered in this presentation.

• Pulse Oximetry:
  • Noninvasive.
  • Reliable.*
  • Inherent limitations.

* Reliable only when properly used and understood.
Pulse Oximetry

• Pulse oximetry allows us to measure ONE aspect of oxygenation, that being SpO2, or pulse oximetry saturation.

• Pulse oximetry has **SEVERAL limitations** and the saturation reading can be **both** falsely alarming or reassuring.

• Pulse oximeter measures saturated vs. unsaturated hemoglobin:
  • Does not differentiate O2Hb from COHb.
  • DO NOT use on a suspected CO poisoning.

• It is ESSENTIAL and IMPERATIVE to relate an SpO2 reading with PATIENT assessment.
“Off hand, I'd say you're suffering from an arrow through your head, but just to play it safe, I'm ordering a bunch of tests.”
Pulse Oximetry

• Valuable time can be wasted on “getting a sat”.
• Needs to be correlated to patient’s clinical condition:
  • Example: Lower SpO2 is normal in state of acidemia.
Pulse Oximetry – Principles of Operation

- Constant “light absorbers” at measuring site:
  - Skin
  - Tissue
  - Venous blood
  - Non-pulsatile arterial blood

- To work out the color of the arterial blood, a pulse oximeter looks for the slight change in the overall color caused by a beat of the heart pushing arterial blood into the sensor site.

- Every patient will display some reading on the pulse oximeter!
  - The practitioner at the bedside needs to assure that the reading is valid.
Pulse Oximetry - Limitations

- **Major Limitation:**
  - Reduced Perfusion:
    - Hypovolemia.
  - Vasoconstriction:
    - Spontaneous.
    - Drug Induced.
  - Can be overcome with varying technologies.
- Motion Artifact – impossible to detect without a plethysmograph.
- COHb, MetHb.
- Contaminants - Methylene Blue (treatment for methemoglobinemia), Indocyanine Green (used to measure CO, hepatic and ophthalmic angiography), Indigo Carmine (used to localize ureteral orifices during cystoscopy).
- Can’t tell if the O2 dissociation curve is altered.
Dangers of the Misuse of Pulse Ox Probes

Probes and sensors are marketed for the use of specific principles:

- **Transmission** – sending signal THROUGH a site.
- **Reflectance** – bouncing signal OFF a site.

- Using a probe on a site not intended for that probe:
  - Results in erroneous and inaccurate results.
The Old “Finger Probe on the Forehead” Trick...

👉“I can’t get a sat on his finger”.
😊“Just put it on his forehead”.
😊“Look, his sat is 100%”.
😊“I don’t know, he doesn’t look so good”.


The Problem...

• Often in critical illness or decompensation, pulse oximeters fail to give us a reading. This leads to:
  • “Creative” or “Off-label use” of probes:
    • Dangerous.
    • Misleading:
      • Incorrect results may lead to more invasive, expensive, and unnecessary tests.
      • Incorrect results may lead to delay in obtaining necessary tests.
  • High probability for clinical error.
The Solution...

• Appropriate sensor application.
• Being satisfied with the possibility that a SpO2 reading may not be obtainable until restoration of proper physiology is achieved.
• Purchasing technology (despite expense) that allows monitoring of alternative sites:
  • Forehead, ear probes.
  • Ability to read through motion artifact and in low perfusion states.
Pulse oximetry remains accurate in these conditions.
Remember, pulse oximetry is a measure of % saturation and **NOT** O2 content.
Oximetry not a useful tool in these conditions.
Anemia

Red Blood Cells

<table>
<thead>
<tr>
<th>Patient One</th>
<th>Patient Two</th>
</tr>
</thead>
<tbody>
<tr>
<td>SpO2 = 100%</td>
<td>SpO2 = 100%</td>
</tr>
</tbody>
</table>
Sickle Cell Anemia
What’s The Best Way To Check A Patient’s Pulse Oximetry Saturation?

• Place the oximeter probe on the site appropriate for that probe.

• Leave the probe in place for several seconds to allow the oximeter to “look” for a pulsatile signal and a strong signal:
  • Oximeters use a combination of LEDs, sound, and waveforms to let you know if the signal is adequate to produce a “believable” result.

• Assure that the pulse rate reading on the oximeter matches the patient’s pulse rate.

• If the signal is inadequate or the pulse rates do not correlate, the saturation is likely not accurate and should not be reported.
SpO2 Value or Clinical Assessment?

Pulse oximeter reading 97%...Hmmmm.
SpO2 Value or Clinical Assessment?

Pulse Oximeter reading 78%...Hmmmm.
What Do I Do When My Patient’s Hypoxemia Becomes Refractory?

- Refractory Hypoxemia: That which becomes unresponsive to maximum conventional oxygen therapy.
- Cardiogenic or Non-cardiogenic?
- What saturation is a “bad” saturation?
- Hypoxemic respiratory failure:
  - How do I know?
  - What therapies to employ?
  - Calling an expert.
What Doesn’t Work...and What Does.

• Doesn’t: Waiting...waiting...and more waiting.
• Does:
  • Recognition of ALI.
  • Aggressive investigation of WHY your patient’s hypoxemia is refractory.
  • Call in an expert.
  • Transfer to an institution for more aggressive investigation.
Hypoxemic Respiratory Failure (Type I Failure)

• Failure to maintain a PO2 >60mmHg on 100% FIO2.
• PO2 < 50mmHg in room air.
• Usually a result of:
  • Acute Pulmonary Edema
  • Acute Lung Injury*/ARDS

*All acute lung injury is now considered ARDS of varying degrees.
V/Q Mismatch vs. Shunt

• V/Q Mismatch:
  • Anesthesia.
  • Pulmonary Embolism.
  • Responds to increased FiO2 (O2 Therapy).

• Shunt:
  • Pneumonia, Atelectasis, Pulmonary Edema.
  • Refractory to O2 Therapy.
Common Causes of Hypoxemic Respiratory Failure

• Acute Lung Injury/Acute Respiratory Distress Syndrome (ARDS)
  • Direct (Intra-alveolar)
  • Indirect (Intra-vascular)
• Chronic bronchitis and emphysema
• Pneumonia
• Pulmonary edema
• Pulmonary fibrosis
• Asthma
• Pneumothorax
Common Causes of Hypoxemic Respiratory Failure

- Pulmonary embolism
- Thromboembolic pulmonary hypertension
- Lymphatic carcinomatosis
- Pneumoconiosis
- Granulomatous lung disease
- Cyanotic congenital heart disease
- Fat embolism
- Pulmonary arteriovenous fistulae
Acute Respiratory Distress Syndrome (ARDS)

• Defining Criteria:
  • Acute Onset (over one week*).
  • Bilateral pulmonary infiltrates on CXR.
  • Absence of evidence of cardiac failure or fluid overload.
  • Mild* – PaO2/FiO2 $\leq 300$ with PEEP $\geq 5$cmH2O
  • Moderate* – PaO2/FiO2 $< 200$ with PEEP $\geq 5$cmH2O
  • Severe* – PaO2/FiO2 $< 100$ with PEEP $\geq 5$cmH2O

* The Berlin Definition
Causes Of ARDS

• Direct (Lung Injury):
  • Common:
    • Pneumonia.
    • Aspiration of gastric contents.
  • Less Common:
    • Inhalation injury.
    • Pulmonary Contusion.
    • Fat Embolism.
    • Near Drowning.
    • Reperfusion injury.
Causes Of ARDS

• **Indirect (Lung Injury):**
  • **Common:**
    • Sepsis.
    • Severe trauma with prolonged hypotension and/or multiple fractures.
    • Multiple blood transfusions.
  • **Less Common:**
    • Acute pancreatitis.
    • Cardiopulmonary bypass.
    • Drug overdose.
    • Burns.
    • Head Injury.
Beware of the Hypoxemic Patient with a “Normal” ABG

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<tr>
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Treatment Strategies for Hypoxemic Respiratory Failure

• Despite the type of failure, goals should include:
  • **Rest**
    • Maximizing oxygenation.
    • Minimizing damage to airways and lung parenchyma (Lung Protective Strategy).

• Strategies should be:
  • Etiology-specific.
  • Patient-specific.
  • Evaluated on an ongoing basis and adjusted according to patient response.
PLAN B

[Diagram of a patient receiving mechanical ventilation with settings for inspiratory pressure and PEEP (Positive End-Expiratory Pressure).]
Non-Invasive Ventilation (NIPPV, NIV)

- Commonly known as Bi-PAP.
- **Must employ early to be effective.**
- **Not entirely effective for “hypoxemic” failure.**
- Requires patient compliance.
- Clinicians must recognize patients whom are “beyond” Bi-PAP.
- Highest success rate in:
  - Cardiogenic Pulmonary Edema.
  - COPD Exacerbation.
Non-invasive Ventilation

• Non-invasive ventilation is that which is provided without an artificial airway (endotracheal tube).

• Most Common Forms of Noninvasive Ventilation:
  • CPAP
  • BiPAP
CPAP, BiPAP, What’s The Difference?

- The application of a **Continuous Positive Airway Pressure (CPAP)** to the airways has historically been shown to enhance diffusion of O2 at the alveolar level and to “recruit” collapsed alveoli in conditions such as RDS and Apnea of Prematurity in the neonate, and atelectasis in adults.
CPAP, BiPAP, What’s The Difference?

• As stated previously, later research showed that CPAP was very useful as an “airway stent” in patients with Obstructive Sleep Apnea. CPAP is now almost exclusively used in this patient population.
CPAP, BiPAP, What’s The Difference?

• BiPAP is Positive Airway Pressure (“PAP”), utilized on both the inspiratory and expiratory (“Bi”-phasic) phases of a spontaneous breath.

• Research has shown BiPAP to be more effective as an acute treatment of respiratory distress than CPAP alone.

• Two settings are required:
  1) IPAP – used to augment the patient’s tidal volume and aide CO2 removal (ventilation).
  2) EPAP – used to distend the distal airways and alveoli to enhance diffusion of O2 (oxygenation).
The BiPAP Breath Cycle

On inspiration, a preset pressure (IPAP) will be delivered to the patient’s airways. At the termination of the inspiratory phase, the patient is allowed to exhale to a set baseline pressure (EPAP, CPAP, or PEEP).
Who Can Benefit?

Patients with the following conditions may benefit from BIPAP:

- Hypoxemic and Mixed Respiratory Failure
  - Pulmonary Edema (hemodynamically stable).
  - Post-operative Respiratory Failure.
  - Post-traumatic Respiratory Failure.
  - Pneumonia.
  - Patients who are not candidates for intubation (DNR or terminal illness with REVERSIBLE cause of acute failure).
What Should I Use For Settings?

• There are no magic settings to start with.
• In general, a conventional level of PEEP/CPAP/EPAP and a level of IPAP to provide a pressure support level of 5 – 12 cmH2O is a good place to start.
• Remember, this is a form of positive pressure ventilation and carries with it all the risks, hemodynamic compromise and barotrauma.
What Should I Use For Settings?

• For the acute ventilatory failure patient, generally settings can be titrated upward at the bedside to a level tolerated by the patient. Blood gases should be done after 30 – 60 minutes of therapy to assess a positive or negative trend.

• Frequent blood gases are usually not necessary once the patient has shown improvement.

• Pulse oximetry is not valuable in assessing ventilation.
What Should I Use For Settings?

• For the patient with refractory hypoxemia, use as much PEEP/EPAP that the patient can hemodynamically and physically tolerate.
• Add an IPAP setting that allows the patient to “feel” the transition in the inspiratory and expiratory cycles. FIO2 of 1.0 is a good starting point.
• Obtain blood gases after 30 – 60 minutes of therapy to assess for positive or negative trend.
• Careful monitoring with pulse oximetry can be used to titrate FiO2.
When maximum conventional O2 therapy and non-invasive ventilation aren’t effective...

It's time to...

And go to...

Plan A
Plan B
Plan C
Overview of Mechanical Ventilation

Mechanical ventilation carries with it a number of inherent risks and side effects, including **renal impairment**, **fluid retention**, **decreased cardiac output**, **barotrauma/volutrauma/biotrauma**, and **ventilator associated pneumonia (VAP)**. While some of these risks occur simply as a result of instituting mechanical ventilation, judicious use of ventilator modes and various settings can dramatically reduce others.
The Ventilator...Friend or Foe?
YOU’RE SCREWED!

GOOD LUCK!
Effect of Positive Pressure on the Cardiovascular System

- Rise in pleural (intra-thoracic) pressure
- Rise in intra-abdominal pressure
- Increased lung volumes (Vt, PEEP, Ti)

All can greatly affect venous return (preload) to the heart and result in decreased cardiac output.
Effect of Positive Pressure on the Cardiovascular System

**Normovolemia and adequate fluid resuscitation:**
- Venous return not (as) compromised
- Intra-thoracic and abdominal pressures match (with exception of patient with open abdomen)
Effect of Positive Pressure on the Cardiovascular System

Hypovolemia and shock:

- Collapse of intra-abdominal veins and SVC
- Results in decreased venous return, RV stroke volume and LV preload
Delivery of a Mechanical Ventilator Breath

• The ventilator is an idiot:
  • Needs to be told HOW and WHEN to deliver the breath:
    • How quickly or slowly.
    • How many times/minute.
  • Needs to be told WHEN to STOP delivering the breath:
    • When to “shut off” the inspiratory cycle and allow the patient to exhale.
Our Job...

• To somehow, without too much harm, marry a machine to a human body.
Ventilation Constants
( Two choices for cycling a breath)

Volume
Setting: Tidal Volume
Volume Cycled
Volume Control

Pressure
Setting: Inspiratory Pressure
Pressure Cycled
Pressure Control
Volume Cycled Ventilation

• A preset Tidal Volume (VT) is the cycling constant. The ventilator will terminate the inspiratory cycle when the set tidal volume has been delivered.

• Airway and Chest Wall Resistance, and Lung Compliance will generate a resultant peak inspiratory pressure (PIP, Paw) as the tidal volume is delivered. **Peak inspiratory pressure will be variable breath to breath.**
Pressure Cycled Ventilation

- A preset Inspiratory Pressure is the cycling constant. The ventilator will terminate the inspiratory cycle when the set pressure is delivered.

- Airway and Chest Wall Resistance, and Lung Compliance will generate a resultant tidal volume as the breath is delivered. **Tidal volume delivery will be variable breath to breath.**
Effect of Decreased Lung and Chest Wall Compliance and Increased Airway Resistance

• In Volume Control:
  ◆ Increased Peak Inspiratory Pressure (PIP).
  ◆ Increased risk of barotrauma (pneumothorax, pneumomediastinum, pulmonary interstitial emphysema).
  ◆ Shearing Injury (alveolar).
    ◆ Volutrauma.

• In Pressure Control:
  ☢ Decreased Tidal Volume delivery.
  ☢ Risk of under ventilation.
How Do I Decide Whether To Use Volume vs. Pressure Ventilation?

**Volume Control**
- “Conventional” (first) choice for ventilation.
- Used in states of relatively compliant lungs and chest wall.
- Used in setting of little risk for large and frequent changes in airway resistance and pulmonary compliance.*

**Pressure Control**
- Optimum choice in settings of poor lung compliance.
- Can lessen the risk of barotrauma in setting of low compliance and/or high airway resistance.

*Newer features such as VC+ and PRVC allow us to use volume control more frequently.*
Conditions of Low Airway Resistance and High Pulmonary Compliance

• Primary Pulmonary Emphysema.
• Those patients with “normal lungs”, ventilated for non-pulmonary disease reasons:
  • Post-operative recovery.
  • Uncomplicated overdose.
  • Over-sedation.
  • Spinal Injury/Neuromuscular disease.
Conditions of High Airway Resistance and Low Pulmonary Compliance

- ARDS
- Pulmonary Edema
- Asthma
- Chronic Bronchitis
- Pneumonia

- Atelectasis
- Pneumothorax
- Bronchogenic Ca
- Pneumoconioses
- Interstitial Lung Disease
Ventilator Induced Lung Injury (VILI)

• Mechanical ventilation induces an inflammatory response (biotrauma).
• This response creates the potential for secondary VILI.
• There are four primary mechanisms in which this injury may occur:
  • Barotrauma.
  • Volutrauma.
  • Atelectrauma.
  • Biotrauma.
Barotrauma

- Lung over-distension caused by excessive peak inspiratory pressures:
  - Can result in:
    - Air leak syndromes.
    - Pneumothorax.
Barotrauma
Volutrauma

• Pulmonary capillary membrane damage caused by over stretching of lung units at the end of inspiration:
  • Results from excessive tidal volume delivery.
  • May result in epithelial and microvascular injury with resultant pulmonary edema.
  • May cause air leak syndromes and pneumothorax.
Atelectrauma

• Shearing injury caused by the repeated opening and closing of under-recruited alveoli:
  • Usually results from inadequate PEEP setting.
Volutrauma, Atelectrauma, and Barotrauma
The Modes

Assist-Control

PRVC  CPAP  PSV

SIMV  VC +

Pressure Control
Ventilator Mode

- Think of the mode as the breathing “pattern”. The ventilator can provide 3 basic patterns:
  1. Ventilator doing ALL of the work = Assist Control (AC).
  2. Ventilator doing SOME of the work = Synchronized Intermittent Mandatory Ventilation with or without Pressure Support (SIMV).
  3. Ventilator doing NONE (I’m using this term loosely) of the work = Continuous Positive Airway Pressure with or without Pressure Support (CPAP).
The Modes

Choose a mode and settings that will meet your patient’s individual minute ventilation, flow, and breath cycle demands!!!!!
So Easy A Monkey Can Do It?
No, that’s why we need to understand it!
A Little History Lesson

Anesthesia Machine Circa 1958
Dr. O'Dwyer says, in commenting on the Fell apparatus: "In the performance of artificial respiration by any

The Fell-O'Dwyer apparatus. This illustration shows an early model; since then the bellows has been improved by the addition of a strong wooden frame which holds it steadily, and is provided with a long arm that acts as a powerful foot-piece for compressing the machine, with the least amount of muscular effort. 
The Modes Through History

Control Mode

- Can utilize volume (VT) or pressure (IP) as the constant for breath delivery.
- Patient will receive the SET volume or pressure at the SET rate (f) ONLY!
- Patient CANNOT trigger additional breaths.
- Frequently associated with “air hunger” and the patient being “out of synch” with the ventilator.
- No longer utilized but the terminology still exists in the anesthesia arena.
Control Mode “Patterns”

CONTROL MODE (VOLUME-TARGETED VENTILATION)

- Time Triggered, Flow Limited, Volume Cycled Ventilation
- Preset $V_t$ Volume Cycling
- Pressure (cm H$_2$O) Dependent on $C_r$ & $R_{aw}$

CONTROL MODE (PRESSURE-TARGETED VENTILATION)

- Time Triggered, Pressure Limited, Time Cycled Ventilation
- Pressure (cm H$_2$O) Set PC Level
- Time Cycling

[Diagram showing pressure, flow, and volume over time for both control modes]
Dyssynchrony in Control mode

Mid-breath, the patient tries to inhale more gas than the ventilator is willing to deliver, generating a negative pressure and some negative flow.

The patient tries to initiate a breath, but the ventilator keeps the inspiratory valve closed, and no flow is generated.

The patient tries to exhale, generating a positive pressure, but the ventilator keeps the expiratory valve closed in a breath hold.
The Modes Through History

Assist – Control Mode

- Can utilize volume (VT) or pressure (IP) as the constant for breath delivery.

- Patient will receive the set VT or IP at the set rate and in addition, may “trigger” an additional breath, receiving the set VT or IP.

- Used in settings with high minute ventilation requirements.
Assist Control “Pattern”

Flow

Pressure

Volume
The Modes Through History

Intermittent Mandatory Ventilation (IMV Mode)

- Can utilize volume or pressure as the constant for breath delivery.

- Patient will receive the set VT or IP at the set rate, and in addition, may take spontaneous breaths (own varying VT) between set breaths.

- Ventilator can “stack” a machine breath on the patient’s spontaneous breath.

- No longer available on newer generation vents.
IMV Mode Pattern
The Modes Through History

Synchronized Intermittent Mandatory Ventilation
(SIMV Mode)

- Same premise as IMV.
- Unlike IMV, the ventilator constantly “watches” the patient’s spontaneous efforts, and WILL NOT “stack” a machine breath on a spontaneous breath...hence the “Synchronized” feature.
- Can utilize volume (VT) or pressure (IP) as the constant for breath delivery.
- Recent literature suggests that SIMV increases oxygen demand and may actually create increased WOB:
  - “…the patient’s inherent neurologic mechanisms may find this alternating pattern disturbing.”*
  - Hering-Breuer reflex.

SIMV “Pattern”
SIMV with Pressure Support
The Modes Through History

Continuous Positive Airway Pressure
(CPAP Mode)

- A completely spontaneous mode of ventilation.
- The patient breathes at their own rate and depth of respiration with a level of baseline PEEP.
- This mode was a precursor to Pressure Support Ventilation.
CPAP “Pattern” Without Pressure Support
The Modes Through History

Pressure Support Ventilation
(PSV Mode)

- A completely “spontaneous” mode of ventilation in which the patient determines their own respiratory pattern.
- Spontaneous breaths are augmented by the setting of a pressure that “kicks in” on the inspiratory portion of the patient’s effort to make their own tidal volumes more effective.
- PSV cannot be set in Control or Assist-Control modes.
- PSV can be set in the SIMV mode, functioning on spontaneous efforts only.
Pressure Support “Pattern”

PRESSURE SUPPORT VENTILATION (PSV)

Pressure (cm H₂O)

Flow (L/min)

Volume (mL)

Time (sec)

Set PS Level

Longer Inspiration

Better Efforts

Flow Cycling

Patient Triggered, Flow Cycled, Pressure limited Mode
Effect of Adding Pressure Support to CPAP Mode
The Modes Through History

Pressure Regulated Volume Control (PRVC) and Volume Control Plus (VC+)

- A Volume Control feature (AC or SIMV mode) in which the ventilator will deliver the set tidal volume while generating the lowest peak airway pressure possible.

- Accomplished by varying the flow rate actively during the inspiratory cycle in order to regulate peak airway pressure.

- Servo™ owns the term “PRVC”.

- Nellcor-Puritan Bennett™ owns the term “VC+”.

PRVC/VC+ “Pattern”

The graph shows the pressure, volume, and flow over time. Each peak represents a breath cycle, with pressure and volume indicating the respiratory pattern. The labels 20, 22, 24, and 27 likely represent specific points or settings in the pattern.
Mechanical Ventilation (Invasive)
Conventional Approach

- Tidal Volume (Vt) set at 6-8 ml/kg ideal body weight.
- Set ventilator rate to “normalize” PCO2.
- Adjust FiO2 to maintain PO2 in the 80 – 100mmHg range (SpO2 ≥ 95%).
Low Tidal Volume Strategy

- Reduces “alveolar stretch” injury induced by large tidal volumes:
  - Reduces alveolar and subsequent systemic inflammatory response.
- Lessens risk of “volutrauma”:
  - Alveolar over inflation exacerbates existing lung injury, causing microvascular injury and worsening pulmonary edema.
  - Microbarotrauma is a result of lung over inflation rather than high airway pressures.
- Set Vt at 4-6 ml/kg ideal body weight.
Permissive Hypercapnia

• A result of attempts to reduce volutrauma and stretch injury:
  • Goal of Low Tidal Volume strategy.
• Deliberately accepting hypoventilation in efforts to reduce alveolar over distension and high transalveolar pressures within compliant (non-collapsed) lung.
• Goal is to preserve oxygenation at this point.
Permissive Hypercapnia

• Tidal volume is gradually reduced to allow PCO2 to rise no more than 10 mmHg/hr, to a maximum of 80–100 mmHg.

• Goal is to keep peak airway pressure < 40 cmH2O and maintain SaO2 > 90% while accepting a pH as low as 7.15 (average of 7.20).
Open Lung Strategy

• Can prevent cyclic atelectasis caused by repeated opening and closing of alveoli at low levels of PEEP (atelectrauma).

• Evidence suggests that maximum airway recruitment and maintenance of FRC occurs when PEEP is set at the lower inflection point on the pressure-volume curve.
Open Lung Strategy
Open Lung Strategy

• Goals:
  • Increased recruitment of FRC.
  • Decreased intrapulmonary shunting.
  • Improved arterial oxygenation.

Open Lung Ventilation = Low Volume Strategy +
PEEP Set Above Lower Inflection Point +
Permissive Hypercapnea.
Alveolar Recruitment

- Forms of lung recruitment:
  1. Sustained inflation technique (most common):
     - “40 for 40” or PEEP 40cmH2O for 40 seconds.
     - “30 for 30”
  2. Incrementally increased PEEP with limited inspiratory pressure.
  3. Escalating PEEP with constant driving pressure.
  4. Prolonged lower pressure recruitment:
     - PEEP elevation up to 15 cmH2O and end inspiratory pauses for 7 sec twice per minute during 15 min.
  5. Intermittent sighs to reach a specific plateau pressure.
  6. Long slow increase in inspiratory pressure up to 40 cmH2O (RAMP).
ARDSnet Recommendations

- Avoid high inspiratory pressures.
- Use Low Tidal Volumes.
- Use levels of PEEP to encourage lung recruitment and avoid cyclic atelectasis.
- Plateau pressure of 25-30cmH2O.
Alternative Approaches

• **I:E Inverse Ratio Ventilation***
  • ARDS and Severe Hypoxemia
  • Prolonged inspiratory time (3:1) leads to better gas distribution with lower PIP
  • Improves alveolar recruitment
*No statistical advantage over PEEP and does not prevent repetitive collapse and reinflation

• **Prone Positioning***
  • Addresses dependent atelectasis
  • Improved recruitment and FRC; relief of diaphragmatic pressure from abdominal viscera; improved drainage of secretions
  • Logistically difficult
*No mortality benefit demonstrated

• **ECHMO**

• **High Frequency Oscillatory Ventilation (HFOV)**
  • High frequency, low amplitude ventilation superimposed over an elevated MAP
  • Avoids repetitive alveolar opening and closing
  • Avoids over distension that occurs at high airway pressures
  • Consistent improvements in oxygenation
  • Logistically difficult
  • **Unclear mortality benefits**
  • Requires neuromuscular paralysis

• **Airway Pressure Release Ventilation**
Inverse Ratio Ventilation

- An alternative strategy in which the inspiratory time is extended, and, in concept, maintains or improves gas exchange at lower levels of PEEP and peak distending pressures.

- There are two methods to administer IRV:
  - Volume-cycled ventilation with an end-inspiratory pause, or with a slow inspiratory flow rate.
  - Pressure-controlled ventilation applied with a long inspiratory time.

- Problems common to both forms of IRV:
  - Excessive gas-trapping.
  - Adverse hemodynamic effects.
  - Need for significant sedation in most patients.
Prone Ventilation

• 50-75% of patients with ARDS (and some other causes of acute respiratory failure) show an improvement in oxygenation when turned prone.

• Probable mechanism is that when patient is turned prone the ventilation to the dorsal atelectatic parts of the lung is improved. However perfusion continues to pass preferentially to these regions and hence shunt is reduced.

• Improvement in gas exchange often persists even patient is returned to supine position. This is probably because once the collapsed alveoli have been recruited in the prone position they can be kept open by PEEP.

• Reduction in thoraco-abdominal compliance thought to play an important part in producing beneficial effects of prone ventilation.

• Most common serious complication of turning prone is accidental extubation.
Closed Loop Ventilation

• Probably the ideal way to go if you can afford the technology.

• Pros:
  • Technology allows the ventilator to deliver ventilation in a pattern that is in response to the clinical dynamics of the patient’s compliance and airway resistance.
  • Senses “readiness” to wean and extubate based on mechanics of breathing.
  • Many different choices for modes.

• Cons:
  • Expensive.
  • Requires complete paradigm shift in clinical management.
Now...”The Big One”!
Barriers & Hurdles To Providing Effective Ventilation

• Lung protective strategies have proven to significantly improve outcomes.

• *Clinical practice frequently deviates from recommended practice.*

• Requires a paradigm shift in behavior of *ALL* relevant clinicians.
  • Reconsider many of the “conventional” RR, Vt, and blood gas goals of mechanical ventilation.
Barriers To Providing Effective Ventilation

• Top Concerns:
  • Patient discomfort and tachypnea.
  • Dyssynchrony.
  • Hypercapnia and acidosis.
Major Hurdles

• **Tachypnea and Patient – Ventilator Dyssynchrony:**
  • Tachypnea related to hypercapnia and acidosis associated with LTVV.
  • Dyssynchrony:
    • Trigger
    • Flow
    • Cycle
The Solutions

• Clinician education.
• Staff/provider accountability.
• **Tolerance** of acidemia, hypercapnea, inherent respiratory drive.
• **Sedation, sedation, sedation!**
Realities About Sedation

• The **MAIN** goal of sedation is to allow the ventilator to do its job!
  • If the chest wall is not made to be compliant, the ventilator has to overcome this resistance:
    • *Often at the expense of patient safety.*

• **Secondary** benefits:
  • Tolerance of endotracheal tube.
  • Necessary rest.
Realities About Sedation

• Nurses and providers have very variable comfort levels regarding sedation.

• Sedative combinations vary from patient to patient.

• Frequently, patients are under-sedated for fear of decreasing blood pressure:
  • Need to remember WHY we are ventilating the patient.
  • Inadequate sedation can have greater consequences than low blood pressure:
    • Need the right nurse, and right medication regimen to balance all needs for sedation and consequences.
MAKE HIM TAKE 15 OF THESE A DAY UNTIL WE FEEL BETTER ABOUT WHAT WE'RE DOING TO HIM!
• Three Phases of Severe ARDS Illness:
  • Initial (Early Exudative) Phase:
    • Lasts 1-7 days.
  • Sub-Acute (Fibroproliferative) Phase:
    • Lasts 7-14 days after onset.
  • Chronic (Fibrotic) Phase:
    • Starts ≈ 14 days after initial insult.

Need for deepest sedation.

Consider weaning sedatives.
Ventilator Effects On Blood Gases

- Dependent on ventilation strategy:
  - Conventional/Uncomplicated – target settings to achieve blood gas values within the limits of normal.
  - Lung Protective – Tolerance of hypercapneic acidosis and mild hypoxemia:
    - pH mean of 7.20.
    - PCO2 mean maximum of 67mmHg.
    - PO2 55-60mmHg or SpO2 88-90%.
**OXYGENATION GOAL:** \( \text{PaO}_2 \geq 55-80 \text{ mmHg or } \text{SpO}_2 \geq 88-95\% \)

Use incremental \( \text{FiO}_2 / \text{PEEP} \) combinations below to achieve goal. Higher PEEP options (lower row) will decrease \( \text{FiO}_2 \) and may be preferred in patients with high \( \text{FiO}_2 \) who can tolerate higher PEEP (stable blood pressure, no barotrauma). Survival is similar with both PEEP approaches.

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**PLATEAU PRESSURE GOAL:** \( \leq 30 \text{ cm H}_2\text{O} \)

Check \( \text{Pplat} \) (0.5 second inspiratory pause), \( \text{SpO}_2 \), Total RR, TV and pH (if available) at least q 4h and after each change in PEEP or TV.

- If \( \text{Pplat} > 30 \text{ cm H}_2\text{O} \): decrease TV by 1 ml/kg steps (minimum = 4 ml/kg).
- If \( \text{Pplat} < 25 \text{ cm H}_2\text{O} \): TV < 6 ml/kg, increase TV by 1 ml/kg until \( \text{Pplat} > 25 \text{ cm H}_2\text{O} \) or TV = 6 ml/kg.
- If \( \text{Pplat} < 30 \) and breath stacking occurs: may increase TV in 1 ml/kg increments (maximum = 8 ml/kg).

**pH GOAL:** 7.30-7.45

**Acidosis Management:** (pH < 7.30)
- If pH 7.15-7.30: Increase RR until pH > 7.30 or \( \text{PaCO}_2 \) < 25 (Maximum RR = 35).
- If RR = 35 and \( \text{PaCO}_2 \) < 25, may give \( \text{NaHCO}_3 \).
- If pH < 7.15: Increase RR to 35.
- If pH remains < 7.15 and \( \text{NaHCO}_3 \) considered or infused, TV may be increased in 1 ml/kg steps until pH > 7.15 (Pplat target may be exceeded).

**Alkalosis Management:** (pH > 7.45) Decrease vent rate if possible.

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**INCLUSION CRITERIA:** Acute onset of
- \( \text{PaO}_2 / \text{FiO}_2 \leq 300 \) (corrected for altitude)
- Bilateral (patchy, diffuse, or homogeneous) infiltrates consistent with pulmonary edema
- No clinical evidence of left atrial hypertension

**PART I: VENTILATOR SETUP AND ADJUSTMENT**

1. Calculate predicted body weight (PBW)
   - Males = 50 + 2.3 [height (inches) - 60]
   - Females = 45.5 + 2.3 [height (inches) - 60]
2. Select Assist Control Mode
3. Set initial TV to 8 ml/kg PBW
4. Reduce TV by 1 ml/kg at intervals \( \leq 2 \) hours until TV = 6 ml/kg PBW.
5. Set initial rate to approximate baseline VE (not > 35 bpm).
6. Adjust TV and RR to achieve pH and plateau pressure goals below.
7. Set inspiratory flow rate above patient demand (usually > 80L/min)
I:E RATIO GOAL: 1:1:0 - 1:3 Adjust flow rate to achieve goal. If \( \text{FiO}_2 = 1.0 \) and PEEP = 24 cm H\(_2\)O, may adjust I:E to 1:1.

PART II: WEANING

A. Conduct a CPAP Trial daily when:
   1. \( \text{FiO}_2 \leq 0.40 \) and PEEP \( \leq 8 \) or, if using the higher PEEP scale and \( \text{FiO}_2 \leq 0.3 \) and PEEP 12-14, slowly reduce PEEP to 8 and increase \( \text{FiO}_2 \) to .4 for 30 min.
   2. PEEP and \( \text{FiO}_2 \) values of previous day
   3. Patient has acceptable spontaneous breathing efforts. (May decrease vent rate by 50% for 5 minutes to detect effort.)
   4. Systolic BP \( \geq 90 \text{ mmHg} \) without vasopressor support.

CONDUCTING THE TRIAL:
Set CPAP = 5 cm H\(_2\)O, \( \text{FiO}_2 = 0.50 \)

If RR \( \leq 35 \) for 5 min., advance to Pressure Support Weaning below:
If RR \( > 35 \) in < 5 min., may repeat trial after appropriate intervention (e.g., suctioning, analgesia, anxiolysis)
If CPAP trial not tolerated: return to previous A/C settings

B. PRESSURE SUPPORT (PS) WEANING PROCEDURE
   1. Set PEEP = 5, and \( \text{FiO}_2 = 0.50 \)
   2. Set initial PS based on RR during CPAP trial:
      a. If CPAP RR < 25: set PS = 5 cm H\(_2\)O and go to step 3d.
      b. If CPAP RR = 25-35: set PS = 20 cm H\(_2\)O then reduce by 5 cm H\(_2\)O at \( \leq 5 \) min. intervals until RR = 26-35 then go to step 3a.
      c. If initial PS not tolerated: return to previous A/C settings.
   3. REDUCING PS: (No reductions made after 1700 hrs)
      a. Reduce PS by 5 cm H\(_2\)O q1-3 hr.

b. If PS \( \geq 10 \text{ cm H}_2\text{O} \) not tolerated, return to previous A/C settings (Reinitiate last tolerated PS level next AM and go to step 3a)
   c. If PS = 5 cm H\(_2\)O not tolerated, return to PS = 10 cm H\(_2\)O. If tolerated, 5 or 10 cm H\(_2\)O may be used overnight with further attempts at weaning the next morning
   d. If PS = 5 cm H\(_2\)O tolerated for \( \geq 2 \) hours assess for ability to sustain unsupported breathing below.

C. UNASSISTED BREATHING TRIAL:
   1. Place on T-piece, trach collar, or CPAP \( \leq 5 \text{ cm H}_2\text{O} \)
   2. Assess for tolerance as below for two hours.
      a. \( \text{SpO}_2 \geq 90\% \) and/or \( \text{PaO}_2 \geq 60 \text{ mmHg} \)
      b. Spontaneous TV \( \geq 4 \text{ ml/kg PBW} \)
      c. RR \( \leq 35/\text{min} \)
      d. pH \( \geq 7.3 \)
      e. No respiratory distress (distress \( \geq 2 \) or more)
         > HR \( > 120\% \) of baseline
         > Marked accessory muscle use
         > Abdominal paradox
         > Diaphoresis
         > Marked dyspnea
   3. If tolerated consider extubation.
   4. If not tolerated resume PS 5 cm H\(_2\)O.

COMPLETE PROTOCOL ONLINE: www.ardsnet.org or from National Auxiliary Pulplications Service (NAPS). To order 15 pages of supplementary material, contact NAPS, c/o Microfiche Publications, 248 Hempstead Tpk., West Hempstead, NY 11552 Document # 05542.

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Weaning From The Ventilator

• What is *weaning*?
• How do I know when my patient is ready to begin the weaning process?
• How is weaning performed?
Have you tried turning it off and on again?
Weaning from the Ventilator

- Weaning may be thought of as reducing the mechanical support and allowing the patient to participate in once again providing their own ventilatory needs.
- The majority of ventilator patients (80%) do not require a weaning process.
- Patients requiring “weaning” (20%) are those who cannot be removed from the ventilator within 12 hours of gradual reduction in support.
- Some basic requirements must be in place before this can occur.
Requirements for Weaning Consideration

- *Correction of the cause for the need of ventilation.*
- Patient’s ventilatory drive in tact.
- Adequate ventilatory “mechanics”.
Ventilatory Mechanics (Weaning Parameters)

- NIF: > 25 cmH2O
- Vt: 5 ml/kg
- f/Vt (RSBI): < 100 (*90)
- VE: < 10 l/min
- VC: 10 – 15 ml/kg
- RR: ≤ 25/min (*30)

- Normal: > 90cmH2O
- Normal: 5 – 7ml/kg
- Normal: < 50
- Normal: 5 – 7 l/min
- Normal: 65-75ml/kg
- Normal: 14-18/min

*Concord Hospital Ventilator Liberation Guideline Value*
Weaning Strategies

• Decreased SIMV rate (f).
• Pressure Support Ventilation.
• Spontaneous Breathing Trials:
  • Reduced PS
  • T-piece
  • Trach collar
Weaning Strategies

• A Daily Spontaneous Awakening Trial (SAT) and Spontaneous Breathing Trial (SBT) should be performed in patients being considered for weaning and/or extubation.
  • SAT – A period of sedation clearance.
  • SBT – A trial of marked reduction in ventilator support.
• Results of the SAT and SBT will support either:
  • Extubation.
  • Weaning trials.
  • Further “rest” on mechanical settings.
Concord Hospital
Ventilator Weaning Assessment Screening

- Performed on night shift.
- RT and RN will assess parameters on the screening tool to establish whether or not the patient qualifies for a SBT on the day shift.
Ventilator Weaning Assessment Screening Tool

- **Oxygenation**
  - Oxygen saturation ≥88%
  - FiO2 < 50%
  - PEEP ≤ 8

- **Sedation/Mental Status**
  - RASS -1 to +3
  - Absence of seizure

- **Hypothermia protocol, if applicable, is completed**

- **No planned operative procedure scheduled within 24H**

- **Cardiovascular stability; no ischemia**

- **Vasopressors may be present ≤ following doses***:
  - Dopamine: 5mcg/kg/min
  - Norepinephrine: 5mcg/kg/min
  - Dobutamine: 5mcg/kg/min
  - Vasopressin: 0.03unit/min

- **Presence of spontaneous respirations ≥ vent rate/ OR in PSV mode**

- **pH >7.20**

* Unless approved by CPM provider.*
Recap...

- There is more to oxygenation than meets the eye.
- Several factors influence external and internal respiration.
- Several normal “defense mechanisms” come into play in conditions of physiologic adversity.
- Clinicians often times fail to recognize these compensatory mechanism and their importance in clinical practice.
- Noninvasive technology has significant limitations.
When Evaluating Whether Your Patient Has an Oxygenation “Problem” or Not...

✓ Is your patient mentating properly?
  ▪ Take into consideration baseline discrepancies.

✓ Is there a compensatory mechanism in play?
  ▪ Allow this to occur.
  ▪ Support the process as able.
  ▪ Try not to “reverse” this process.

✓ How is end-organ function?
  ✓ Urine output.
  ✓ Capillary refill.
  ✓ Blood pressure.
  ✓ Cardiac aberration.
Solve The Case #1

47 YO male; 48 hours post abdominal surgery. Clinical picture includes decreased breath sounds at the lung bases with the following vital signs and ABG in room air:

**ABG**
- pH 7.51
- PaCO2 29 mmHg
- PaO2 52 mmHg
- HCO3- 22 mEq/l
- SaO2 86%

**VS**
- B/P 140/90
- RR 24/min
- HR 106/min
- Temp 39°C
Solve The Case #1

1. Interpret ABG.
2. List Abnormalities.
3. Differential Diagnosis.
4. Assessment.
5. Treatment.
Solve The Case #1

47 YO male; 48 hours post abdominal surgery. Clinical picture includes decreased breath sounds at the lung bases with the following vital signs and ABG in room air:

1. Uncompensated Respiratory Alkalosis with moderate hypoxemia.
2. Fever, decreased basilar breath sounds, tachypnea, tachycardia, hypertension.
3. Atelectasis vs. Pulmonary Embolus.
5. Oxygen therapy, incentive spirometry, C&DB, ambulation, CXR.

ABG
- pH 7.51
- PaCO2 29 mmHg
- PaO2 52 mmHg
- HCO3- 22 mEq/l
- SaO2 86%

VS
- B/P 140/90
- RR 24/min
- HR 106/min
- Temp 39°C
55 YO female; post cardiothoracic surgery. She appears pale and uncomfortable. She is receiving 60% O2. Her ABG, available labs, and VS are:

**ABG**
- pH 7.30
- PaCO2 37 mmHg
- PaO2 94 mmHg
- HCO3- 20 mEq/l
- SaO2 95%

**Lab**
- THb 5.0 g%
- WBC 8000 mm3

**VS**
- BP 130/85
- HR 120/min
- RR 25/min
- Temp 37°C
Solve The Case #2

1. Interpret ABG and Labs.
2. List abnormalities.
3. Assessment/Diagnosis.
4. Treatment/Recommendations.
Solve The Case #2

55 YO female; post cardiothoracic surgery. She appears pale and uncomfortable. She is receiving 60% O2. Her ABG, available labs, and VS are:

1. Uncompensated metabolic acidosis with normoxemia; however, PO2 only 94 on 60% O2. Hemoglobin 1/3 of normal.
2. Tachycardia, hypertension, tachypnea, pallor.
3. Consistent with severe anemia; significant decrease in O2 content; despite acceptable SaO2, patient has severe anemic hypoxia, producing metabolic (lactic) acidosis.
4. Transfusion, breath sound assessment, CXR.

ABG
- pH 7.30
- PaCO2 37 mmHg
- PaO2 94 mmHg
- HCO3- 20 mEq/l
- SaO2 95%

Lab
- THb 5.0 g%
- WBC 8000 mm3

VS
- BP 130/85
- HR 120/min
- RR 25/min
- Temp 37°C
What Would You Do? #1

56 YO male with CAP and ARDS. Requiring paralysis and sedation for means of pressure control, low tidal volume strategy and 1:1 ratio ventilation. While you are caring for this patient, you observe his SpO2 drop to 85% without any change in vital signs.

A. Increase FIO2.
B. Suspect patient has too much PEEP.
C. Increase paralytic.
D. Nothing immediately, continue to monitor for other clinical changes or worsening SpO2.

Current ABG
- pH 7.30
- PCO2 77 mmHg
- PO2 66 mmHg
- HCO3- 29
- SaO2 87%

Current Settings
- PC 36 cmH2O
- F 20
- Ti 1.5 seconds
- VTE 500 ml
- FIO2 0.60
- PEEP 12
What Would You Do? #2

26 YO female asthmatic refractory to continuous nebulized bronchodilators. Intubated and on pressure control ventilation with target VT of 400ml. Initially stable but suddenly drops SpO2 to 60% and VT now only 200 on same PC setting.

A. Increase PC to 40 cmH2O to increase VT back to target goal.
B. STAT nebulized Albuterol.
C. Increase FIO2 to 1.0 (100%); check breath sounds; suspect barotrauma; order CXR STAT.
D. Increase FIO2 to 0.8 (80%).

Current ABG
- pH 7.35
- PCO2 55 mmHg
- PO2 70 mmHg
- HCO3- 27
- SaO2 92%

Current Settings
- PC 36 cmH2O
- F 25/min
- FIO2 0.60
- PEEP 5 cmH2O
That’s It!
You did it!
Congratulations!
Any Questions?
Good job!
Thanks for coming!
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