The delivery of oxygen to all body tissues is the essence of critical care. Patients in respiratory distress/failure come easily to mind as the ones that require oxygen therapy as their initial therapeutic agent. However, all patients in the PICU may require oxygen during their disease course. It is with this viewpoint in mind that I will address the concept of oxygen delivery and the rationale use of the different forms of oxygen administration.

Let’s first review the key factors which affect adequate oxygenation for patients with lung disease. This entails the transfer of oxygen molecules from the atmosphere to the blood (external respiration). Alveolar oxygen tension is the major limiting factor in the oxygenation of desaturated blood. The four factors that determine alveolar oxygen tension are: (1) the fraction of inspired oxygen (FiO2); (2) the alveolar gas exchange; (3) the mixed venous oxygen content; and (4) the distribution of ventilation to perfusion.
The oxygen tension drops from 159mmHg just outside the mouth to 101mmHg in the alveolus in 100mm air at sea level. This decrease is due to several factors: (1) the addition of water vapor; (2) the addition of a volume of carbon dioxide as well as the removal of a volume of oxygen from the alveolus; and (3) the incomplete gas exchange with every breath.

Increasing the FiO$_2$ will obviously cause an increase alveolar oxygen-tension which may be calculated as follows:

$$PAO_2 (Pb-PH_2O) \times FiO_2 - PaCO_2 \times (1.25)$$

$PAO_2 = $ Alveolar O$_2$ tension
$Pb = $ Barometric pressure (760mmHg at sea level)
$PH_2O = $ Water vapor tension (47mmHg)
$FiO_2 = $ fraction of inspired oxygen
$PaCO_2 = $ arterial carbon dioxide tension

Before progression further with the other factors affecting alveolar oxygen tension, I would like to review the ways of providing oxygen in a non-intubated patient. When choosing a form of oxygen treatment, one needs a consistent O$_2$ concentration in the range of desired FiO$_2$'s. What one hopes to achieve is a sense of the significance of the pulmonary process. This is most easily achieved by knowing the FiO$_2$ that a particular pO$_2$ and/or saturation is based upon. The following oxygen-hemoglobin dissociation curve demonstrates a ballpark figure for a pO$_2$ at a particular saturation.
Another way to demonstrate this important fact is by way of the following shunt curve which will be further explained when we talk about ventilation-perfusion relationships.

Therefore, it is important to know the actual FiO₂ provided to a patient. This is easy on a ventilator. It is simply dialed in. It becomes much more difficult in a non-intubated patient. There are two types of oxygen delivery setups - a low flow system or a high flow system. A high flow system is a setup that provides the patient’s total minute ventilatory flow requirements. In a low flow setup, the patient receives some of his flow requirements from room air. The patient’s actual flow requirements are 3-4 times his calculated minute ventilation depending on his I:E ratio.

\[
\text{Minute Ventilation} = \text{Tidal Volume} \times \text{Respiratory Rate}
\]

Therefore, for a 10kg child breathing 60 times a minute with an assumed I:E ratio of 1:2, his Minute Ventilation would be 3.6 l/min.
Minute ventilation = (10kg) (6cc/kg) x 60 = 3600 l/min

But since he only inhales from the flow one-third of the time, 3.6 l/min must flow past his airway to receive his required minute volume ie **10.8 l/min**. This becomes even more significant in a 50kg asthmatic breathing 30 times per minute with an I:E ratio of 1:3.

ie: (50kg) (6cc/kg) x 30 = 9 liters

9 liters x 4 = 36 l/min flow requirement since only inhaling one-fourth of the time.

The most common oxygen delivery systems used in pediatrics are nasal cannulas, venturi masks, non-rebreather masks, and high flow mask/hood set-ups and are ordered as such. Let us first look at the capabilities of the venturi mask system. The amount of total flow provided is determined by the amount of FiO₂ ordered and its corresponding colored venturi device.

### VENTURI FLOW

<table>
<thead>
<tr>
<th></th>
<th>FiO₂</th>
<th>O₂ Flow</th>
<th>Total Flow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue</td>
<td>24%</td>
<td>4 L</td>
<td>105 L</td>
</tr>
<tr>
<td>Yellow</td>
<td>28</td>
<td>6</td>
<td>68</td>
</tr>
<tr>
<td>White</td>
<td>31</td>
<td>8</td>
<td>63</td>
</tr>
<tr>
<td>Green</td>
<td>35</td>
<td>10</td>
<td>56</td>
</tr>
<tr>
<td>Pink</td>
<td>40</td>
<td>12</td>
<td>50</td>
</tr>
<tr>
<td>Orange</td>
<td>50</td>
<td>12</td>
<td>33</td>
</tr>
</tbody>
</table>

Let’s again use an asthmatic as an example for the use of this system. How about CW? Let’s assume
a tidal volume of 350cc with a respiratory rate of 40 and an I:E ratio of 1:3. Her minute ventilatory requirement is calculated as follows:

\[ 40 \times 350 = 14 \text{ l/min} \times 4 = 56 \text{ l/min} \]

Since only breathing in 1/4 the time with an I:E ratio of 1:3

Therefore, you may find that she may saturate worse as you go from 35% to a 50% venturi setup since the flow from the system drops from 56 liters (which she requires) down to 33 liters which may actually produce an FiO\(_2\) less than the 35% you started with since she must now obtain the remaining from room air.

From this example you can see how important it is to have a reasonable idea of one’s minute ventilatory requirement as well as their flow requirements in mind when ordering any form of oxygen therapy.

When ordering a high flow system in a hood or mask setup, make sure you ask the respiratory therapist what the total flow is at the FiO\(_2\) ordered. This will allow you to better assess ones oxygen requirement if you know that you are providing ones total minute ventilatory requirements based upon your own calculations of the patient’s requirement. This is the most common mode of oxygen delivery to a patient in respiratory distress in the PICU.

A non-rebreather mask should rarely, if ever, be used in the PICU. It is only appropriate for the emergency use of a potentially high concentration of oxygen. As long as the reservoir bag does not collapse on inspiration then you can be assured of providing upwards of around 90% oxygen if using 100% O\(_2\) as the wall source for the gas flow.

Finally, I would like to review the use of the nasal cannula and the FiO\(_2\) that it provides. This is the most commonly used form of O\(_2\) therapy in pediatrics due to its ease of use and comfort to the patient but it is also the most commonly misunderstood in terms of the actual FiO\(_2\) delivered.

There are three different areas of flow that take part in the actual flow presented to the alveolus in this form of a “low” flow system. These include the inspiratory time flow, the anatomic reservoir flow, and the flow from room air.
The variables that determine the FiO₂ delivered by a nasal cannula include: 1) the capacity of the available oxygen reservoir, 2) the oxygen flow; and 3) the patient’s breathing pattern.

The anatomic reservoir is 1/3 of the anatomic dead space. Since the anatomic dead space is 2cc/kg, then the capacity of the reservoir is 2/3 cc/kg.

Oxygen flow is expressed in cc per second when calculating the FiO₂.

\[ \text{X L/min} = \text{X} \frac{1000cc}{60\text{sec}} = \text{X} 16.7cc/sec \]

The breathing pattern determines the inspiratory time in seconds. The filling time of the anatomic reservoir is 1/4 of the expiratory time since there is essentially no flow of outward gases during the last 1/4 of expiration. This filling time is also determined by the breathing pattern.

Let us now use these definitions and principles in the calculation of the FiO₂ of a 2 liter nasal cannula provided to a 5kg infant breathing 40 times per minute.

**First, Calculate:**

- **Tidal volume**: 
  \[ 6cc/kg \times 5kg = 30cc \]

- **Inspiratory time (secs)**: 
  \[ 1/3 \text{ of Cycle Time} = 1/3 \text{ of } 1.5 \text{ secs} = 0.5 \text{ sec} \]

- **Anatomic Reservoir Capacity (cc’s)**: 
  \[ 2/3cc/kg \times 5 = 3.3cc \]

- **Filling Time (secs)**: 
  \[ 1/4 \text{ exp. time } = 1/4 (1sec) = 0.25 \text{ sec} \]

- **Oxygen flow (cc/sec) (y L/min)**: 
  \[ y (16.7cc/sec) = 2 (16.7cc/sec) = 33.4 cc/sec \]

  \*Cycle time = Time it takes for both inspiration and expirations ie 60 ÷ RR

**Second, Multiply:**

- **Inspiratory Time x Flow**: 
  \[ 0.5 \text{ sec } \times 33.4cc/sec = 16.7 \text{ cc} \]

- **Filling time x Flow = maximum reservoir O₂ volume**: 
  \[ 0.25 \text{ sec } \times 33.4cc/sec = 8.34 \text{ cc/sec} \]

  \[ \text{but maximal } = 3.3 \text{ cc} \]

**Third, Subtract**

- **and from tidal volume giving** 
  \[ 30 \text{ cc } - (16.7 + 3.3) = 10cc) \]

- **Room air volume**
Fourth, Multiply

Room air volume x 0.21 = room air O₂ volume
(in cc’s) = 10 x 0.21 = 2.1 cc

Then, add

Î Inspiratory O₂ volume = 16.7 cc plus
Î Reservoir O₂ volume = 3.3 cc plus
D Room air O₂ volume = 2.1 cc

= 22.1

Finally:

Finally, divide total by tidal volume to achieve FiO₂

\[
\frac{22.1}{30cc} = 74\%
\]

If this infant breaths at a respiratory rate of 30, then he/she will receive 91% O₂. Make the child 4kgs with a tidal volume of 24cc and he/she will be receiving virtually 100% O₂ in both instances. This is why one must be careful with the use of nasal cannula O₂ in young infants. They may require 2l to keep them saturated when breathing 60 times per minute when they are in respiratory distress which may be a non-toxic amount of oxygen. Allow them to go to sleep and now breath 30-40 times per minute and you may now be giving toxic amounts of oxygen producing irreversible lung damage.

You may also think that you need to provide higher flows of oxygen in an infant with a snotty nose. This may not always be true. Whenever there is a potential to be giving toxic amounts of oxygen to a child, substantiate the safety by seeing how much FiO₂ you are actually giving by placing the patient in a high flow hood system and measuring the actual FiO₂ required to give you similar saturations with both systems.

Let us now calculate the amount of FiO₂ provided by giving 2l by nasal cannula to a 50kg adolescent breathing 20 times per minute, ie. one in none to minimal respiratory distress.

<table>
<thead>
<tr>
<th>Tidal volume</th>
<th>300cc</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Time</td>
<td>1 sec</td>
</tr>
<tr>
<td>Reservoir Capacity</td>
<td>33cc</td>
</tr>
<tr>
<td>Filling Time</td>
<td>0.5 sec</td>
</tr>
<tr>
<td>O₂ flow</td>
<td>33.4cc/sec</td>
</tr>
<tr>
<td>1) I Time</td>
<td>33.4 cc</td>
</tr>
<tr>
<td>2) Reservoir</td>
<td>16.7cc</td>
</tr>
<tr>
<td>3) Room Air</td>
<td>52.5</td>
</tr>
<tr>
<td></td>
<td>102.6 ÷ 300 = 34%</td>
</tr>
</tbody>
</table>
This is why there is no harm in providing this minimal amount of $O_2$ to adolescent patients who might benefit, ie. DKA patients, ingestions, etc. ie. large patients with the potential for CNS, cardiac, or respiratory decompensation.

**Alveolar Gas Exchange**

When does the majority of oxygenation take place, inspiration or expiration? One’s first thought may be inspiration. However, with the concept of FRC, (the amount of air left in the lungs at the end of expiration) and the I:E ratio (with expiration being longer than inspiration) then one can logically see that the majority of oxygenation takes place during expiration since respiration occurs continuously with each beat of the heart. The degree of replenishment of alveolar gas on inspiration will depend upon the resistance to gas flow in and out of the alveolus and the elastic properties of that alveolus. It should be obvious that the greater the alveolar gas exchange, the greater delivery of oxygen molecules to the alveolus per unit time which is why a greater flow is required of oxygen delivery devices with higher respiratory rates. Therefore, a major factor in determining the alveolar $O_2$ tension would be the degree of alveolar gas exchange.

**Mixed Venous Oxygen Content**

Any situation that may cause a decrease in the mixed venous $O_2$ saturation may contribute to a drop in the $PAO_2$. Severe anemia, a decreased cardiac output, shock, and an increased metabolic requirement are all situations where the tissue utilization of oxygen that has been delivered may increase leading to a decreased mixed venous $O_2$ sat. Instead of the venous sat being 70-75% ie normal which leads to a pulmonary capillary arterial saturation of 98 - 100%, a reduced venous sat in the range of 60% will lead to a decreased pulmonary capillary arterial sat ie 88-92% range which will then be associated with an overall decreased arterial $O_2$ saturation. This assumes a constant FiO$_2$, a constant volume of alveolar gas exchange and a constant pulmonary blood flow. An increased pulmonary blood flow may outstrip the amount of oxygen available in the alveol which may also lead to a reduced pulmonary capillary $O_2$ sat in that alveolar capillary lung unit.

“Uneven distribution of ventilation with respect to the perfusion is the most common clinical phenomenon responsible for hypoxemia that is responsive to oxygen therapy (Shunt Effect)”
The C. Shunt Unit above plays the most important role in hypoxemia. Desaturated blood remains desaturated when mixed with saturated blood the saturation increases but not to normal. If a 50% shunt occurs then 50% of blood dumping into the LA will be approximately 70% saturated and 50% will be 100% saturated with a resultant saturation of 85% or a PAO$_2$ of about 50 mmHg. Figure 4.13 on page 3 reveals the other PAO$_2$'s at various shunts and FiO$_2$'s. A shunt greater than 15-20% implies significant lung disease and the potential need for intubation. This degree of shunt reflects a PAO$_2$ of about 80-120 mmHg on 60% O$_2$. 
Deadspace Unit B. With only ventilation and no perfusion only affects the inability of removal of CO₂ whereas the Silent Unit D with no perfusion or ventilation affects neither pCO₂ or pO₂. Obviously, there is a continuum between these units with varying affects on pCO₂ and O₂. Treatment of these processes through mechanical ventilation will be discussed in the next section.