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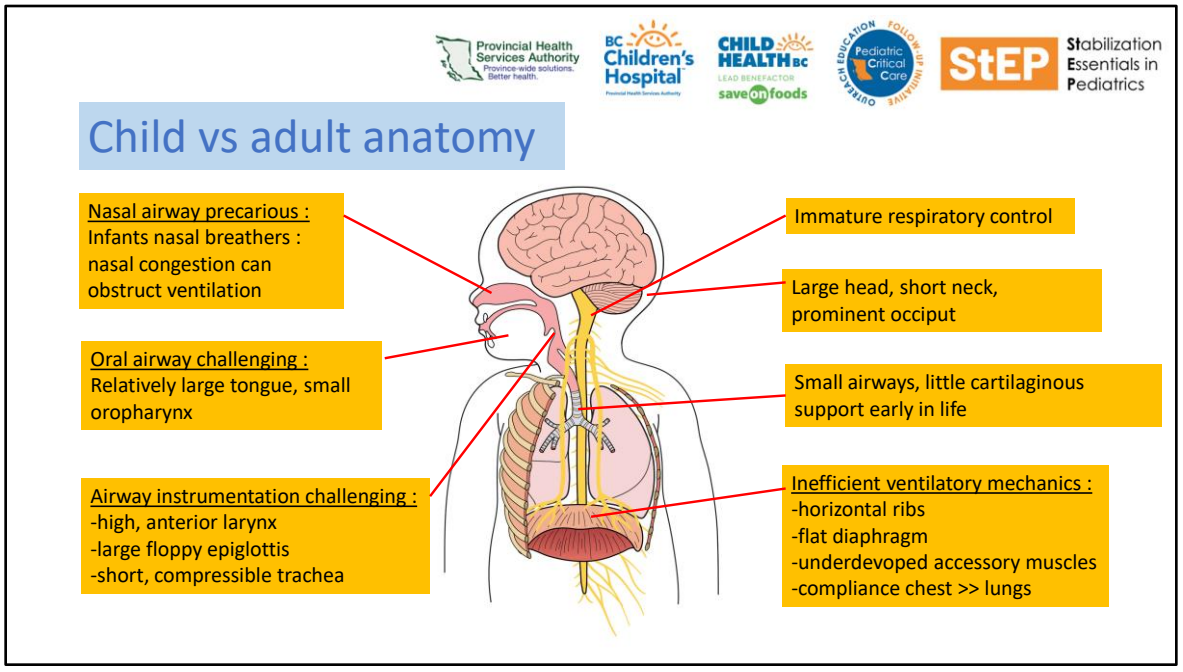
Pediatric Critical Care
OUTREACH EDUCATION FOLLOW-UP INITIATIVE

StEP
Stabilization Essentials in Pediatrics

Acute Respiratory Failure

Objectives for pre-Course reading

- Describe the similarities and differences between adults, children and infants' anatomy and their physiological responses to critical illness
- Identify clinical signs of respiratory distress and of impending respiratory failure in pediatric patients



In comparison to adults, the pediatric airway **more prone to obstruction**, **more difficult to maintain,** and **more technically challenging to intubate**.

Their **respiratory mechanics are inefficient** due to a combination of relatively horizontal ribs, flatter diaphragm shape and under-developed accessory muscles. Leads to **smaller tidal volumes** and a **higher respiratory rate** to maintain minute ventilation.
Infants also have an **extremely compliant chest wall** which **surpasses their lung compliance** leading to a greater **tendency for lungs to collapse** and **decreased functional residual capacity (FRC) which is often near or below closing capacity (CC**). This results, at **baseline,** in **increased atelectasis and V/Q mismatch (V= Ventilation and Q=Pulmonary Blood Flow) and lower arterial partial pressure of oxygen**.

Child vs. adult physiology

FRC / Closing volume in infants

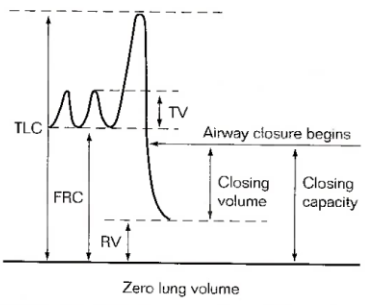


Figure 11.6 Static lung volumes and closing lung volumes. Spirogram to demonstrate normal static lung volume nomenclature. The *y* axis is volume and the *x* axis is time during two normal breaths, a maximum inspiration followed by maximal expiration. The closing volumes are included to help one to visualise how the closing volume to FRC ratio may vary as it does between children and adults. TLC – total lung capacity, FRC – functional residual capacity, RV – residual volume, TV – tidal volume. (With permission from Nunn JF 1993. *Applied Respiratory Physiology* Heinemann Educational Publishers; p. 83.)

Pediatric patients under a year of age are called infants - and they have precarious ventilatory physiology when well, let alone when an opportunistic respiratory virus happens along

This graph show the lung volume on the vertical axis, and the graph line shows what happens with normal tidal ventilation (TV).
The lung volume at the end of a normal, unforced expiration is the FRC. = functional residual capacity
The lung volume at the end of a forced expiration is called the residual volume (RV)

During expiration, the peripheral airways start to close somewhere below FRC : as the peripheral airways close, the alveoli distally collapse causing atelectasis
In infants (and old adults) the **closing volume** is the point at which peripheral airways start to close, and it is perilously close to the FRC – the lung volume at the end of expiration in **normal** breathing
That means the even in a well infant , it doesn't take much before atelectasis starts to occur.
Superimpose RSV or any other respiratory virus on this picture with a mix of

inflammation in the lower airways and alveolar bed, and a bunch of increased secretions in the airways, and FRC and closing volume are pushed closer together. This has the effect of creating multiple parts of the lung with atelectasis causing areas of both VQ mismatch and shunt resulting in hypoxemia and hypercapnia.

It doesn't take a lot of imagination to see why PEEP helps kids with bronchiolitis : it doesn't allow FRC to get anywhere close to closing volume, and it helps resolve both the VQ mismatch and shunt.

More about VQ mismatch and Shunt in the next slide
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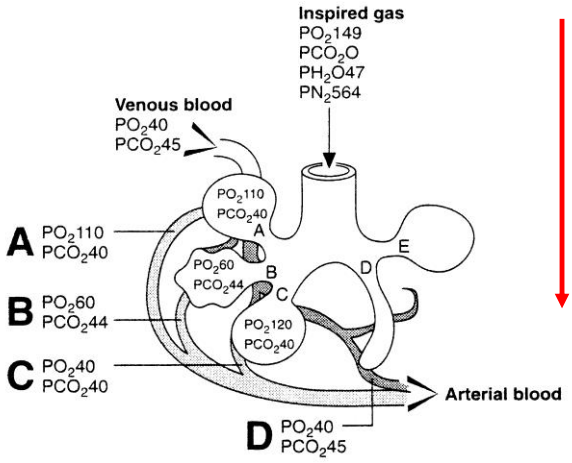
Ventilation (V) perfusion (Q) relationships in the lung



A Normal
B VQ mismatch
C Diffusion block
D Shunt
E Dead space

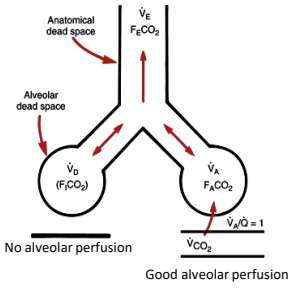
ALL of these variants of ventilation / perfusion matching may be present in the lungs in varying proportions at any time

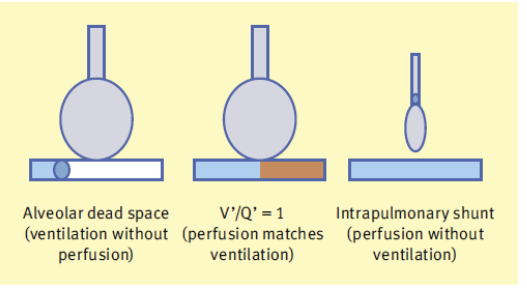
So, what are VQ mismatch and shunt ?

This slide shows mixed venous blood from the right ventricle perfusing five different areas of lung (A-E) each with a different ventilation –perfusion (V-Q) relationship
Understanding A, B, D, and E will help in a big way to understanding what causes respiratory failure and how we go about treating it from a physiological perspective
PO2 is the partial pressure of oxygen that is dissolved in the blood. This is measured on a blood gas.
Mixed venous blood typically has a PO_2 = 40 and a PCO2 = 45
Lung area A represents Normal VQ matching and the blood exiting that area has oxygenated well and decarboxylated into the alveoli so that the blood leaving has a PO2 = 110 and a PCO2 = 40
Lung area B represents VQ mismatch as seen in atelectasis : perfusion is good but ventilation is poor : oxygenation is poor and CO2 clearance poor
Lung area C represents diffusion block and is rare in pediatrics where oxygen cannot pass from the alveoli into the blood : it is characterised by good perfusion and ventilation but very poor oxygenation
Lung area D represents a shunt which is still perfused, but the lung is not ventilated and there is no gas exchange at all
Lung area E represents Dead Space which is lung that sees ventilation but is not perfused
Be aware that all of these VQ variants may be present in different areas of the lungs in varying proportions at any time : how efficiently the lungs work will be dependent on the proportions of each variant, and this will determine the final mixed pulmonary venous blood PaO2 and PaCO2 entering the systemic

circulation

Shunt /dead space / V/Q mismatch





V/Q > 1

V/Q < 1

May have all present in same patient at same time (bronchiolitis, ARDS)

As a recap:
With most lung units have a ventilation/perfusion ratio near to 1

V/Q >1 i.e. ventilation > blood flow (true dead space = ventilation w/o blood flow)

Dead space ventilation does not contribute to hypoxia but can contribute to hypercarbia

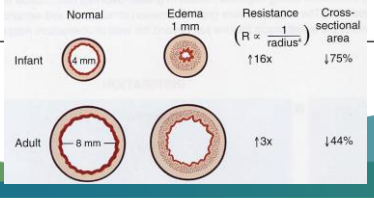
V/Q < 1 i.e. decreased ventilation with normal perfusion (shunt physiology is the extreme with no ventilation but continuing blood flow)

Child vs Adult anatomy and physiology

Table 1. Differences in Pediatric and Adult Physiology

Feature	Child	Adult
Airway cartilage formation	Incomplete	Complete
Airway resistance	Greater increase in airway resistance with reduction in airway radius	Smaller increase in airway resistance with reduction in airway radius
Chest-wall compliance	Greater compliance in view of incomplete ribcage ossification	Less compliant in view of ribcage ossification
Alveolar maturation and impact on FRC	20–300 million alveoli (age-dependent); lower FRC	300 million mature alveoli; higher FRC
Respiratory muscle reserve	More reliant on diaphragm	Less reliant on diaphragm
Risk of pulmonary vascular remodeling	Greater due to higher pulmonary vascular resistance during perinatal transition	Lower
Metabolic requirements	Higher	Lower

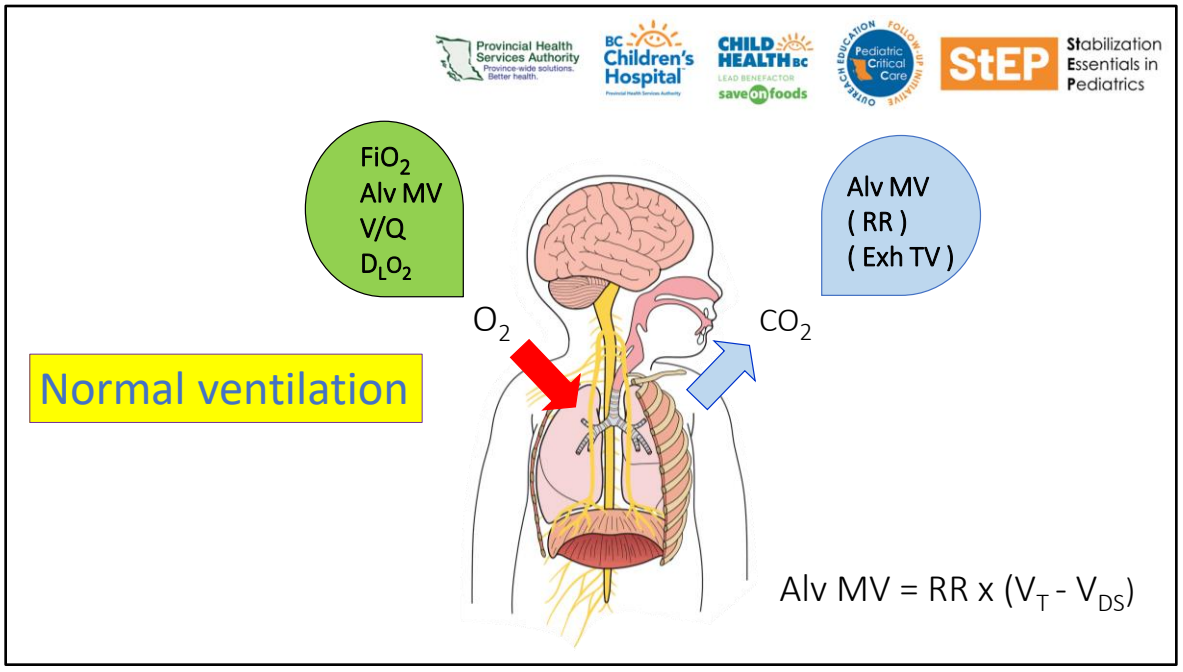
FRC = functional residual capacity



Features of pediatric respiratory system:
Airway is small and therefore small absolute changes in radius (due to oedema or secretions) result a large relative change in cross sectional area and increase resistance.
Clinically significant up to age of 8-9 years
Younger children have relatively little cartilaginous support of the airways.
Dynamic compression during high expiratory flow may occur.
Neonates chest wall extremely compliant – substantial recession, easily overinflated with mech vent, little opposition to deflating tendency of lungs leading to low functional residual capacity and rapid desaturation during apnea (eg during intubation)
In early infancy ribs are relatively horizontal with the result that the respiratory system is very dependent on diaphragmatic function. In presence of hyperinflation or abdominal distension the diaphragmatic function may be restricted with severe respiratory embarrassment

Infants and children have limited respiratory reserve and decompensate rapidly in the face of respiratory compromise……

Let's review the physiology to understand why



R**espiratory Physiology**: The major function of the lung is to get oxygen in and carbon dioxide out

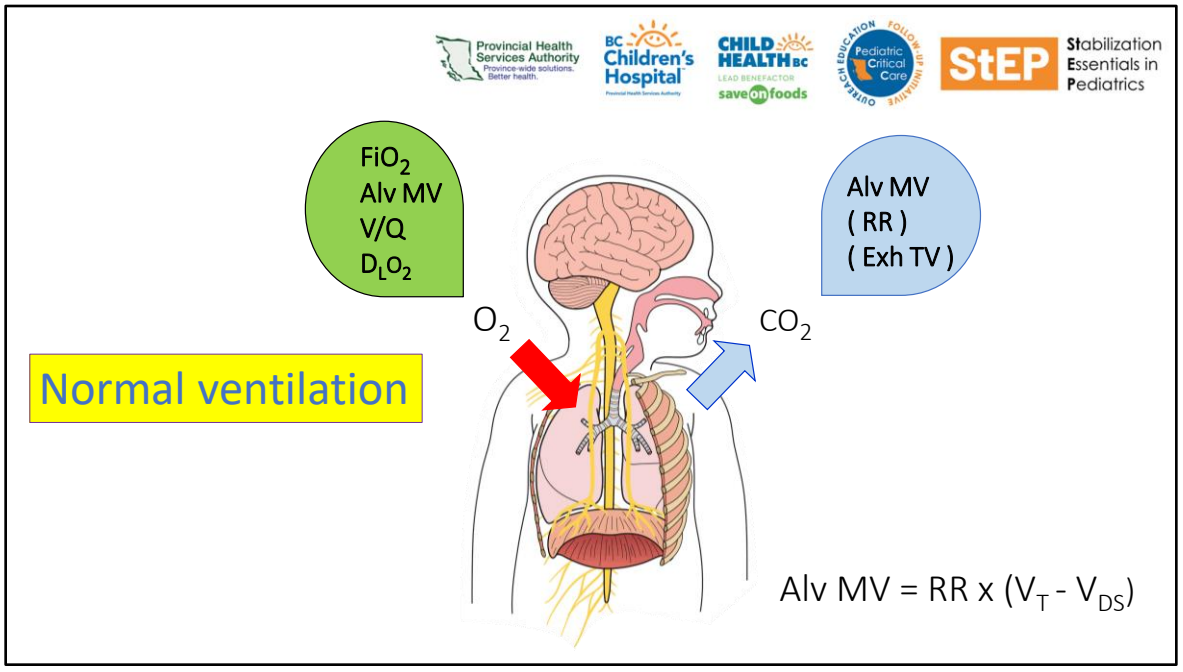
<u>Oxygen in :</u>

The **FiO2** of the inhaled gas mixture is the major determinant of the PAO2, the partial pressure of oxygen in the alveoli, but the partial pressure of CO2 also plays a role, as we will see shortly. Hyperventilation, lowering the PACO2, will increase the PAO2, and this is what enables climbing purists to climb Everest without oxygen tanks.

Alv MV = Alveolar minute ventilation

V/Q matching : as we have already seen, the ventilation : perfusion relationships in the alveolar bed can vary and optimal oxygenation occurs only when V and Q are perfectly matched

D_LO_2 = Diffusing capacity: how well gas moves from alveoli to blood : <u>very rare for this to be an issue in pediatric patients</u>



Continued from previous slide :

Carbon dioxide removal is largely dependent on alveolar ventilation which, in turn, is dependent on the respiratory rate and the exhaled tidal volume. The latter will vary, again depending on ventilation perfusion matching as a determinant of dead space

There is nothing we can do to treat CO2 retention other than taking over ventilation for the patient and controlling the respiratory rate and tidal volume.

It is not easy to detect ventilatory failure unless you very astute in clinical exam or have the help of a blood gas

Carbon dioxide removal

- Largely dependent on alveolar ventilation
- Alveolar ventilation = Resp rate X exhaled tidal volume (TV)
- Exhaled TV = Total tidal volume (6-10 ml / Kg)– dead space volume (3 ml / Kg)

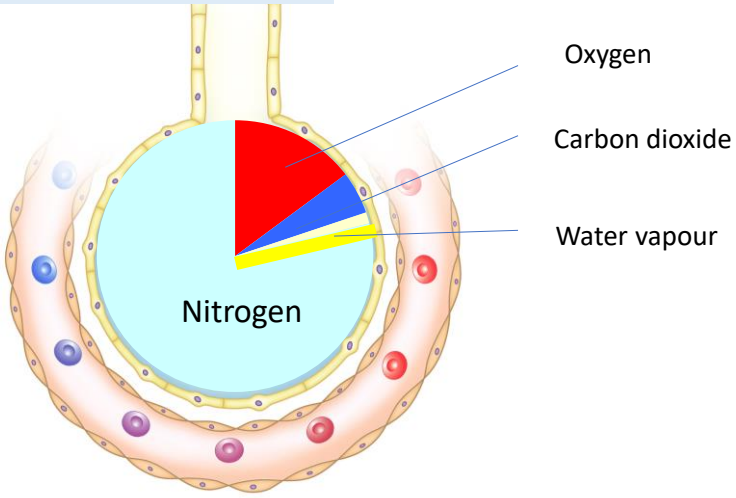
- Anatomical dead space is constant
- Physiological dead space variable dependent on VQ matching
- Equipment dead space

Carbon dioxide removal is largely dependent on alveolar ventilation, which, in turn, is dependent on the respiratory rate and the exhaled tidal volume. The latter will vary, again depending on ventilation perfusion matching as a determinant of dead space.
There is nothing we can manipulate to treat CO2 retention other than taking over ventilationfor the patient and controlling the respiratory rate and tidal volume

It is not easy to detect ventilatory failure unless you are very astute in clinical examination OR have the help of a blood gas

CO2 is closely related to work of breathing and respiratory reserve

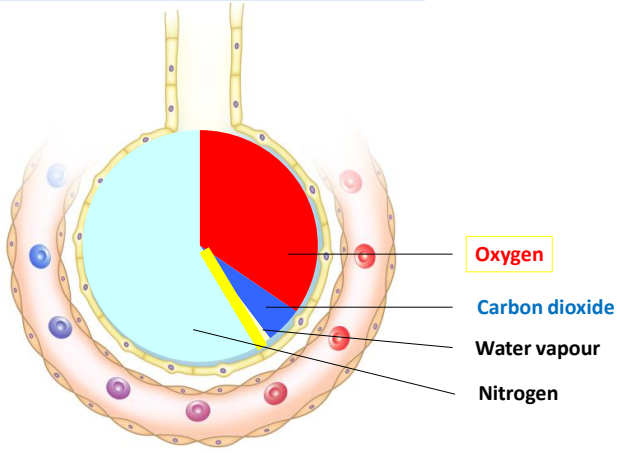
Normal alveolar gas mixture



Alveolar pressure = $P_AO_2 + P_ACO_2 + P_AH_2O + P_AN_2$

The alveolar pressure is equal to the sum of the partial pressures of the gases within the alveolus.
The partial pressure of each gas is proportional to the concentration of the gas. An increase in alveolar pressure will therefore result in a proportionate increase in the partial pressure of all of those gases

Alveolar gas with supplemental FiO_2



Alveolar pressure = $P_AO_2 + P_ACO_2 + P_AH_2O + P_AN_2$

When oxygen is delivered (ie through nasal prongs) the P_AO_2 increases, the P_ACO_2 remains constant and the P_AN_2 decreases meaning increased oxygen saturations and unchanged ventilation and CO2

Alveolar Gas Equation

$P_AO_2 = [FiO_2 \times (P_{atm} - P_{H2O})] -- (P_ACO_2/RQ)$

$PACO_2 = PaCO_2$ Resp Quotient (RQ) = 0.8

Patm = 760mmHg P_{H2O} = 47mmHG

$P_AO_2 = FiO_2 \times (760 - 47mmHg) - P_aCO_2/0.8$

-PLEASE DO NOT THINK THAT YOU HAVE TO MEMORISE THIS : IT IS HERE ONLY TO HELP YOU UNDERSTAND
NORMAL PHYSIOLOGY AND EXAMPLES OF COMMON CLINICAL SCENARIOS

Alveolar gas equation

-PAO2 (is the alveolar partial pressure of oxygen) depends on:

-Oxygen (FiO_2)
-atmospheric pressure (P_{atm})
-water vapor partial pressure of water (P_{H2O})
- CO2 partial pressure in the alveoli (P_ACO_2)
-**Respiratory coefficient (RQ)** (ratio of CO2 eliminated divided by the O2 consumed, usually 0.8)

Practical applications of the Alveolar Gas Equation

Alveolar gas equation: $PAO_2 = FiO_2 * (Patm - PH2O) - (PaCO2/RQ)$

Breathing in RA

- PaCO2= 40, FiO2 =0.21
- PAO2= 0.21 * (760-47) –(40/0.8) = 150-50 = 100 (measured sat of 100%)

Breathing 100% O_2

- P_AO_2 = 1.00 x 713 – (40/0.8) = 663mmHg (measured sat of 100%)

Rise in PaCO2 from hypoventilation, with consequent hypoxemia

- PaCO2= 80, FiO2= 0.21
- PAO2 = 0.21 (760-47) –(80/0.8) = 150-100 = 50 (measured sat approx 80%)

Hypoxemia from hypoventilation easily overcome with small amount of oxygen

- PaCO2= 80, FiO2= 0.30
- PAO2 = 0.30 (760-47) –(80/0.8) = 213-100 = 114 (measured sat of 100%)

A-a (O_2) Gradient

Estimate difference between alveolar partial pressure O_2 (PAO_2) and arterial PaO_2

A-a (O_2) Gradient = $PAO_2 - PaO_2$

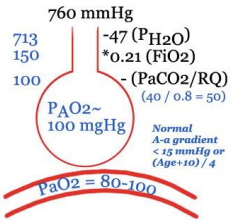
- PAO2= (P_{atm}–P_{H2O}) x FiO2 –(PaCO2 / RQ)= (760 –47) x 0.21 –(40 / 0.8)(713 x 0.21) –50
- PAO2= 149 –50 = 99= 352 – 60 = 292
- Normal PaO2= 70 –100

Normal A –a gradient= 99 –80 = ~ 20 mm Hg (or **0 –30 mm Hg**) (or Age + 10 /4)

Example : Patient has PaO_2 60 on FiO_2 0.6 and $PaCO_2$ is 60

PAO_2 = (713*0.6) – (60/0.8) = 427-75 = 352

A-a gradient = 352 – 60 = 292



AGAIN, NOT TO BE MEMORISED BUT USEFUL TO EXPLAIN CLINICAL SCENARIOS

Alveolar gas estimation minus the measured arterial PaO2.
Not really a gradient, but a difference

If A-a gradient elevated then oxygenation of the alveolus happens BUT the passage of oxygen to the body does not. Pathology of the alveolar-capillary unit will result in a high A-a gradient
Patients with hypoxemia due to hypoventilation will have an A-a gradient within normal limits

Acute Respiratory Failure

- Hypoxaemic respiratory failure:
  $PaO_2 \leq 60mmHg$ (approxmate
- Hypercarbic respiratory failure:
  $PaCO_2 \geq 50mmHg$
- Common to have features of both in respiratory failure

We have now reviewed the important aspects of how the lungs work with normal physiology ,Ie, in the absence of disease

Now it is time to look at what happens in a variety of pathophysiological states caused by a variety of diseases, where respiration fails and our patients have either failure of oxygenation or accumulation of carbon dioxide, or both

From a Pathophysiology perspective, we avoid talking about type 1 or type 2 respiratory failure – it is preferable to describe the actual pathology

Common causes of hypoxemia

- Ventilation – perfusion mismatch
- Shunt
- Hypoventilation with high P_aCO_2
- Low inspired FiO_2 - altitude
- Alveolar diffusion abnormality – rare in children

Far and away, the most common causes of hypoxemia that you see clinically in pediatric patients will be due to a mix of VQ mismatch and shunt caused by acquired lung disease like bronchiolitis and pneumonia

Next in likely frequency will be scenarios where the primary problem is accumulation of carbon dioxide - and the hypoxemia in that setting is a relatively easy fix

Although, in theory, acute respiratory failure may result from a low inspired PO2 this is rarely a problem in Intensive Care except in locations at high altitude or during transport air evacuation

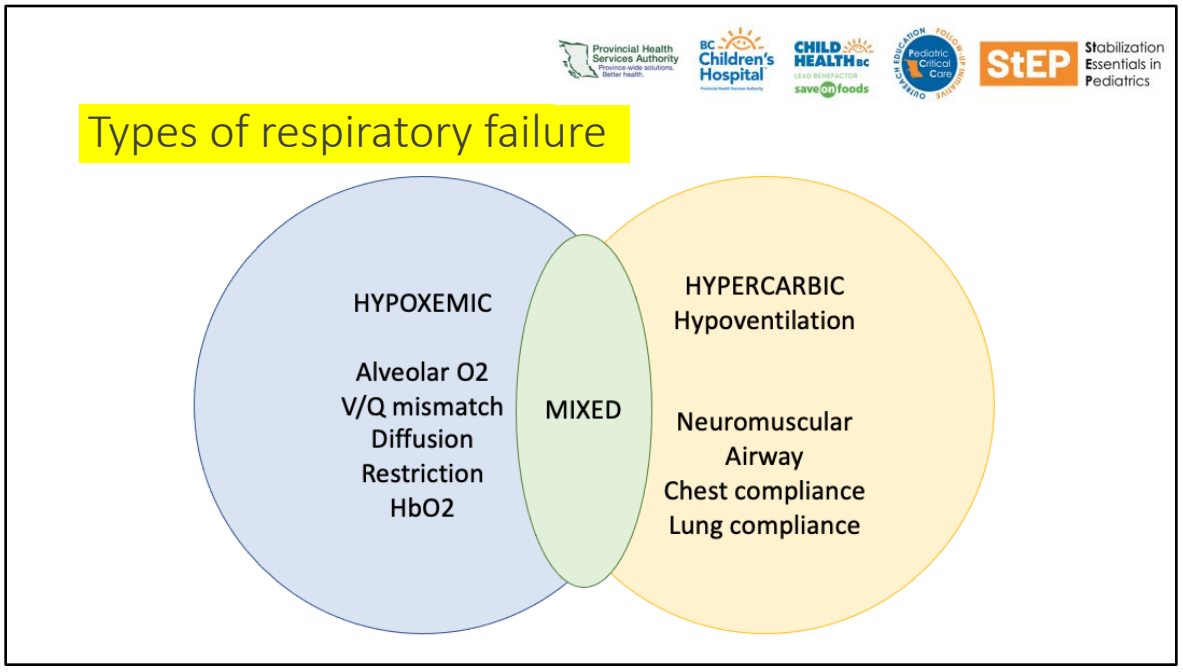
If you are skiing in Aspen Colorado with atmospheric pressure being 550 mmHg pAO2 in RA is 65. At 6000ft in a modern commercial airplane, the pressure is 600, giving a pAO2 of 76

Diffusion abnormalities can result from a failure of diffusion across the alveolar membrane or a reduction in the number of alveoli resulting in a reduction in the alveolar surface area : they are quite rare in pediatric patients

Common causes of hypoventilation / hypercarbia

- Hypoventilation from CNS depression
  - CNS disease, medication, trauma, vascular
- Hypoventilation due to disease determined weakness
  - Congenital, acquired, medication
- Obstructed airway
- Chest compliance
  - Scoliosis, trauma
- Lung compliance
  - Restrictive lung disease
- Bronchiolitis

Hypoventilation : obtundation from meningitis/ encephalitis,secondary to intended sedation (anesthesia), secondary to unintended drug effects (opiate crisis), TBI, intracranial bleeds/strokes
HypoV due to motor weakness : botulism, GuillainBarre, muscular dystrophies
Airway obstruction from disease (croup) , bacterial tracheitis, FB, Trauma involving face/neck



Many lung pathologies present with a mixed hypoxemic and hypercarbic respiratory failure

Dead space = apparatus and physiologic
- Physiologic dead space is much higher in infants and can reach 50%
- Dead space = ventilated but not perfused (PE)

- Shunt = perfused but not ventilated (consolidation, atelectasis etc.)

Approach to managing hypoxemic respiratory failure

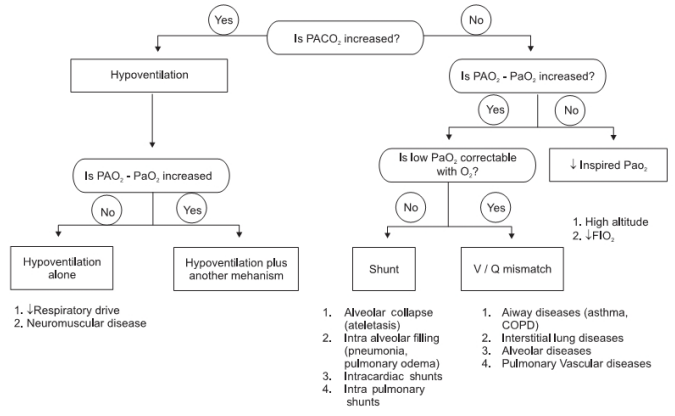


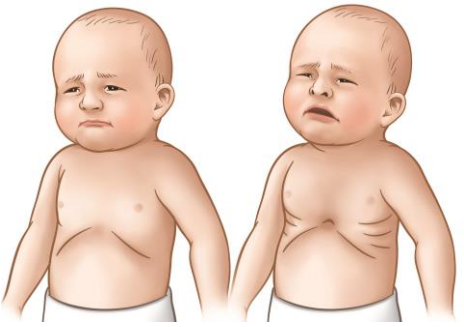
Figure 2: Flow diagram showing approach to hypoxemic respiratory failure

All of this physiology review explains what happens in different contexts of respiratory failure with hypoxia.
Most of the time each pathology has a combination of the physiological causes.
Here is the diagram that summarize what we have seen so far.

Please note : In the algorithm, on the right hand side , under "Is PAO2 – PaO2 increased ?, the "No" should read **Decreased Inspired PAO2**

Clinical signs of respiratory distress

1. Respiratory compensation
   - Tachypnea
     - Very rapid rates can be seen!
   - Noisy breathing
   - Work of breathing
     - Accessory muscle use
     - Nasal flaring (increased nasal air flow)
     - Recession/intercostal retractions
     - Grunting (auto-PEEP)
     - Active exhalation with abdo muscles
     - "See-saw" breathing
     - Head bobbing



Tachypnoea and tachycardia are early and universal signs of respiratory distress.

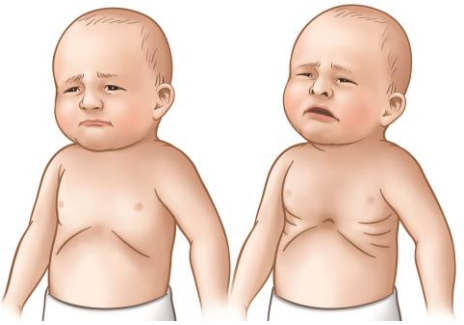
Remember that infants and younger children have **relatively inefficient respiratory mechanics** and it's difficult for them to increase their tidal volume with normal lungs, let alone lungs that are stiff and non-compliant due to acquired disease. They rely on increasing their **respiratory rate** in order to increase their minute volume.

An absence of noisy breathing

Clinical signs of respiratory distress
When do we call it failure ?

Frequent clinical reassessments are **key** to recognizing progressive deterioration

Maintaining longitudinal recording of **vital signs** , **oxygen saturations** , changes in **ventilatory support**, **blood gases** and **Xrays** may be helpful



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An absence of noisy breathing

Warning signs that the need for ventilation support is approaching

- Be concerned if
  - Extreme RR (age dependant, > 50-60), apnea/variable pattern
  - **Cyanosis** or SpO_2 < 90%, despite O_2 support
  - Rising $PaCO_2$
  - CVS deterioration, bradycardia
  - Agitated, confused or comatose
  - Deteriorating despite therapy

Remember that applying oxygen therapy in patients with primarily hypercarbic respiratory failure has the potential to mask secondary hypoxemia