ADVANCED ASSESSMENT
Ventilation & Oxygenation

2007 Ontario Base Hospital Group
# ADVANCED ASSESSMENT

**Ventilation & Oxygenation**

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Learning outcomes

At the conclusion of this presentation, the learner will be able to:

- explain the difference between ventilation and oxygenation
- define ventilation to perfusion ratio (V/Q)
- define and describe shunting
- define and describe dead space ventilation
- list examples of shunting and dead space ventilation
- describe oxygen transport in terms of the Bohr effect
- explain why having a patient re-breath through a paper bag during hyperventilation may be detrimental
- explain why hyperventilation of the head injured patient may be detrimental
Lung anatomy

Inhaled air passes through the mouth and nose
↓
trachea
↓
right & left mainstem bronchi
↓
secondary & tertiary bronchi, etc
↓
Gas exchange region
Lung anatomy

- Gas exchange takes place in the respiratory bronchioles, the alveolar ducts and the alveoli.
- Gas exchanging surface area is approximately 70 m².
- Alveoli have a rich capillary network for gas exchange.
Relevant lung volumes/values

- Respiratory rate: number of breaths per minute (bpm)
- Tidal volume ($T_v$): volume of air inhaled in one breath - exhaled tidal volume can also be measured
- Minute volume ($V_m$) = R.R. x tidal volume
Ventilation vs. Oxygenation
Ventilation vs. Oxygenation

- Often thought of as synonymous
- two distinct processes
Ventilation vs. Oxygenation

What is PaCO$_2$ and PaO$_2$?

- PaCO$_2$ is the partial pressure exerted by dissolved CO$_2$ in arterial blood. Normal PaCO$_2$ is: 35-45 mmHg
- PaO$_2$ is the partial pressure exerted by dissolved O$_2$ in arterial blood. Normal PaO$_2$ is: 80-100 mmHg
- Think of RBC’s as a magnet that is affected by blood ph
Ventilation vs. Oxygenation

- PaCO₂ is affected by ventilation

e.g. if you were to hyperventilate someone without supplemental O₂, you would notice that their PaCO₂ level would drop (below 35 mmHg), while their PaO₂ would remain unchanged or increase marginally.
Ventilation vs. Oxygenation

- PaO₂ is affected by providing supplementation oxygen

E.g., if you were to provide the patient with supplemental O₂, now you would see their PaO₂ begin to rise significantly (above 100 mmHg). Meanwhile, if their R.R. and tidal volume remained unchanged, you would see no change in their PaCO₂.
Exception

If there is a ventilation to perfusion (V/Q) mismatch that can be improved by providing positive pressure ventilation (e.g. ventilating a patient who has pulmonary edema), this may help to increase the PaO$_2$.

Positive pressure ventilation does this by opening up a greater number of alveoli and increasing the surface area across which oxygen can diffuse.

…more about this later
Ventilation to perfusion ratio:
V/Q Review
V/Q ratio

GOOD AIR ENTRY

GOOD BLOOD FLOW

= V/Q MATCH
Let’s look at some examples of V/Q mismatch,
i.e. **shunting** and **dead space** ventilation
WHAT IS A SHUNT?

Results when something interferes with air movement to the gas exchanging areas
WHAT IS A SHUNT?

EXAMPLES?

- F.B.O.
- bronchospasm
- mucous plugging
- pneumonia
- pulmonary edema
- hypoventilation
- positional
- etc.
WHAT IS DEAD SPACE?

ANATOMICAL

- air passages where there is no gas exchange
- mouth & nose, trachea, mainstem bronchi, secondary, tertiary, etc
- ~ 150 cc in the adult

PATHOLOGICAL

- pulmonary embolus
- Shock (vasoconstriction)
Pathological dead space

Normal blood flow

Blocked or impaired blood flow
Mixed shunt & dead space

EXAMPLE
- emphysema
- pulmonary edema

Shunt from fluid in the airways

Dead space: interstitial edema separates the airways from the capillaries
SUMMARY

Shunt vs. Dead Space

- Results from anything that interferes with the movement of air down to the gas exchanging areas

- Non-gas exchanging areas - or
- Areas of the lungs normally involved in gas exchange, however blocked or impaired blood flow preventing this.
Once again...

IMPORTANT CLINICAL NOTE

Ventilation alone has virtually no effect on PaO$_2$ - except where there is a shunt and when positive pressure ventilation can help to open up additional alveoli to ↑ the surface area for oxygen diffusion
FLOW OF OXYGEN

O₂ crosses the alveolar-capillary membrane
then dissolves in plasma
then binds to hemoglobin (98%) - 2% remains dissolved in blood plasma
O₂ bound to hemoglobin then comes off hemoglobin, dissolves in plasma and diffuses to the tissues
NOTE: Pulse oximetry (SpO₂) is the measurement of O₂ bound to hemoglobin - i.e. the percentage of hemoglobin saturated with O₂ molecules.

Whatever O₂ is not bound to hemoglobin gets transported in its dissolved form in blood plasma – this ↑ the PaO₂
Hemoglobin’s affinity for oxygen

...It’s like a magnet
Before we begin discussion of the Bohr effect, we need to review a little about blood pH

- normal blood pH is 7.35 to 7.45
- a pH below 7.35 is called an acidosis, while a pH above 7.45 is called an alkalosis
- CO$_2$ is part of the carbonic acid buffer equation - when CO$_2$ is blown off, it’s like blowing off acids - therefore the blood pH shifts toward the alkaline side. CO$_2$ diffuses 20 times more readily than O$_2$ - i.e. 20:1 ratio.
Bohr Effect

- The Bohr effect describes the body’s ability to take in and transport oxygen and release it at the tissue level.
- According to the Bohr effect, hemoglobin is like a magnet that becomes stronger in an alkaline environment and weaker in an acidotic environment.

Let’s look now at how the Bohr effect is put in to practice in the process of breathing...
Bohr Effect

- when we exhale, we blow off CO\textsubscript{2}. This shifts the blood pH toward the alkaline side. Hemoglobin becomes a stronger magnet and attracts O\textsubscript{2} as air is inhaled.

- At the tissue level, CO\textsubscript{2}, a by-product of cellular metabolism, diffuses from the tissue to the blood. This shifts the blood pH toward the acidic side, weakening hemoglobin’s hold on O\textsubscript{2} (weaker magnet), and releasing O\textsubscript{2} to the tissues.

- This occurs on a breath by breath basis
Clinical application - Bohr Effect

- If you over-zealously hyperventilate a patient, they will become alkalotic.

- When the blood pH becomes persistently alkalotic, hemoglobin strongly attracts $O_2$ at the level of the lungs, but doesn’t release it well at the tissue level.

- i.e. blowing off too much $CO_2$ may result in impairment of oxygenation at the tissue level.
Clinical application - Bohr Effect

- What did they teach you to do when you encounter a patient who is “hyperventilating”?

1. Don’t give them oxygen “?”
2. Coach their breathing to slow them down
Ventilation: Clinical Issues

When the *patient* hyperventilates

When *we* hyperventilate the patient
When the patient hyperventilates

Benign or life-threatening

- When you first encounter a patient who is hyperventilating, always assume there's an underlying medical condition responsible.

Differential:
Acute RDS, asthma, atrial fibrillation, atrial flutter, cardiomyopathy, exacerbated COPD, costochondritis, diabetic ketoacidosis, hyperthyroidism, hyperventilation syndrome, metabolic acidosis, myocardial infarct, pleural effusion, panic disorder, bacterial pneumonia, pneumothorax, pulmonary embolism, smoke inhalation, CO poisoning, withdrawal syndromes, drug overdose (e.g. salycilates)…
When the patient hyperventilates

- too much CO₂ is blown off
- respiratory alkalosis
- potassium and calcium shift intracellular
  - tetany
  - vessel spasm
When the patient hyperventilates

- withholding oxygen from someone who is hyperventilating serves no benefit and may be harmful
- making the patient re-breath their own CO₂ can be dangerous and even fatal
When the patient hyperventilates

Shift in thinking

1. don’t judge the patient
2. give them all oxygen
3. don’t coach their breathing - at least not at first
4. don’t use a paper bag or oxygen mask (without oxygen)
5. begin assessment on the assumption that there is an underlying metabolic (or structural) cause
Brain Injury: Increased ICP

- CO₂ is a potent vasodilator
- Hyperventilating the patient causes cerebral vasoconstriction which helps decrease ICP
- Good in theory – not so good in practice
When we hyperventilate the patient

Secondary Brain Injury: Watershed

- The vessels within the injured area are damaged
- Constricting the vessels surrounding the damaged area from overzealous hyperventilation results in blood flow into the damaged area resulting in worsened edema and further secondary brain damage.
SUMMARY

Ventilation & Oxygenation

Not the same thing
Question # 1

What does the term “minute volume” mean?

A. a very small volume
B. the amount of air inhaled with each breath \( (T_V) \)
C. the volume of inhaled air over one minute \( (R.R. \times T_V) \)
D. a quiet sound
Question # 1

What does the term “minute volume” mean?

A. a very small volume
B. the amount of air inhaled with each breath ($T_V$)
C. the volume of inhaled air over one minute ($R.R. \times T_V$)
D. a quiet sound
Question # 2

The gas exchanging areas of the lungs include:

A. The mouth, nose and trachea
B. The respiratory bronchioles, alveolar ducts and alveoli
C. The mainstem bronchi
D. The lining of the stomach
Question # 2

The gas exchanging areas of the lungs include:

A. The mouth, nose and trachea
B. the respiratory bronchioles, alveolar ducts and alveoli
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Question # 3

What blood gas component does ventilation affect?

A  PaO₂

B  PaCO₂
Question # 3

What blood gas component does ventilation affect?

A  PaO₂
B  PaCO₂

Ventilation affects primarily the PaCO₂ level

e.g. if you were to hyperventilate someone without supplemental O₂, you would notice that their PaCO₂ level would drop (below 35 mmHg), while their PaO₂ would remain unchanged or increase only marginally.
Question # 4

The short form V/Q stands for:

A. vintage quality
B. various quantities
C. verbal question
D. ventilation to perfusion ratio
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Question # 5

All of the following are examples of a shunt except:

A. pulmonary embolus
B. foreign body obstruction
C. bronchospasm
D. mucous plugging of the terminal bronchioles
Question # 5

All of the following are examples of a shunt except:

A. pulmonary embolus
B. foreign body obstruction
C. bronchospasm
D. mucous plugging of the terminal bronchioles

Pulmonary embolus is the only one from the list that is not an example of a “shunt”. It is an example of dead space ventilation.
Question # 6

When you exhale, blood pH in the pulmonary capillaries shifts toward the:

A  acidic side
B  alkaline side

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Question # 6

When you exhale, blood pH in the pulmonary capillaries shifts toward the:

A. acidic side
B. alkaline side
Question # 7

When the blood pH is acidotic, hemoglobin’s affinity for oxygen is:

A  unaffected
B  stronger
C  weaker
D  none of the above
Question # 7

When the blood pH is acidotic, hemoglobin’s affinity for oxygen is:

- [A] unaffected
- [B] stronger
- [C] weaker
- [D] none of the above
Question # 8

At the tissue level, if the blood pH is too alkalotic, hemoglobin will:

- [A] hold onto oxygen more tightly
- [B] release oxygen more readily
- [C] destroy oxygen molecules
- [D] none of the above
Question # 8

At the tissue level, if the blood pH is too alkalotic, hemoglobin will:

A. hold onto oxygen more tightly
B. release oxygen more readily
C. destroy oxygen molecules
D. none of the above
Question # 9

A shunt means that ventilation is:

A. less than perfusion
B. the same as perfusion
C. greater than perfusion
D. all of the above
Question # 9

A shunt means that ventilation is:

- **A** less than perfusion
- **B** the same as perfusion
- **C** greater than perfusion
- **D** all of the above
Question # 10

Air entry to the lungs in a patient who has a massive pulmonary embolism is most likely to be:

A. absent
B. markedly diminished
C. normal
D. absent on one side only
Question # 10

Air entry to the lungs in a patient who has a massive pulmonary embolism is most likely to be:

- A. absent
- B. markedly diminished
- C. normal
- D. absent on one side only

When an embolus blocks blood flow, air entry into the lungs will be unaffected.
Well Done!

Ontario Base Hospital Group
Self-directed Education Program
SORRY,
THAT’S NOT THE CORRECT ANSWER