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Gas Exchange

John G. Younger, MD
Associate Professor
Department of Emergency Medicine
A Tour of the Lecture

• Some perspective

• Some physics and biochemistry
  – Convection and Diffusion
  – Hemoglobin
  – Pressure, Content, and Transport

• The lung as a gas exchanger
  – Carbon dioxide handling and dead space
  – Oxygen handling
    – The four causes of hypoxia
  – Quantifying gas exchange
The citric acid cycle, also known as the Krebs cycle, is a series of biochemical reactions that take place in the mitochondrial matrix of eukaryotic cells. It is a central metabolic pathway that produces energy in the form of ATP, and is involved in the metabolism of carbohydrates, fats, and proteins.

The cycle begins with the degradation of pyruvate to acetyl-CoA, which then enters the cycle. Acetyl-CoA is converted to oxaloacetate, forming citrate through the action of citrate synthase. Citrate is then converted to cis-aconitate, which can either be converted to malate or isocitrate.

Malate is converted to oxaloacetate, completing the cycle, while isocitrate is converted to α-ketoglutarate, which is then converted to succinyl-CoA. Succinyl-CoA is then converted to fumarate, which is converted back to oxaloacetate, completing the cycle.

The cycle is driven by the oxidation of NADH and FADH2, which are produced during the reactions of the cycle. These electrons are transferred to the electron transport chain, ultimately leading to the production of ATP.
Mitochondria are where respiration occurs. The ‘respiratory system’ is really a bulk transport system for an incoming oxidizer (O$_2$) and outgoing gas (CO$_2$) and heat.
Bulk Transport:
Getting Molecules from One Point to Another

• Convection
  – Bulk movement of gas due to pressure gradients
  – Requires mechanical power input
  – Transport from the environment to the terminal bronchioles
  – Transport of erythrocytes between pulmonary capillaries and peripheral capillaries

• Diffusion
  – Transport based on random motion of thermally energetic particles situated in a concentration gradient
  – Requires thermal input
  – Transport from terminal bronchioles to erythrocytes
  – Transport between erythrocytes and peripheral mitochondria
Transport by Convection

• The movement of particles by convection is driven by:
  – The pressure gradient between the atmosphere and terminal bronchioles
  – Thus, this is in the realm of what’s typically referred to as pulmonary mechanics

• The mechanical work required to get this job done is a function of:
  – Resistance of the transport path to air flow
    • Itself a function of the effective cross-sectional area of the airways
  – The viscosity and density of the air being moved
  – The compliance of the lung and chest wall
Transport by Diffusion

• The movement of particles by diffusion is driven by:
  
  – Concentration gradient
    • For physiological purposes, usually reported as differences in partial pressure
    • Gradient may exist in either gas phase (e.g., bronchiole->alveolus) or liquid phase (e.g., plasma->RBC membrane)
    • Note: Although concentrations are often reported in partial pressures, at it’s heart this is a Brownian Motion based phenomenon!

  – Diffusivity
    • A measure of the tendency of a molecule to avoid getting ‘hung up’ in the surrounding media
    • Specific for solute, solvent, and temperature
    • In part is a function of molecular mass
    • E.g., the diffusivity of oxygen in air is different than in plasma, and the diffusivity of oxygen in plasma is different than the diffusivity of carbon dioxide
Diffusion is A Random Walk in Space
Bulk Transport by Diffusion

- The Brownian movement of particles over a distance is proportional to the square root of time.

- At scales more than 50 microns or so, diffusion is a uselessly slow process!

- The keys to diffusing large amounts of gas (e.g., hundreds of milliliters per minute) are:
  - a very large surface area for gas particles to randomly walk across.
  - A very short diffusion path.

![Graph showing distance versus time with the source undetermined.](source-undetermined.png)
- In adult humans, ~23 generations of airway bifurcations
- 2.5 – 7.5 x 10^6 alveoli
- Total surface area of ~ 130 square meters
- Corresponding generations of both pulmonary arteries and pulmonary veins
• Arthur Ashe Stadium
  – Each player brings a respiratory surface area comparable to the area of the court
  – At capacity, the stadium has a respiratory surface area of $2.8 \times 10^6 \text{ m}^2$ and is diffusing more than 4,400 liters of $\text{O}_2$ per minute
Pulmonary Gas Diffusion in Health and Disease

- Factors Adversely Affected by Illness
  - Diminished concentration gradient between alveoli and pulmonary capillaries
    - Due to convective failure
  - Distance to be Traveled
    - Membrane thickness
    - Perivascular edema or fibrosis
  - Surface Area
    - Loss of alveoli, alveolar flooding

- At some level, many therapies for lung disease strive to reverse these physical issues and enhance diffusive transport
Pressure, Content, and Transport
Simple enough: Pressure is the force over an area applied perpendicular to that area’s surface. Both gas diffusion and gas convection can exert pressure.

- Two useful laws for today’s discussion – One applies to gas phase only, both apply to gases dissolved in liquid
  - Dalton’s Law: The total pressure of a gas mixture is the sum of the partial pressures of its constituents:
    \[ P_{\text{total}} = \sum_i P_i \]
  - Henry’s Law: The partial pressure of a gas in equilibrium with a volume of liquid is proportional to the amount of gas dissolved in the liquid:
    \[ P = kc \]

where \( k \) is a constant for a particular gas-solvent-temperature combination and \( c \) is the concentration of the gas.

Beware: This is also often written as \( c = kP \), where the relationship is the same, but the value of \( k \) is the reciprocal of the one noted above.
Consequences and Caveats about Dalton’s and Henry’s Laws

- If total pressure is held constant (such as being held at atmospheric pressure), the partial pressure of one gas in a gas mixture or solution can only change if the partial pressures of one or more other gases change.

- Both Dalton’s and Henry’s Laws assume that the gases under discussion (either as gases or dissolved in liquid) are inert – they don’t interact with one another or chemically with the media which they’re in.
  - Both of these are not true in the case of oxygen and carbon dioxide.
Content: Dissolved and Hemoglobin-Bound Oxygen
Implications and Limitations of Henry’s Law

• Henry’s Law relates partial pressure of oxygen to concentration of dissolved oxygen in plasma as follows:

\[ [O_2] = 0.003 \times PO_2 \]

where \([O_2]\) is in ml/dl, \(PO_2\) is in mmHg and \(k\) is in ml/dl/mmHg, and physiological temperature (37°C) is assumed.

• For typical \(PO_2\) values (~100 mmHg or so), this is a very modest amount of dissolved oxygen and would require a tremendously high cardiac output to deliver enough oxygen per minute to the periphery.
The Only Way to Make Gas Transport Work in Large Creatures is with a Dedicated Oxygen Carrier

• Some useful facts about hemoglobin*
  – Concentration in blood is very high (~15 g/dl), constituting about a third of the mass of an erythrocyte, and about 15% of the mass of blood
  – Each gram of hemoglobin can bind about 1.34 ml/g of O_2 **
  – Always a tetramer, there are a variety of subunits that come to the fore at different points during development (including \( \alpha, \beta, \gamma, \delta, \epsilon, \) and \( \zeta \)). In large part, these provide different ‘tunings’ of the oxygen-hemoglobin dissociation curve
  – A number of physical features of the local environment also serve to tweak the loading and unloading of oxygen from hemoglobin
  – Hemoglobin is also an important carrier of carbon dioxide
    • carbaminohemoglobin

* By useful, I mean of course testable.
** Note: This number is convention – it’s certainly an overestimate of the actual figure
Important Facts of O$_2$ Binding by Hemoglobin

- Binding is allosterically cooperative
  - $O_2$ binding to any heme group changes the structure of the entire molecule
  - Each $O_2$ bound promotes binding of the next $O_2$
  - The result is a very steep Hgb-$O_2$ dissociation curve in the physiologically useful range
Relationship Between Oxygen Tension and Hemoglobin Saturation

- Under resting conditions, hemoglobin leaves about 25% of its oxygen in the periphery.

- With exercise, greater and greater amounts of oxygen are extracted, with progressively deoxygenated hemoglobin returning to the lungs.

- Acidosis and increased temperature* tend to move the curve to the right, facilitating better O₂ unloading.

* At heavy exercise, peripheral venous pH and temperature may reach 7.25 and 40°C.
Conditions Affecting Oxygen-Hemoglobin Dissociation Kinetics

• A shift to the right
  – For a given PO$_2$, hemoglobin will be less saturated
  – For a given drop from arterial PO$_2$, more oxygen will have been unloaded
  – Useful for unloading hemoglobin

• A shift to the left
  – For a given PO$_2$, hemoglobin will be more saturated
  – For a given drop from arterial PO$_2$, less oxygen will have been unloaded
  – Useful for loading hemoglobin

• Factors shifting to the right
  – High Temperature
  – High PCO$_2$
  – Low pH
  – 2,3 diphosphoglycerate
  – Adult (versus fetal) hemoglobin

• Factors shifting to the left
  – The inverse of those above, plus
  – Carbon monoxide
  – Methemoglobinemia
An Important Application of a Curve Shifted to the Left

- Maternal-fetal oxygen transfer
  - The affinity for oxygen of hemoglobin on the fetal side of the circulation must, at a given $\text{PO}_2$, be higher than the hemoglobin oxygen affinity on the maternal side
• Christian Bohr:
  – Acidosis decreases affinity of hemoglobin for $O_2$

• J. S. Haldane:
  – Low $P_O_2$ increases the affinity of hemoglobin for $CO_2$
Content and Transport: The link between the cardiovascular and respiratory systems
Oxygen Content and Transport

• Oxygen content is the total amount of oxygen within a volume of blood
  – Determined by concentration of hemoglobin, its extent of saturation, and the partial pressure of oxygen
  – Typically abbreviated as $C_xO_2$ (Arterial: $C_AO_2$  Venous: $C_VO_2$)
  – Expressed in volume/volume (usually, clunkily, in ml/dl)

$C = ([Hb] \times \text{Saturation} \times 1.34) + (0.003 \times PO_2)$

- Hemoglobin-Bound Oxygen
  - Hemoglobin: g/dl
  - Saturation: %/100
  - $C$: ml/g

- Dissolved Oxygen
  - $PO_2$: ml/dl/mmHg
Oxygen transport is the rate at which oxygen is being moved by the circulatory system from the lungs to the periphery
- Determined by oxygen content and cardiac output
- Typically referred to as oxygen delivery, $DO_2$
- Expressed in volume/minute or volume/minute/surface area

$$DO_2 = \text{Cardiac Output} \times C_AO_2$$

- $l/min$ or $l/min/m^2$
- $ml/dl$
Oxygen consumption is the rate at which oxygen is utilized by the periphery.
- Defined as the difference between the oxygen content in arterial blood and in mixed venous blood in the pulmonary artery.
- Expressed in volume/minute or volume/minute/surface area.

\[ VO_2 = \text{Cardiac Output} \times (C_{AO_2}-C_{VO_2}) \]

- \( l/min \) or \( l/min/m^2 \)
- \( ml/dl \)
Pressure, Content, and Transport

• Ultimately, respiratory failure is an issue of reduced oxygen transport.

• Clinical assessment of transport adequacy typically concentrates on pressure and content. Measuring blood flow requires more invasive methods than measuring hemoglobin saturation or the partial pressure of oxygen.

• Physiological compensation and medical therapy are directed against each element of the content and transport equations
  – Increased \( \text{PO}_2 \)
  – Increased hemoglobin concentration
  – Increased cardiac output
‘Nonrespiratory’ Situations May Have Large Impact on Oxygen and CO₂ Transport

• Anemia
  – Decreased oxygen content regardless of the extent of saturation

• Congestive heart failure
  – Decreased blood flow regardless of content

• Living at altitude or in artificial atmospheres
  – Low ambient oxygen tension
The Lung as a Gas Exchanger
The Human Lung as Gas Exchanger: System Requirements

• Oxygen uptake
  – Measured as volume of oxygen consumed per minute (VO$_2$)
  – At rest, VO$_2$ ~ 200 ml/min
  – At exercise, VO$_2$ ~ 3 l/min for 8 minute mile

• Carbon dioxide clearance
  – Commensurate with amount of oxygen consumed

• Extensive reserve
  – In health, for exercise
  – In illness, for ‘wiggle room’
The Human Lung as Gas Exchanger: System Requirements

• **The basic idea**
  – Bring deoxygenated blood and well-ventilated alveoli as close to one another as you can. This is called ventilation:perfusion matching
  
  – Ventilation is controlled ‘globally’ by CNS respiratory regulation in conjunction with the chest wall and diaphragm
  
  – Perfusion is controlled globally by regulation of cardiac output, and locally by hypoxic pulmonary vasoconstriction

• **How efficiency is lost**
  – Malfunctioning alveoli are not appropriately ventilated
  
  – Local blood flow regulation fails to re-route blood around hypoxic alveoli
  – Diffusion distance is increased
  – Diffusion surface area is reduced
Highest VO₂ Measured in Human:
VO₂: 6.61 l/min
Respiratory Rate: 62
Tidal Volume: 3.29 l

John Slade (Wikipedia)
CO$_2$ Handling in the Lung
Carbon Dioxide Handling in the Lung

- CO$_2$ is ~ 20x more soluble than O$_2$ in plasma
- CO$_2$ transfer is therefore much less susceptible than oxygen transfer to changes in disease-related loss of diffusion ability
- For the sake of discussion, if CO$_2$ rich venous blood gets to an alveolus, the PCO$_2$ in blood and gas will quickly equilibrate
Changes in arterial PCO2 are, practically speaking, a result of changes in CO2 production by tissues or changes in alveolar ventilation.
Functional Compartments in the Lung: Anatomic Dead Space

- Only a portion of the respiratory system participates in gas exchange (i.e., diffusion)
  - Respiratory Bronchioles -> Alveoli

- A portion of the system is only needed to move tidal breaths (i.e., convection)
  - Pharynx -> Bronchioles are ‘conducting airways’
  - ‘Anatomic dead space’
Functional Compartments in the Lung: Physiologic Dead Space

- Anatomic dead space, plus...
- Ventilated areas receiving no blood flow
Ventilation

• Ventilation is the movement of fresh gas from the environment down to the alveoli and, conversely, the movement of hypoxic, hypercarbic gas from the alveoli back to the environment.

• Typically categorized into 3 component rates, each described as volume per time, derived from 3 anatomic compartments in the lung:
  – Minute ventilation: Tidal volume x Breaths per minute
  – Dead space ventilation: Dead space x Breaths per minute
  – Alveolar ventilation: (Tidal volume – Dead space) x Breaths per minute
Why Dead Space is Important

• To move air requires power
  – (i.e., work over time)

• To meet oxygen delivery and CO₂ removal demands, a certain amount of fresh gas *must* be moved per minute

• As dead space increases, more air has to be moved to maintain the same alveolar ventilation

• Therefore, increased dead space means decreased efficiency and increased work of breathing
Conditions Associated with Increased Dead space

- Chronic obstructive disease
  - Obliteration of capillaries

- Pulmonary embolism
  - Occlusion of vessels to ventilated alveoli

- Endotracheal incubation
  - Length of tube beyond the lips represents additional ‘anatomic’ dead space
Conditions Associated with Changes in Minute Ventilation

• Hyperventilation
  – Compensation for hypoxia
  – Compensation for metabolic acidosis
  – Anxiety
  – Intoxication (e.g., salicylates, pretty uncommon)

• Hypoventilation
  – Obstructive Sleep Apnea
  – CNS and Peripheral Neuromuscular Disease
  – Intoxication
  – Airway Obstruction
  – COPD
Oxygen Handling
The Problem with Oxygen

• Transfer into the plasma from the alveoli by diffusion is less rapid
  – Oxygen is less soluble
  – Diffusion is more strongly impacted by distances and surface area

• A lot of oxygen must be transferred, so the time required to ‘load up’ becomes important
Significant Time is Needed to Fully Oxygenate Blood Entering the Lung
Diffusion of Gas from an Alveolus into Capillary Blood
Diffusion into capillaries: Impact of Short Transit Time
Impact of Supplemental Oxygen

- Supplemental oxygen increases the oxygen gradient from alveoli to capillaries

- Flux into capillary blood increases
  - Plasma and hemoglobin load more quickly
  - Blood can become fully oxygenated despite diffusion limitation

Source Undetermined
The Four Riders of the Apocalypse: The Causes of Hypoxia

• Hypoventilation
• Diffusion Block
• Shunt
• V/Q Mismatch
A Quick Diversion: Normal Partial Pressures of Various Gases in the Lung and Blood

- **Alveolar Values**
  - $\text{PH}_2\text{O}$ ~47 mmHg
  - $\text{PO}_2$ ~150 mmHg
  - $\text{PCO}_2$ ~35-45 mmHg

- **Arterial Values (at Rest)**
  - $\text{PO}_2$ ~90-100 mmHg
  - $\text{PCO}_2$ ~35-45

- **Mixed Venous Values (At Rest)**
  - $\text{PO}_2$ ~40 mmHg
  - $\text{PCO}_2$ ~45 mmHg
I. Hypoventilation

- Failure to bring fresh gas into the lung will decrease the arterial $pO_2$.

- Hypoventilation causes hypoxia by displacing alveolar $O_2$ with $CO_2$ -- the alveolar-capillary partial pressure gradient goes down, so diffusion is reduced.
II. Diffusion Block

• Direct impairment of gas transfer across the alveolar membrane

• Seen in any disease that lengthens the gas diffusion path
  – Fibrotic disease
  – Lung edema

• Or that significantly reduces surface area
  – COPD
III. Venous Admixture (Shunt)

\[ pO_2 = 100 \]

\[ pO_2 = 40 \]

\[ pO_2 = ? \]
Venous Admixture and Arterial Oxygen Tension

• Key fact #1: The higher the proportion of flow through the shunt to the total flow in the lung, the lower the PaO$_2$. 
Venous Admixture and Arterial Oxygen Tension

- Key Fact #2: Hypoxia caused by shunt cannot be overcome with supplemental oxygen. A portion of blood passing through the lung *never* encounters a ventilated alveolus.
More on Venous Admixture

- **Physiologic**
  - Bronchial veins -> drain to pulmonary vein
  - Thebesian veins -> drain to left ventricle

- **Pathologic**
  - Intracardiac, R->L shunts
  - Intrapulmonary AV malformations
  - Totally unventilated alveoli
    - e.g., Collapsed lobe due to obstructing endobronchial cancer
IV. Ventilation-Perfusion Inequality

- Pure dead space and pure shunt are not commonly seen clinically.
- Some ‘blend’ of these phenomena is the most common cause of hypoxia.
- A mix of dead space and shunt physiology.
- Requires thinking in a ‘multi-alveolar’ way.
Multiple Alveolus Model
Character of Pulmonary Venous Blood

The Dead Space Alveolus. In theory, no blood leaves this unit. As V/Q approaches $\infty$, in pulmonary venous blood:

$$\text{PaO}_2 \rightarrow (P_{\text{atm}} - P_{\text{H2O}}) \times \text{FiO}_2$$
$$\text{PaCO}_2 \rightarrow 0 \text{ mmHg}$$

The Ideal Alveolus. V/Q $\sim 1$. In pulmonary venous blood:

$$\text{PaO}_2 \rightarrow [(P_{\text{atm}} - P_{\text{H2O}}) \times \text{FiO}_2] - (\text{PCO}_2 / \text{RQ})$$
$$\text{PaCO}_2 = 40 \text{ mmHg}$$

The Shunt Alveolus. No fresh gas is delivered to blood. As V/Q approaches 0, in pulmonary venous blood:

$$\text{PaO}_2 \rightarrow \text{PvO}_2$$
$$\text{PaCO}_2 \rightarrow \text{PvCO}_2$$
Diseases Associated with V/Q Inequality

- Pretty much *everything* except pure shunt or pure hypoventilation will produce V/Q inequality
  - Pneumonia
  - Obstructive disease
  - Fibrotic disease
  - Pulmonary embolism
Diffusion Block
Hypo-Ventilation
Shunt
V/Q Inequality
Fibrosis

- Diffusion Block
- Hypo-Ventilation
- Shunt
- V/Q Inequality
COPD

Hypo-Ventilation

Diffusion Block

V/Q Inequality

Shunt

J. Younger
The 5th Cause: Low Inspired Oxygen Concentration

- Altitude
- Commercial air travel
  - Cabin pressurized to ~ 8,000 ft altitude
- Errors regarding supplemental oxygen
Thinking Clinically about Gas Exchange

• Clinically, quantifying gas exchange is used to evaluate the function of the lung

• If you know:
  – Gas partial pressures (tensions) in the alveoli
  – Gas tensions in the blood

• Then you can calculate the A-a gradient
  – This allows you to make some good guesses about:
    • Ventilation, perfusion, and diffusion phenomena across the alveolar membrane.

• In critical care and research settings, you can get fancier
  – $\text{PaO}_2/\text{FiO}_2$ ratio
  – Oxygenation Index
    • $(\text{Paw x FiO}_2) / \text{PaO}_2$
The Alveolar Gas Equation

• The basis for calculating the alveolar-arterial $O_2$ difference (A-a gradient)

• The A-a gradient is a really useful way to ask the general question: How effectively is oxygen brought into the lung making it into the bloodstream?

• Simply pregnant with testing possibilities
To Evaluate the Performance of the Lung as a Gas Exchanger, Begin with the Gas Tensions in The Alveoli

• Measuring alveolar gas concentrations is not simple and is usually not done except in pulmonary function or research laboratories

• Guessing about alveolar gas concentrations usually suffices

• The ‘guess’ that is used is the alveolar gas equation
Nitrogen
Oxygen
Water Vapor
Carbon Dioxide
Trace Gases
The Alveolar Gas Equation

Water Vapor: 47 mmHg

Everything else entering the lung: \((P_{atm} - P_{H2O})\)

Oxygen entering the lung: \((P_{atm} - P_{H2O}) \times FiO_2\)

Oxygen in the alveoli: \([ (P_{atm} - P_{H2O}) \times FiO_2 ] - PaCO_2/RQ\)

Carbon dioxide in the alveoli: Assumed equal to arterial PCO_2
The Respiratory Quotient

• The ratio of the amount of CO$_2$ produced to the amount of O$_2$ consumed

• Typical values range from 0.7 to 1.0

• Obviously dependent on relative consumption of carbohydrate, protein, and lipid substrates

• For many calculations, a value of 1.0 is reasonable

• Assumes metabolic equilibrium
  – Vigorous exercise is a great example of disequilibrium
  – During exercise beyond the anaerobic threshold, the value can take on values greater than 1.
The alveolar-arterial (A-a) oxygen gradient

- The difference between the oxygen partial pressure in alveoli and the pressure in blood
- Higher gradients mean worse performance
A-a Gradient and the 4 Causes of Hypoxia

- Hypoventilation
  - A-a Gradient should be normal
  - Hypoventilation simply increases the PCO$_2$ term in the alveolar gas equation

- A-a Gradient will not differentiate:
  - Diffusion block
  - V/Q inequality
  - Shunt
Figuring Out Which of the 4 Causes Affects a Given Patient

• History and Physical
  – i.e., the rest of the respiratory sequence
• Evaluate for obstructive physiology
• Test for diffusion abnormalities (DLCO)
• Intervene and see what happens
  – Supplemental oxygen
  – Augmented ventilation
    • Bronchodilators
    • Mechanical support
A Teaser for Next Week: Oxygen Delivery versus Mitochondrial Accessibility

• DO2 is a global, ‘gross’ measure of a patient’s ability to delivery oxygen to tissues

• Relying on DO2 clinically assumes that downstream vascular ‘plumbing’ is working appropriately
  – Regional vascular autoregulation

• Shock states (e.g., sepsis, trauma) are associated with dysfunctional autoregulation at the tissue level, making DO2 an imperfect parameter

• Sometime in our lifetime, practical tissue O2 sensors will be available to monitor oxygen delivery at the tissue or cellular level
Important Things to Walk Away With

• Differences between convection and diffusion
• Meaning of tension, saturation, and content and associated calculations
• Impact of alveolar ventilation and dead space on carbon dioxide handling
• The 4 primary causes of low arterial PO$_2$ and the physiology of each
• The alveolar gas and A-a gradient equations
Question to Ponder Tonight:

Which would most adversely affect arterial oxygenation?

1. An acute pneumonia completely involving the left lower lobe such that no gas exchange could occur
2. Surgically removing a normal left lower lobe
At altitude, some degree of hypoxia is a common occurrence. If someone were hypoxic at a given altitude, how could you tell whether or not there lungs were exchanging gas properly?
Question to Ponder Tonight

For each measurement, which would take longer to reach equilibrium following a change?

- Arterial oxygen content, following a sudden decrease of atmospheric FiO$_2$ from 0.21 to 0.16

- Arterial carbon dioxide tension, following a sudden decrease in minute ventilation by 15%?

- Hint: At this moment, which gas are you carrying around more of, oxygen or carbon dioxide? Where do you store those gases?
Some seals, walruses, and whales can stay submerged for extended periods of time (approaching an hour). How is this possible?
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