PHYSIOLOGY TEAM 435

9 Control of Breathing

- Very important
- Extra information
- Terms

Always bear in mind, that your own resolution to succeed is more important than anything.

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Objectives

- Understand the role of the medulla oblongata in determining the basic pattern of respiratory activity.
- List some factors that can modify the basic breathing pattern like e.g.
  A- The Hering-Breuer reflexes
  B- The proprioreceptor reflexes
  C- The protective reflexes, like the irritant, and the J-receptors.
- Understand the respiratory consequences of changing PO2 , PCO2 , and PH.
- Describe the locations and roles of the peripheral and central chemoreceptors.
- Compare and contrast metabolic and respiratory acidosis and metabolic and respiratory alkalosis.

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Controls of rate and depth of respiration

- **Arterial PO2**
  - When PO2 is VERY low (Hypoxia), ventilation increases to give more O2.

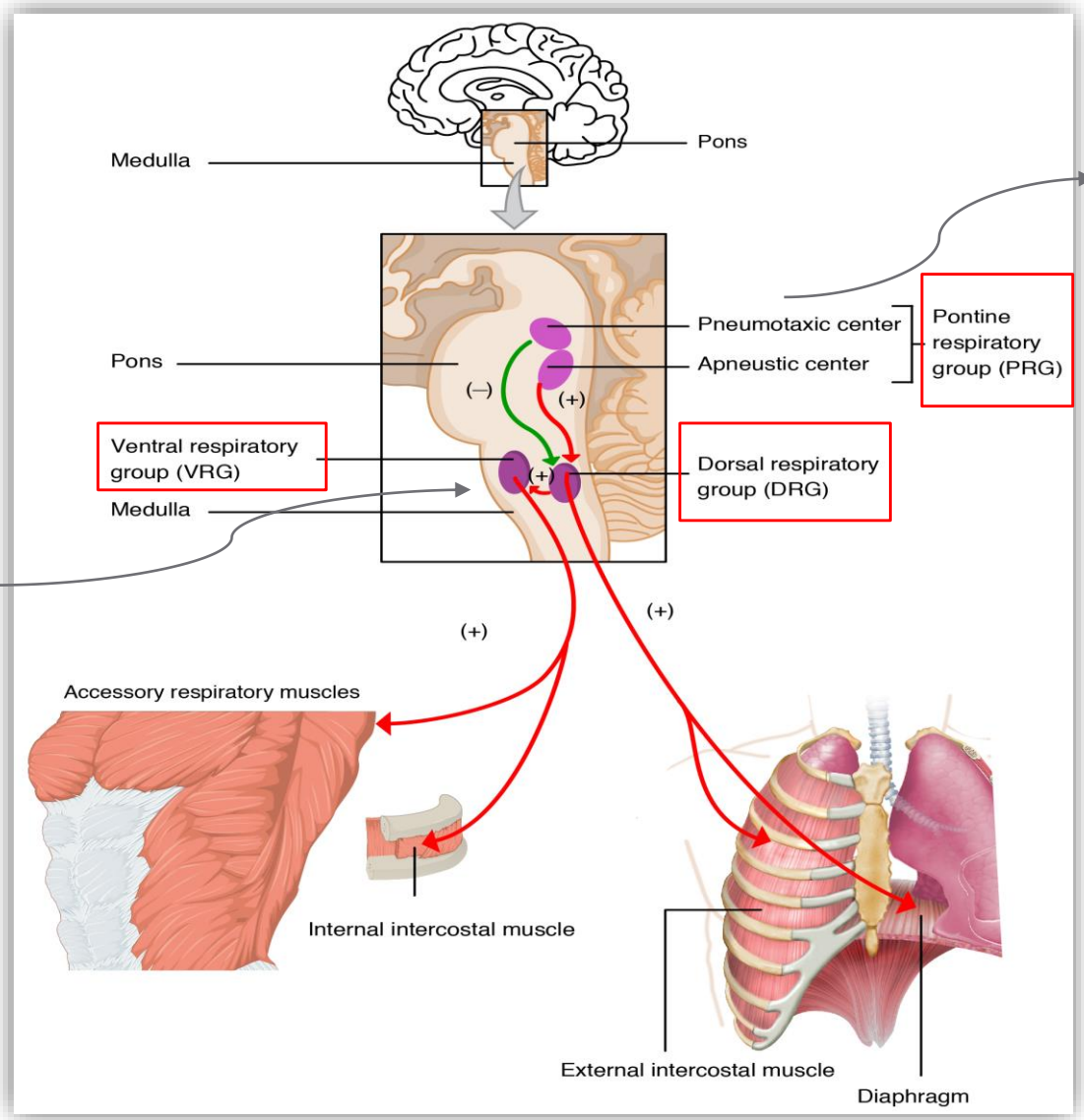
- **Arterial PCO2**
  - Small increases in PCO2, greatly increases ventilation to excrete more CO2.

- **Arterial pH**
  - As hydrogen ions increase (acidosis), alveolar ventilation increases to maintain pH "7.41".

"The most important regulator of ventilation is PCO2"

Arterial PO2	Decrease O2	"Hypoxia"	**Increase Ventilation**
Arterial PCO2	Increase CO2	"Hypercapnea"	
Arterial PH	Increase H ions	"Acidosis"	

Respiratory Centres



- Pneumotaxic centre (-) Inhibition(DRG)
- Apneustic centre (+) Stimulation(DRG)

(DRG) (+) Stimulation of (VRG)

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Medullary Respiratory centres

The respiratory centre is composed of several groups of neurons located bilaterally in the medulla oblongata and pons of the brain stem. It is divided into three groups:

(1) Dorsal respiratory group.

located in the dorsal portion of the medulla, which mainly causes inspiration.

(2) Ventral respiratory group.

located in the ventrolateral part of the medulla, which mainly causes expiration.

(3) Pontine respiratory group (Pneumotaxic & Apneustic centres)

located dorsally in the superior portion of the pons, which mainly controls rate and depth of breathing.

- Smooth Respiratory Rhythmic-

Inspiratory area (Dorsal Respiratory Group) DRG	Expiratory area (Ventral Respiratory Group) VRG
-Determines basic rhythm of breathing. -Causes contraction of diaphragm and external intercostals.	-Inactive during normal quiet breathing. -Activated by inspiratory area (DRG) during forceful breathing. -Causes contraction of internal intercostals and abdominal muscles.

- The medullary respiratory center stimulates basic inspiration for about 2 seconds and then basic expiration for about 3 seconds (5sec/ breath = 12 breaths/min).

Pontine Respiratory centres

Its function is transition between inhalation and exhalation.

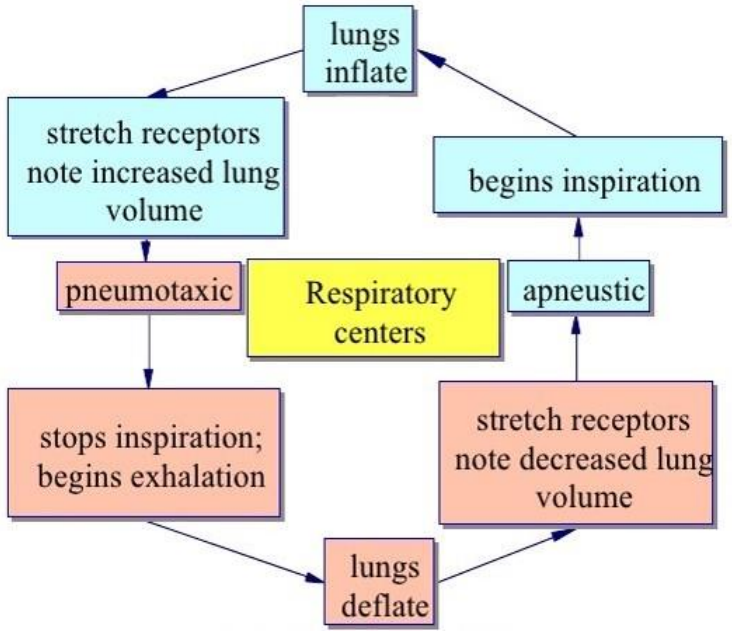
And it's divided into 2 areas :

1. Pneumotaxic area

- Inhibits inspiratory area of medulla to stop inhalation.
- Breathing is more rapid when pneumotaxic area is active.
- increasing the rate of breathing because limitation of inspiration also shortens expiration and the entire period of each respiration.
- Strong pneumotaxic signal can increase the rate of breathing to 30 to 40 breaths per minute, whereas a weak pneumotaxic signal may reduce the rate to only 3 to 5 breaths per minute.

2. Apneustic area

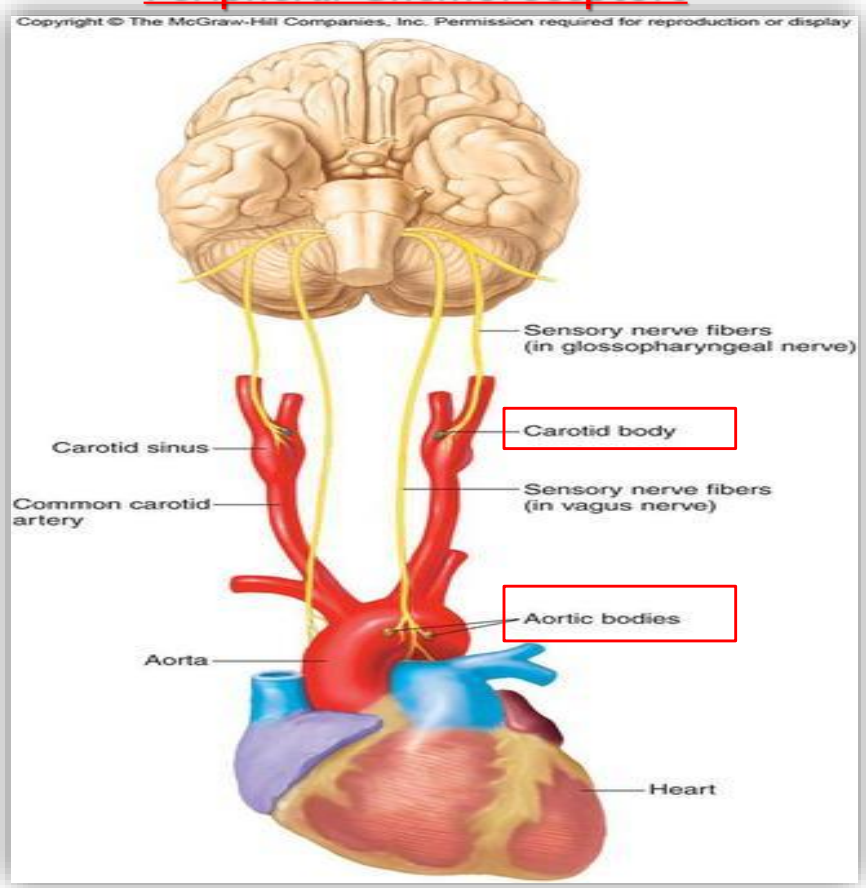
- Stimulates inspiratory area (DRG) of medulla to prolong inhalation.
- It is inhibited by pulmonary stretch receptors.



Pneumotaxic area (Active)	Apneustic area (Active)
Low inspiration (1s)	High inspiration (4s)
Low expiration (2s)	High expiration (5s)
Short time for one breath. (3s/breath)	Long time for one breath. (9s/Breath)
Normal is (5sec/breath)	
Rapid breathing	Slower breathing.

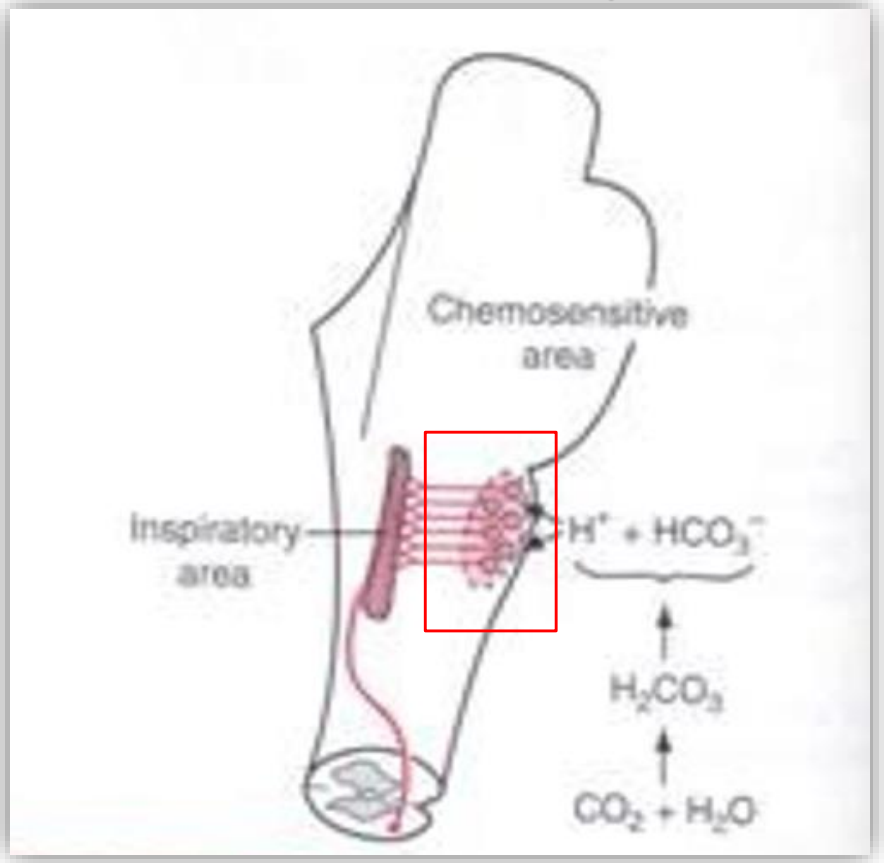
Chemoreceptor Control of Breathing

Peripheral Chemoreceptors



Most of the chemoreceptors are in the carotid bodies. However, a few are also in the aortic bodies, and a very few are located elsewhere in association with other arteries of the thoracic and abdominal regions.

Central chemoreceptors



Are found on the medulla oblongata.

Chemoreceptor Control of Breathing

➢ **Chemoreceptors represent Chemical Control of Respiration.**

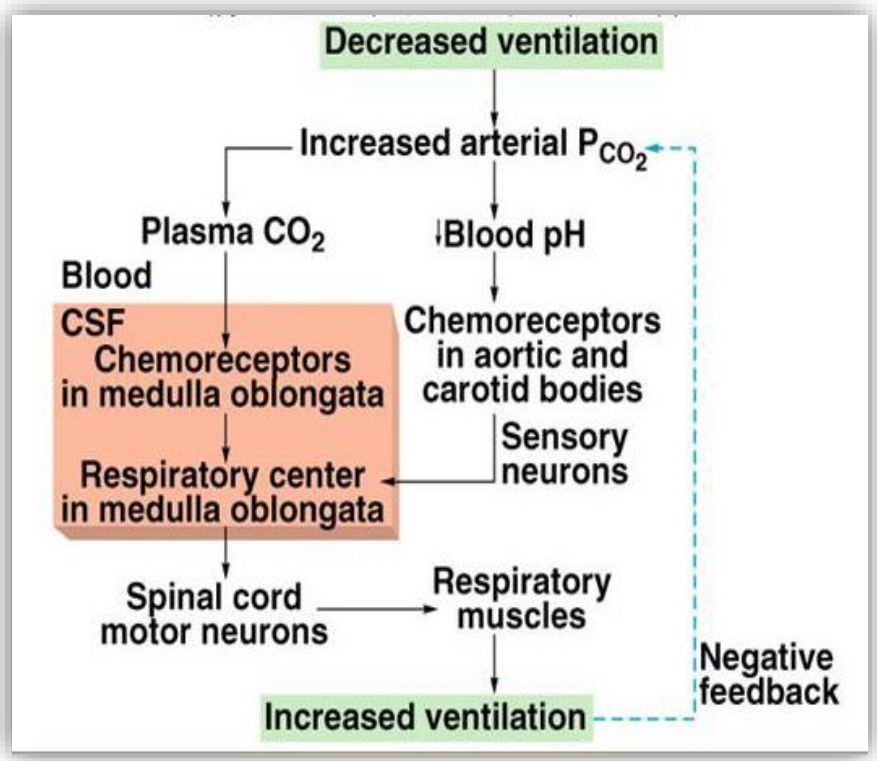
- O2 does not have a significant direct effect on the respiratory center of the brain in controlling respiration. It acts on peripheral chemoreceptors located in the carotid and aortic bodies.

- CO2 have a significant direct effect.

Extra explanation

O2	CO2
Acts : on peripheral chemoreceptors only.	Acts : on Both central and peripheral chemoreceptors.
Stimulate only in "Severe decrease of O2"	Stimulate in any mild change in the CO2 level.
Weak regulator of ventilation.	Strong regulator of ventilation.

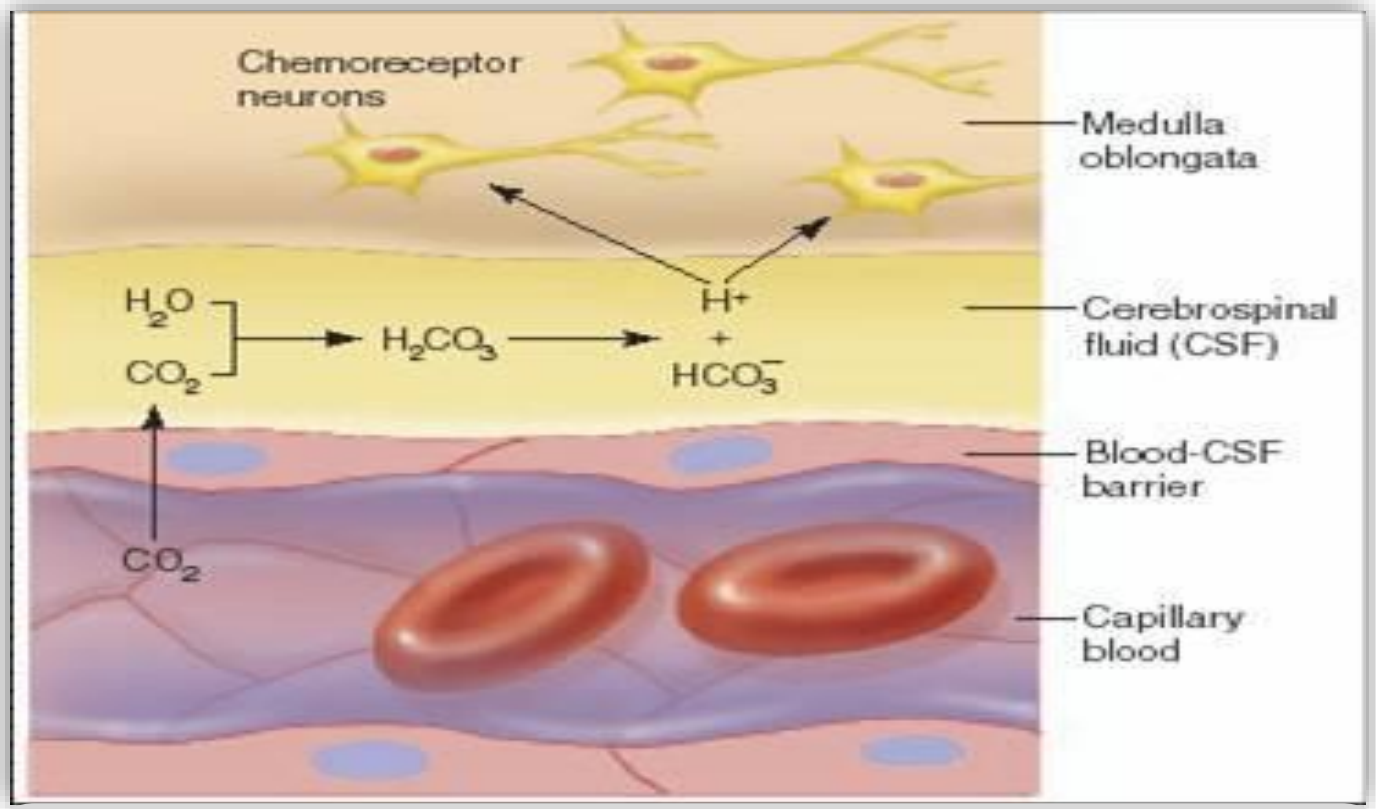
Role of CO2 Mechanism :



Effect of blood CO_2 level on central chemoreceptors

1. **Increase blood CO2 → stimulate breathing indirectly by decrease pH of two things: blood and CSF.**
2. **Stimulate chemoreceptor neurons in the medulla oblongata.**

- **Decrease pH = Increase H^+ concentration.**



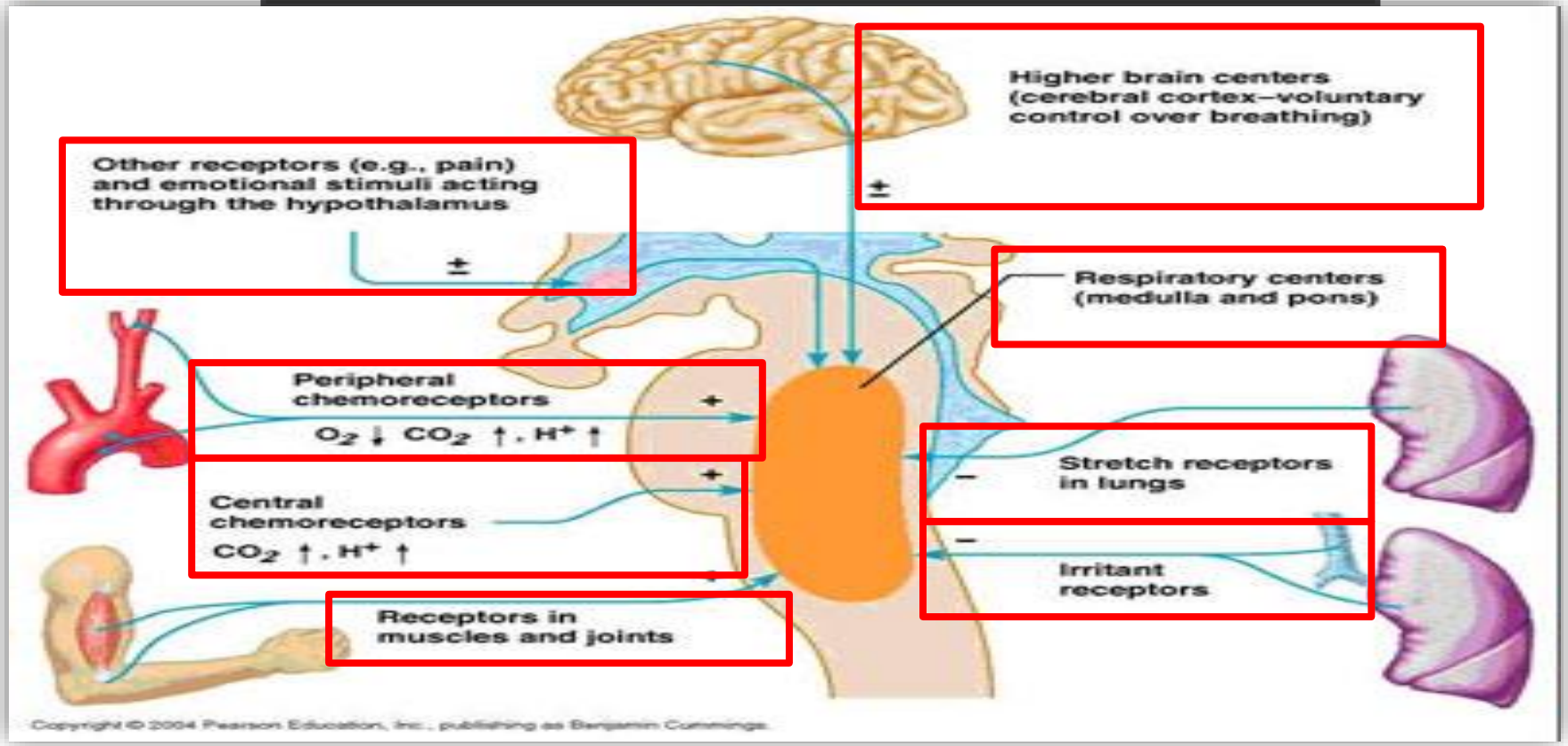
- **High H+ in cerebrospinal fluid** - **High CO2 in Blood**	Direct central response of H+ ,because it's available near the receptors
- High H+ in Blood. - High CO2 in Blood.	- **Peripheral** response of H+ - **Central** response of CO2

- الMedulla لاتستطيع التعرف على الCO2 بشكل مباشر فقط بشكل غير مباشر .. الCO2 له خاصية التحول إلى أيونات الهيدروجين. إذا زاد CO2 في الدم يدخل BBB وذلك لأن ذائبيته عالية.. ثم يتحد مع الماء في الMedulla وبالتالي يتحول إلى كربونيك أسيد وهيدروجين. وزيادة الهيدروجين تحفز المستقبلات في الMedulla لأنها تستطيع التعرف عليها فتستجيب لها.
- **Okay, why we don't say "H ions is the more regulator instead of CO2"?**

Because the H+ is poorly diffusible, so, it can't cross BBB and will not stimulate the central chemoreceptor in medulla. But, CO2 is more diffusible. That's why we said the CO2 is "Strong regulator".

يعني حتى لو زادت أيونات الهيدروجين في الدم مارح تقدر تعطيني Central action فقط تعطي Peripheral action

Factors Influencing Respiration



Some factors that can alter respiratory rates:

1. Pain and emotions.
2. Irritating physical or chemical stimuli in the respiratory tract.
3. Overstretch of the lungs -- activates lung stretch receptors which act on brainstem centers to cause exhalation and prevent inhalation (the Hering-Breur reflex) "Next slide"
4. A decrease in BP can result in an increase in respiration rate/depth
5. Changes in plasma and CSF PCO2, PO2, and PH.

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Hering-Breuer inflation reflex

- When the lung becomes overstretched (tidal volume is 1 L or more) – Normal is 500ml-
- Stretch receptors located in the wall bronchi and bronchioles transmit signals through vagus nerve to DRG producing effect similar to pneumotaxic center stimulation.
- Switches off inspiratory signals and thus stops further inspiration.
- This reflex also increase the rate of respiration as does the pneumotaxic center.

❑ DRG = Dorsal Respiratory Group of neurons.

Effect of Irritant receptors in the airways

- Irritant receptors supply the epithelium of trachea, bronchi and bronchioles.
- Stimulated by irritants that enter respiratory airways.
- Causing: Sneezing, coughing, bronchoconstriction, bronchial asthma & emphysema.

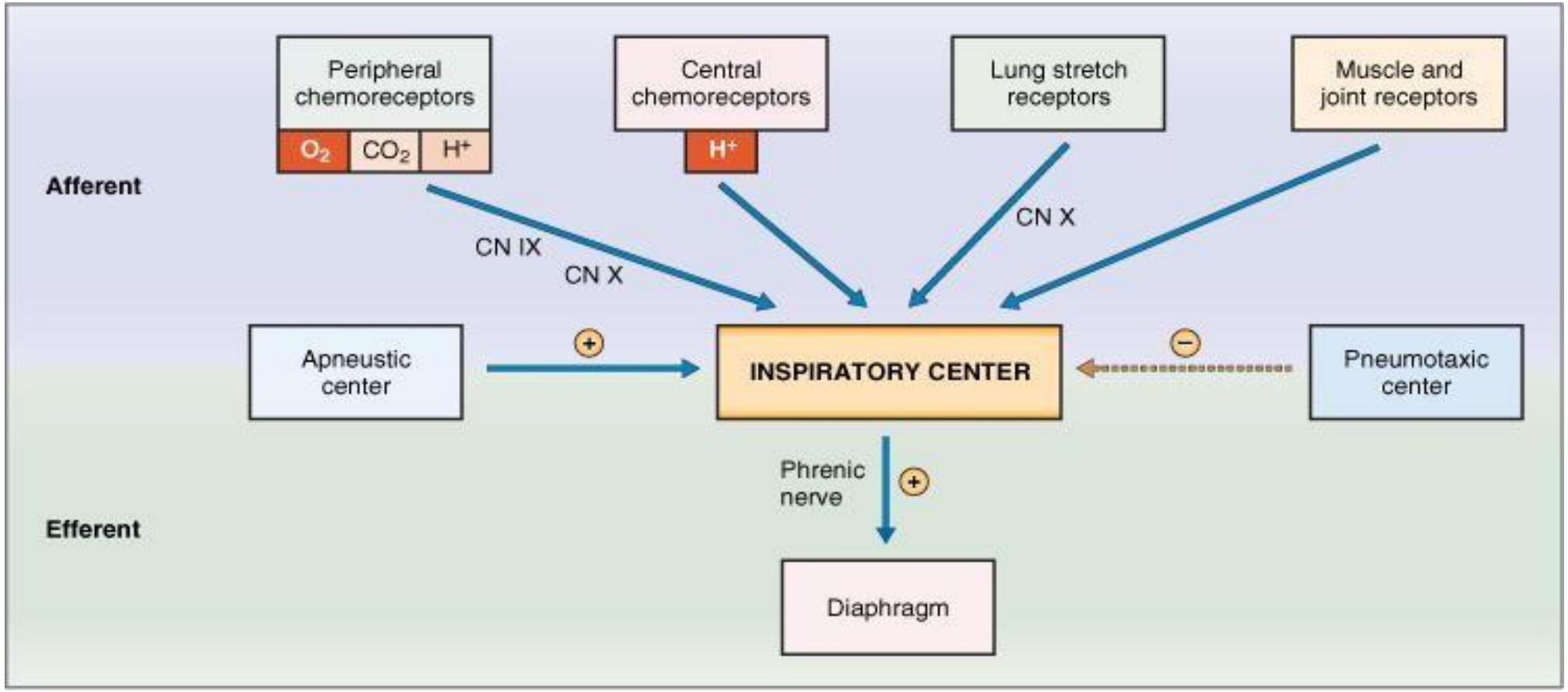
Function of lung J receptors.

- Located in the wall of the alveoli in juxta position to pulmonary capillaries.
- Stimulated by engorged pulmonary capillaries by blood or pulmonary edema (CHF).
- Causing: feeling of dyspnea.

Juxta = (placed close together)
CHF = (Congestive heart failure)

Factors Influencing Respiration

❖ **Other illustration to understand the factors that can alter respiratory rates.**



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- **Afferent : from Receptors to CNS.**
- **Efferent : from CNS to Effectors (Mainly diaphragm to descent or ascent)**

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Acidosis *Vs* Alkalosis

Respiratory acidosis.	Respiratory alkalosis.
Hypoventilation. Accumulation of CO2 in the tissues. PCO2 increases – H+ Increase pH decreases.	Hyperventilation. Excessive loss of CO2 . PCO2 decreases (↓ 35 mmHg). – H+ decrease pH increases.

Metabolic acidosis.	Metabolic alkalosis.
- Ingestion, infusion, or production of a fixed acid. - Decreased renal excretion of hydrogen ions. - Loss of bicarbonate or other bases from the extracellular compartment. - pH decreases.	- Excessive loss of fixed acids from the body - Ingestion, infusion, or excessive renal reabsorption of bases such as bicarbonate. - pH increases.

- **The respiratory system can compensate for (يُعوض) metabolic acidosis or alkalosis by altering alveolar ventilation.**
- **Metabolic acidosis > Hyperventilation.**
- **Metabolic alkalosis > Hypoventilation.**

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10

Low and high altitude

- Very important
- Extra information
- Terms

(وَبَشِّرِ الصَّابِرِينَ)

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Objectives

By the end of this lecture you should be able to:

1. Describe the effects of exposure to low and high barometric pressures on the body.
2. Describe the body acclimatization to low barometric pressure.
3. Define decompression sickness and explain how it can be avoided.
4. Understand the effects of high nitrogen pressure, and nitrogen narcosis.

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EFFECT OF INCREASED BAROMETRIC PRESSURE (DEEP SEA DIVING)

1 When humans descend below the sea, the pressure around them increased.

2 To prevent the lungs from collapsing, the air must be supplied also under high pressure.

3 This exposes the blood in the lungs to extremely high alveolar gas pressure (hyperbarism)

4 Under certain limits these high pressures cause tremendous alterations in the physiology of the body.

5 The surrounding pressure increases by 1 atmosphere for every 10 meter (33 feet) of depth in sea water.

6 Therefore at a depth of 31 meter (100 feet) in the ocean the diver is exposed to a pressure of 4 atmospheres.

(3atm for 31 meter + 1Atm in air = 4atm)

7 These problems confront SCUBA (Self Contained Under water Breathing Apparatus)

- **Hyperbarism :** a condition due to exposure to ambient gas pressure or atmospheric pressures exceeding the pressure within the body.

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DEEP SEA DIVING (HIGH PRESSURE)

Relationship of pressure to sea depth:

+ 33 feet
+ 1atm

A person at 33 feet is exposed to 2atm pressure

+ 33 feet
+ 1atm

At 66 feet, the pressure is 3atm

And so on..

- كل مازاد العمق كل مازاد الضغط.
لأنه تحت الماء سيكون الضغط عبارة عن مجموع ضغط الهواء الجوي + ضغط الماء فوق الغواص.

EFFECTS OF DEPTH

ON THE VOLUME OF THE GASES:

is the compression of gases to smaller and smaller volumes.

1L (sea level)→1/2 L at 33 feet and so on.

- كل مازاد الضغط على الغاز كل ماقلت المساحات بين جزيئات هذا الغاز بالتالي ممكن يتحول من غاز إلى سائل -

ON DENSITY OF GASES:

increase in density of gas and hence increased work of breathing.

كل مازاد العمق كل مازادت لزوجة الغاز نظراً لقلة حجمه وتحوله من حالة لأخرى بالتالي يزيد من مقاومة جدران الأوعية التنفسية ويزيد معدل التنفس تبعاً لذلك.

HIGH NITROGEN PRESSURE

Has 2 effects:

- Nitrogen narcosis "anesthetic effect"
- Decompression thickness.

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Nitrogen effect at high nitrogen pressure

A. NITROGEN NARCOSIS[1] AT HIGH NITROGEN PRESSURE:

- About four fifths of the air is nitrogen.
- At sea-level pressure, the nitrogen has no significant effect on bodily function but at high pressures it can cause varying degrees of narcosis.

HOW?

1. Nitrogen like most other anesthetic gases, dissolve freely in the fats of the body including the membranes and other lipid structures of the neurons.
2. This lead to alteration of the electrical conductance of the membranes, reduces their excitability and subsequent necrosis develops.

Nitrogen narcosis has characteristics similar to those of alcohol intoxication:

- **At 120 feet:** the diver lose many of his cares.
- **At 150 feet:** there is a feeling of euphoria (happiness) + drowsiness and impaired performance.
- **At higher pressure:** loss of coordination and finally coma might develop.

[1] **Narcosis, not necrosis.** It means anesthetic and has an effect similar to that produced by alcohol or drugs.

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Nitrogen effect at high nitrogen pressure Cont…

B. DECOMPRESSION SICKNESS (CASSION'S DISEASE):

It is a syndrome caused by a decrease in the ambient (surrounding) pressure which occur in animal and men when the tissues of the body contain an excess of physically inert gas.

A. **During descent,** the high partial pressure of nitrogen (encountered when breathing compressed air at depth) forces the nitrogen to absorbed by fat (it has a high N2 solubility).

B. **On ascending,** due to higher partial pressure of N2 in the fat than in the blood, the faster the ascent the less time there is for absorbed gas to be offloaded safely through the lungs, causing these gases to come out of solution (gaseous phase) and form "micro bubbles" in the blood.

RAPID

RAPID DECOMPRESSION:
bubbles of gaseous nitrogen are released in tissues and blood, causing the symptoms of decompression sickness (the bends or caisson disease)

SLOW ASCENT:
N2 is slowly removed from the tissues since the partial pressure there is higher than that in the arterial blood and alveolar gas.

SLOW

When a person breathes air under high pressure for a long time, the amount of nitrogen dissolved in the body fluids increases. The reason for this is that blood owing through the pulmonary capillaries becomes saturated with nitrogen to the same high pressure as that in the alveolar breathing mixture, and over several more hours, enough nitrogen is carried to all the tissues of the body to raise their tissue nitrogen partial pressure to equal the nitrogen pressure in the breathing air.

Because **nitrogen is not metabolized by the body**, it remains dissolved in all the body tissues until the nitrogen pressure in the lungs is decreased back to some lower level, at which time the nitrogen can be removed by the reverse respiratory process; however, this removal often takes hours to occur and is the source of multiple problems, collectively called *decompression sickness*.

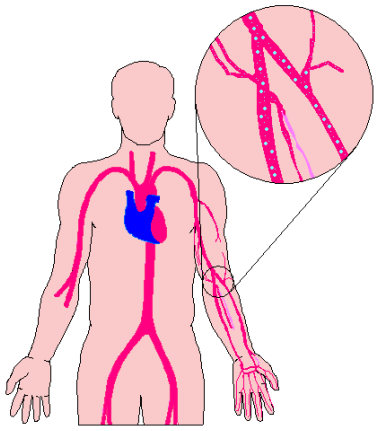
If a diver has been beneath the sea long enough that large amounts of nitrogen have dissolved in his or her body and the diver then suddenly comes back to the surface of the sea, significant quantities of nitrogen bubbles can develop in the body fluids either intracellularly or extracellularly and can cause minor or serious damage in almost any area of the body, depending on the number and sizes of bubbles formed. **The bubbles may not appear for many minutes to hours because sometimes the gases can remain dissolved in the "supersaturated" state for hours before bubbling**

Study smart .. Read it!

Nitrogen effect at high nitrogen pressure Cont…

A. The mildest form of DS* is fatigue or drowsiness and local skin itch.

B. Other sever symptoms may include:



SIGNS AND SYMPTOMS OF DECOMPRESSION SICKNESS

Musculoskeletal	sever pain particulaty around the joints due to trapped bubbles in the tissues.
Skin	Iching usually around the face and neck and arms, and Edema may be prominent.
Neurologic	Parasthesia, Paralysis, inner ear disturbance causing Dizziness and vertigo (could also cause deafness)
Pulmonary	thoracic pain ,Dry cough, Dyspnea, substernal pain, cyanosis (Pulmonary edema > hypoxia > death)
Vascular	Bubbles in the coronary arteries may cause myocardial Damage

Decompression sickness Shock:

Capillaries become permeable to plasma and hypovolemia (Decrease of blood plasma volume) rapidly develop and usually complicated by pulmonary edema.

DS = Decompression sickness

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Nitrogen effect at high nitrogen pressure Cont…

❑ TREATMENT OF DECOMPRESSION SYMPTOMS:

Rapid recompression in a pressure chamber is applied, followed by slower decompression.
This reduces the volume of nitrogen bubbles and force them back to the solution.

Put the diver into a pressurized tank to lower the pressure gradually back to normal atmospheric pressure, by using this time schedule.

- 10 minutes at 50 feet depth
- 17 minutes at 40 feet depth
- 19 minutes at 30 feet depth
- 50 minutes at 20 feet depth
- 84 minutes at 10 feet depth

❑ PREVENTION OF DECOMPRESSION SICKNESS:

Helium-O2 mixture is more desirable than Nitrogen-O2 Mixture in deep dives.

BECAUSE:

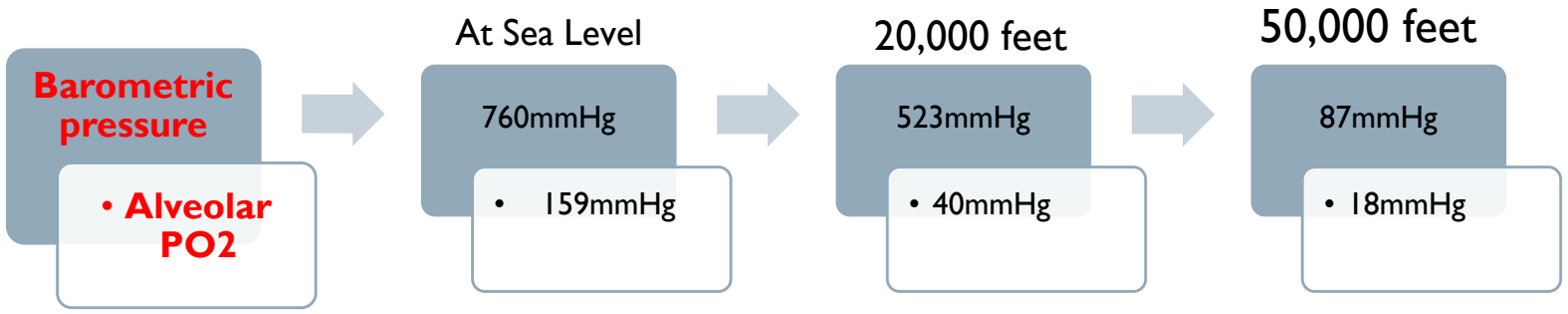
- 1\4 – 1\5 the narcotic effect of Nitrogen on CNS.
- 1\7 the molecular weight of nitrogen.
- Less airway resistance due to it's low density.
- High diffusion through tissues.
- 1\2 solubility of Nitrogen in body fluid, so that's reduces the quantity
- of bubbles formed in tissues.

We can prevent N2 toxicity but not O2 toxicity > free radicals > for this reason we cannot give a patient O2 directly w/o being thinned with another gas ex, **Helium.**

BOYS SLIDE

Effects of low oxygen pressure on the body (aviation ascend to high altitude)

- At the sea level the barometric pressure is 760mmHg, and it decreases as we go higher.
- As the barometric pressure decreases, PO2 (oxygen partial pressure) decreases proportionally to reach even less than 21% of the total:



This decrease in barometric pressure is the basic cause of all the hypoxia problems in high altitude in physiology.

- Even at high altitude, CO2 is continuously excreted from the pulmonary blood to the alveoli + water vaporizes into the inspired air from the respiratory surfaces (participate in low PO2).

Therefore, these two gases dilute oxygen and reduce its concentration in the alveoli which lead to Hypoxia.

Effects of low oxygen pressure on the body (cont.)

- **EFFECTS OF ACUTE HYPOXIA:**

Some of the important acute effects of hypoxia begin at an altitude of approximately 12,000 feet, and worsen with height.

At 12,000 feet

Acute effects:
E.g.:
- Drowsiness
- Lassitude.
- mental and muscle fatigue.
- Headache.
- Occasionally.
- nausea and sometimes euphoria

At 18,000 feet

Acute symptoms progress to a stage of twitching or convulsions

–Spontaneously contraction-

Above 23,000 feet

the un acclimatized person can enter into coma.

Acclimatized=متأقلم

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Effects of low oxygen pressure on the body (cont.)

ACCLIMATIZATION TO LOW PO2:

- A person remaining at high altitudes for days , weeks or years becomes more and more acclimatized to low PO2.

- So that it causes fewer deleterious effects on the body and it becomes possible for the person to work harder without hypoxic effects or to ascend to still higher altitude.

PRINCIPLE MEANS OF ACCLIMATIZATION:

1- Increase in pulmonary ventilation.

2- Increased red blood cells. (stimulation of erythropoietin secretion)

3-Increased diffusing capacity of the lungs. (Like what we do during exercise)

4- Increased vascularity of the tissues. (By doing new vacuolization)

5-Increased ability of the cells to utilize oxygen despite the low PO2. (by increasing the enzymes that helps the tissue utilize the oxygen)

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Principle means of acclimatization Summary & Revision.

1-A great increase in pulmonary ventilation:

low Po2 stimulates the arterial chemoreceptors, and this increases alveolar ventilation to a maximum of about 1.65 times normal then lead to reducing the Pco2 and increasing the pH of the body fluids - inhibit the brain stem respiratory center -oppose the effect of low Po2 - after 2 to 5 days inhibition fades away.

2- Increase in Red Blood Cells and Hemoglobin Concentration:

exposed to low oxygen for weeks at a time, the hematocrit rises slowly from a normal value of 40 to 45 to an average of about 60, with an average increase in whole blood hemoglobin concentration from normal of 15 g/dl to about 20 g/dl the blood volume also increases, often by 20 to 30 percent.

3- Increased diffusing capacity of the lungs:

normal diffusing capacity of oxygen through the pulmonary membrane is about 21 ml/mm Hg/min at high altitude increased similar to exercise performance--increased pulmonary capillary blood volume --expands the capillaries and increases the surface area of oxygen can diffuse into the blood. Another part results from an increase in lung air volume, which expands the surface area of the alveolar-capillary . A final part results from an increase in pulmonary arterial blood pressure; this forces blood into greater numbers of alveolar capillaries.

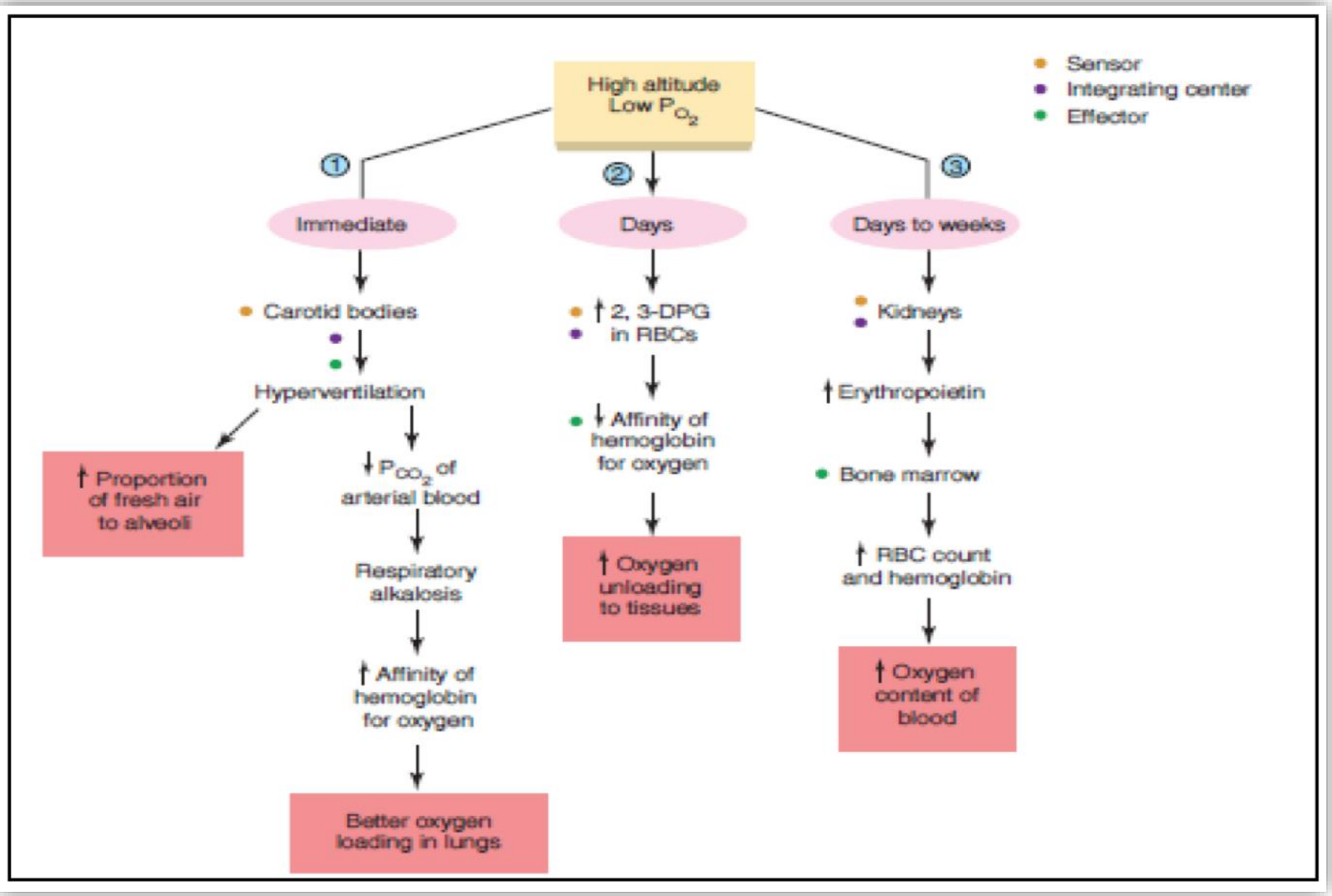
4- Increased vascularity of the peripheral tissues:

cardiac output increases as much as 30 percent growth of increased numbers of systemic circulatory capillaries (or angiogenesis)

5- Increased ability of the tissue cells to use oxygen despite low Po2:

In animals native to altitudes of 13,000 to 17,000 feet, cell mitochondria and cellular oxidative enzyme systems are slightly more plentiful than in sea-level inhabitants.

BOYS NOTES


Sensor
Integrating center
Effector
High altitude
Low P_{O_2}
①
②
③
Immediate
Days
Days to weeks
Carotid bodies
↑ 2, 3-DPG in RBCs
Kidneys
Hyperventilation
↓ Affinity of hemoglobin for oxygen
↑ Erythropoietin
↑ Proportion of fresh air to alveoli
↓ P_{CO_2} of arterial blood
↑ Oxygen unloading to tissues
Bone marrow
Respiratory alkalosis
↑ RBC count and hemoglobin
↑ Affinity of hemoglobin for oxygen
↑ Oxygen content of blood
Better oxygen loading in lungs

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Videos

1- Control of Breathing.
2- Respiratory centers.

Quiz L9

https://www.onlinequizcreator.com/l8-control-of-breathing/quiz-150181

MCQ's L10

Q1: what's make the partial pressure increased under the sea?
1- Temperature
2- Water weight
3- Deficiency of air
4- Depth

Q2: the density of the gases decrease under high pressure?
1- True
2- False

Q3: what is the depth that the diver starts feeling of euphoria?
1- 3.5 feet
2- 120 feet
3- 150 feet
4- More than 150 feet

Q4: rapid changes from high to normal pressure cause Decompression sickness?
1- True
1- False

Q1: 2
Q2: 2
Q3: 3
Q4: 1

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MCQ's L10

Q5: the cause of bubbles in the tissue and blood is?
1- Hilum & O2 mixture
2- Nitrogen solvent
3- CO2
4- Water

Q6: these are Decompression sickness symptoms & signs, except?
1- Paresthesia
2- Inner ear disturbances
3- Swollen
4- Paralysis

Q7: hilum has less density under high pressure comparing with nitrogen?
1- True
2-False

Q8: stay for few days in gradual high altitudes, called?
1- Ventilation
2- Acclimatization
3- Utilization
4- Hyperventilation

Q9: increase 2,3-DPG in RBCs is the first step of facing low PO2?
1- True
2- False

Q10: what is the stage which stopped "hyperventilation"?
1- Carotid bodies
2- Erythropoietin
3- Respiratory alkalosis
4- Bicarbonate formation

Q10: 3
Q9: 2
Q8: 2
Q7: 1
Q6: 3
Q5: 2

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MCQ's L10

Q11: decrease Hb binding with O2, will increase O2 diffusion through the tissue?

1- True
2- False

Q12: to stop formation of respiratory alkalosis, are kidneys needed to increase?

1- Erythropoietin
2- 2,3-DPG in RBCs
3- Bicarbonate
4- Carotid bodies

SAQ's L10

Case: A 27 old military diver was admitted to the nearest clinic to the beach, with skin itching, inner ear disturbances, sever pain around the joint, dyspnea and cough.

Q1: with no tests done, what would be your diagnosis?

Decompression sickness "rapid ascent".

Q2: what is the best treatment for him?

Rapid recompression followed by slower decompression.

Q: knowing that the diver will face high-depth, let you replace nitrogen with hilum in the oxygen tube, why?

1- 1/5 narcotic comparing with nitrogen.
2- Less molecular weight which mean less density which lead to less resistance.
3- High diffusion through tissue which mean as easy to enter the tissue as easy to exit it.
4- Less soluble by ½ in body fluids which mean a little amount only will dissolve in the fat, so, when the pressure returns to normal the bubbles number won't be high "the body can control it".

Q11: 1
Q12: 1

PHYSIOLOGY TEAM 435

Physiology Team

Leaders:

- Omar AlOtaibi
- Samar AlOtaibi

Girls members:

- Khawla Alammari
- Sara Alenezy
- Nouf Alrushaid
- Nouf Alabdulkarim
- Shadn Alomran
- Reem Alageel
- Nurah Alqahtani
- Malak Alsharif
- Ghaida Aljamili
- Monirah Alsaloli
- Lojain Alsiwat

Boys members:

- Rawaf Alrawaf
- Abdulaziz Alghanaym
- Abdulrahman Albarakah
- Abdullah Aljaafar
- Adel Alshihri
- Abdulmajeed Alotaibi
- Khalil Alduraibi
- Hassan Albeladi
- Omar Alshehri
- Abdulrahman Thekry
- Abdulaziz Alhammad

THANK YOU FOR CHECKING OUR WORK

For any correction, suggestion or any useful information, please contact us: Physiology435@gmail.com