وَقُل رَّبِّ زِدْنِي عِلْمًا

RESPIRATORY SYSTEM

HAYAT BATCH

SUBJECT : Physiology

LEC NO. : Lecture 9

DONE BY : Zeyad tareq

Control of Ventilation
(Regulation of Respiration)

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Lecture objectives:

1. Describe the general organization of the respiratory control system.
2. Identify the regions in the central nervous system that play important roles in the generation and control of cyclic breathing.
3. Describe the central and peripheral chemoreceptors and their role in the control of ventilation.
4. List the anatomical locations of chemoreceptors sensitive to changes in arterial PO_2, PCO_2, and pH that participate in the control of ventilation.
5. Describe the effects of alterations in body oxygen, carbon dioxide, and hydrogen ion levels on the control of breathing and understand the integrated responses to CO_2, hypoxia, pH and identify the relative importance of each in sensing alterations in blood gasses.
6. List the cardiopulmonary and other reflexes that influence the breathing pattern.
7. State the ability of the brain cortex to override the normal pattern of inspiration and expiration temporarily.
8. Discuss the regulation of ventilation in chronic respiratory failure.

قبل ما نبدا المحاضرة الدكتور حكى انو هاي المحاضرة عليه كمية اسالة اكثر عالفاينل

بهاي المحاضرة رح نعرف كيف يتم تنظيم عملية التنفس من hyperventilation and hypoventilation وكيف نحافظ على PO2 and PCO2 بالشرايين

كلو يتم بمركز بالدماغ اسمو respiration center فرح نتعرف ان شاء الله عليه ومن ايش يتكون

The Respiratory Center

*Ventral → Anterior

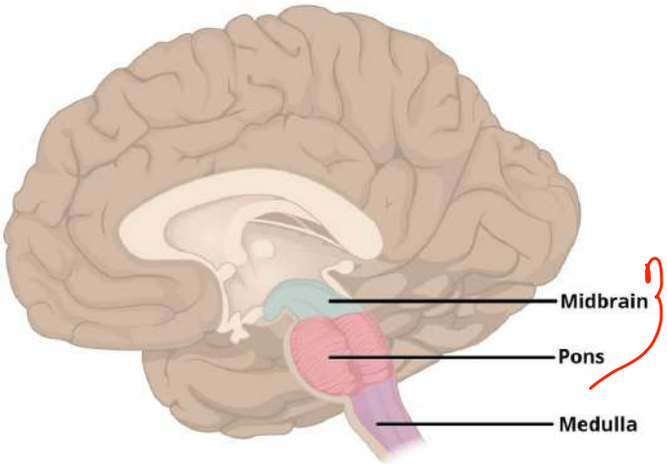
Function: The respiratory center adjusts alveolar ventilation to meet the demands so PO_2 and PCO_2 in the arterial blood are held constant even during exercise.

Physiological anatomy: The respiratory center is composed of groups of neurons located bilaterally in the medulla oblongata and pons. These groups are;

- Dorsal respiratory group (DRG) (causes inspiration)
- Ventral respiratory group (VRG) (mainly causes expiration)
- Pneumotaxic center (controls the rate and depth of breathing). Located dorsally in the superior portion of the pons
- Apneustic center

Note: The pons, hypothalamus, reticular activating system (RAS), and the cerebral cortex as well as afferents from the vagus, glossopharyngeal, and somatic nerves influence both the DRG and VRG.

* كل هدول بسيطرو على VRG DRG .



Here respiratory center is located.

* Upper regions control lower regions in nervous system. eg ⇒ Cerebral cortex have control in all parts of nervous system

* Groups of respiratory center :-

[1] - DRG ⇒ Cause action potential that transfered to diaphragm by phrenic nerve & cause inspiration

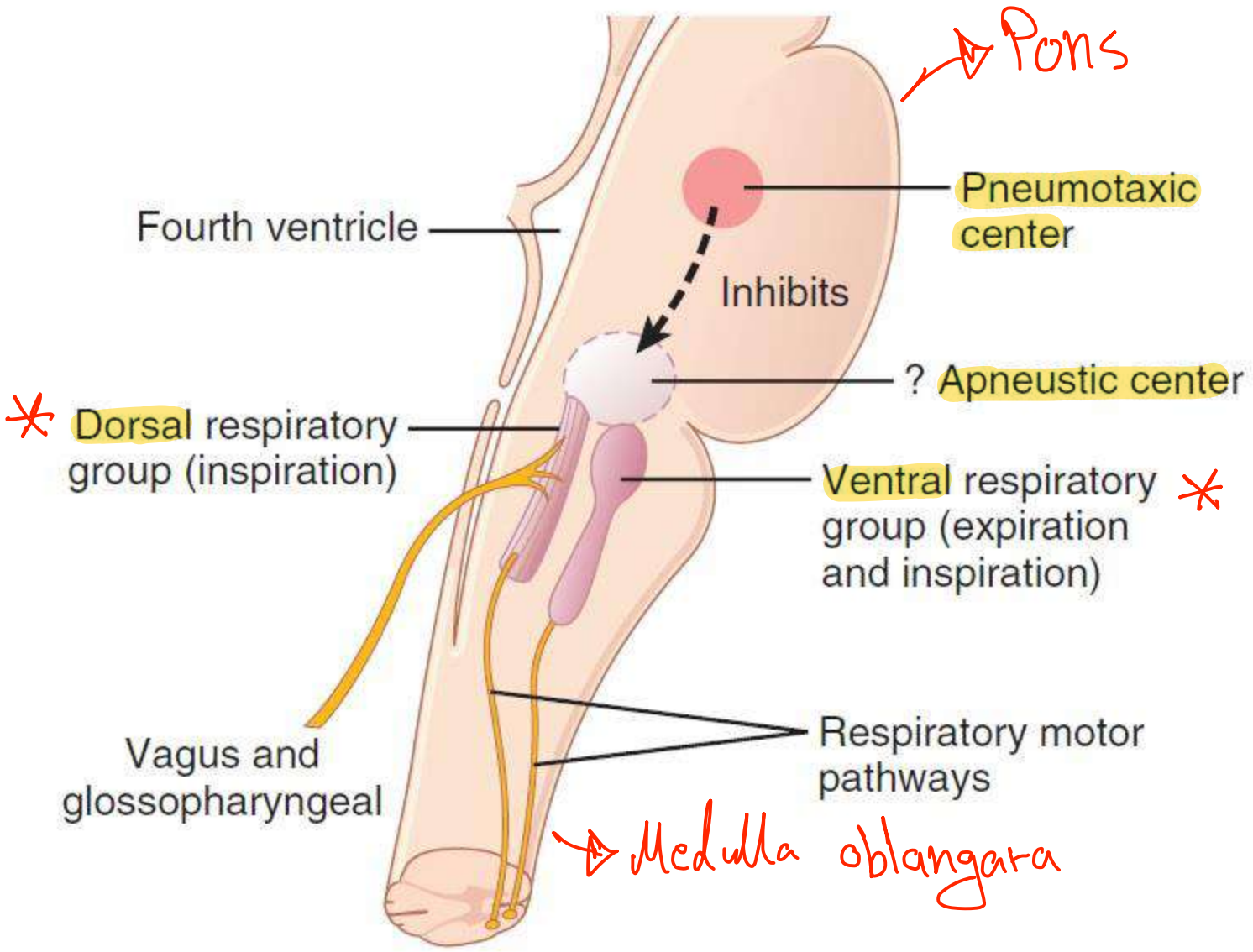
[2] - VRG ⇒ From the point the expiration is passive process, so this group just active during exercise

[3]- Pneumotaxic center ⇒ Control * Rate ⇒ Control of respiration per minute

* Depth ⇒ Control of respiratory tidal volume

Depth ← هي انك توخذ نفس طويل ولا قصير ، يعني Tidal volume

[4]- Apneustic center ⇒ Presents in animal , but in human no any one can make researches & Know their function



Organization of the respiratory center

Dorsal respiratory group (DRG):

- Neurons are almost located within the *nucleus of the tractus solitarius*. The neurons extends most of the length of the medulla.
- The nucleus of the tractus solitarius is the terminal station for the sensory part of the vagus and glossopharyngeal nerves (from chemoreceptors, baroreceptors, and other receptors in the lung).
- The DRG is the **pacemaker** for the basic respiratory rhythm. The intrinsic inspiratory rhythm starts with a latent period of several seconds during which there is no neuronal activity.
- Then bursts of action potentials begin to appear and are transmitted to the primary **inspiratory muscles** (through the phrenic nerve to the diaphragm) in a "**Ramp or Crescendo**" manner → No gasps. * We can't inspire after this point
- The ramp has a rate and limit (span) that can be controlled. The greater the increase in the rate the greater is the filling of the lung. Whereas, the shorter the limit the shorter is the respiratory cycle → higher respiratory rate.

The DRG is the **pacemaker** for the basic respiratory rhythm.
The intrinsic inspiratory rhythm starts with a latent period of several seconds during which there is no neuronal activity.

*After expiration there is a latent period (no activity), after this period A.P starts. A.P starts gradually to prevent gasps in every inspiration

↓

Ramp or cresendo

هاي المنطقة هي pacemaker يعني هي اللي تنشا السيال العصبي عشان تتحكم بالinspiration ryth

كلمة ryth يعني كل قديش بصير inspiration،،،،،هي توصف بانها latent period يعني مش لما يتنهي expiration بسرعة يصير inspiration،،،،في فترة انتظار بعد respiration يعني مرحلة ما فيها اي سيال عصبي.
لانو لو كانو ورا بعض رح يصير شهقة (gasps)

طيب هاض السيال العصبي لما ينشا كيف ينشا؟
ينشا بطريقة تدريجية (ramp or crescendo) صعود تدريجي،،يعني لو كان الهدف ١٠٠ سيال عصبي ما بصيرو ال١٠٠ بلحظة واحدة،،،بصيرو شوي شوي

طب ليش؟!

لانو لو صارو بلحظة وحدة رح يصير inspiration باقل من ثانية وبالتالي بصير زي كانو شهقة وبالتالي فش منو فايدة،،،فعشان هيك ينشا السيال العصبي بطريقة تدريجية عشان يكون inspiation تدريجي،،،بس هاض ramp A.P الو نهاية بوقف عندها،،مين اللي بتحكم فيه؟
ال pneumotaxic center

Pneumotaxic center limits DRG & therfor limits

inspiration .ie⇒limits tidal volume

The Pneumotaxic Center:

- Located dorsally in the *nucleus parabrachialis* of the upper pons.
- It controls the "switch-off" point of the inspiratory ramp in DRG (i.e. inhibits inspiration) and thus:
  - Regulate inspiration volume (tidal volume)
  - Controls respiratory rate.
- The tidal volume is inversely related to the respiratory rate.
- Strong stimulation of the pneumotaxic center can shorten inspiration for as little as 0.5 second.
- The function of the pneumotaxic center is primarily to limit inspiration, leading to a shallower and more rapid respiratory pattern.

Note:

It is believed that the pneumotaxic center is responsible for the "fine-tuning" of respiratory rhythm because a normal rhythm can exist in the absence of this center.

*If switch off occurs before end of inspiration, what happens?!

Decrease of inspiration & expiration time, this will lead to increasing respiration rate.* So if we need to increase the respiration rate the pneumotaxic center will act alittle bit before end of inspiration

هاض مو pacemaker وبالتالي ما بعمل A.P،،،بستقبل اوامر من DRC
في حال بدنا نزيد الrespiration في حالة الرياضة

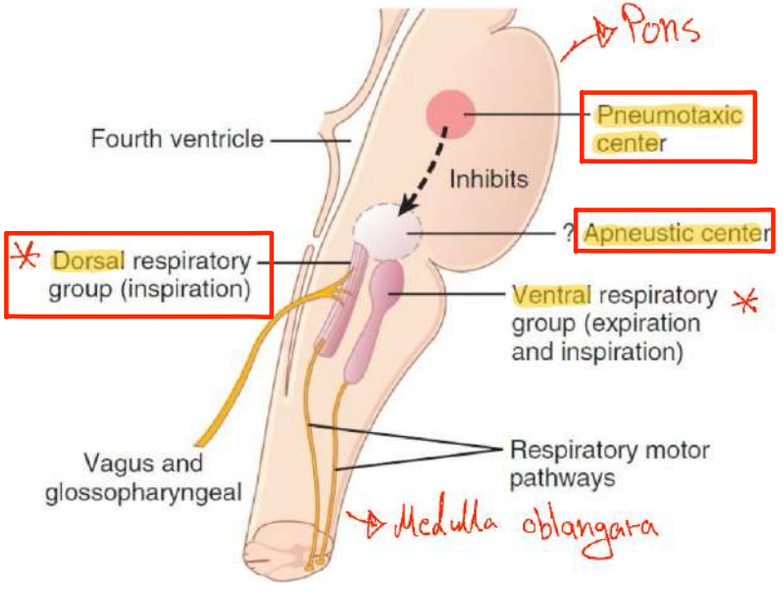
Ventral respiratory group (VRG):

- Neurons are located in the *nucleus ambiguus* and *nucleus retroambiguus*.
- VRG remains almost totally inactive during normal quiet respiration. Therefore, The ventral respiratory neurons do not appear to participate in the basic rhythmical oscillation that controls respiration.
- VRG has no pacemaker activity, but activated by the dorsal respiratory group to act and assist the dorsal group when greater pulmonary ventilation is needed (e.g. exercise).
- VRG supplies expiratory signals to the abdominal muscles during very heavy expiration.

* Expiratory muscle

The apneustic center:

- Located in the lower part of the pons.
- In experimental animal it inhibits the switch-off of the inspiratory ramp signals → increase tidal volume and duration of inspiration (prolonged inspiratory gasps or apneuses interrupted by transient expiratory efforts).
- In experimental animal impulses from this center prolong the ramp action potentials.
- Its activity is inhibited by the pneumotaxic center and by vagal impulses.
- Its importance in human respiration is ?????



هسا احنا حكينا انو "بالجهاز العصبي المنطقة اللي فوق تسيطر على اللي تحت"

* لو وقفنا ال Pneumotaxic رح يشتغل بارادتو ، وكونو هو فوق DRG فرح يتحكم فيها ، كونو Pneumotaxic بطل الها سيطرة فرح Apneustic يمنع انو يصير ↑ Vt & inspiration time وبالتالي . DRG ل switch-off

* هاي التجارب بالحيوان ، بالإنسان لهسا مو معروف

Vagal signals (Hering-Breuer Inflation Reflex or the inspiratory inhibitory reflex)

هاض الreflex انو لما تنفخ الرئتين كثير فيه streach receptors بالقصبات
هاي receptors تطلع امر للدماغ عن طريق vagus n عشان يعمل inhibition ل DRG
عشان هيك انت ما تقدر توخذ inspiration طويل كثير،،،اكثر اشي انو سوصل Vt ١.٥ لتر

- Stimulation of bronchial and bronchiolar stretch receptors → afferent signals through the vagus nerve via large myelinated fibers → inhibition of the DRG (similar to the signals from the pneumotaxic center).
- This reflex operates when tidal volume exceeds 1.5 L (i.e. three times the normal value). It is a protective mechanism.

Reticular Activating System (RAS):

* Keeps you wakeup, any impulse stimulate it

The RAS stimulates the respiratory centers to increase respiratory drive.

During sleep, when RAS activity diminishes, there is a decrease in alveolar ventilation that leads to a slight elevation of arterial CO_2 tension.

هاض RAS بستقبل اي impulse ،،،،هاض المستقبل بحفز التنفس عندك،،،فلما تكون قاعد فش اي impulse فرح تنام،،،فاي محفز لالك وانت نايم رح يصحيك لانو رح يحفز RAS
فلما تكون نايم فش محفز لالك فرح يقل alveolar ventilat وبالتالي رح يرتفع PCO2 شوي

Physiological Control of Respiration

The intensity of the respiratory control signals is increased or decreased to match the ventilatory needs of the body in many physiological and pathological states. Exercise; hypoxia; and metabolic acidosis or alkalosis are examples of such conditions.

The control mechanisms can be divided into **two** groups. These are;

Main *

1. Chemical control of respiration
2. Non-chemical (Neural) control of respiration

B. Chemoreceptors for CO2, H+, and O2 (Table 4-7)

* معقول من كتاب Costanzo لتسهيل الموضوع

1. Central chemoreceptors in the medulla

■ are sensitive to the pH of the cerebrospinal fluid (CSF). Decreases in the pH of the CSF produce increases in breathing rate (hyperventilation).

■ H+ does not cross the blood-brain barrier as well as CO2 does.

a. CO2 diffuses from arterial blood into the CSF because CO2 is lipid-soluble and readily crosses the blood-brain barrier.

b. In the CSF, CO2 combines with H2O to produce H+ and HCO3−. The resulting H+ acts directly on the central chemoreceptors.

c. Thus, increases in PCO2 and [H+] stimulate breathing, and decreases in PCO2 and [H+] inhibit breathing.

d. The resulting hyperventilation or hypoventilation then returns the arterial PCO2 toward normal.

2. Peripheral chemoreceptors in the carotid and aortic bodies

■ The carotid bodies are located at the bifurcation of the common carotid arteries.

■ The aortic bodies are located above and below the aortic arch.

a. Decreases in arterial PO2

■ stimulate the peripheral chemoreceptors and increase breathing rate.

■ PO2 must decrease to low levels (<60 mm Hg) before breathing is stimulated. When PO2 is less than 60 mm Hg, breathing rate is exquisitely sensitive to PO2.

b. Increases in arterial PCO2

■ stimulate peripheral chemoreceptors and increase breathing rate.

■ potentiate the stimulation of breathing caused by hypoxemia.

■ The response of the peripheral chemoreceptors to CO2 is less important than the response of the central chemoreceptors to CO2 (or H+).

c. Increases in arterial [H+]

■ stimulate the carotid body peripheral chemoreceptors directly, independent of changes in PCO2.

The respiratory receptors (The chemoreceptors):

1. Central chemoreceptors: are chemosensitive neurons located bilaterally, and are lying 0.2 millimeter beneath the ventral surface of the medulla in the vicinity of the exit of the 9th and 10th nerves.

   These neurons are sensitive to changes in either **PCO_2 or H^+** concentration in the surrounding interstitial fluid → Stimulation of the respiratory center.

2. Peripheral chemoreceptors: are located in the **carotid** and **aortic bodies**. They mainly detect changes in **Oxygen** in the blood, and also respond to a lesser degree to changes in P_aCO_2 and H^+.

   Signals from the carotid and aortic bodies are transmitted through the glossopharyngeal nerve to the dorsal respiratory area of the medulla.

   The aortic bodies signals are sent through the vagi also to the dorsal respiratory area.

هسا بدنا نحكي عن انواع الـ Chemoreceptors :-

1- Central ← موجودات بالدماغ وبس بحسو تركيز H^+ فقط ، طب كيف بتحس فيه؟ الـ CO_2 تعبر الـ "BBB" ، فلما يزيد او يقل CO_2 بالدم و اصلاً H^+ ما يعبر "BBB" ؟!

رح يزيد او يقل في CSF . الـ CO_2 دخل على الدماغ ، رح يرتبط مع H_2O و يكون H_2CO_3 و يتفكك إلى $H^+ + HCO_3^-$ عشان هيك تركيز CO_2 يُعبِّر عن تركيز H^+

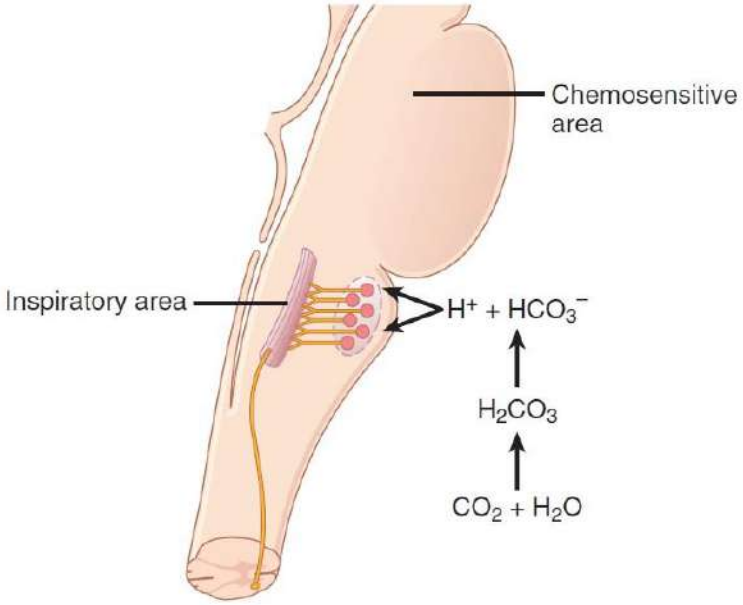
2- Peripheral * الي جاي من CO_2 V.I.P

↑CO_2 in blood → ↑H^+ in CSF → Stimulate central chemoreceptor

→ Hyperventilation & Vice versa

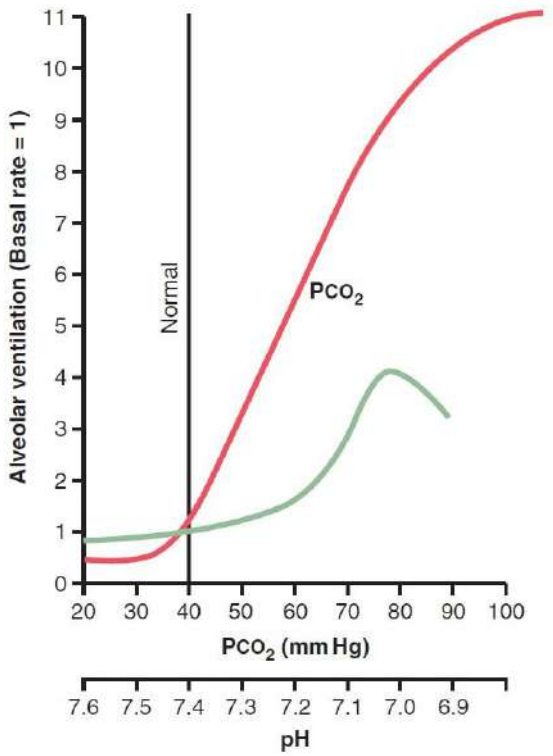
Chemical control:

1. Central chemoreceptors:
   - Hydrogen ions of the CSF are believed to be the only direct stimulus to the central receptors (i.e. the chemosensitive neurons).
   - Changes in H^+ concentration in the blood have less stimulatory effect as H^+ does not easily cross the blood-brain barrier.
   - Changes in blood CO_2 can affect these receptors indirectly as blood-brain barrier is completely permeable to CO_2. Carbon dioxide reacts with H_2O in the CSF $\rightarrow$ H_2CO_3 $\rightarrow$ $H^+ + HCO_3^-$. The H^+ then stimulates the central chemoreceptors.



Chemical control (cont.):

- Any increase in PCO_2 → ↑ strength of both the inspiratory and expiratory motor signals to the respiratory muscles.
- The initial **acute** increase in the respiratory drive when PCO_2 increases declines over the next 1-2 days because of the renal readjustment of the H^+ concentration (pH of the brain extracellular fluid returns to near normal in spite of a raised PCO_2). And only a weak **chronic** effect remains after a few days' adaptation.
- Approximately 85% of the resting, chemical drive of respiration results from the stimulatory effect of CO_2 on central chemoreceptors.
- Oxygen changes have no direct effect on central chemoreceptors. Therefore, CO_2 is the major controller of respiration, not oxygen.



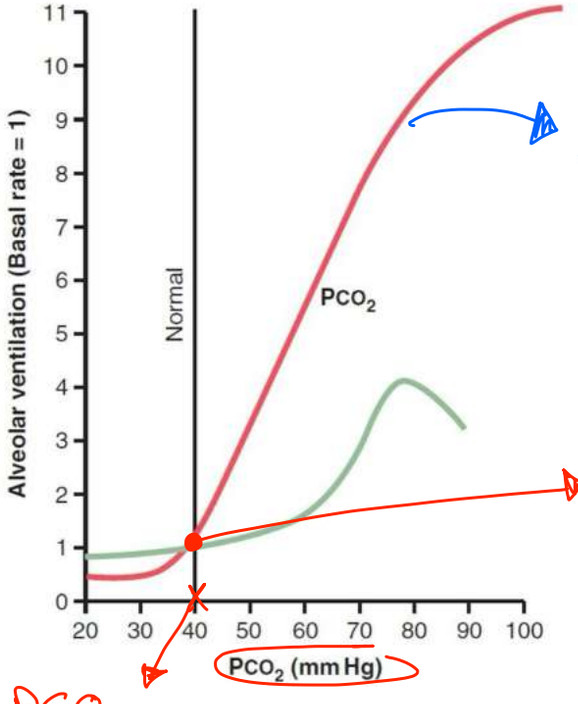
Effects of increased arterial blood P_{CO_2} and decreased arterial pH (increased hydrogen ion concentration) on the rate of alveolar ventilation.

PCO2 and Respiratory Muscles: An increase in the partial pressure of CO2 (PCO2) strengthens the motor signals to the respiratory muscles, enhancing both inhalation and exhalation efforts.

Renal Readjustment: Initially, a rise in PCO2 boosts respiratory drive, but this effect diminishes over 1-2 days due to the kidneys adjusting the hydrogen ion (H+) concentration. This renal compensation helps return the pH of the brain's extracellular fluid closer to normal despite elevated PCO2 levels.

Central Chemoreceptors: Central chemoreceptors, which are primarily responsible for the chemical drive of respiration at rest, are stimulated by CO2. About 85% of this drive is attributed to CO2's effect on these chemoreceptors.

CO2 as the Main Respiratory Controller: Unlike oxygen, CO2 changes directly affect central chemoreceptors. Therefore, CO2 is considered the main regulator of respiration, not oxygen.



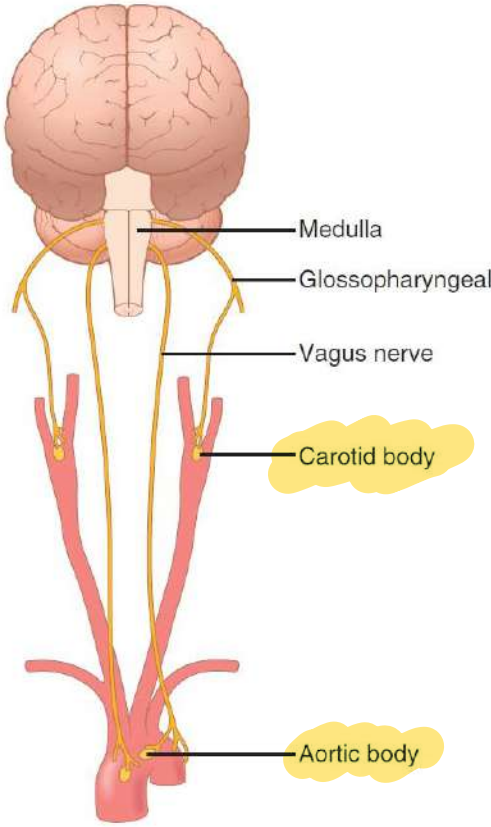
لما يزيد PCO_2 ، رح يزيد Ventilation

لما يكون PCO_2 طبيعي يكون Ventilation = 1

PCO_2 الطبيعي في الدم.

2. Peripheral chemoreceptors:

- They are the only site that detect changes in O_2 in the arterial blood, they respond to a lesser extent to changes in CO_2 and H^+.
- Decrease in PO_2 (below 70 mmHg) → stimulation of peripheral chemoreceptors → stimulation of DRG → increased rate and depth of respiration.
- An increase in either CO_2 or H^+ → stimulation of peripheral chemoreceptors → **Rapid** stimulation of respiration (as much as five times as rapidly as central stimulation).

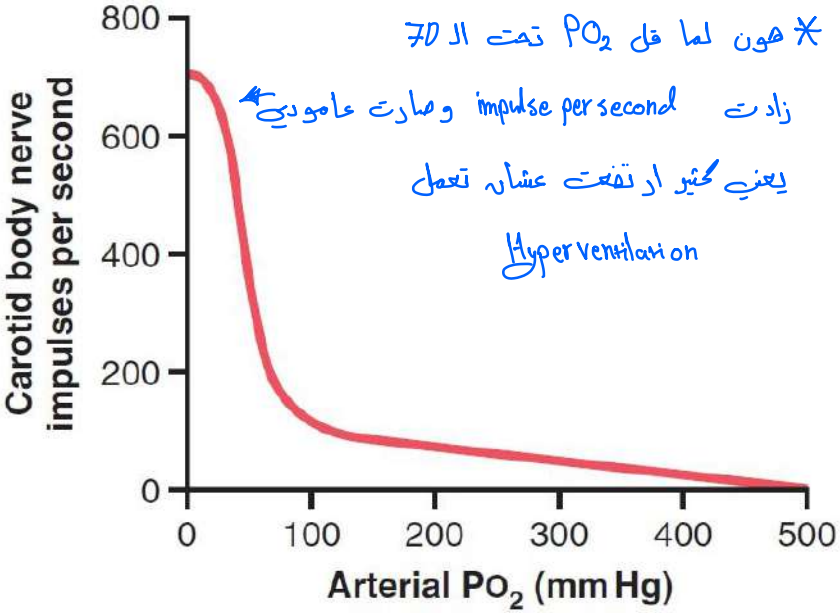


Respiratory control by peripheral chemoreceptors in the carotid and aortic bodies.

مشروح لتحت

2. Peripheral chemoreceptors:

- The increase in ventilatory drive due to arterial hypoxemia is multiplied if central chemoreceptors lose its sensitivity (because of adaptation). This is the basis of acclimatization to the low atmospheric oxygen concentration.
- (Acclimatization = deeper breathing 2-3 days after slow ascending of a mountain)



Note: The response of peripheral chemoreceptors is to the PO_2, not the oxygen concentration. Therefore, anemia and CO poisoning do not stimulate peripheral chemoreceptors.

2) Peripheral ⇐ موجودات عند تفرع الـ aeorta وبحس بتركيز O_2 mainly .

They detect the PO_2 in arterial blood & less extent CO_2 & H^+.

* Detects Pressure not concentration, therfore they can't detect hypoxia in anemia because in anemia hemoglobin is low, but PO_2 normal

$\downarrow O_2$, $\uparrow CO_2$, $\uparrow H^+$ ⇒ All need to hyperventilation

So metabolic acidosis is detected by peripheral chemoreceptors.

V.I.P

Stimulation of hyperventilation by peripheral is faster than central by 5 times

طلعت على جبل قل تركيز o2 مين حس فيه ؟ peripheral
بدو يعمل hyperventilation

مع ال hyperventilation قل تركيز الCO2 مين حس فيه؟ central
بدو يعمل hypoventilation

طب لمين الكلمة بالنهاية ؟
سؤال بالامتحان

Ventilatory Drive: This refers to the body's mechanism to regulate breathing. When there's arterial hypoxemia, which is a lower than normal level of oxygen in the blood, the body increases the ventilatory drive to compensate for the lack of oxygen.

Central Chemoreceptors: These are located in the brain and are sensitive to the levels of carbon dioxide (CO2) in the blood. When they lose sensitivity due to prolonged exposure to high CO2 levels, the body's response to hypoxemia is amplified.

Acclimatization: This is the process by which the body adjusts to lower oxygen levels in the atmosphere, such as at high altitudes.

الجهاز التنفسي والكلية ثنينهم بشتغلو على **Acid-Base balance**

التغيرات بتركيز ال**CO2** رح يصيرلها **buffer** عن طريق الكلية

لما يزيد تركيز **CO2** رح يصير **acidosis** رح تعمل الكلية **buffering** عن طريق طرح **HCO3-** فرح يقل تركيز **H+**

فتاثير **central** رح يقل لانو قل **H+** فهون رح يشتغل **peripheral**

يعني حالة التكيف هي انو تاثير ال**CO2** يقل بتاثير عمل الكلية

Respiratory regulation during exercise:

- During exercise the increase in O_2 consumption and CO_2 formation can rise to as much as 20-fold. Yet in the healthy athlete, alveolar ventilation ordinarily increases almost exactly in step with the increased level of oxygen metabolism. The arterial PO_2, PCO_2, and pH remain almost exactly normal.
- The immediate increase in ventilation on the initiation of exercise, before blood chemical changes have time to develop, suggests that the respiratory changes are due to;
  1. Stimulatory impulses from higher centers of the brain to both the respiratory muscles and respiratory center.
  2. Proprioceptive stimulatory reflexes to the respiratory center.

في حالة الرياضة عنا hyperventilation فكانوا يفكروا العلماء انو رح يقل O2 ويزيد CO2

لكن لما فحصوا الانسان اثناء الرياضة لقوا فيه hyperventilation لكن PO2,PCO2 and hydrogen ion are constant

السبب انو فيه حالة الرياضة رح يزيد الventilation قبل ما يصير تغيير بضغط O2,CO2,H+

طب ايش اللي زاد الventilation قبل ما يتغيرو التراكيز؟
لحد هسا العلماء مو عارفين كل اللي موجود هو عبارة عن theories اللي هما نقطة ١و٢ شرحهم اوضح بالسلايد اللي تحت

Stimulatory Impulses from the Brain: These are signals from the higher brain centers that activate both the respiratory muscles and the respiratory center in the brainstem, preparing the body for increased activity.

Proprioceptive Stimulatory Reflexes: These are reflexes initiated by proprioceptors, which are sensors in the muscles, joints, and tendons that provide information about body position and movement. They send signals to the respiratory center to adjust breathing based on the level of physical activity

Non-chemical factors that affect respiration:

معناها انو تقدر تقطع نفسك بارادتك بسبب سيطرة cortex لكن هاي السيطرة محدودة

1. Voluntary control of respiration: The cortex can override the function of the brainstem within limits. The nervous pathway passes directly from the cerebral cortex and other higher centers through the cortico-spinal tract.
2. Other parts of the brain, such as the limbic system and hypothalamus, can alter the pattern of breathing, for example, in emotional states such as **rage** and **fear**.
3. Coughing, sneezing, and even breath-holding (i.e. apnea): due to stimulation of **pulmonary irritant receptors**. These receptors are stimulated by noxious gases, cigarette smoke, inhaled dusts, and cold air. The response can also cause mucus secretion and bronchospasm as in asthma and emphysema.
4. The feeling of dyspnea when the pulmonary capillaries become engorged with blood (as in left heart failure) or when pulmonary edema occurs: is due to stimulation of **J receptors**, nonmyelinated sensory nerve endings positioned in the pulmonary interstitium that are in juxtaposition to the pulmonary capillaries. Their stimulation → tachypnea.
5. Anesthesia: can cause respiratory depression and respiratory arrest in overdosage.
6. Hering-Breuer reflex

2. Other parts of the brain, such as the limbic system and hypothalamus, can alter the pattern of breathing, for example, in emotional states such as **rage** and **fear**.

الlimbic system هو جهاز العواطف والانسان هو المخلوق الوحيد اللي الو سيطرة عليه

مثال حطيت اكل لبسة وانت عند الاكل رح تضل خايفة لحد ما انت تروح والغريزة اللي عندها تخليها ترجع توكل

اما الانسان لو راح عالمطعم وجابولوه الاكل ورموه رمي قدامو ما رح ياكل

① اسباب dyspnea

4. The feeling of dyspnea when the pulmonary capillaries become engorged with blood (as in left heart failure) or when pulmonary edema occurs: is due to stimulation of **J receptors**, nonmyelinated sensory nerve endings positioned in the pulmonary interstitium that are in juxtaposition to the pulmonary capillaries. Their stimulation → tachypnea.

② شعور انك تتنفس بس حاس انو فش اكسجين هو dyspnea

هاي dyspnea تحفز j-receptors فلما يتحفزو يعملو

Tachypnea:which is an abnormally rapid breathing rate

فيه عامل يوقف التنفس هو لما تكون توكل وتبلع اللقمة لحظة البلع يوقف التنفس للحظات لحد ما تكمل بلع اللقمة

هي عملية فشل الجهاز التنفسي اما بتشبع الدم
بالاكسجين او طرح co2
تتصنف الى

Chronic respiratory failure:

- Chronic respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions: oxygenation and carbon dioxide elimination. It is classified as either hypoxemic or hypercapnic respiratory failure. The failure may be complicated by polycythemia or cor pulmonale.
- Hypoxemic respiratory failure is characterized by an arterial oxygen tension (PaO_2) lower than 60 mmHg with a normal or low arterial carbon dioxide tension ($PaCO_2$). This is the most common form of respiratory failure.
- Hypercapnic respiratory failure is characterized by a $PaCO_2$ higher than 50 mmHg. Hypoxemia is common in patients with hypercapnic respiratory failure who are breathing room air. The patient suffers from respiratory acidosis.
- Respiratory acidosis occurs due to;

1. Generalized hypoventilation and results from failure of the controller to respond to carbon dioxide (e.g., during anesthesia, following brain injury, and in some patients with chronic obstructive lung disease).
2. Obstructive lung disease. When these patients breathe room air, hypercapnia caused by reduced alveolar ventilation is accompanied by significant hypoxia and acidosis.

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* اسباب الـ Respiratory acidosis :-

1) Hypoventilation ⇐ سببها بكون عدم قدرة الجسم انو يعمل Hyperventilation بسبب ارتفاع CO_2 . يعني في حالة Brain injury مارح يقدروا Central receptors انهم يتعاملوا مع $\uparrow CO_2$

2) Obstructive lung diseases ⇐ هون لما يتنفس المريض هواء الغرفة رح يقل Alveolar ventilation

$\Rightarrow \uparrow CO_2$ & acidosis

Test Question:

Q. Concerning the respiratory centers?

A. The normal rhythmic pattern of breathing originates from neurons in the motor area of the cortex.
B. During quiet breathing, expiratory neurons fire actively.
C. Impulses from the pneumotaxic center can stimulate inspiratory activity.
D. The cortex of the brain can override the function of the respiratory centers.
E. The only output from the respiratory centers is via the phrenic nerves.