# **Regulation (Control) of Respiration**

الجزء الثاني من المحاضرة اللي هو الية التحكم بالتنفس وتنيمها من خلال الدماغ

- طبعا عنا اليتين الية عصبية والية كيميائية . Neural Mechanism (CNS).
  - 1. Voluntary control by stimulating the Cerebral cortex:
  - Activated only when you need voluntarily to increase the respiration rate and depth.
     هون لما انت تقرر بشكل ارادى انك تتنفس
  - 2. Automatic control Pons and medulla(vegetative function) ( BRAIN STEM):
  - Activated all the time, and responsible of our unconscious breathing. وهون التنفس
     الطبيعي اللارارادي

#### 2- Chemical control:

Respond to changes in CO2 and O2 levels by sense it by Chemoreceptors (Central (in the brain) & Peripheral (in blood vessels))

هون التحكم الناتج عن الاستجابة للتغيرات بنسبة الغازات بالاخص يادة ثاني اكسيد الكربون

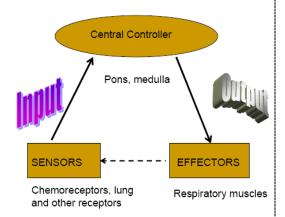
## Main Goals of the Respiratory Control System:

- General.: An alveolar ventilation sufficient to maintain normal blood gases.
- Adapt to changing environments or metabolic needs (e.g. exercise).

طبعا اهم وظيفة هي انه يعمل تنفس قادر على اعطاء الجسم كمية كافية من ااكسجين وإذا حصل اي تغير يعدله.

# عملية التحكم راح تتم بالمخ بالاخص في جذع الدماغ .Control of Respiration

- Central controller or Respiratory center in the brain.
- Sensors: we have many types
- Effectors
  - Ventilation (muscles)
    - diaphragm
    - intercostal muscles
    - · abdominal muscles



accessory muscles

# **Chemical Control of Respiration:**

any increase or decrease in those 3 molecules will lead to stimulate the respiratory centers to make a response:

- 1. Carbon Dioxide
- 2. Oxygen
- 3. Hydrogen ions

عنا نوعين من المستقبلات اللي بحسو التغير عن :Types of receptors in the respiratory system الطبيعي للغازات مركزية في الدماغ وطرفية بجدار اوعية دموية مهمة، وطبعا بتم تقسيمها لمستقبلات كيميئاية.

- 1. Mechanoreceptors.
- 2. Chemoreceptors.

### **Mechanoreceptor:**

- It's Slowly adapting stretch receptors (SARs) in bronchial airways, which mean it prevent overstretching of the lung and prevent rupture, terminate the inspiration, in addition to make an expiration process.
- This mechanism called HERING-BREUR REFLEX. هون هاي مستقبلات بتمنع الرئة تتوسع زيادة عن اللزوم حتى ما تنفجر، فمنطقيا راح توقف الشهيق وتبدا عملية الزفر.

Significance of Hering-Breuer: its significant only in the situation of increase inspiration or diseases.

- Normal adults. Receptors aren't activated at end normal tidal volumes.
  - Become Important during exercise when tidal volume is increased.
  - Become Important Chronic obstructive lung diseases when lungs are more distended.
  - To inhibit over inflation of lungs

# والمستقبلات الكيميئاية وهي الاهم المسؤولة عن احساس اي تغير في الغازات وضغطها.

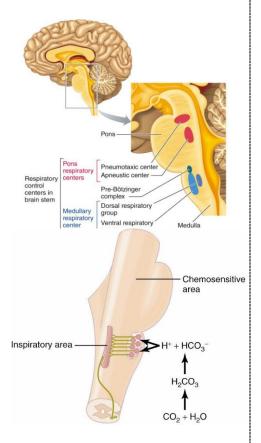
### **Chemoreceptors:**

- 1. Peripheral Chemoreceptors: طبعا عنا نوعين نوع طرفي ونوع مركزي
- Composed of:
- a) Carotid bodies are located in the carotid sinus
- b) Aortic bodies are located in the aortic arch
- Sensitive to O2,CO2 AND H+ changes.
- More sensitive to CO2 changes, small increase in CO2 activate he receptor and send message to respiratory center in the brain to increase the respiration.
- As we know CO2 bind with water and make H2co3, which dissolve into H+ + HCO3-
- Increase CO2 level, will increase H+ level which bind to the receptors and send the message to the brain.
- So co2 indirectly activate the receptor by the H+ IONS
- If the co2 increased, respirartory center activated, and the respiration increase in number and depth.
- Co2 convert to H+ beside the receptors, but plasma H+ have minimal effect on it because H+ can't cross blood brain barrier.

طبعا ثاني اكسيد الكربون بحفزها بشكل غير مباشرة عن طريق الهيدروحين اللي بنتج منه ، بس هذا الهيدروجين بتحول داخل المستقبلات من ثاني اكسيد الكربون بينما الهيدروجين اللي بالدم ما باثر لانه ما بقدر يدخل الدماغ اصلا



- located in the brain stem:
- a) pons: contain هسا هذا الجزء فقط بنظم شغل النخاع المستطيل اللي تحته واللي هناك فيه المركز الرئيسي للتحكم للتحكم
  - 1. **pneumotaxic center:** send impulses to dorsal respiratory group in the medulla and its function as switch of inspiratory neurons.



- 2. Apneustic center.
- Both control the main respiratory centers in medulla only.
- b) **medulla oblongata:** main respiratory center located in it, contain: هون مركز التحكم الرئيسي
  - 1. **Dorsal respiratory group (DRG)**: respiratory pacemaker for normal respiration. هذا مركز التنفس العادي البسيط
  - In resting state, it is responsible of generating action potential to the inspiratory muscles(diaphragm), to make a contraction and increase the thoracic cavity>> normal quite inspiration.
  - Inhibition of dorsal respiratory group lead to relaxation of inspiratory muscles>> leading to elastic recoil of inspiratory muscles>> normal quite passive expiration.
  - Generates 12-15 electrical signals each minute, and this is the normal respiratory rate.
  - It controls the rate and magnitude of the breathing, by controlling the frequency of stimulation.
  - Note: contract the diaphragm muscle, which is sufficient to make an inspiration.



- 2. Ventral respiratory group(VRG): هذا مركز التحكم بالتنفس القوي بحالات الرياضة والجهد
- for active forceful respiration, when doing exercise.
- Its stimulates both inspiratory and expiratory group muscles to increase both depth and rate, so this will lead to active inspiration.
- Voluntarily increase the depth of breathing>> lead to send a message to the ventral center and lead to activate muscle contraction and relaxation.
- Other receptors (e.g., pain) and emotional stimuli acting through the hypothalamus

  Peripheral chemoreceptors

  O2 \( \dagger \cdot \cdot \cdot \cdot \text{H\$^+\$} \)

  Central chemoreceptors

  CO2 \( \dagger \cdot \text{H\$^+\$} \)

  Receptors in muscles and joints
- When you prepare for doing exercise, the joints and muscles send a message to the ventral center and increase the rate and depth of respiration.

So:

- Normal inspiration and expiration: dorsal respiratory center.
- Active forceful inspiration and expiration: both dorsal and ventral groups.

## Effect of CO2 and PH AND O2 on the respiration:

- O2, CO2, H+ affect the respiration, but CO2 has the strongest effect then H+.
- 4. Effect of H+: يعبر الحاجز باشكل رئيسي على المستقبلات الطرفية لانه ما بقدر يعبر الحاجز الدماغي.
- H+ stimulates the peripheral chemoreceptors more than central one(in the brain),
   because the blood brain barrier prevent H+ from entering the brain.
- CO2 passes to the brain through blood brain barrier, it bind to H20>> H2CO3>> H+ AND HCO3-.
- H+ bind strongly to the central chemoreceptor and stimulate more breathing in order to increase respiration and decrease CO2 levels.

### 2. Effect of CO2:

- When partial pressure of CO2 increase it will affect both peripheral and center chemorecetors:
- **Peripheral:** weak effect, so inside the carotid body, it converts into H+ and stimulate respiratory center in medulla oblongata.
- Medullary respiratory center: increase PP of CO2 will lead to more H+ which stimulate both ventral and dorsal groups, and increase the respiration process, in order to decrease the co2 level.
- Increase CO2 level chronically lead to inhibition of respiratory center instead of activation.
- When PP of CO2 increase to 70-80 MMHG, this will lead to increase H+ which will decrease the PH and lead to acidosis and depreesion of the respiratory centers.

هون في معلومة مهمة واللي هي ببساطة انه زيادة ثاني اكسيد الكربون بتخلي التنفس يزيد حتى نتخلص من ثاني اكسيد الكربون ولكن اذا زاد الى حد كبير بادي لاثر عكسى وبدمر مراكز التحكم بالتنفس التنفس بتوقف وبموت الانسان.

### 3. Effect of O2:

- On peripheral: stimulate it only when it is fall less than 60 mmhg( o2 dissolved in plasma that make the partial pressure of o2).
- Effect of central: depressed the central chemoreceptor when decrease below 60 mmhg.
- At the height of 5 km the o2 levels will fall and the human body can't tolerate this decrease in oxygen.

#### Clinical correlation:

• CO poisoning: co gas has an affinity to bind hemoglobin 100 times more than o2, so when CO present in the room people will die, because the brain monitors the

dissolved O2 in plasma which is not affected by CO not that bind to hemoglobin, so O2 hemoglobin levels decrease until the patient die without any reaction from the brain. ومن بدنا نعرف انه لما نترك صوبة الغاز مفتوحة عند اولاد صغار بغرفة مغلقة برتفع عندهم غاز اولى العام عند الكربون اللي حيرتبط بدل الاكسجين مع الهيموغلوبين لانه حب ارتباطه اكبر بكثير ولكن الدماغ ما حيكتشف هذا الانخفاض لانه الدماغ بفحص بس الاكسجين الذائب بالدم اللي بشكل 3% بس وهذا ما بتاثر من تسمم اول اكسيد الكربون فبعد فترة قليلة راح يموت الطفل بدون اي احساس.

A TABLE 13-8 Influence of Chemical Factors on Respiration		
Chemical Factor	Effect on the Peripheral Chemoreceptors	Effect on the Central Chemoreceptors
$\downarrow$ $P_{0_2}$ in the Arterial Blood	Stimulates only when the arterial $P_{O_2}$ has fallen to the point of being life threatening (< 60 mm Hg); an emergency mechanism	Directly depresses the central chemoreceptors and the respiratory center itself when < 60 mm Hg
$\uparrow$ $P_{\text{CO}_2}$ in the Arterial Blood ( $\uparrow$ H <sup>+</sup> in the Brain ECF)	Weakly stimulates	Strongly stimulates; is the dominant control of ventilation (Levels > 70–80 mm Hg directly depress the respiratory center and central chemoreceptors)
↑ H+ in the Arterial Blood	Stimulates; important in acid-base balance	Does not affect; cannot penetrate the blood-brain barrier