# **Regulation Of Respiration**

# **10- Non-Chemical Control Of Respiration**

# By Dr. Nour A. Mohammed Mutah school of medicine

#### **Non-chemical regulation 1. Afferents from the respiratory system**

### A. From the lung

	Lung stretch receptor (Herring Breuer inflation reflex)	Lung irritant receptors	J-receptors (Pulmonary chemoreflex)
Receptors	Stretch receptors in the bronchi.	Bronchi & Bronchioles.	Close to alveoli (Juxta capillary)
Stimulus	Inflation of the lung	Irritants as: Cigarette.	Pulmonary <u>E</u> dema & <u>E</u> mboli.
Afferent	Vagus.	Vagus.	Vagus.
Response	<ul> <li>a) Inhibit DRG &amp; inhibit</li> <li>apneustic center.</li> <li>b) Bronchodilatation.</li> </ul>	<ul><li>a) Cough.</li><li>b) Bronchoconstriction.</li></ul>	a) Apnea <sup>Then rapid</sup> breathing b) Hypotension. c) Bradycardia.

## **B.** From the upper respiratory passages

	Sneezing	Cough	Swallowing
Stimulus	Irritation of nose.	Irritation of bronchi.	Irritation of pharynx.
Afferent	Trigeminal. (V)	Vagus. (X)	Glossopharyngeal. (IX)
Response	Deep inspiration Followed by forced expiration Against opened glottis.	Deep inspiration Followed by forced expiration Against closed glottis with sudden opening.	Swallowing apnea (stoppage of respiration) and closure of glottis.

## 2. Afferent from the cardiovascular system

	Arterial baroreceptors	Atrial baroreceptors
Stimulus	û ABP & û pulse pressure.	℃ VR.
Afferent	Vagus & glossopharyngeal.	Vagus.
Response	Inhibit respiration.	Stimulate respiration.

- ✓ Adrenaline apnea: Injection of large dose of adrenaline ⇒ VC ⇒ ①ABP
- $\Rightarrow$  stimulate arterial baroreceptors  $\Rightarrow$  reflex apnea.

### **3. Afferents from higher centers**

- A. Limbic cortex & Hypothalamus:
- Mild pain & emotions: ⇒ tachypnea via sympathetic.
- Severe pain & emotions: ⇒ inhibition of respiration.
- Hot as fever: ⇒ stimulation of respiratory center( as apart of thermoregulatory function of hypothalamus)
- Panting: shallow rapid breathing in dogs (no sweat glands) ⇒ heat loss
- B. Cerebral cortex (motor areas) : (Voluntary control)
- I. Voluntary apnea (breath holding):
- Temporary stoppage of breathing till the breaking point.
- Voluntary apnea  $\Rightarrow$   $\textcircled{1}CO2 \& \textcircled{1}H + \& \textcircled{0}O2 \Rightarrow$  stimulate respiration.
- Breaking point is delayed by:
- a) Previous hyperventilation  $\Rightarrow$   $\bigcirc$  CO2.
- b) Breathing 100% O2 before apnea ⇔ û O2.
- c) Holding the breath in full inspiration  $\Rightarrow$  inhibition of respiration.
- d) Swallowing (deglutition) ⇒ inhibition of respiration

## **Types(causes )of apnea:**

1)Voluntary apnea may occur during speech, blowing, suckling, childbirth, micturition and defecation.

- 2) Apnea follows the voluntary hyperventilation.
- 3) Adrenaline apnea.
- 4) Swallowing apnea.
- 5) Hypocapnia(after long period of hyperventilation).
- 6) Severe hypoxia (direct inhibition of respiratory center ).
- 7) Depression of respiratory center by certain drugs e.g. Morphine toxicity
- 8) Inhalation of 100% O2 during deep anesthesia.
- 9) Chyne-stockes respiration.

## //. Voluntary hyperventilation

Increase in depth and rate of respiration  $\rightarrow \downarrow PCo_2$  from 40 to 15 mmHg (hypocaphia),  $\uparrow$  PO<sub>2</sub> from 95 to 130 mmHg. and  $\downarrow$  H<sup>+</sup> (alkalosis)  $\rightarrow$ inhibition of respiration  $\rightarrow$  apnea  $\rightarrow \downarrow O_2$  &  $\uparrow Co_2 \rightarrow$  stimulate respiration  $\rightarrow$  hyperventilation and the cycle is repeated, then PCo<sub>2</sub> return to normal level and breathing becomes normal. This alternate hyperventilation and apnea is called (periodic breathing) or (chynestokes respiration)

## **Causes of chyne-stokes respiration ( Periodic respiration)**

- 1. After voluntary Hyperventilation.
- 2. High altitude (hypoxia).
- 3. Heart failure ⇒ prolongation of lung-brain circulation, so changes in arterial gas tension at lung takes longer time to affect the brain center.
- 4. Live failure & kidney failure ⇒ inhibition of DRG by toxic substances.
- 5. Narcotics & morphine  $\Rightarrow \hat{T}$  sensitivity of chemoreceptors to CO<sub>2</sub>.

### 4- Afferents from skeletal muscles, joints and skin

(a) From muscle spindle of the intercostal muscle and the diaphragm to regular the depth of respiration.

(b) From the proprioceptors: During muscle movements, afferent from tendons, ligaments and joints to stimulate the respiratory center  $\rightarrow$  exercise hyperventilation (c) From the skin: Exposure to cold leading to initial apnea followed by deep inspiration

# **5) Respiratory components of the other visceral reflexes**

### A. Swallowing and vomiting: Apnea to prevent aspiration of food or vomitus.

B. **Hiccup:** Sudden spasmodic contraction of diaphragm  $\Rightarrow$  sudden forced inspiration with sudden closure of the glottis  $\rightarrow$  producing characteristic sound.

- It occurs reflexly by afferent vagal fibers to respiratory center as a result of irritation of diaphragm or upper abdominal viscera.
- It is treated by inhalation of CO2 gas mixture .

C. Yawning: is infectious respiratory act characterized by deep inspiration& stretching of the chest wall to help :

a) Open alveoli to prevent collapse b) ↑ venous return

It is usually associated with a desire to sleep.

# Effect Of Muscular Exercise On Respiration

### **During muscular exercise:**

- The O2 consumption increase: from 250 ml/min to 4000 ml/min.
- The CO2 production increase: from 200 ml/min to 8000 ml/min.

### **Changes during exercise:**

### **1.** Acceleration of respiration:

- This increases the pulmonary ventilation from 6 L/min at rest to 100 L/min with exercise .
- This occurs due to stimulation of respiratory centers .

At onset of exercise (Neural control): (Increase in depth > rate of respiration).

- 1) Impulses from cerebral cortex stimulate the RC.
- 2) Impulses from proprioceptors in joints , ligaments & tendons stimulate the RC.

### **During exercise (Humoral control):** (Increase in rate of respiration).

- Decreased O2 due to increased O2 consumption by the active muscle causes stimulation of respiration via *peripheral chemoreceptors*
- 2) Increased CO2 & H+ due to increased metabolic processes in muscle cause stimulation of *central & peripheral chemoreceptors*.
- 3) Increase temperature of blood causes stimulation of RC via

hypothalamus(heat regulating center )

### **2.** *Increase in the coefficient of O2 utilization by tissues:*

Active tissue is given **14 ml O2** from **every 100 ml blood** instead of **5 ml O2** given to tissue during rest (increase **Coefficient of O2**: from **25% to 75%** increases O2 supply **3 times**).

#### This occur because:

1) PO2 in venous blood from exercising muscle fall to about zero

Creating high difference between PO2 in alveolar air & venous blood increasing diffusion **C**apacity of O2 from alveoli.

2) Increase in COP: from 5 to 30 L/min. increases O2 supply 6 times.

3) V.D of skeletal m. blood vessels (sympathetic vasodilator fibers & metabolites) increases blood flow to exercising muscles

**3.** The O2 dissociation curve is shifted to the right:

Due to:  $\downarrow$  O2 &  $\uparrow$  CO2 &  $\uparrow$  H+ &  $\uparrow$  Temperature &  $\uparrow$  2,3 DPG.

•  $\downarrow$  Affinity of Hb to O2 causing unloading so,  $\uparrow$  O2 supply to active muscle.

# Causes of hyperpnea (Rapid breathing )

- 1) Muscular exercise
- 2) Certain emotions (by signals from limbic system)
- 3) Mild pain (by signals from hypothalamus)
- 4) Exposure to heat
- 5) Hypotension (due to hemorrhage)through stimulation of peripheral chemoreceptors by hypoxia & acidosis
- 6) Hypercapnia
- 7) Acidosis as in diabetic ketoacidosis
- 8) High altitudes due to hypoxemia

