

# Regulation OF RESPIRATION

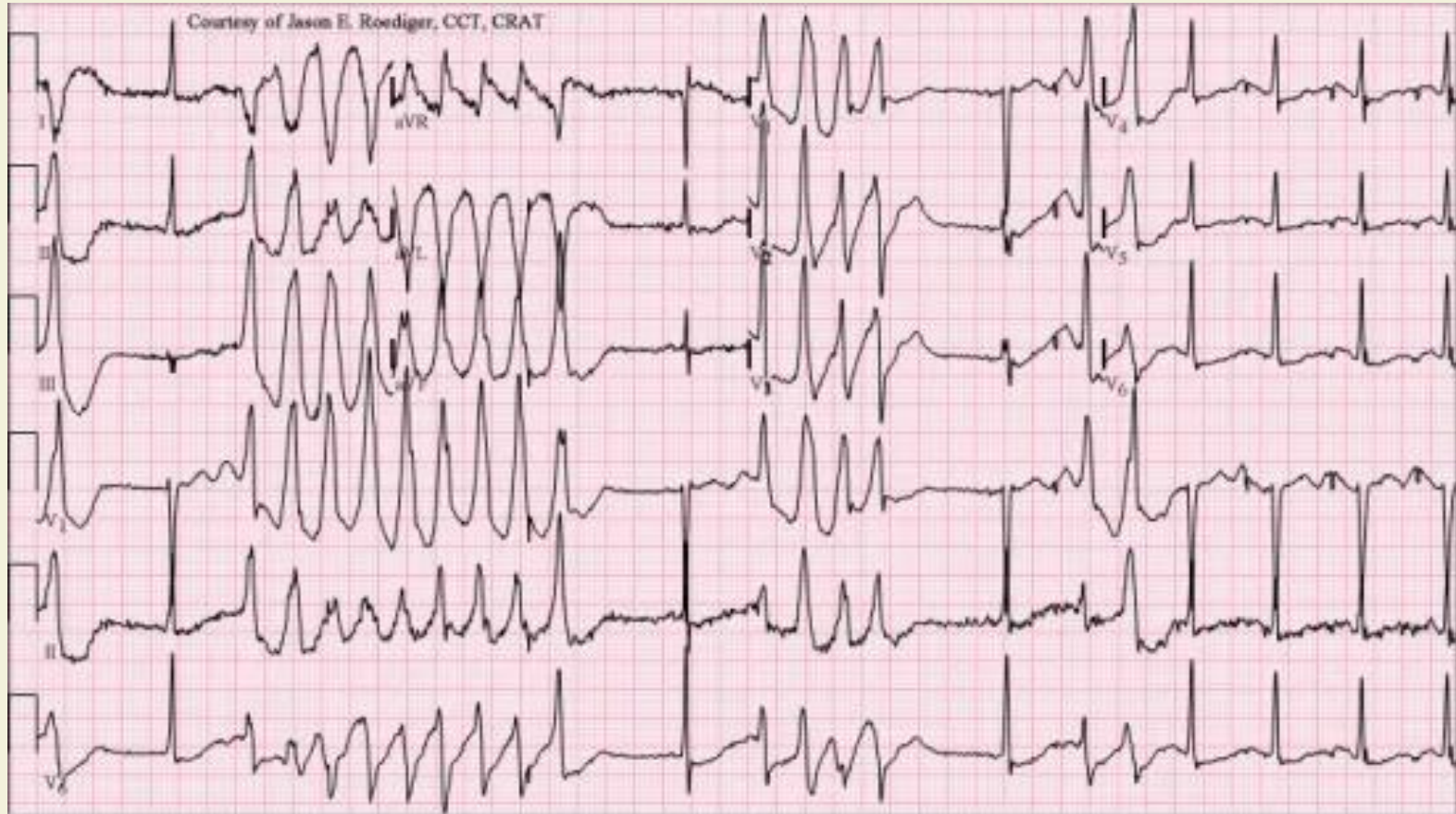
By

**Dr Gul Muhammad**

# Remaining points in CVS

- Torsades de pointis
- Carotid massage
- Pulsus paradoxus
- Cardiac tamponade
- Electrical alternans
- Akinesia and aneurysm of the left ventricle
- CABG/By pass surgery
- haemodynamics in each valvular disease
- Nitrous Oxide effect in local blood flow to treat ED and PH

# Torsades de pointis



# Regulation OF RESPIRATION

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# Learning Outcomes

- Define respiratory regulation
- Name the types of respiratory regulation
- Describe the components of nervous regulatory system
- Describe the chemical regulation of respiration
- Name the components of chemical regulation of respiration
- Name Other factors which take part in regulation of respiration

# INTRODUCTION

- Like heartbeat, breathing must occur in a continuous, cyclic pattern to sustain life processes.
- Inspiratory muscles must rhythmically contract and relax to alternately fill the lungs with air and empty them.
- The rhythmic pattern of breathing is established by cyclic neural activity to the respiratory muscles.

- The nerve supply to the respiratory system is absolutely essential in maintaining breathing and in reflexly adjusting the level of ventilation to match changing needs for O<sub>2</sub> uptake and CO<sub>2</sub> removal.
- Respiratory activity can be voluntarily modified to accomplish speaking, singing, whistling, playing a wind instrument, or holding one's breath while swimming.

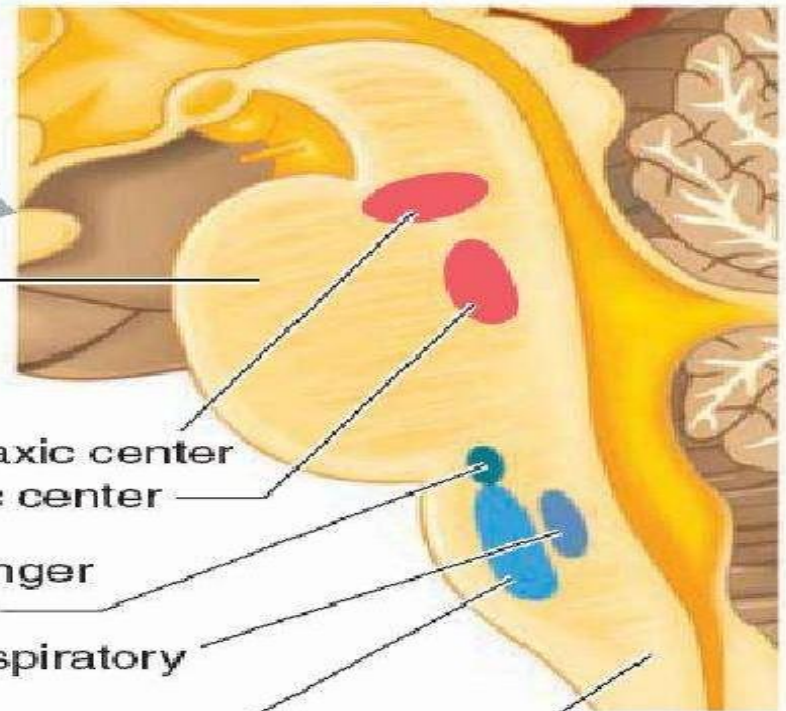
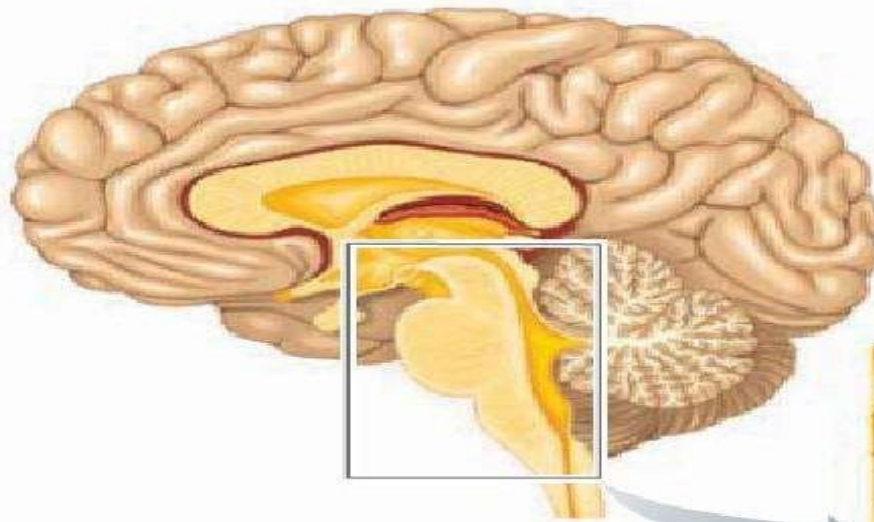
# COMPONENTS OF NEURAL CONTROL OF RESPIRATION

- Three distinct components:
  - 1) Factors that generate the **alternating inspiration/expiration rhythm**
  - 2) Factors that regulate the **magnitude of ventilation** (that is, the rate and depth of breathing) to match body needs,
  - 3) Factors that **modify respiratory activity** to serve other purposes.



# NEURAL CONTROL

- The primary respiratory control center,
- ***The Medullary Respiratory Center***
  - *consists of several* aggregations of neuronal cell bodies within the medulla that provide output to the respiratory muscles.
- Two other respiratory centers lie higher in the brain stem in the pons
- ***The Pneumotaxic Center and Apneustic Center***
  - *These pontine* centers influence output from the medullary respiratory center



Pons

**Pons  
respiratory  
centers**

Pneumotaxic center  
Apneustic center

Pre-Bötzinger  
complex

Dorsal respiratory  
group

Ventral respiratory  
group

Medulla

Respiratory  
control  
centers in  
brain stem

**Medullary  
respiratory  
center**

- The **medullary respiratory center** consists of **two neuronal** clusters known as the
  - *Dorsal Respiratory Group (DRG)*
  - *Ventral Respiratory Group (VRG)*

- The **dorsal respiratory group (DRG)** consists **mostly of** *inspiratory neurons whose descending fibers terminate on the* motor neurons that supply the inspiratory muscles.
- When neurons fire, inspiration takes place;
- When they cease firing, expiration occurs.

- The **ventral respiratory group (VRG)** is composed of *inspiratory neurons and expiratory neurons, both of which remain inactive* during normal quiet breathing.
- This region is called into play by the DRG as an “overdrive” mechanism during periods when demands for ventilation are increased.
- It is especially important in active expiration.

# THE PNEUMOTAXIC AND APNEUSTIC CENTERS

- The respiratory centers in the pons exert “fine-tuning” influences over the medullary center
- The **pneumotaxic center** sends impulses to the DRG that help “switch off” the inspiratory neurons, limiting the duration of inspiration.
- The **apneustic center** prevents the inspiratory neurons from being switched off , thus providing an extra boost to the inspiratory drive.

- In this check-and-balance system, the pneumotaxic center dominates over the apneustic center, helping halt inspiration and letting expiration occur normally.
- Without the pneumotaxic brakes, the breathing pattern consists of prolonged inspiratory gasps abruptly interrupted by very brief expirations. This abnormal breathing pattern is known as **apneusis**

# REFLEX MODIFICATION OF BREATHING

## HERING–BREUER REFLEX

- **When the tidal volume is large** (greater than 1 liter), as during exercise, the **Hering–Breuer reflex is triggered to prevent overinflation of the lungs.**
- **Pulmonary stretch receptors** within the smooth muscle layer of the airways are activated by stretching of the lungs at large tidal volumes.
- Action potentials from these stretch receptors travel through afferent nerve fibers to the medullary center and inhibit the inspiratory neurons.



# JOINT RECEPTORS

- Impulses from **moving limbs** reflexly **increase breathing**
- Probably contribute to the **increased ventilation during exercise**

## DEFLATION REFLEX

- The deflation reflex is stimulated when the lungs are compressed or deflated, causing an increased rate of breathing.
- Precise mechanism for this reflex is not known.

## IRRITANT REFLEX

- When the lungs are exposed to toxic gases, the irritant receptors may be stimulated.
- These irritant receptors are subepithelial mechanoreceptors located in the trachea, bronchi, and bronchioles.
- When these receptors are activated, a reflex response causes the ventilatory rate to increase as well as cough and bronchoconstriction.

# JUXTAPULMONARY-CAPILLARY RECEPTORS

- J receptors are located in the interstitial tissues between the pulmonary capillaries and the alveoli.
- When these J receptors are stimulated, a reflex response triggers rapid, shallow breathing.
- J receptors are activated by:
  - pulmonary capillary congestion
  - capillary hypertension
  - edema
  - lung deflation
  - serotonin
  - emboli

# FACTORS THAT MAY INCREASE VENTILATION DURING EXERCISE

- ❑ Reflexes originating from **body movement**
- ❑ Increase in body **temperature**
- ❑ **Adrenaline** release
- ❑ Impulses from the **cerebral cortex**
- ❑ **Later**: accumulation of **CO<sub>2</sub>** and **H<sup>+</sup>** generated by active muscles

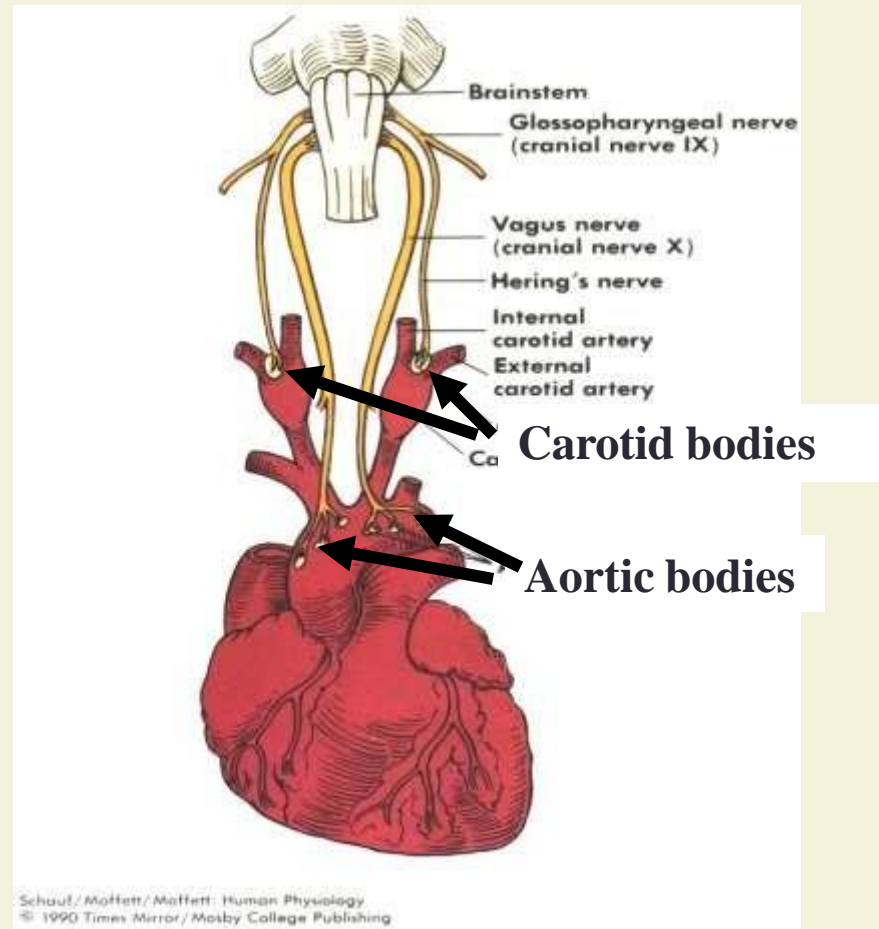
# INFLUENCE OF CHEMICAL FACTORS ON RESPIRATION

- The magnitude of ventilation is adjusted in response to three chemical factors:
  - *$P_{O_2}$ ,  $P_{CO_2}$ , and  $H$ .*
- Arterial blood gases are maintained within the normal range by varying the magnitude of ventilation (rate and depth of breathing) to match the body's needs for  $O_2$  uptake and  $CO_2$  removal.

# CHEMICAL CONTROL OF RESPIRATION

- ❑ An example of a **negative feedback control system**
- ❑ The controlled variables are the blood gas tensions, **especially carbon dioxide**
- ❑ **Chemoreceptors** sense the values of the gas tensions

# PERIPHERAL CHEMORECEPTORS



Sense tension of **oxygen and carbon dioxide;**  
**and [H<sup>+</sup>]** in the blood

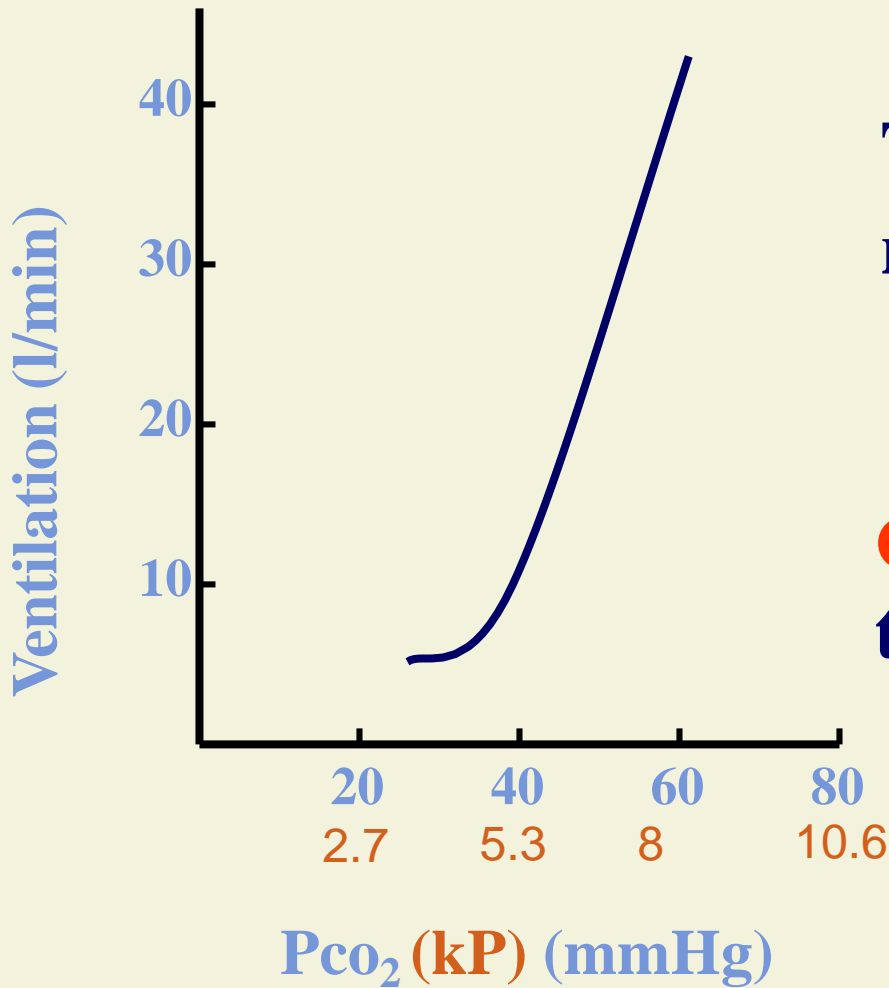


# CENTRAL CHEMORECEPTORS

- ❑ Situated near the surface of the **medulla** of the brainstem
- ❑ Respond to the **[H<sup>+</sup>]** of the cerebrospinal fluid (CSF)
- ❑ CSF is separated from the blood by the **blood-brain barrier**
  - ❑ Relatively **impermeable to H<sup>+</sup>** and HCO<sub>3</sub><sup>-</sup>
  - ❑ **CO<sub>2</sub> diffuses readily**
  - ❑ **CO<sub>2</sub> will work for first 2 to 3 days, then kidney compensation will occur through HCO<sub>3</sub> retention.**
- ❑ **CSF** contains less protein than blood and hence is **less buffered than blood**



# HYPERCAPNIA AND VENTILATION



The system is very responsive to P<sub>CO<sub>2</sub></sub>

CO<sub>2</sub> generated H<sup>+</sup> through the central chemoreceptors

# HYPOXIC DRIVE OF RESPIRATION

- ❑ The effect is all via the **peripheral chemoreceptors**
- ❑ Stimulated only when arterial **P<sub>O<sub>2</sub></sub>** falls to low levels
- ❑ Is **not important** in normal respiration
- ❑ May become important in patients with **chronic CO<sub>2</sub> retention** (e.g. patients with COPD)
- ❑ It is important at **high altitudes (acclimatization)**

# THE $H^+$ DRIVE OF RESPIRATION

- The effect is via the **peripheral chemoreceptors**
- **$H^+$  doesn't** readily cross the blood brain barrier ( **$CO_2$  does!**)
- The **peripheral chemoreceptors** play a major role in adjusting for acidosis caused by the addition of **non-carbonic acid  $H^+$**  to the blood (e.g. lactic acid during exercise; and diabetic ketoacidosis)
- Their stimulation by  **$H^+$**  causes **hyperventilation** and **increases elimination of  $CO_2$  from the body** (remember  **$CO_2$**  can **generate  $H^+$** , so its increased **elimination** help **reduce the load of  $H^+$**  in the body)
- This is important in acid-base balance

# Other factors in regulation of respiration in abnormal condition

- Brain edema    Cheyne - Stokes breathing
- Sleep apnoea
  - Obstructive sleep apnoea
  - Central sleep apnoea
- ❖ CPAP (continuous positive airway pressure )
- ❖ BiPAP (bi-level positive airway pressure)

# INFLUENCE OF CHEMICAL FACTORS ON RESPIRATION

CHEMICAL FACTOR	EFFECT ON THE PERIPHERAL CHEMORECEPTORS	EFFECT ON THE CENTRAL CHEMORECEPTORS
$\downarrow P_{O_2}$ 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 <60mm Hg
$\uparrow P_{CO_2}$ in the Arterial Blood ( $\uparrow H^+$ 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)
$\uparrow H^+$ in the Arterial Blood	Stimulates; important in acid–base balance	Does not affect; cannot penetrate the blood–brain barrier

THANK YOU