CONTROL OF RESPIRATION

(REGULATION OF RESPIRATION)

EFFECT OF HIGH ALTITUDE AND DEEP SEA DIVING ON RESPIRATION

Dr. R. Prasad,
Assistant Professor,
Department of Zoology,
Eastern Karbi Anglong College

The basic process of respiration changes with metabolic demand e.g., at rest body cells use about 200 mL of O_2 each minute while during strenuous exercise, it can increase by 15-30 fold in normal healthy adults & several times more (up to 30 fold) in endurance-trained athletes. Therefore, regulation of respiration assumes great importance.

Respiration is regulated by two types of mechanisms - the nervous mechanism and chemical mechanism.

Nervous regulation: Nervous system regulates the rate of alveolar ventilation in response to altered level of pO_2 and pCO_2 during physical exercise and other stress conditions. This control mechanism operates through a widely dispersed group of neurons known as **"respiratory centre"**, located bilaterally in the reticular substance of medulla oblongata & pons in the brain stem. Therefore, medulla has inspiration-expiration centre to regulate respiration process.

Nervous system has 3 major areas on the basis of their functions (Fig. 5.14):

1. **The medullary rhythmicity area** in the medulla oblongata (controls the basic rhythm of respiration). It is further made up of two groups of neurons :a) Dorsal Respiratory Group of neurons / DRG or **Inspiratory Area** and b) Ventral Respiratory Group/ VRG or **Expiratory Area**

2. **The pneumotaxic area** in the upper pons (by transmitting **inhibitory** impulses prevents overinflation of lungs).

3. **The apneustic area** in the lower pons(sends **stimulatory** impulses to Inspiratory Area and in turn inhibits expiration).

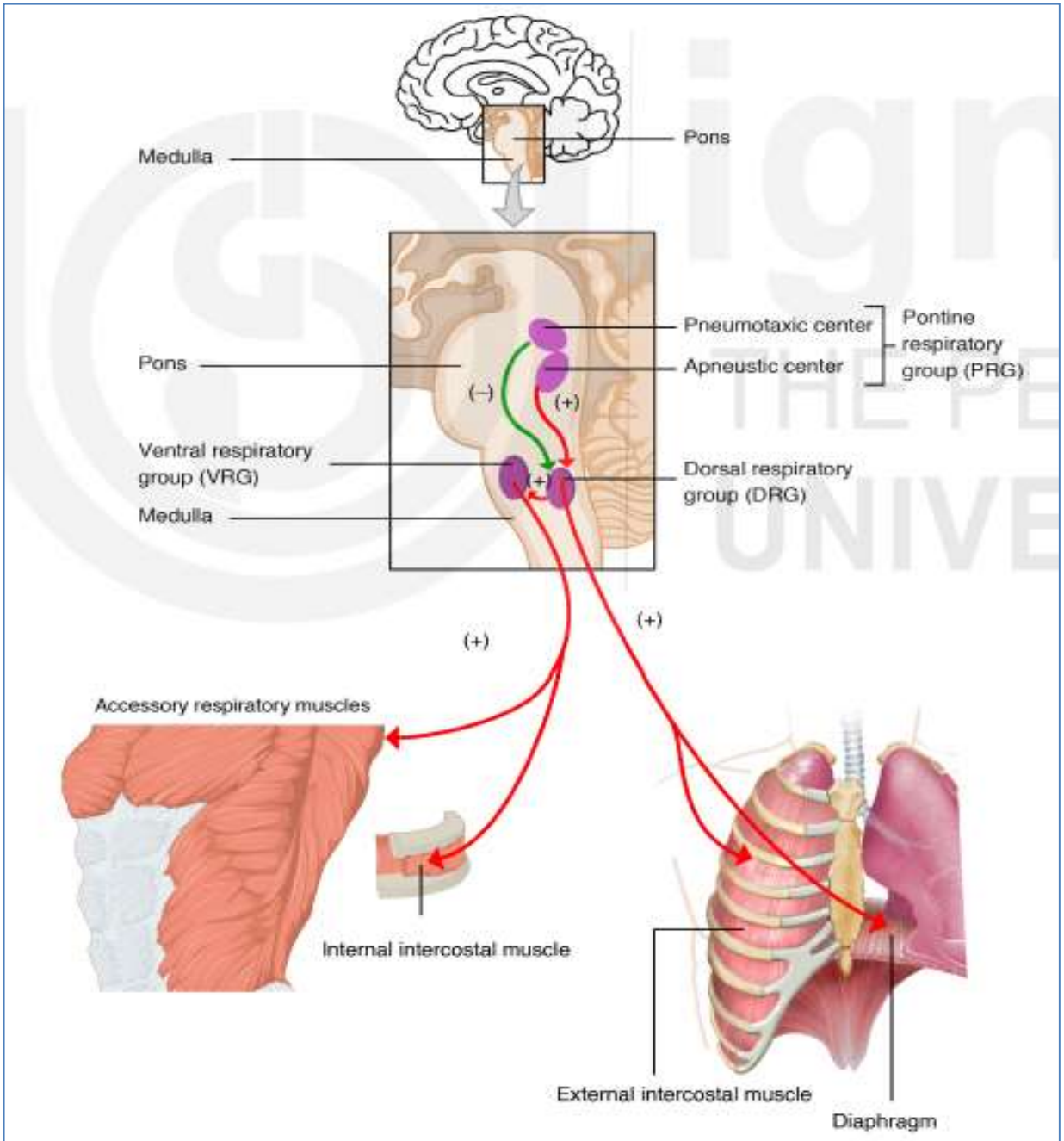


Fig. 5.14: Overview of regulation of Respiratory system (Image courtesy: Anatomy & Physiology http://cnx.org/content/col11496/1.6/

The function of the medullary rhythmicity area is to control the basic rhythm of respiration. It is the **inspiratory area which plays a more fundamental role** in the control of respiration. The excitement of its neurons generates nerve impulses for about 2 seconds. The neuronal activity seems to result from an inherent intrinsic excitability of inspiratory neurons themselves; therefore, they are thought to be **pacemaker neurons.** These impulses stimulate the inspiratory muscles causing their contraction and bring about **Inspiration** for 2 seconds. Then the neuronal signal suddenly comes to halt for 3 seconds bringing about **Expiration**. This is the basic rhythm of respiration.

The neurons of the **expiratory area** remain dormant during normal quiet respiration as expiration is passive. However, during forceful breathing they become active & send nerve Impulses. Impulses from the expiratory area cause contraction of the intercostal & abdominal muscles bringing about forceful expiration.

Chemical regulation

The sensors detecting chemical fluctuations are known as **chemoreceptors**. There are two types of chemoreceptors found in the body:

(1) **The central chemoreceptors** located bilaterally & ventrally in the reticular substance of medullary region of brain, are highly sensitive to changes in either blood CO_2 or H^+ concentration. It has excitatory effects on the Inspiratory Area ,increasing both the rate and depth of Inspiratory signal. And,

(2) **The peripheral chemoreceptors** found in the aortic arch & common carotid arteries, are more sensitive to pO_2 in the blood.

Other factors which influence respiration include; Inflation reflex/Herring-Breuer reflex (mechanical regulation of respiration), pulmonary irritant reflex, hypothalamic controls, cortical controls, temperature, pain, blood pressure & proprioreceptor stimulation etc.

EFFECT OF HIGH ALTITUDE AND DEEP SEA DIVING ON RESPIRATION

Classification of heights

* High altitude = 1,500–3,500 metres (4,900–11,500 ft)
* Very high altitude = 3,500–5,500 metres (11,500–18,000 ft)
* Extreme altitude = above 5,500 metres (18,000 ft)

* The death zone - altitudes above a certain point where the amount of oxygen is insufficient to sustain human life. This point is generally tagged as 8,000 m (26,000 ft) [less than 356 millibars of atmospheric pressure]

ACCLIMATIZATION AT HIGH ALTITUDES

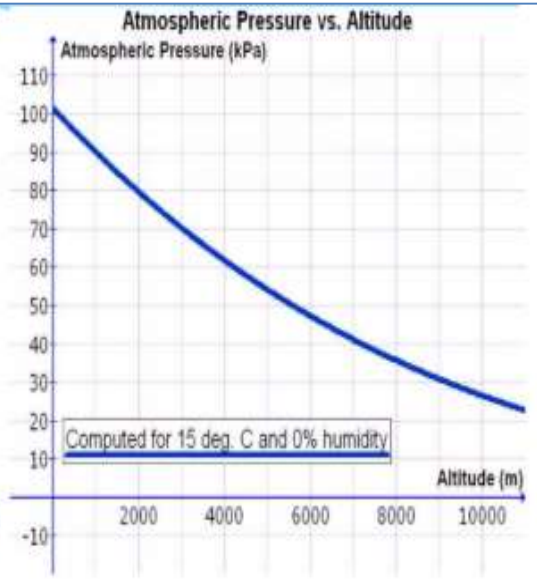
HYPERVENTILATION:

* Most important mechanism
* In normal ventilation

  PCO_2 = 40mm Hg, respiratory exchange ratio = 1, PO_2 = 3mm Hg.
* In hyperventilation – PCO_2 = 8mm Hg, alveolar PO_2 = 35mm Hg.
* *Mechanism* – hypoxic stimulation of peripheral chemoreceptors – decreased PCO_2 and alkalosis – inhibits hyperventilation – inhibition removed by excretion of excess HCO_3 by kidneys.
* Increased sensitivity of carotid bodies to hypoxia.

* Atmospheric pressure decreases with increase in altitude.
* At 5000m it is only half the normal pressure –

  ½ X 760=380mm Hg.
* So PO_2 of inspired gas = (380-47)*0.2093 = 70mm Hg



POLYCYTHEMIA

* Increased RBC concentration – increased Hb concentration – increases O_2 carrying capacity.
* Mechanism : hypoxemia – stimulates erythropoietin secretion from kidneys – stimulates bone marrow – polycythemia.
* Disadvantage – increases blood viscosity

ACCLIMATIZATION AT HIGH ALTITUDES

OTHER CHANGES:

* ODC –> right (at moderate heights) –d/t increased 2,3 DPG and respiratory alkalosis – promotes O2 release.
* ODC –> left (at very high altitudes) – d/t alkalosis – Increased O2 uptake form pulmonary capillaries.
* Number of capillaries per unit volume in peripheral tissues increases and changes occur in the oxidative enzymes in cells.
* Increase in maximum breathing capacity because the air is less dense, but O2 uptake declines above 4600m.
* Alveolar hypoxia – pulmonary vasoconstriction – increased pulmonary arterial pressure – increased work of right heart – hypertrophy.
* Increased pulmonary arterial pressure – pulmonary edema – d/t uneven arteriolar constriction and leakage from unprotected and damaged capillaries. The fluid has increased proteins

ACUTE MOUNTIAN SICKNESS

* Usually above 8,000 feet (2,400 meters) in un-acclimatized climbers.
* The faster you climb to a high altitude, the more likelihood of acute mountain sickness.
* Symptoms include – nausea, vomiting, headache, fatigue, dizziness, palpitation, loss of apetite and insomnia.
* Mechanisms postulated are cerebral edema and alkalosis due to hypoxemia (hypoxemia – arteriolar dilatation – limit of cerebral autoregulatory mechanisms – cerebral edema due to fluid transudition.)
* In more severe cases – high altitude pulmonary edema and high altitude cerebral edema develops.
* Symptoms – reduced by large doses glucocorticoids (decreases cerebral edema) and acetazolamide (decreases alkalosis –by inhibiting carbonic anhydrase).
* If not treated – may lead to ataxia, disorientation, coma and finally death – d/t tentorial herniation of brain tissue.

* Keys to preventing acute mountain sickness include:
  * Climb the mountain gradually
  * Stop for a day or two of rest for every 2,000 feet (600 meters) above 8,000 feet (2,400 meters)
  * Sleep at a lower altitude when possible
  * Learn how to recognize early symptoms of mountain sickness

RESPIRATORY CHANGES DURING DEEP SEA DIVING

* Diving – pressure increases by 1 atmosphere for every 10m (33ft) of descent – a non communicating gas cavity such as lung, middle ear or intracranial sinus – pressure difference causes compression on descent or over expansion on ascent.

* Hence scuba divers should exhale as they ascend to prevent over inflation and possible rupture of lungs.

* Increased density at depth increases the work of breathing – CO_2 retention

DECOMPRESSION SICKNESS

* Diving – high partial pressure of N_2 – forces poorly soluble N_2 into tissues (esp fat). The diffusion is slow because of the low solubility of N_2 and equilibration takes hours.

* Ascend – N_2 diffuses out from the tissues. Rapid ascend – bubbles of N_2 form – pain at the joints (bends), neurological symptoms – deafness, impaired vision and even paralysis may occur.

High altitude and deep sea diving both present unique challenges to the respiratory system due to altered pressures and gas concentrations. At high altitude, the primary challenge is reduced partial pressure of oxygen, leading to hypoxia and increased respiratory rate. Deep sea diving, on the other hand, involves increased pressure, which can lead to nitrogen narcosis, oxygen toxicity, and decompression sickness.

High Altitude:

Hypoxia:

Reduced atmospheric pressure at high altitude leads to lower partial pressure of oxygen, causing hypoxia (oxygen deficiency).

Hyperventilation:

The body initially compensates for hypoxia by increasing respiratory rate (hyperventilation) to increase oxygen intake.

Acclimatization:

With time, the body undergoes acclimatization, which includes increased red blood cell production and other physiological changes to better utilize available oxygen.

Respiratory Effects:

Increased respiratory rate, potential for dehydration due to increased insensible fluid loss, and possible bronchial irritation or asthma-like symptoms from breathing dry, cold air.

Altitude Sickness:

Rapid ascent to high altitudes can lead to acute mountain sickness (AMS), high-altitude pulmonary edema (HAPE), or high-altitude cerebral edema (HACE).

Deep Sea Diving:

Increased Pressure:

At depth, the pressure increases significantly, affecting gas solubility and partial pressures.

Nitrogen Narcosis:

Nitrogen, a common component of breathing gas, can become narcotic at depth, causing impaired judgment and coordination.

Oxygen Toxicity:

Breathing high concentrations of oxygen at pressure can lead to oxygen toxicity, damaging cells.

Decompression Sickness:

Ascending too quickly from a dive can cause dissolved nitrogen to form bubbles in the blood and tissues, leading to decompression sickness (the bends).

Increased Work of Breathing:

The pressure of the water and the resistance of the breathing apparatus increase the work of breathing.

Pulmonary Function:

Repeated deep dives can potentially lead to long-term adverse effects on the lungs, including small airways disease and accelerated loss of lung function, according to the European Respiratory Society.