Control of ventilation

Under construction / Forgotten

This article was marked by its author as *Under construction*, but the last edit is older than 30 days. If you want to edit this page, please try to contact its author first (you fill find him in the history (https://www.wikilectures.eu/index.php?title=Control_of_ventilation&action=history)). Watch the as well. If the author will not continue in work, remove the template `{{Under construction}}` and the page.

Last update: Tuesday, 02 Feb 2021 at 9.54 pm.

Control of Ventilation (by Coiled)

CNS control of ventilation: The control of breathing by the CNS can be split into three main groups: Those controlled by the group of structures located in the Medulla (the Dorsal Respiratory Group and the Ventral Respiratory Group), and structures located in the Pons (Pneumotaxic Centre, and Apneustic Centre), and finally those control centers that are located above the brainstem. REMEMBER, the most important two clusters of controllers are in the PONS and MEDULLA 1) Structures located in medulla: Dorsal Respiratory Group (DRG) and Ventral Respiratory Group (VRG) DRG: DRG is the crucial structure for spontaneous inspiration. Located at the dorsal part of the medulla. Uses a “ramp signal” to stimulate diaphragm and other respiratory muscles (You can imagine ramp signal as a burst of action potentials. The frequency of action potentials progressively increases with the depth of the breath. After the burst ceases, the expiration fallows.). This is a pretty good mechanism how to control the length and depth of inspiration. The ramp signal is controlled by the respiratory centres located higher in the brain stem. Receives many peripheral sensory inputs - centripetal fibers of n. glossopharyngeus and n. vagus end at this group and thus bring input from peripheral chemoreceptors, stretch receptors, baroreceptors, irritant receptors etc. VRG: This group is practically inactive at rest. Is activated during intensive breathing (exercise etc.). Causes increase in both, expiration and inspiration, although expiration support seems to dominate. Located in ventral part of medulla. Receives signals from DRG.

2) Structures located in pons: Pneumotaxic and Apneustic centers

Pneumotaxic center: - Located in upper pons. - Shuts off the ramp signal of DRG and thus ends the inspiration and starts expiration. - Its stimulation of DRG increases frequency of ventilation. - Prevents over-inflation of the lungs. - Massive activation of this centre can increase the respiratory rate to 40 breaths per minute.

Apneustic center: - Only hypothetical. Nobody has actually proved it exists! - Its location is assumed to be in lower pons. - Regulates the amount of air inspired, making sure that a 2/3 ratio of inspiration to expiration is maintained. - Hypothetically this centre would take over after the superior control from pneumotaxic center has failed. When the apneustic centre takes the breathing pattern changes to prolonged inspiratory gasps.

3) CNS structures located above brainstem: Limbic system – emotions affect ventilation, also regulation of ventilation through ANS Hypothalamus – thermoregulation, regulation of ANS Cerebral Cortex – voluntary control of respiratory muscles Cortical-bulbar tract Cortical-spinal tract – direct control of ventilation

Odine’s curse (congenital central hypoventilation syndrome (CCHS) or primary alveolar hypoventilation) This can be congenital or acquired (due to trauma, stroke or tumour of the Dorsal Respiratory Group and other centres in brainstem). The patient has to think about his breathing, and also during sleep artificial ventilation must be provided. Note that there is no damage to the cortical spinal tract. A new therapy for such an illness is the stimulation of phrenic nerves via an implant.

Sensory input from Lungs also affects the ventilation:

Stretch – inflation and deflation These receptors protect lungs against possible rupture in over-inflating and against collapse of the lungs in deflation. Hering-Breuer reflex: Stretch receptors in lungs -> Vagus -> respiratory groups in brainstem - this turns off ramp signal -> expiration starts. This reflex seems to only be protective, not a normal control of breathing, because it is activated only after the volume of inflation exceeds 3 times the normal tidal volume. J receptors J stands for juxtaposition – they lie next to alveolar capillaries. Some sources say, that these receptors trigger the dyspneic feeling during lung oedema. Irritant receptors To cough out solid pieces we inhale. Chemoreceptors in bronchi These sense toxic gasses and, in order to prevent further inhalation of the noxious gasses are able to stop breathing at once!

Chemical control of ventilation:

Peripheral chemoreceptors: Location – carotid bodies (more important), aortic bodies. The centripetal information from aortic bodies comes into the brainstem through fibers of the cranial nerve X (Vagus), the information from carotid bodies comes through fibres of cranial nerve IX (Glossopharyngeal). The primary function of these receptors is to monitor pO2!!!, but also pH!!! (acidosis stimulates ventilation), as well as pCO2 (although the increase has to be higher than for central chemoreceptors) and also Potassium concentration in blood. Note that the receptors only

become massively activated once the pO2 falls below 60 mmHg (NB: In case of people with COPD, this fall has to be even below 40 mmHg, before any notable stimulation occurs.) Actually you are already unconscious when the hypoxic drive takes over!!!! In contrast with the central chemoreceptors, we can basically say, that these receptors will not get adapted (see below). In other words the low pO2 will stimulate the respiratory drive on and on… NB: In natives living for centuries in lower partial pressures (Andes, Himalayeas) the respiratory response to hypoxia/hypoxemia is blunted…its genetics…

Central chemoreceptors: Central chemoreceptors are located in chemosensitive area, which is located just beneath the ventral surface of upper medulla. There is a direct connection with DRG (inspiration group). Central chemoreceptors are continuously and very precisely controlling ventilation and nature made it so, that pCO2 is the major regulator of ventilation, not pO2 (The pO2 is a strong stimulator of ventilation only after it falls below 60 mmHg in arterial blood – this is done through peripheral chemoreceptors, not the central ones – see below). The central receptors are sensitive to pH of CSF (which basically means arterial pCO2 – this molecule can very easily pass through the BBB – then in CSF reacts with water with the assistance of carboanhydrase – this will change the pH to which central chemoreceptors are sensitive to. Thus you can say, that central chemoreceptors are monitoring arterial pCO2 (the more precise answer is that they are sensitive to protons in CSF – in tests, you have to carefully read the question – sometimes they ask only if pO2 or PCO2, than obviously in connection with central chemoreceptors, you will answer pCO2, sometimes they go more deeply into the matter and ask about pH. NB: CSF is more acidic and has no proteins as buffers (that means that pH changes are normally larger then it would be in blood, just because of the lower buffer capacity of CSF) – thus any bleeding (haemoglobin) or infection (proteins) can buffer the pH changes in CSF and thus impair the sensitivity (in another words, function) of central chemoreceptors. The central chemoreceptors are very sensitive to acute pCO2 changes in blood and instantly change the respiratory rate to normalize the pCO2 levels. This precise control is impaired, if the pCO2 levels are continuously increased (due to hypoventilation – as in COPD etc.) for longer then 3 days. This process of central chemoreceptor impairment is called “adaptation”. This adaptation process is due to slow penetration of protons, which are increased in CSF and are then able to slowly penetrate through BBB to blood and also thanks to bicarbonate, which also slowly penetrates from blood to CSF – the outcome is that although the levels of pCO2 are increased in blood, the pH in CSF is normalized and the ventilator drive is turned off. Thus central stimulation through increased CO2 works very fast, but will get very soon turned of through adaptation. (NB: this is actually the reason, why you are not suppose to give 100% oxygen to a person with COPD because they have already adopted their central chemoreceptors and they breathe only thanks to hypoxic/hypoxemic drive through the peripheral chemoreceptors (this is not as efficient and thus COPD patients have higher levels of pCO2). If you give them 100% oxygen, then you will turn of also this peripheral drive and the person will stop breathing – thus their levels of pCO2 can rise to dangerous levels! This theory was abandoned!!!!

Special types of intoxication in connection with ventilator response:

Intoxication with CO: Already small doses of CO will intoxicate you – but these small doses will not change the partial pressures of pO2 or PCO2, thus already toxic levels of CO will not trigger immediately the respiratory drive through chemoreceptors.

Intoxication with cyanide: Cyanide blocks the respiratory chain in mitochondria – thus the receptors will think that there is absolute lack of oxygen in the blood and will start massive ventilation.