CONTROL OF BREATHING

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Anaesthesia affects respiratory function in different ways. Knowledge of respiratory physiology is necessary to understand these effects.

Physiological control systems involving the nervous system usually have three components. These are:

- a central controlling area
- an afferent pathway and
- an efferent pathway.

The neurones (nerve cells) of the controlling area integrate the information from other parts of the body and produce a coordinated response. This response from the central controlling area is carried to the various organs and muscles along efferent pathways. The input to the central controlling area is from the various sensors via the afferent pathways.

Central Controlling Area

The central controlling area for breathing, called the respiratory centre, is in the lower part of the brain stem, in the medulla oblongata. There are "inspiratory neurones" which are active during inspiration and inactive during expiration. Other neurones are active during expiration but not inspiration-the "expiratory neurones". These two groups of neurones automatically maintain a rhythmic cycling pattern of inspiration and expiration. This automatic rhythm can be modified by the afferent information.

Afferent Supply

(1) Central chemoreceptors. Chemoreceptors are cells that respond to chemical stimuli. There are cells in the floor of the fourth ventricle (part of the brain stem) that respond to the acidity of the cerebrospinal fluid (CSF) and the output from these cells influences breathing. The acidity of any fluid is measured by the pH; this is related to the number of hydrogen ions in the solution. The normal pH of the body is 7.4; values higher than this represent alkaline conditions in the body, with a lower hydrogen ion concentration, and values of pH less than 7.4 represent acidic conditions, with a higher hydrogen ion concentration. The cells in the floor of the fourth ventricle respond to the pH of the CSF. An acidic CSF causes hyperventilation this is the reason for dyspnoea with conditions such as diabetic ketoacidosis. An alkaline CSF inhibits the respiratory centre. Carbon dioxide in the blood can rapidly diffuse across into the CSF, and there is a balance between the level of carbon dioxide, hydrogen ion and bicarbonate ion in the CSF. If the carbon dioxide in the blood increases (eg following exercise), then the carbon dioxide, hydrogen ion and bicarbonate ion concentrations increase correspondingly in the CSF. This increase in CSF acidity causes hyperventilation which lowers the carbon dioxide concentration in the blood. A low blood carbon dioxide level (hypocarbia) has the opposite effect and may occur, for example, following controlled ventilation during anaesthesia. This may delay the return of spontaneous breathing at the end of surgery.

(2) Peripheral chemoreceptors. The carotid and aortic bodies are small pieces of tissue that contain chemoreceptors which respond to the oxygen and carbon dioxide concentrations in arterial blood. The carotid body is the more important of the two and is situated at the division of the common carotid artery into the external and internal carotid arteries in the neck. The aortic body is found on the aortic arch. The information from the carotid body is carried along the glossopharyngeal nerve (the ninth cranial nerve) and the information from the aortic body is along the vagus nerve (the tenth cranial nerve), to the respiratory centre. The output from the carotid body is thought to provide information to allow immediate regulation of breathing, breath by breath, by the respiratory centre. In normal people, if the arterial blood reaching the carotid body has a partial pressure of oxygen of 10kPa (80mmHg) or a carbon dioxide partial pressure of more than approximately 5kPa, (40mmHg), then there is an immediate and marked increase in breathing. These limits can be modified by disease or age; for example, people with chronic bronchitis may tolerate an increased concentration of carbon dioxide or a decreased concentration of oxygen in the blood.

(3) Brain. Breathing can be influenced by other parts of the brain. We can all consciously breathe deeply and more rapidly (called hyperventilation), and this can happen, for example, before starting strenuous exercise. Intensely emotional situations, for example, distressing sights, will also cause hyperventilation. Hyperventilation is also part of the response to massive blood loss. This response is co-ordinated by the autonomic system in the hypothalamus and the vasomotor centre in the brain stem.

(4) Lung. There are various receptors in the lung that modify breathing. Receptors in the wall of the bronchi respond to irritant substances and cause coughing, breath holding and sneezing. In the elastic tissues of the lung and the chest wall are receptors that respond to stretch. The exact function of these receptors is not fully understood but are thought to be responsible for various reflexes that have been discovered in laboratory

studies of animals. There are stretch responses that occur when the lung and chest wall are distended and inhibit further inspiration. This is an obvious safety mechanism to avoid overdistension. Conversely, when the lung volume is low, then there are opposite reflexes. A small increase in lung size may stimulate stretch receptors to cause further inspiration. This can sometimes be seen in anaesthetised patients who have been given an opioid; spontaneous breathing may be absent or very slow, but if the patient is given a small positive pressure breath by the anaesthetist, then inspiration is stimulated and the patient takes a deep breath. This reflex may also have some function in newborn babies just after delivery, when small breaths may stimulate further inspiration.

There are also stretch receptors in the blood vessels in the lung. If these are stretched, as in heart failure, the response is to hyperventilate. The information from these receptors in the lung is carried to the respiratory centre along the vagus nerve.

Efferents

The efferent nerves from the respiratory centre pass down the spinal cord to the diaphragm, intercostal muscles and accessory muscles of inspiration in the neck. The diaphragm is supplied by the phrenic nerve that is formed in the neck from the spinal nerves, C3, 4 and 5. The intercostal muscles are supplied by the segmental intercostal nerves that leave the spinal cord between TI and TI2. The accessory muscles in the neck are supplied from the cervical plexus. During normal breathing, inspiration is an active muscular process. Expiration is passive and relies on the natural elasticity of the tissues to deflate the lung. The most important muscle for inspiration is the diaphragm. Any disease that affects the efferent pathways from the respiratory centre to C3, 4 and 5 and then the phrenic nerve to the diaphragm, may cause severe difficulty in breathing. Trauma to the cervical cord, above C3, is normally fatal for this reason.

Anaesthetic drugs and respiration

Opioid drugs, such as morphine or fentanyl, depress the respiratory centre's response to hypercarbia. These effects can be reversed by naloxone. Volatile anaesthetic agents depress the respiratory centre in a similar fashion, although ether has less effect on respiration than the other agents. Volatile agents also alter the pattern of blood flow in the lungs, resulting in increased ventilation/perfusion mismatch and decreasing the efficiency of oxygenation. Nitrous oxide has only minor effects on respiration.

The depressant effects of opioids and volatile agents are additive and close monitoring of respiration is necessary when they are combined. When oxygen is not available respiration should always be supported during anaesthesia.