Effects of a change in posture on ventilatory function.

Gravity has a major influence on the distribution of ventilation and perfusion, therefore with the change to supine postion the posterior areas of the lung receive increased perfusion and are more compressed. During normal breathing there is minimal change in V/Q ratios however in healthy lungs because both ventilation and perfusion undergo similar changes . Change from erect to supine results in an increase in pulmonary blood volume by almost a third due to return of blood from the periphery. Of particular importance is the effect of the abdominal contents in the supine postion. These push the diaphram superiorly and subsequently cause a reduction in the FRC. This is becomes important with respect to the closing capacity which increases with age, closing capacity will exceed FRC at the age of 44 in average subjects. The tendency to airway closure however may be counteracted by increased diaphram stretch capacity. Change in posture typically result in a decrease of anatomical deadspace of one third. This results in a volume of VD/VT ratio change from 34% erect to 30% when supine (improved ratio). Diffusing capacity is substantially improved when in the supine position despite decreased lung volume, this is likely because of the increased pulmonary blood volume.

GRAVITY
- Ventilation base → posterior
- Perfusion base → posterior
- V/Q ratio unchanged in healthy patients
- Blood volume ↑ by a third
- Improved diffusing capacity (because ↑BV)

ABDOMINAL PRESSURE
- Reduces FRC by compressing diaphram

ANATOMICAL DEADSPACE
- Decreased by a third
- Improves VD/VT from 34% to 30%

Table 28.1 Relationship of temperature and saturated vapour pressure

Temperature (°C)	Saturated vapour pressure (mmHg)	Saturated vapour pressure (kPa)	Absolute humidity (g/m^3)
0	4.6	0.6	4.8
10	9.2	1.2	9.3
20	17.5	2.3	17.1
30	31.3	4.2	30.4
34	39.9	5.3	37.5
37	47.1	6.3	43.4
40	55.3	7.4	51.7
46	78.0	10.4	68.7

Humidification

The upper airway normally warms, moistens and filters inspired gas. When these functions are impaired by disease, or when the nasopharynx is bypassed by endotracheal intubation, artificial humidification of inspired gases must be provided.

Absolute humidity (AH) – the total mass of water vapour in a given volume of gas at a given temperature (g/m3)

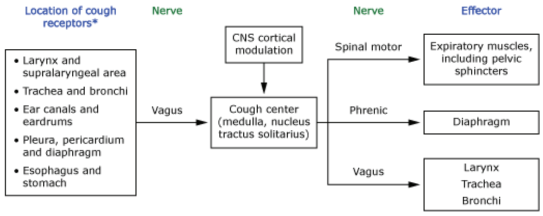
Relative humidity (RH) – the actual mass of water vapour (per volume of gas) as a percentage of the mass of saturated water vapour, at a given temperature.

Saturated water vapour exerts a saturated vapour pressure (SVP). As the SVP has an exponential relation with tempera ture, addition of further water vapour to the gas can only occur with a rise in temperature (see adjacent table and note that at 37 degrees the SVP is 47)

Latent heat of vapourisation is the energy absorbed per gram in the phase change from liquid to gas ($\mathcal{L}_{H_2O}$ = 540 cal.g^{-1})

Heating and humidification of dry gas are progressive down the airway, with an isothermic saturation boundary (i.e. 100% RH at 37 C or AH of 43 g/m3) just below the carina. Under resting conditions, approximately 250 ml of water and 1.5 kJ (350 kcal) of energy is lost from the respiratory tract in a day. A proportion (10–25%) is returned to the mucosa during expiration due to condensation. The need for humidification during endotracheal intubation and tracheostomy is unquestioned. As the upper airway is bypassed, RH of inspired gas falls below 50% with adverse effects, including: 1. increased mucus viscosity 2. depressed ciliary function 3. cytological damage to the tracheo-bronchial epithelium, including mucosal ulceration, tracheal inflammation and necrotising tracheobronchitis 4. microatelectasis from obstruction of small airways, and reduced surfactant leading to reduced lung compliance 5. airway obstruction due to tenacious or inspissated sputum with increased airway resistance

The **cough reflex** is a protective reflex which is initiated as a protective mechanism against chemical and mechanical irritants. It derives primarily from airway receptors in the larynx, trachea and bronchi although other locations have been identified. The information is fed back to the cough centre in the medulla via the vagus nerve, where cortical input may modify or partial initiate the reflex. Information is then sent through the effector nerves which consist of the spinal, phrenic and vagus. The muscles of the cough reflex then coordinate in a three phase process. The first stage involves inspiration of a suficient amonut of air for the expiratory activity. The compressive phase involves expiration against a closed glottis, cuasing a transient increase in pressure of the thorax, arterial blood and the CSF of upn to 300mmHg. The final stage is the expulsive stage in which the glottis opens allowing rapid expiratory flow through the respiratory tract.



HORMONAL CHANGES
- PROGESTERONE - sensitises central chemoceptors
  - decreased PCO2 (30mmHg)
  - increased PO2 (103mmHg)
- OESTROGEN - increased blood volume
  - greater oxygen delivery

MECHANICAL CHANGES
- DIAPHRAM DISPLACED - decreased FRC
  - Less O2 reserve

During **pregnancy** there are significant changes to respiration which is a result of both the mechanical changes of childbearing (which therefore are most significant in the third trimester) and hormonal changes which occur through-out the pregnancy. One of the most important changes in the sensitisation of chemoreceptors by progesterone which increases six-fold during pregnancy. The sensitisation of chemoreceptors leads to three fold increase in the CO2/ventilation response curve and a two-fold increase in the hypoxic response curve. Whilst RR is unchanged the tidal volume increases up to 40% at full term. The consequence of this increased ventilation is resting PCO2 levels decrease to around 30mmHg (35 normally), and PO2 levels increase to around 103 (95). Oestrogen increases the blood volume and this leads to increased Oxygen delivery. In the final trimester the diaphram is displaced cephalad by the expansion of the uterus and the abdominal contents and this is exacerbated in the supine position. The result of the diaphram displacement is a substaintal reduction in FRC, this is particularly important as it removes an important store of O2 and may be crucial in the setting of anaesthesia.

Non respiratory functions of the lung

can be seperated into three main categories. The pulmonary circulation undertakes two important roles. Firstly the lungs can act as an important reservoir of blood through mainly distension but also recruitment of vessels, increasing its volume without major increases in pulmonary pressures. The second role involves the extensive filtration of the blood removing material in the blood stream which would have very deleterious effects in the arterial system such as thrombo-emboli reaching the brain. The lung also acts as an important defence against inhaled substances. This is achieved through mechanical means such as the action of the cilliated epithelium and the production of mucus, and chemical means through antimicrobial peptides in the airway lining fluid and surfactant. Lastly immune systems provide defences from a hummoral perspective with IgA and the cellular immunity of macrophages and immunologically active epithelial cells lining the airway. The third main category of non-respiratory functions of the lung is the processing of endogenous and exogenous compounds. This includes the inactivation of amines such as noradrenaline, the activation of peptides such as angiotensin I and inactivation of bradykinin and the activation of arachidonic acid derivatives into eicosanoids such as leukotrienes involved in bronchoconstriction. The lung also has an effect on exogenous drugs but usually through disposition (retention in the lungs) rather than actual metabolism.

PULMONARY CIRCULATION
- Blood Reservoir
- Blood Filter

DEFENCE AGAINST INHALED SUBSTANCES
- Mechanical - mucus, cillia
- Chemical - antimicrobials, sufactant
- Immune - IgA, Macrophages

METABOLIC FUNCTIONS
- Activation -angiotensin I, leukotrienes,
- Inactivation -bradykinin, noradrenaline

The respiratory effects of altitude.

As a patient ascends from sea level the atmospheric pressure decreases. The partial pressure of O2 (21%) remains the same however at around 5500m above sea level the atmosphere is halved (760 down to 380). The resultant decrease in PO2 is from 160 to 80. This is complicated at the alveolar by the SVP of 47mmHg at body temp. The formula for Alveolar PO2 is (Barometric Pressure - 47)0.21, therefore as the BMP drops the SVP becomes an increasingly important component and at 19200m the BMP = 47mmHg therefore the Alveolar PO2 is 0. Ascent to altitude presents three main challenges to the respiratory system. The first is the most important and is the increasing hypoxia, the next is the decrease humidity and the third is extreme cold which occurs in outdoor environments. The bodies response to hypoxia is marked by three phases, the first is the acute response which involves a rapid increase in ventilation due to carotid body feedback. This increases for about 5-10 minutes. The second phase is the hypoxic ventilatory decline which last 10-20 minutes until it reaches a plateau still above resting ventilation. The third phase is a gradual increase in ventilation to a new increased minute ventiation baseline over eight hours if the patient remains at elevation. Whilst PCO2 decrease blunts this response there is a reset of the central chemoceptors which leads to a lower baseline PCO2. There is an initial DPG driven right shift of the Hb-O2 curve to improve O2 offloading at the tissues. Ongoing ascent eventually leads to central hypoxic depression of the respiratory centres and ultimately apnoea and death. Long term adaptation in addition to the increased MV is polycythemia, left shift of the Hb-O2 curve to encourage O2 uptake and inceased vascularity to the heart and striated muscles. Less beneficial changes include hypoxic pulmonary vasoconstriction and associated pulmonary hypertension, which in addition to the increased viscosity (polycythemia) leading to to right heart strain and RVH.

High pressure and diving

At a depth of 10 metres the atmospheric pressure is doubled (2ATA), at 20 metres it is three times that of the surfance and so on. The consequence of this in a significant increase in partial pressures of gases such that the SVP become less significant with regards to alveolar air. From a mechanical perspective increased pressure leads to increased peripheral blood return adn therefore increased pulmonary blood volume, often leading to the ANP driven diuresis noted in divers. As the pressure increases the denisty of gas also increases greatly increasing the work of breathing due to resistance to turbulent flow (this is the main benefit of helium). With respect to oxygen there are several important consequences. Even at constant normobaric 100% O2 there is a risk in the long term of pulmonary toxicity which leads to pulmonary absorption collapse and ulitmately may lead to acute lung injury. Oxygen levels above 2ATA (10ATA on air) may lead to oxygen convulsions (Paul Bert Effect) which are poorly understood but believed to be related to GABA and NO. Nitrogen is a narcotic at increased pressures and may lead to narcosis, it is also very dense and increases work of breathing, finally its solubility means that it is deposited in tissues and difusses out at a decreased rate leading to nitrogen bubbles in the blood stream and the risk of decompression illness.