Hyperventilation (HVS) Effects on pH & CO2

- HVS produces far-ranging physiological effects via alteration of pH and depletion of CO2, resulting in respiratory alkalosis - acute or chronic.
- Alkalosis disrupts calcium ion balance, producing symptoms of hypocalcaemia. (Seifter 2011)
- Respiratory alkalosis is the most frequent acid-base disturbance encountered in clinical practice. (Biff 2012)

Definitions

**Hypocapnia:** Deficiency of CO2 in the blood, resulting from hyperventilation, leading to respiratory alkalosis - induces vasoconstriction, leading to a 20% decrease in microcirculatory blood flow, and a 9% reduction in tissue oxygen pressure (pt02) - in local skeletal muscle. (Gustafsson et al 1993)

**Hypoxia:** Reduction of oxygen (O2) supply to tissue, below physiological levels, despite adequate perfusion of the tissue by blood. (cf. Anoxia)

**Hyperventilation** (“the induction and/or maintenance of levels of CO2 tension in the arterial blood, below the normal range”) lowers intracranial pressure by the induction of cerebral vasoconstriction, with a subsequent decrease in cerebral blood volume ...... [possibly} to ischemic levels.”(Stocchetti et al 2005)

Respiratory or metabolically induced alkalosis, results in altered vascular smooth muscle tone, as does the resulting change in calcium concentration. (Stocchetti et al 2005)

**Smooth-muscle cells are also found in fascia** – as noted by Staubesand & Li (1996), who documented their presence in human fascia profunda (lower leg). They also noted the rich presence of sympathetic nerve fibers in these tissues.

Simons & Mense (1998) observe that **fascial stiffness plays a major role in EMG-silent, resting, muscle tone**

Masi & Hannon (2008) have confirmed that human resting muscle tone may be significantly influenced by changes in **fascial stiffness.**

Hyperventilation sequence:

- Reduced vasomotor tone (cerebral, coronary, cutaneous)
- Diminished O2 availability
- Increased neuronal excitability
- Loss of buffer base (HC03 excretion)
- 02 uptake impaired (Bohr effect)
- CO2 threshold reset lower (Nixon 1993,Wilkins et al 2000)
- Neuronal hyperexcitability, paraesthesias (hands, trunk, mouth)
- Hypertonicity, cramps, carpopedal spasm: possibly due to lower serum phosphate or hypocalcemia (Magarian 1982)
- Cardiac muscle hyperexcitability may cause arrhythmia
- PaCO₂ reduction may lead to coronary spasm
- Chest pain via aerophagia pressure on diaphragm (Evans & Lum 1981)
- Intercostal muscle fatigue produces dull chest pain (Evans & Lum 1981)
- Retrosternal pain into neck & arms (pseudoangina) (Magarian 1982)
- Headache/nasal congestion (Bartley 2005)
- Esophageal reflux

Thoracic breathing leads to lower CO₂ levels, and lower scores on Functional Movement Screening (Bradley H Esformes J 2014)

**Assessment** is via observation, use of Nijmegen questionnaire and/or capnography (van Dixhoorn & Folgering 2015)

**Breathing Rehabilitation** (Hodges et al 2013)

**Improve Respiration**
- Reduce tonic/excessive muscular activity
- Maintain deep muscle activity during respiration
- Gradually increase inspiratory volume to threshold
- Optimize breathing pattern (slow exhalation)

**Improve Posture**
- Retrain neutral posture (functional retraining)

**Optimize Thoracic Dynamic Control**
- Thoracic spine, rib cage mobility/motor control
diaphragm – through exercise & manual therapy

**Enhance Efficiency of Breathing Pattern**
- Optimize movement and muscle activity
- Increase fitness – pulmonary rehabilitation

Retrain breathing pattern with motor control progressions

**Osteopathic Approach**
- Address efficiency of all fascial, muscular and osseous features
- Mobilize thoracic spine and rib articulations
- Attention to diaphragm & pelvic floor
- Deactivate myofascial trigger points
- Educational & Retraining exercises
- Ergonomics, posture, diet, sleep, exercise, stress management
References