The Influence of Respiration on Muscle Tone
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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 O_2 availability
- Increased neuronal excitability
- Loss of buffer base ($HC0_3$ excretion)
- 0_2 uptake impaired (Bohr effect)
- CO_2 threshold reset lower *(Nixon 1993,Wilkins et al 2000)*

- Neuronal hyperexitability, parasthesias (hands, trunk, mouth) hypertonicity, cramps, carpopedal spasm : possibly due to lower serum phosphate or hypocalcemia *(Magarian 1982)*
- Cardiac muscle hyperexcitability may cause arrhythmia
- $PaCO_2$ 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

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