

Research & Thoracic Structure & Function

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HVS & BPD defined

HVS (hyperventilation syndrome) occurs when the rate of breathing exceeds metabolic demands, resulting in hemodynamic and chemical changes that produce characteristic symptoms.

BPD (breathing pattern disorders) include all modifications of 'normal' breathing, with HVS as the extreme of this.

Respiratory alkalosis is one of the first changes to manifest with arterial pH rising further into the alkaline range (normal is ± 7.4).

Two Additional Key Definitions

hypocapnia

Deficiency of carbon dioxide in the blood, resulting from hyperventilation and eventually leading to respiratory alkalosis.

hypoxia

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

O₂-CO₂ balance

"Maintaining O₂ and CO₂ within balanced limits is a complex task for the body, because the supply of each gas fluctuates with each breath. This tidal oscillation must be smoothed out so that the brain and bodily tissues receive a steady supply of O₂, and also so that CO₂ in the body remains at a stable level.....We live in a narrow zone of homeostasis, bordered on both sides by physiological disaster.

Much of what goes wrong with breathing involves attempts to avoid this disaster."

Gilbert C. in Chaitow, Bradley & Gilbert, 2002, Multidisciplinary Approaches to Breathing Pattern Disorders. Churchill Livingstone, Edinburgh

Just one sigh...

Careful inquiries as to the precipitating causes of episodes of HVS helps both with the diagnosis and focusing on choice of treatment.

Nixon (1993) suggests that there are often attacks where there is no preceding stressful event.

In chronic hyper-ventilators the respiratory centre may have been reset to tolerate lower than normal partial pressure of arterial carbon dioxide (PaCO₂).

In such patients a single sigh, or one deep breath, may reduce the PaCO₂ enough to trigger symptoms.

Nixon P G F .1993. The grey area of effort syndrome and hyperventilation : from Thomas Lewis to today. Journal of the Royal College of Physicians of London. Vol 27: no 4:377-383

How widespread is HVS/BPD?

Acute hyperventilation represents only approximately 1% of all cases of hyperventilation, well outnumbered by chronic hyperventilation.

Chronic HVS can present with a myriad of respiratory, cardiac, neurologic, or GI symptoms, without any clinically apparent overbreathing by the patient. In the US as many as 10% of patients in a general internal medicine practice are reported to have HVS as their primary diagnosis.

•Lum L 1987 Hyperventilation syndromes in medicine and psychiatry J. Royal Society of Medicine 229-231

•Newton E 2001 Hyperventilation Syndrome
<http://www.emedicine.com/>

HVS/BPD and Gender

HVS/BPD is female dominated, ranging from a ratio of 2 :1 to 7 :1

Women may be more at risk because of hormonal influences, since progesterone stimulates respiratory rate, and in the luteal (post ovulation/pre-menstrual) phase, CO₂ levels drop on average 25%. Additional stress can then, "increase ventilation at a time when carbon dioxide levels are already low."

Damas-Mora J, Davies L, Taylor W, Jenner F A. 1980
Menstrual Respiratory Changes and Symptoms. British Journal of Psychiatry. 136, 492-497

Symptoms of HVS/BPD

"HVS patients are often pursued relentlessly with every investigative device known to modern science, and end up with the label of "anxiety state" and the implication that they are inadequate or in some way inferior. Symptoms may be resulting from a deficiency of carbon dioxide, bicarbonate, oxygen, and calcium ions; to name but a few of the well-known biochemical disturbances which accompany acute hypocapnia."

HVS Symptoms

CARDIOVASCULAR: palpitations, tachycardia, precordial pain, Raynaud's phenomenon

NEUROLOGICAL: Central: dizziness, disturbed consciousness/vision.

PERIPHERAL: Paraesthesia, tetany (rare)

RESPIRATORY: Shortness of breath, "asthma", chest pain

GASTROINTESTINAL: Globus, dysphagia, epigastric pain

MUSCULOSKELETAL: Muscle pains (particularly thorax), tremors, tetany

PSYCHIC: Tension, anxiety

GENERAL: Fatigability, weakness, exhaustion, sleep disturbance, nightmares

Lum L.C. 1988 Hyperventilation : The tip and the iceberg. Applied Respiratory Psychophysiology.

Understanding Anxiety

Despite advances in the understanding of the nature of anxiety-related responding during periods of elevated bodily arousal, it is not evident by what psychological mechanisms anxiety is produced and maintained. To address this issue researchers have increasingly employed biological challenge procedures to examine how psychological factors affect anxious responding, during elevated bodily arousal. Of the challenging procedures, hyperventilation and inhalations of carbon dioxide-enriched air have been among the most frequently employed, and a relatively large body of literature using these procedures has now accumulated. Overall, we conclude challenge research is a promising paradigm to examine the influence of psychological variables in anxious responding, and that such work will likely be enhanced with greater attention to psychological process issues

Zvolensky M Eifert G 2001 A review of psychological factors/ processes affecting anxious responding during voluntary hyperventilation and inhalations of carbon dioxide-enriched air Clinical Psychology Review 21(3) 375-400

BPD effects on deconditioned individuals

Nixon & Andrews (1996) have summarized the emerging symptoms resulting from hypercapnoea in a deconditioned individual, as follows ::

"Muscular aching at low levels of effort; restlessness and heightened sympathetic activity; increased neuronal sensitivity; and, constriction of smooth-muscle tubes (e.g. the vascular, respiratory and gastric-intestinal) can accompany the basic symptom of inability to make and sustain normal levels of effort."

Nixon P Andrews J 1996 A study of anaerobic threshold in chronic fatigue syndrome (CFS) Biological Psychology 43(3):264

Habitual BPD - pure habit ?

Lum (1984) discussed the reasons for people becoming hyperventilators

"Neurological considerations can leave little doubt that the *habitually* unstable breathing is the prime cause of symptoms. Why they breathe in this way must be a matter for speculation, but manifestly the salient characteristics are *pure habit*."

Is breathing retraining an answer ?

Breathing retraining has been used to correct hyperventilation. Lum reported that more than 1000 anxious and phobic patients were treated using breathing retraining, physical therapy and relaxation. Symptoms were usually abolished in one to six months with some younger patients requiring only a few weeks.

At 12 months 75% were free of all symptoms, 20% had only mild symptoms and about one patient in twenty had intractable symptoms.

Lum L 1984 Editorial : Hyperventilation and anxiety state. Journal Royal Society of Medicine January p1-4

Core stability, the diaphragm and BPD

Diaphragmatic and transversus abdominis tone are key features in provision of core stability (Panjabi 1992, O'Sullivan 1997).

McGill (1995) notes reduction in support offered to the spine occurs if there is both a load challenge to the low back, combined with a breathing challenge. "Modulation of muscle activity needed to facilitate breathing may compromise the margin of safety of tissues that depend on constant muscle activity for support".

After approximately 60 seconds of hypercapnoea, the postural (tonic) and phasic functions of both the diaphragm and transversus abdominis are reduced or absent (Hodges 2001)

*Hodges P et al 2001 Postural activity of the diaphragm is reduced in humans when respiratory demand increases. Journal of Physiology 537(3): 999-1008

*McGill S et al 1995 Loads on spinal tissues during simultaneous lifting and ventilatory challenge. Ergonomics. 38(9):1772-1792

*O'Sullivan P et al 1997. Altered patterns of abdominal muscle activation in patients with chronic low back pain. Australian Physiotherapy 43, 91-98.

*Panjabi M 1992. The stabilizing system of the spine. Part 1. J Spinal Disorders 5:383-389

Respiratory alkalosis

The result of increased ventilation, during which the rate of CO₂ exhalation exceeds the rate of its accumulation in the tissues, is **respiratory alkalosis**, characterized by the decrease in CO₂ and an increase in pH.

This induces vascular constriction, decreased blood flow as well as inhibition of oxygen transfer from haemoglobin to tissue cells (due to the **Bohr effect**). (Pryor & Prasad 2002)
This leads to an accumulation of incompletely oxidised products of metabolism due to the activation of anaerobic energy pathways. The products of the anaerobic pathway are acids (lactic acid, pyruvic acid). (Fried 1987)

•Fried R 1987 Hyperventilation Syndrome John Hopkins University Press
•Pryor J Prasad S 2002 Physiotherapy for respiratory and cardiac problems (3rd edition) Churchill Livingstone Edinburgh

Fluctuating Alkalosis

Lum notes, "Alkalosis alone cannot fully explain the symptoms. Altitude adaptation allows residents of high altitudes to remain well, despite chronic respiratory alkalosis. In symptomatic hyperventilation however, the PCO₂ fluctuates, often wildly, *causing constantly changing pH in nerve cells and tissue fluid to which no adaptation is possible*....Significant amounts of CO₂ can be lost in a few minutes of overbreathing, immediately causing respiratory alkalosis. Compensation, by excretion of bicarbonate, is relatively slow and may take hours or days."

Lum L 1994 Hyperventilation Syndromes In : Timmons B Ley R. (eds) Behavioral and Psychological Approaches to Breathing Disorders. Plenum Press New York

Bohr effect

The Bohr effect states that an increase in alkalinity (decrease in CO₂) increases the affinity of Hemoglobin for O₂. (Note : the lungs are more alkaline than the rest of the body - enhancing O₂ uptake). The O₂ molecule is therefore less likely to release its oxygen in an alkaline environment

Increased O₂-Hemoglobin affinity also leads to changes in serum calcium & red cell phosphate levels (Levitsky 1995, George 1964)
The resulting hypoxia shifts the system from oxidative to anaerobic metabolism, resulting in increased lactic acid.

Muscles become prone to fatigue, dysfunction (e.g. cramp), and trigger point evolution.

Loss of intra-cellular Mg occurs as part of the renal compensation mechanism for correcting alkalosis.
Supplementary magnesium can correct a tendency to hyperventilation. (Pereira 1988)

•George S 1964 Changes in serum calcium, serum phosphate and red cell phosphate during hyperventilation. New Engl J Med, 1964; 270:726-728

•Pereira O 1988 The Hazards of Heavy Breathing, New Scientist, Dec: 46-48

•Levitsky 1995 L Pulmonary Physiology, McGraw Hill, 4th Edition.

Physiological responses to Bohr effect

•Bicarbonate is excreted to balance pH, depleting plasma buffer capacity.

•A shift occurs in the electrolytic makeup of the extracellular fluid, involving loss of cations Ca, K, Mg.

•As bicarbonate levels in the plasma changes, there is disruption of the formation of proteins, nucleic acids, lipids & carbohydrates, and a change in the carboxylation intensity.

•As the Bohr effect comes into operation, ischemia increases, oxygenation decreases, as does the formation of ATP.

•Smooth muscle constriction occurs

Courtney R 2002 The Buteyko Method, an osteopathic approach to asthma. Osteopathy Today July 2002 pp14-19

Physiology of smooth muscle constriction

"Sympathetic activity resulting in catecholamine release is termed adrenergic stimulation.... In general alpha-adrenergic receptors are found in *smooth-muscle* (gut, blood vessel walls etc) and stimulation of those receptors produces constriction of the smooth muscle. **In the blood vessels this means they get narrower, and blood flow is restrained.** Beta-adrenergic receptors are found in some blood vessel smooth muscle (and in heart muscle), in those cases, adrenergic stimulation will inhibit muscle constriction, allowing dilation...and less restrained blood flow" (Naifeh 1994)

Naifeh K 1994 Basic Anatomy and Physiology of the Respiratory System, In : Timmons B Ley R. (eds) Behavioral and Psychological Approaches to Breathing Disorders. Plenum Press New York

Colonic spasm

Symptoms attributable to hyperventilation are common among patients with the irritable bowel syndrome. A study was conducted to assess the effects of hyperventilation on colonic tone and motility and to discover if hypocapnia was critical to elicit the response.

Hypocapnic hyperventilation (low CO₂ blood levels) unlike eucapnic hyperventilation (normal CO₂ blood levels) produces an increase in colonic tone and phasic contractility in the transverse and sigmoid regions.

The findings are consistent with inhibition of sympathetic innervation to the colon, or direct effects of hypocapnia on colonic smooth muscle, or both. These physiological gut responses suggest that some of the changes in colonic function are caused by altered brain or autonomic control mechanisms.

Ford MJ, Camilleri MJ, Hanson RB, 1995

Hyperventilation, central autonomic control, and colonic tone in humans Gut 37:499-504

Pseudo coronaries and HVS

A study evaluated a series of 45 patients with chest pain, who had normal coronary arteries on angiography, and who were ultimately diagnosed with HVS/BPD.

Over a 3.5 year average follow-up 67% had made subsequent emergency visits for chest pain, and 40% had been re-admitted to rule out myocardial infarction. (Newton 2000)

The implication is that many individuals with HVS/BPD experience severe and genuinely distressing symptoms, and represent a considerable medical expense in excluding more serious pathology

Newton E 2001 Hyperventilation Syndrome
<http://www.emedicine.com/>

Smooth Muscle (and fascial) contraction

•Staubesand & Li (1996, 1997) studied fascia in humans with electron photomicroscopy and found smooth muscle cells widely embedded within the collagen fibres. They describe a rich intrafascial supply of capillaries, autonomic nerves and sensory nerve endings and concluded that these intrafascial smooth muscle cells enable the autonomic nervous system to regulate a fascial pre-tension, independently of muscular tonus.

•They suggest that this understanding of fascia as an actively adapting organ may have far reaching clinical implications.

•Schleip (2002) notes that elevated pH (alkalinity), resulting from hyperventilation, would produce smooth muscle contraction and even spasm in fascial tissues

•Schleip R 2003 Fascial plasticity - a new neurobiological explanation Journal of Bodywork and Movement Therapies 7(1):11-19

•Staubesand J, Li Y 1996 Zum Feinbau der Fascia cruris mit besonderer Berücksichtigung epi- und intrafaszialer Nerven. Manuelle Medizin 34:196-200

•Staubesand J, Li Y 1997 Begriff und Substrat der Faziensklerose bei chronisch-venöser Insuffizienz. Phlebologie 26: 72-79

Other SMC sites in connective tissue

There is increasing interest on the possible effects that active SMC contractility may have in the many fascial/connective tissue sites in which their presence has now been identified, including cartilage, ligaments, spinal discs and the lumbodorsal fascia.

•Ahluwalia S 2001 Distribution of smooth muscle actin-containing cells in the human meniscus Journal of Orthopaedic Research 19(4):659-664

•Hastreite D et al 2001 Regional variations in cellular characteristics in human lumbar intervertebral discs, including the presence of -smooth muscle actin. Journal of Orthopaedic Research 19(4):597-604

•Meiss RA 1993 Persistent mechanical effects of decreasing length during isometric contraction of ovarian ligament smooth muscle. J Muscle Res Cell Motil 14(2): 205-18

•Murray, M Spector, M 1999 Fibroblast distribution in the anteromedial bundle of the human anterior cruciate ligament: the presence of alpha-smooth muscle actin-positive cells Journal of Orthopaedic Research 17(1):18-27

•Yahia L, Pigeon P DesRosiers E 1993 Viscoelastic properties of the human lumbodorsal fascia Journal of Biomedical Engineering 15:425-429

BPD The fatigue - pain cascade

Progression from acute to chronic dysfunction resulting from, or aggravated by, breathing pattern disorders:

- Increased oxygen requirements are not met & CO2 levels drop
- Oxygen delivery is retarded (due to Bohr effect & smooth muscle contraction)
- Metabolic waste retention occurs
- Protein synthesis disturbed
- Degenerative fibre damage starts
- Trigger points evolve
- Tendon overload & pain evolve
- Periosteal pain points develop
- Joint crowding occurs
- Firing sequences are disturbed
- Active trigger points produce pain & dysfunction
- Global patterns become disturbed (posture, respiration) creating more myofascial distress



Physiologically unsustainable adaptive demands lead to chronic myofascial and joint problems.

Add to this increased smooth muscle tonus, sustained sympathetic arousal, lowered pain thresholds, calcium and magnesium deficits, chronic fatigue, 'brain fog'

Ischemia and the Evolution of pain

Persistent ischemia seems to account for reduced O2 tension at Myofascial trigger point (MTrP) sites (Bruckle 1990)

Hypoxia is a potent stimulator of bradykinin release, encouraging perpetuation of MTrP sensitization and persistence of pain (Baldry 2001)

A combination of circulatory stasis and hypoxia is likely responsible for the presence of 'ragged red' fibres found in the vicinity of MTrPs (Bengtsson 1986)

Such evidence is found in both MTrP pain syndrome and FMS, apparently resulting from hypoxic induced alteration in ATP production (Henriksson & Mense 1994)

•Bruckle W et al 1990 Gewebe-po2-messung in der verspannten rückenmuskulatur. Zeitung Rheumatol. 49:208216

•Baldry P 2001 Myofascial pain and fibromyalgia syndromes Churchill Livingstone Edinburgh

•Bengtsson A et al Muscle Biopsy in primary FMS Scand.Jnl. Rheumatology 15:1-6

•Henriksson K Mense S 1994 Pain & Nociception in FMS Pain Reviews 1:245-260

Summary

Structural modifications of spinal and thoracic structures, including the accessory respiratory muscles (upper fixators of the shoulders) and the diaphragm, follows inevitably from changes in normal respiratory function - as demonstrated in breathing pattern disorders (BPD).

Normalization of dysfunctional breathing habits, and the myriad associated symptoms, via breathing rehabilitation, is possible through relearned habits of use.

Evidence suggests that rehabilitation is dramatically enhanced and maintained when structural features (muscles, joints etc) are mobilized prior to, and during during the course of, self-applied breathing retraining exercises.

The process of rehabilitation takes anything from 3 to 6 months