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.”


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.


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
**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, "increased ventilation at a time when carbon dioxide levels are already low."


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**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 hypcapnia.

**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


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**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.


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**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.


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**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."


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**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).


**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). (Prasanth S 2002 Physiotherapy for respiratory and cardiac problems (3rd edition) Churchill Livingstone Edinburgh)

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**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 over-breathing, immediately causing respiratory alkalosis. Compensation, by excretion of bicarbonate, is relatively slow and may take hours or days.”

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**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 mechanism for correcting alkalosis.

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

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**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

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**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).


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**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.

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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.


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 infratascial supply of capillaries, autonomic nerves and sensory nerve endings and concluded that these infratascial 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.


-Baudsens et al 1996 Zum Feinbau der Fascia cruris mit besonderer Berücksichtigung epi- und intrafaszialer Nerven. Manuelle Medizin 34:196-200


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.


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 extanted due to Bohr effect & smooth muscle contraction
- Metabolic waste retention occurs
- Protein synthesis disturbed
- Oxygengenerative fibre damage starts
- Trigger points evolve
- Tendon overload & pain evolve
- Periostial 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 (MTJP) sites (Bruckel, 1990)

-Hypoaxia is a potent stimulator of bradykinin release, encouraging perpetuation of MTJP sensitization and persistence of pain (Baldry, 2001)

-A combination of circulatory stasis and hypoaxia is likely responsible for the presence of ‘ragged red’ fibres found in the vicinity of MTJPs (Bengtsson, 1986)

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


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


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 retreating exercises.

The process of rehabilitation takes anything from 3 to 6 months.