Contribution

Breathing pattern disorders, motor control, and low back pain

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Abstract

Motor control is a key component in injury prevention. Loss of motor control involves failure to control joints, commonly because of incoordination of the agonist-antagonist muscle co-activation. Three subsystems work together to maintain spinal stability: The central nervous subsystem (control), the osteoligamentous subsystem (passive), and the muscle subsystem (active).

There is evidence that the effects of breathing pattern disorders, such as hyperventilation, result in a variety of negative psychological, biochemical, neurological and biomechanical influences and interferences, capable of modifying each of these three subsystems. Breathing pattern disorders (the extreme form of which is hyperventilation), automatically increase levels of anxiety and apprehension, which may be sufficient to alter motor control and to markedly influence balance control. Hyperventilation results in respiratory alkalosis, leading to reduced oxygenation of tissues (including the brain), smooth muscle constriction, heightened pain perception, speeding up of spinal reflexes, increased excitability of the corticospinal system, hyperirritability of motor and sensory axons, changes in serum calcium and magnesium levels, and encouragement of the development of myofascial trigger points – all or any of which, in one way or another, are capable of modifying normal motor control of skeletal musculature.

Diaphragmatic and transversus abdominis tone are key features in provision of core stability, however it has been noted that reduction in the support offered to the spine, by the muscles of the torso, may occur if there is both a load challenge to the low back, combined with a breathing challenge. It has been demonstrated that, after approximately 60 seconds of hypercapneoa, the postural (tonic) and phasic functions of both the diaphragm and transversus abdominis are reduced or absent. Smooth muscle cells, now known to be widely embedded in connective tissues (including spinal discs, and lumbar fascia) constrict during periods of respiratory alkalosis, with as yet undetermined effects on joint stability and fascial tone. Breathing rehabilitation offers the potential for reducing the negative influences resulting from breathing pattern disorders.

Keywords: breathing pattern disorder, hyperventilation, respiratory alkalosis, motor control, musculoskeletal pain

INTRODUCTION

Motor control is a key component in injury prevention and loss of motor control involves failure to control joints, commonly because of incoordination of the agonist-antagonist muscle co-activation[1]. According to Panjabi[2] three subsystems work together to maintain spinal stability:

- the central nervous subsystem (control)
- the osteoligamentous subsystem (passive)
- the muscle subsystem (active).

Anything that interferes with any aspect of these features of normal motor control, may contribute to dysfunction and pain.

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An increased rate of ventilation, such as prevails with hyperventilation, during which the rate of carbon dioxide (CO_2) exhalation exceeds the rate of its accumulation in the tissues, produces respiratory alkalosis, characterised by the decrease in CO_2 and an increase in pH. This induces vascular constriction, decreasing blood flow, as well as inhibiting transfer from haemoglobin, of oxygen, to tissue cells (due to the Bohr Effect).[3]

The Bohr effect states that an increase in alkalinity (decrease in CO_2) increases the affinity of haemoglobin (Hb) for oxygen (O_2). The Hb molecule is therefore less likely to release its oxygen in tissues that have become increasingly alkaline due to overbreathing.[4] Increased O_2-Hb affinity also leads to changes in serum calcium and red cell phosphate levels.[5] Additionally there is a loss of intra-cellular Mg^{2+} as part of the renal compensation mechanism for correcting alkalosis.[4,5] Muscles affected in this way inevitably become

prone to fatigue, dysfunction (e.g. cramp), and trigger point evolution.[6]

Acute episodes of hyperventilation represent only approximately 1% of all cases, far outnumbering chronic patterns.[7] Chronic hyperventilation leads to hypocapnoea (reduced levels of carbon dioxide), and can present with a myriad of respiratory, cardiac, neurological or gastrointestinal symptoms, without any clinically apparent overbreathing by the patient. In the United States as many as 10% of patients in a general internal medicine practice are reported to have HVS as their primary diagnosis.[7,8]

Studies show that, relative to men, women have a higher rate of respiration and a greater tendency to respiratory alkalosis, which is exaggerated during the luteal (progesterone) phase of the menstrual cycle.[9] Hyperventilation syndrome (HVS) and breathing pattern disorders (BPD) are therefore female dominated, with a female:male ratio ranging from 2:1 to 7:1. Women may be more at risk because of hormonal influences, since progesterone stimulates respiration, and in the luteal (post ovulation/pre-menstrual) phase, CO_2 levels drop on average 25%. Additional stress can subsequently, "increase ventilation at a time when carbon dioxide levels are already low".[10]

Lum[11] points out that there are many people with BPD who have been labelled as asthmatics. "Thirty percent of cases of asthma are known to be induced by emotion or exercise, and many symptoms are common to hyperventilation and to asthma: intermittent, labored breathing; relief from bronchodilators (transient in hyperventilation); exercise; cough; fear, anxiety and panic. It is thus a matter of individual preference whether the clinician calls such cases asthma or hyperventilation. The distinction is important. Treatment of hyperventilation cures the patient. The asthmatic is condemned to a life of medication."

While investigation as to the precipitating causes of episodes of hyperventilation may help with both the diagnosis and choice of treatment, Nixon[12] suggests that there are often attacks where there is no preceding stressful event. In chronic hyperventilators the respiratory centre may have been reset to tolerate a lower than normal partial pressure of arterial carbon dioxide ($PaCO_2$). In such patients a single sigh, or one deep breath, may reduce the $PaCO_2$ sufficiently to trigger symptoms.

Lum[7] has 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."

Respiratory alkalosis and its effects

Discussing hyperventilation syndrome and its links to vasospasm, Castro et al[13] observe that both the acute and chronic forms of the syndrome are characterized by hypocapnoea and respiratory alkalosis. "The chronic form has a blood pH closer to the normal range, and is usually more symptomatic, in that only mild hyperventilation is necessary to produce a substantial increase in the degree of hypocapnoea … The underlying mechanisms of the syndrome are cerebral vasoconstriction, due to hypocapnoea and a decrease in the delivery of oxygen by haemoglobin."

Respiratory alkalosis 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 such as lactic acid. and pyruvic acid.[14] This acidification is more extreme in deconditioned individuals. When ATP production is supplemented by anaerobic glycolysis, lactate accumulates in muscle cells and the bloodstream – reducing pH. Relative acidosis then encourages bicarbonate retention, resulting in increased CO_2 production, stimulating a more rapid breathing rate, leading to the respiratory threshold being breached. In a deconditioned individual this threshold is lower, resulting in dyspnoea and fatigue early in aerobic activity. The deconditioned individual relies more on anaerobic metabolism for energy supply.

Outcomes of deconditioning include:

1. Loss of muscle mass

2. Decreased ability to use energy substrates efficiently

3. Decreased neuromuscular transmission

4. Decreased efficiency in muscle fibre recruitment with indications of disruption of normal motor control being apparent.[15]

Nixon and Andrews[16] have summarised the emerging symptoms resulting from hypocapnoea 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."

Lum[7] notes, "Alkalosis alone cannot fully explain the symptoms [of chronic hyperventilation]. Altitude adaptation allows residents of high altitudes to remain well, despite chronic respiratory alkalosis. In symptomatic hyperventilation however, the PCO_2 fluctuates, often wildly, causing constantly changing pH in nerve cells and tissue fluid to which no adaptation is possible…significant amounts of CO_2 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."

Low back pain, balance and anxiety

Anxiety and apprehension are closely associated with altered breathing patterns, and breathing pattern disorders are in turn exaggerated by anxiety and apprehension.[17,18] Maintaining body balance and equilibrium is a primary role of functionally coordinated muscles, acting in task specific patterns, and this is dependent on normal motor control.[19]

Balaban and Theyer [20] have examined the neurological basis of links between balance control and anxiety, based upon neural circuits that are shared by pathways that mediate autonomic control, vestibulo-autonomic interactions, and anxiety: "The core of this circuitry is a parabrachial nucleus network, consisting of the parabrachial nucleus and its reciprocal relationships with the extended central amygdaloid nucleus, infralimbic cortex, and hypothalamus. Specifically, the parabrachial nucleus is a site of convergence of vestibular information processing, and somatic and visceral sensory information processing, in pathways that appear to be involved in avoidance conditioning, anxiety, and conditioned fear."

Klein[17] reports that hyperventilation, and resultant alkalosis, is capable of triggering anxiety and/or panic (and associated balance control changes) when (as is commonly the case) it is interpreted by the individual as representing a danger of suffocation.

Abnormal breathing patterns such as hyperventilation lead to elevated reports of somatic symptoms, including disorientation. There is evidence that the central changes that accompany hyperventilation may influence balance system functioning. Healthy individuals exhibit a substantial increase in sway following voluntary hyperventilation, and this postural instability may be linked to peripheral and central changes in somatosensory function.[21]

Low back pain often involves altered muscle length relationships, postural changes, muscular imbalances, variations in location of the centres of mass and of pressure.[22,23] Unsurprisingly, in the presence of such changes, associated with chronic low back pain, the speed and intensity of muscular contractions are commonly altered[24] with deep segmentally related muscles losing both contraction speed and intensity, while over activity and tonic contraction occurs in the larger multi-segmental muscles.[25,26] All these changes lead to low back pain patients moving differently, compared to healthy individuals.[27]

Increased anxiety levels, caused or aggravated by disordered breathing patterns, such as hyperventilation, are capable of amplifying many of these changes. Put simply, the responses of the motor system alter under conditions of pain and anxiety, due to modified cerebral processing.[28] The amygdala appear to play a pivotal role in the transmission and interpretation of fear and anxiety. The neuronal interactions between the amygdala enable the individual to initiate adaptive behaviours to threat, based upon the nature of the threat and prior experience. There is mediation between the efferent pathways involving the amygdala, locus coeruleus, hypothalamus, and autonomic, neuroendocrine, and skeletal-motor responses associated with fear and anxiety.[29]

Anxious, apprehensive thoughts have been shown to have an effect on the functioning of muscles. Lotze et al[30] using functional MRI scans have demonstrated that the cortical activity involved in thinking about a movement is similar to the cortical activity associated with the movement itself. It appears that simply talking about painful experiences increases activity in associated muscles in chronic low back pain patients.[31] Therefore, there is ample evidence that anxiety regarding movement, pain and re-injury can all modify motor behaviour.[32,33]

Anxiety and other emotions have also been shown to encourage recruitment of a small number of motor units that display almost constant, or repeated, activity when influenced psychogenically. In one study, low amplitude myoelectric activity (measured using surface electromyography) was evident even when muscles were not being employed in situations of mental stress.[34] "A small pool of low-threshold motor units may be under considerable load for prolonged periods of time…motor units with Type 1 [postural] fibres are predominant among these. If the subject repeatedly recruits the same motor units, the overload may result in a metabolic crisis." This aetiology parallels the proposed evolution of myofascial trigger points, as suggested by Simons et al.[6]

Neuronal excitability

There appear to be both biochemically induced, as well as psychological effects, deriving from breathing pattern disorders. Mogyoros[35] states: "The thresholds of human sensory and motor axons are altered during hyperventilation. Hyperventilation does not alter conduction velocity, refractoriness or super-normality, implying that the hyperventilation-induced increase in excitability is not the result of conventional depolarization, as seems to occur during ischaemia. These results suggest that hyperventilation has a rather selective action on the threshold channels... The greater expression of threshold channels in sensory [rather] than in motor fibres, can explain why hyperventilation induces paraesthesiae before fasciculation, and why only paraesthesiae occur during ischaemia." [35]

Seyal et al[36] note that hyperventilation increases the excitability of both cutaneous and motor axons, and that in experimental animals, HVS increases excitability of hippocampal neurons. Their research, involving healthy humans, demonstrates that hyperventilation increases the excitability of the human corticospinal system.

Respiratory alkalosis, resulting from low $PaCO_2$, which is almost always the result of hyperventilation, automatically lowers calcium ion levels in the plasma, precipitating hyperirritability of motor and sensory axons [37] Lum [38] reports,: "During moderate hyperventilation, loss of CO_2 ions from neurons stimulates neuronal activity, causing increased sensory and motor discharges, muscular tension and spasm, speeding of spinal reflexes, heightened perception (photophobia, hyperacusis) and other sensory disturbances. More profound hypocapnoea, however, increasingly depresses activity. This parallels the clinical state: initial alertness with increased activity, progressing through decreased alertness, to stupor and coma."

Combinations of inflammatory mediators, together with altered tissue pH, effectively induce sensitisation more markedly than chemical mediators alone.[39] Fluctuations in $PaCO_2$, resulting from overbreathing, can have a

destabilising effect on the autonomic nervous system, leading to sympathetic dominance, with patients often in a state of arousal.[40] Mean urinary excretion of adrenaline in hyperventilators may up to three times greater than normal.[41]

Influence of myofascial trigger points

Myofascial trigger points are commonly a source of pain and dysfunction in the low back.[42,43,6] There appear to be a variety of possible influences operating:

Simons and Travell[6] have noted that ischemia is a precursor to the evolution of myofascial trigger points (MTrPs). Persistent ischemia, such as prevails with respiratory alkalosis, seems to account for reduced O_2 tension at MTrP sites[44] They further report that, "a muscle that contains an active trigger point shows electromyographic activity 'at rest' when it is stretched to, or beyond, the point of pain."

Baldry[45] observes that hyperventilation induced hypoxia, is a potent stimulator of bradykinin release, encouraging perpetuation of MTrP sensitisation, and persistence of pain.

Using a novel microdialysis technique Shah et al[46] have shown that at the nidus of an active trigger point, bradykinin levels are significantly higher (as were substance P, calcitonin gene-related peptide, norepinephrine, tumor necrosis factor-alpha, and IL-1) compared with latent trigger points and normal tissue.

An altered pH in the local chemical environment of peripheral nociceptors, such as occurs with respiratory alkalosis, helps to induce mechanical sensitisation and ischaemic pain.[47,48]

Bengtsson[49] has suggested that a combination of circulatory stasis and hypoxia is probably responsible for the presence of 'ragged red' fibres in the vicinity of MTrPs. Such tissues, found in both MTrP pain syndrome and fibromyalgia, apparently result from hypoxia induced alteration in ATP production[50]

Brucini et al[51] have shown that trigger points, "increase motor unit activity of muscles in both the pain and reference zone"

More recently Lucas[52] has shown that the presence of latent trigger points alters activation (firing) sequences in entire kinetic chains, for example involving latent trigger points in upper trapezius, on abduction at the shoulder joint.

A relevant question might be posed as to whether trigger points can at times be functional (to induce stabilisation of hypermobile structures, for example) in local and/or target tissues[53] since they represent an energy efficient means of assisting sustained increased contracture, a chemical rather than action potential-mediated shortening of the muscle fibers?[54]

The diaphragm

It seems likely that habitual, chronic, breathing pattern disorders interfere with normal function of key stabilizing muscles such as transversus abdominis and the diaphragm.

Hypercapnoea (increased levels of CO_2) can be induced by having the subject inhale through a long tube, increasing the dead space in the lungs, or by having the subject breath air containing higher than normal levels of CO_2. Either method appears to be preferable to voluntary hyperventilation which can have unpredictable outcomes. Hypercapnoea triggers an artificially rapid breathing rate, the effects of which can then be studied.

Using a 10% CO_2 gas mixture to elevate breathing, McGill[55] noted that reduction in the support offered to the spine, by the muscles of the torso, may occur if there is both a load challenge to the low back, combined with a breathing challenge (shovelling snow is given as an easily understood example in real-life rather than under research conditions). "Modulation of muscle activity needed to facilitate breathing may compromise the margin of safety of tissues that depend on constant muscle activity for support".

Hodges[56] demonstrated (using a long-tube breathing method) that after approximately 60 seconds of hypercapneoa the postural (tonic) and phasic functions of both the diaphragm and transversus abdominis are reduced or absent. "The present data suggest that increased central respiratory drive may attenuate the postural commands reaching motoneurons. This attenuation can affect the key inspiratory and expiratory muscles, and is likely to be co-ordinated at a pre-motoneuronal site." Hodges further hypothesises: "Although investigation of spinal mechanics is required to confirm the extent to which spinal control is compromised by increases in respiratory demand, it is hypothesised that such a compromise may lead to increased potential for injury to spinal structures and reduced postural control. During strenuous exercise, when the physical stresses to the spine are greater, the physiological vulnerability of the spine to injury is likely to be increased."

Fascial considerations

Staubesand and Li[57] studied fascia in humans using electron photomicroscopy and found smooth muscle cells (SMC) widely embedded within the collagen fibres. They describe a rich intrafascial supply of capillaries, autonomic 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.

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 ligaments, [58] menisci,[59] spinal discs[60] and, as suggested by the research of Yahia et al,[61] on the lumbodorsal fascia, which has been shown by Barker and Briggs[62] to extend from the pelvis to the cervical area: "Both superficial and deep laminae of the posterior layer are more extensive superiorly than previously thought."

One result of respiratory alkalosis, with an as yet unspecified degree of impact on low back pain and function, as pH rises markedly, involves the potential for increased contractility of SMC. The research of Yahia et al[59] suggests the possibility of (smooth) muscle cells in fascia offering a protective role, although at the time there was no histological proof of their

presence in these tissues. They have demonstrated a progressive stiffening of lumbar fascia (human cadaver specimens) when subjected to repetitive isometric strain forces. Yahia et al[59] also cite research[63,64,65] into the effects of alterations in pH on modification of the viscosity of connective tissue (the 'swelling rate'), another phenomenon with a possibly protective, and certainly an influential, role in low back stability.

SMC contractility directly impacts on circulation to muscle and brain tissues, by reducing blood vessel diameter and therefore oxygenation, leading to increased likelihood of fatigue.[66]

A further connective tissue consideration involves hypermobility which has been shown to be a major risk factor in the evolution of low back pain.[67] Breathing pattern disorders have been found to be much more common in hypermobile individuals (where fascial stability is most needed) -often associated with chronic pain syndromes.[68,69,70]

A pertinent question arises: In a hypermobile individual who hyperventilates, is the altered breathing pattern functional – a means of increasing tone and stability in lax connective tissue structures, via the effect of respiratory alkalosis on contractile smooth muscle cells?

Breathing retraining

Reducing levels of apprehension, anxiety and fear may be seen to have the potential for allowing a variety of features, including motor control, to improve. Breathing retraining is one way of achieving this objective. There is good evidence that breathing rehabilitation is a useful method for achieving reduced anxiety/panic levels and for improving postural control and somatic complaints, such as low back pain.[16,71,72,73]

Nixon and Andrews[16] suggest that recovery from BPD depends upon: "Due attention to the restoration of proper sleep, the modulation of arousal, the recovery of natural breathing, a salutary balance of rest and effort, and the subject's achievement of self-regulation and autonomy".

Breathing retraining has been used to successfully correct hyperventilation. In one study[7]more than 1000 anxious and phobic patients were treated using a combination of 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.

In another study[72] breathing therapy was evaluated in patients with HVS in which most of the patients met the criteria for an anxiety disorder. The diagnosis was based on the presence of several stress related complaints, reproduced by voluntary hyperventilation, patients with organic diseases having been excluded. Therapy was conducted in the following sequence:

1. Brief, voluntary hyperventilation to reproduce the complaints in daily life
2. Reattribution of the cause of the symptoms to hyperventilation
3. Explaining the rationale of therapy—reduction of hyperventilation by acquiring an abdominal breathing pattern, with slowing down of expiration
4. Breathing retraining for 2 to 3 months by a physiotherapist

After breathing therapy, the sum scores of the Nijmegen Questionnaire[74,75] were markedly reduced. A canonical correlation analysis relating the changes of the various complaints to the modifications of breathing variables showed that the improvement of the complaints was correlated mainly with the slowing down of breathing frequency. The Nijmegen questionnaire provides a non-invasive test of high sensitivity (up to 91%) and specificity (up to 95%).[75] This easily administered, internationally validated[74] diagnostic questionnaire is the simplest, kindest and to date most accurate indicator of acute and chronic hyperventilation. The questions enquire as to the following symptoms, and their intensity:

- constriction in the chest,
- shortness of breath,
- accelerated or deepened breathing,
- inability to breathe deeply,
- feeling tense,
- tightness around the mouth,
- stiffness in the fingers or arms,
- cold hands or feet,
- tingling fingers,
- bloated abdominal sensation,
- dizzy spells,
- blurred vision,
- feeling of confusion or losing touch with environment.

Breath work can also be seen to offer prophylactic benefits. Aust and Fischer[73] investigated whether psychophysical breath work influences postural control. The method used involved optical patterns being projected onto a video screen, the test subjects having been instructed to shift their centre of gravity according to the patterns projected. The patterns consisted of a line which had to be followed in the anterior-posterior and lateral plane, and a circle to be followed clockwise and counter-clockwise. The results showed that those participants with some experience of breath training had significantly better results in the posturographic test with visual feedback. Additionally, the posturographic results immediately following one hour of breath work demonstrated clear improvements in body equilibrium suggesting that breath work leads to a general improvement in maintaining equilibrium, which remains stable over time.

There is also evidence of a degree of entrainment between active movement and respiratory rate, suggesting that

rhythmic slow movements (such as performed during Tai chi exercise) can assist in reducing respiratory rate.[76] Jasinskas[77] reports that, "results strongly support the existence of entrainment, and provide evidence for neurogenic input to ventilatory control during steady state work."

The respiratory (and cardiovascular) effects of rosary prayer ('Ave Maria' in Latin) and recitation of a yoga mantra have been assessed.[78] Results were similar for both methods, showing a slowing of respiration to approximately 6cpm, and synchronisation of all cardiovascular rhythms, Traube-Hering-Meyer oscillations, representing blood pressure, heart rate, cardiac contractility, pulmonary blood flow, cerebral blood flow and movement of cerebrospinal fluid). This positive influence on autonomic activity, may offer great benefits toward normalisation of sympathetic arousal and abnormal neural function resulting from BPD.

Biochemical influences on BPD, including allergy and pseudo-allergy

Lum[79] reports that more than one third of patients suffering from chronic hyperventilation have associated conditions that frustrate efforts to correct breathing. He reports that:

- "Allergies (e.g. hay fever) may keep patients sniffing and coughing for half the year, perpetuating irregular thoracic inspirations"
- Food intolerance, with bloating after meals, may 'splint' diaphragmatic movement. Such cases need an expert in dietary management.
- "Pseudo-allergy is common; many patients falsely attribute symptoms to an allergy to particular foods. In two-thirds of such cases of pseudo-allergy, the symptoms have been shown to be due to a conditioned reflex of hyperventilation on exposure. A similar mechanism is common in allergy to perfumes, and industrial gases."[80]
- Progesterone is a respiratory stimulant, making patients with BPD most vulnerable during the post-ovulation phase of the menstrual cycle.[10]
- Blood sugar levels are, "clinically the most important of these non-ventilatory factors. When blood glucose is below the middle of the normal range (i.e. below 4.4 mmol/L) the effects of overbreathing are progressively enhanced at lower levels." [81]

SUMMARY POINTS

- Chronic BPD such as hyperventilation is widespread, more frequent in females, and leads to respiratory alkalosis, constriction of smooth muscles, and a variety of neurological, cardiac, gastrointestinal and emotional symptoms.
- Reduced CO_2 levels (hypocapnoea), involving respiratory alkalosis, causes smooth muscle constriction, reduced blood, and therefore reduced oxygen, delivery to tissues, and this is more pronounced in deconditioned individuals.
- Breathing pattern disorders are associated with anxiety, and anxiety is associated with altered neuronal (including motor) function, muscular imbalances, disturbed postural balance, and the enhanced evolution of myofascial trigger points.
- BPDs, such as hyperventilation, induce biochemical changes that increase neuronal excitability, enhance sensitisation processes, and destabilize the autonomic nervous system.
- BPDs encourage trigger point evolution, and trigger points can have a profound influence on motor function and pain.
- Core stabilising muscles are compromised by hypercapnoea – an induced rise in breathing rate that leads to respiratory alkalosis – compromising key core muscles involved in spinal stability.
- SMC contractility, and its widespread presence in connective tissues, appears to have a relevance to stability, however the precise relationship with conditions such as low back pain remains to be established, as does the connection between hyperventilation and hypermobility.
- Breathing retraining can have a positive effect in normalising BPD as well as associated neural dysfunction.
- There appears to be an overlap between functional, habitual BPD and breathing pattern disorders associated with allergy.

CONCLUSION

It seems very likely that chronic BPD negatively influences motor control, neurological sensitisation, muscle behaviour, pain threshold and balance. There is evidence that breathing rehabilitation can reverse these tendencies and restore more normal breathing patterns in many individuals. As with most features and functions not directly associated with the symptoms, unless BPDs are looked for and evaluated, they are unlikely to be recognized in a manual medicine setting. While seldom causative, BPD can be seen to potentially be a major factor in encouraging and maintaining musculoskeletal dysfunction in general, and back pain in particular.

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